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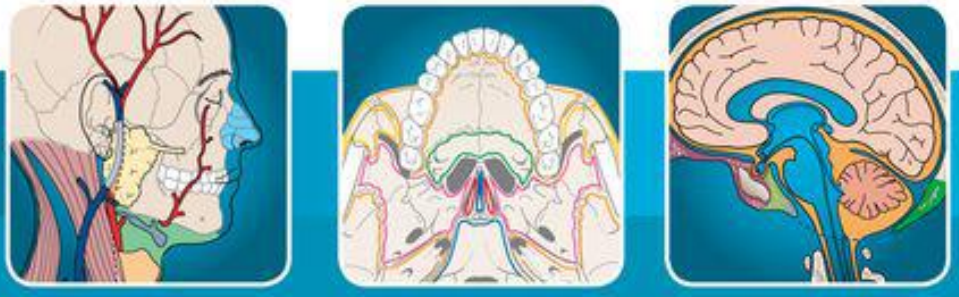
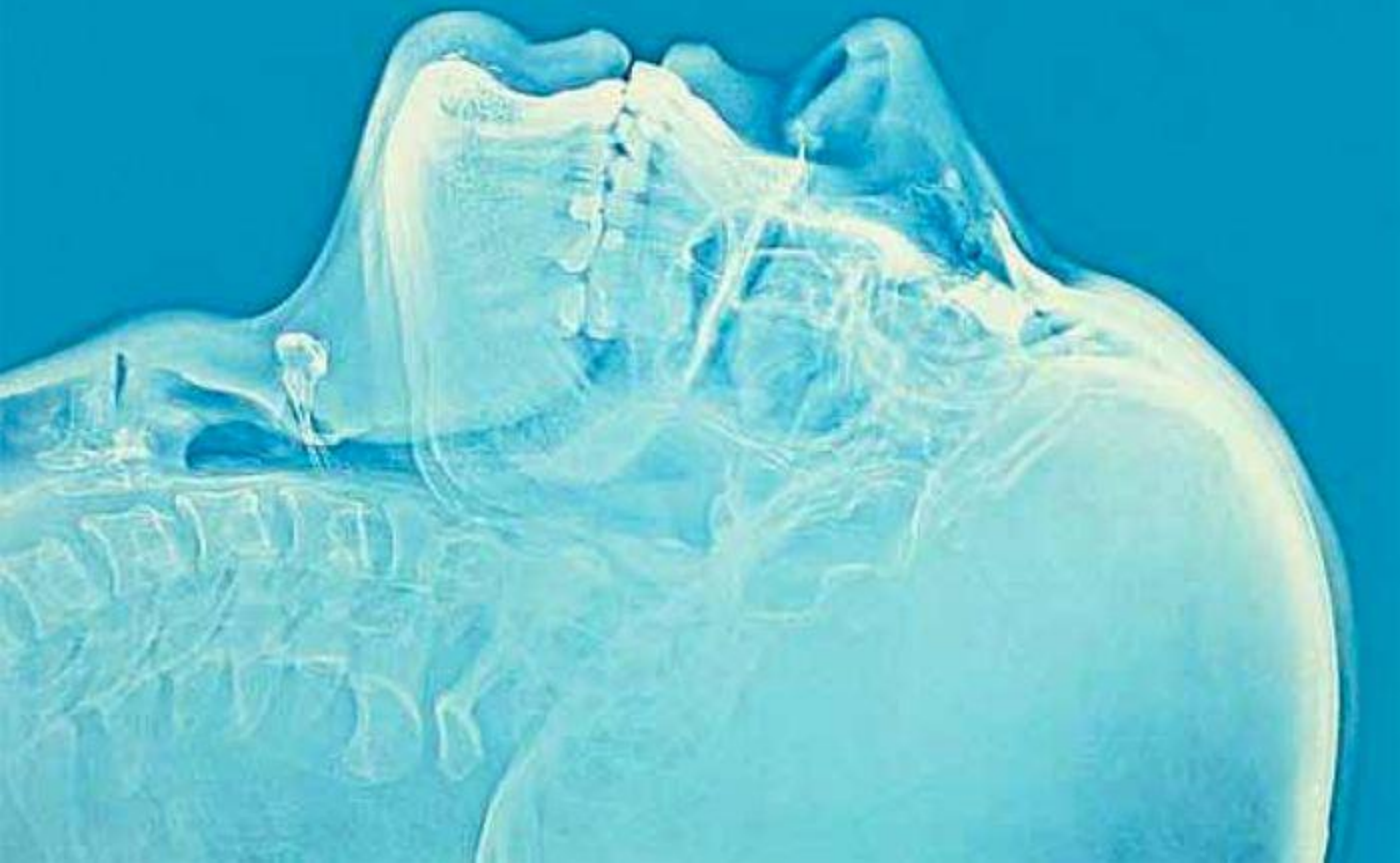
Anatomy for Dental Students



Fourth edition

Martin Atkinson



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Anatomy for dental students

FOURTH EDITION

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Preface to fourth edition of *Anatomy for Dental Students*

I was delighted to be asked to edit the fourth edition of *Anatomy for Dental Students* by Oxford University Press. It brought things full circle for me. Jim Moore, one of the original authors alongside David Johnson, was one of my excellent anatomy teachers at Birmingham University and was instrumental in guiding me into a career in anatomy. It is fitting that I can repay that debt by editing "Johnson and Moore".

Reading the preface to the first edition published almost thirty years ago shows that many aspects of dental education are still much the same. Development of dental course delivery and assessment continues in many dental schools and the introduction of integrated curricula blur or demolish traditional subject boundaries. Why then is there still a need for a "single subject" book in this brave new world? David Johnson and Jim Moore hit the bull's eye with their first aim in the original preface—that all health care professionals need a sound working knowledge of the structure and function of the human body and its application to their particular clinical area. This is paramount whether students study anatomy as a named subject or whether it is integrated into wider units of the curriculum. Three editions of *Anatomy for Dental Students* have provided a concise and precise account of the development, structure and function of the human body relevant to dental students and practitioners and it is my hope that the fourth edition will continue in that role.

Anatomy and publishing technology have advanced considerably since the last edition in 1997. The fourth edition has an entirely different style and presentation which will make it easier to use. One new feature of the fourth edition is the use of text boxes; 'clinical' boxes emphasise the application of anatomical information to clinical practice and 'sidelines' boxes contain additional interesting material not necessarily required in all dental courses. Colour illustrations are used much more extensively; all the figures have been expertly redrawn by David Gardner but the majority are based on the original drawings of Anne Johnson. David redrew Figures 3.2, 5.1, 5.3, 5.4, 14.1, 15.19, 17.1, 17.2, 18.5, 20.5, 24.6, 26.2, 26.1, 27.8, 28.6, 28.11, 28.14 and 32.17 from illustrations published in *Basic Medical Science for Speech and Language Therapy Students* by Martin Atkinson and Stephen McHanwell; I am grateful to Wiley-Blackwell for permission to use them.

The entire book has been edited and reordered to bring it into line with the requirements of students studying dental courses today. Section 1 on the basic structure and function of systems pertinent to dental practice has been expanded to benefit students who enter dental school without a biological background and also those who have studied one of the myriad modular higher level biology courses where vital material on human biology often falls through the gaps. Section 1 should create a level playing field for everyone irrespective of their previous biological experience. An appreciation of the nervous system, especially the cranial nerves, is fundamental to understanding the head and neck; the section on the nervous system therefore now precedes the section on head and neck anatomy. The head and neck section has been substantially reordered to describe the anatomy from the superficial to deep aspects of the head and then down the neck, the sequence of dissection usually followed by those who still have the opportunity to carry it out. An innovative approach to the study of the skull is used in chapter 22. The skull is assembled bone by bone so that the relationships and contributions of each bone to different subdivisions of the skull can be appreciated. The requisite detail of specific bones is then described with reference to soft tissue anatomy in chapters 23 onwards, each covering a particular region of the head and neck or their development. All the chapters on the nervous system and embryology and development have been rewritten to incorporate recent advances in these subjects; the developmental chapters have been integrated with the pertinent anatomy.

I wish to thank my colleagues Keith Figures and Adrian Jowett for their helpful discussions on various clinical aspects of anatomy and current guidelines to clinicians issued in the UK; I am also grateful to Keith for reading various clinically related sections and giving me extremely useful comments. Nevertheless any errors in the book are entirely my responsibility. Martin Payne kindly provided some of the radiographs used in chapter 31. Thanks also to Martin and Jane Wattam for introducing me to the wonders of cone beam computerized tomography. I am indebted to Geraldine Jeffers, my editor at Oxford University Press—the most exacting but also the most encouraging and supportive editor I have ever worked with—great *craic* Geraldine. I must also thank Hannah Lloyd and Abigail Stanley who played a significant part in bringing this edition to fruition. Diana—thanks as ever for your support, encouragement, and input throughout this venture. Can life return to normal now?

M.E.A.
Sheffield
June 2012

Table of contents

Abbreviations and symbols

β	beta
∘	degree
%	percent
Ach	acetylcholine
AE	anterior extension
ANS	autonomic nervous system
AV	atrioventricular
BA	basicranial
BMP	bone morphogenic protein
Ca^{++}	calcium ion
CHL	conducting hearing loss
Cl^{-}	chloride ion
cm	centimetre
CN	cranial nerve
CNS	central nervous system
CPR	cardiopulmonary resuscitation
CSF	cerebrospinal fluid
CT	computed-assisted tomography
CVA	cerebrovascular accident
DPT	dental panoramic tomograph
ECM	extracellular matrix
ECO	endochondral ossification
e.g.	***exempli gratia*** (for example)
FGF	fibroblastic growth factor
fMRI	functional magnetic resonance imaging
g	gram
GAG	glycosaminoglycan
GIT	gastrointestinal tract
h	hour
Hz	Hertz
ICP	intracranial pressure
ID	inferior dental (block)
IMO	intramembranous ossification
K^{+}	potassium ion
LRT	lower respiratory tract
m	metre
µm	micrometer
MRI	magnetic resonance imaging
mV	millivolt
Na^{+}	sodium ion
NA	noradrenalin
nm	nanometer
PM	premotor (cortex)
PNS	peripheral nervous system
®	registered trademark
RA	retinoic acid
SA	sinoatrial
SEA	spheno-ethmoidal angle
SHH	sonic hedgehog
SMA	supplemental motor area
SNHL	sensorineural hearing loss
TCMS	transcutaneous magnetic stimulation
TMJ	temporomandibular joint
TSNC	trigeminal sensory nuclear complex
UK	United Kingdom
URT	upper respiratory tract
VPL	ventroposterolateral
VPM	ventroposteromedial

Online Resource Centre

To help you consolidate your knowledge and revise for exams, we have provided interactive learning resources on the following site: http://www.oxfordtextbooks.co.uk/orc/atkinson/

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Test yourself with over 50 revision questions in single best answer and multiple choice styles. These questions apply to all four sections of the book to give you comprehensive coverage of the content.

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Selected figures from the book are available for you to test your knowledge with interactive 'drag-and-drop' labels. With over 30 figures from across the four sections, the drag-and-drop exercises are a great way to revise complicated anatomical structures.

How to use this book

This book has been developed not only with hundreds of colour illustrations, but also several learning features to enhance your understanding.

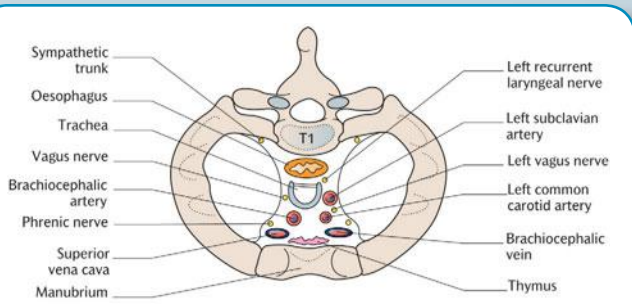

Fig. 12.2 Structures in the superior mediastinum at the level of the first rib.

Illustrations

This book is illustrated throughout with over 300 clear, colourful, and high-quality line drawings. The captions and accompanying text have been carefully written to take you through complex structures step-by-step.

Box 2.1 The clinical importance of periosteum

Periosteum is clinically important during operations on bone. It must be carefully reflected off the bone surface and then carefully replaced. Periosteum is the source of osteoblasts essential for repair of bone. It is also the main route for nutrition of bone; blood vessels passing over the bone give branches to the periosteum that then penetrate into the bone to supply it; if peri-

Clinical applications boxes

These blue boxes demonstrate how the form and function of anatomy might have consequences for clinical practice.

Box 2.3 The evolution of bone

The two types of bone formation have a long evolutionary history. A skeleton based on calcium rather than silicon appeared in the Cambrian geological period (between 545 and 510 million years ago), presumably because of a change in the chemistry of the ocean or the physiology of the creatures which lived in it. The first vertebrates had an exoskeleton consisting of bony

Sidelines boxes

Deepen your understanding with green boxes that take a closer look at selected anatomical structures.

Anatomical terms

The following list is composed of an anatomical term, a plural form (pl.) where this is in common use or unusual, the language of origin, and a short explanation or translation.

Alar (L) From ala = a wing, hence wing-like
Alveolus pl. alveoli (L) A cavity or socket
Amygdala; amygdaloid (G) An almond; resembling an almond

Glossary

Glossary terms are highlighted in bold and collected at the back of the book, forming a great revision aid to help you master anatomical vocabulary. The glossary includes a short list of common suffixes and prefixes and explains the Latin or Greek roots of the terms.

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Section 1
Introduction and developmental anatomy

Section contents

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1 The study of anatomy

Chapter contents

1.1 Introduction

Human anatomy concerns the structure of the human body. Anatomy is often interpreted as the study of only those structures that can be seen with the naked eye (**gross anatomy**). Anatomy also covers the study of structure at the cellular (histology) and subcellular level (ultrastructure). The formation (**embryology**) and growth of anatomical structures (**developmental anatomy**) influence their organization, appearance, and their relationship to other structures and often explain gross anatomical arrangement.

Historically, physiology (the study of the function of the body) was regarded as a separate subject from anatomy but the relationships between structure and function (**functional anatomy**) is critical to understanding how the body works at all levels. Most modern dental curricula now have some degree of integration between anatomy and physiology to emphasize their interrelationship in the study of the human body. It is impossible to recognize changes in structure brought about by disease and their clinical manifestations and effects on function without an understanding of healthy structure and function. It is impossible to use any surgical procedures effectively and safely without a good working knowledge of the anatomy of the relevant part of the body. In clinical work, internal structures often need to be located accurately even when they cannot be visualized directly. A good example of this is the need to be able to locate the nerves supplying the teeth in order to deliver local anaesthetic accurately prior to carrying out a restoration or extraction. Fortunately, most structures have a fairly constant relationship to surface features (**surface anatomy**) to allow their position to be determined with considerable accuracy. Information about deep structures can also be obtained by the use of imaging techniques such as X-rays or scanning technology. Interpretation of radiographs and scans requires knowledge of the radiographic appearance of normal body structures (**radiological anatomy**). Surface and radiological anatomy are obviously of great practical importance and are covered in the relevant sections of the book.

The principal aim of this book is to provide you with sufficient practical information about the anatomy of the human body to form a basis on which to build your clinical skills and practice. Gross anatomy, including functional, clinical, surface, and radiological anatomy will be covered, together with embryology and developmental anatomy where relevant. Histology and ultrastructure will be only included where they aid understanding of structure and function.

Gross anatomy can be studied in two ways. One method is to take each region of the body in turn and examine all the structures found there and their relationships to each other; this is regional or topographical anatomy. It is the anatomy that surgeons need to know so that they are always aware of the structures they will encounter in the area of the body in which they specialize. The second method is to deal with all aspects of each of the body systems in turn; this is systemic anatomy. Ideally, systemic and regional anatomy go hand in hand; systemic anatomy gives a whole picture of several structures forming a system and regional anatomy examines the structures from different systems contributing to a particular region. For example, when you encounter a blood vessel in one region, you would need to know where it came from and where it was going to beyond that immediate region before subjecting it to any surgical procedure; you could then assess the likely consequences of your actions elsewhere in the body. In this book, the areas of the body most important to the practice of dentistry are considered on a **regional anatomy** basis. However, it is easier to understand the anatomy of a specific area if you build up in your mind a picture of the **systemic anatomy** of the structures you find there. In other words, *try and discover the plan or pattern of an area before studying the detail*.

As a prelude to the important aspects of regional anatomy, **Section 1** presents brief descriptions of the major body systems relevant to the practice of dentistry to enable you to see the overall pattern of the body. These chapters are also a useful orientation for students entering dental schools without a biological background. This introductory section concludes with a brief outline of early embryological development. The relevant developmental anatomy of specific systems and regions will be included in the corresponding sections of the book.

Section 2 covers the anatomy of the thorax. Diseases of the chest are frequent; many common drugs used to treat illnesses of this region have systematically acting effects and may have implications in the planning of dental treatment.

Section 3 deals with the nervous system. Some knowledge of the structure and function of this system is essential for anyone concerned with the diagnosis and treatment of disease. It is also vital to gain an overall understanding of the cranial nerves, their function, and distribution as they are the basis for the structure and function of the head and neck. The cranial nerves are one of the cardinal areas where an understanding of the general pattern and distribution aids the detailed understanding of the regional anatomy.

Section 4 focuses on the head and neck—that part of the body in which, as dentists, you will spend most of your working life.

1.2 How to approach anatomy

Anatomy can be quite daunting to start with. More or less as soon as you start to examine a given structure, you will find you need some information on other structures or distant parts of the same structure. Try to see the overall pattern first and worry about individual detail later. As your knowledge increases, the jigsaw will start to come together and the whole picture will begin to emerge.

However anatomy is taught to you, you will be convinced that your teachers are talking a language foreign to most of you. To some extent, they will be because the naming of bodily structures is historically based on ancient Greek and Latin (see Glossary). Many structures were named because, in the mind of early anatomists, they bore a resemblance to everyday objects such as drinking vessels and fruits. If you understand why

a particular Latin or Greek term is used, this often aids understanding and memory of anatomical terminology. However, when you look for the resemblance yourself, you may well conclude that some of the pioneer anatomists must have had very vivid imaginations. To help with terminology, a **glossary** of the meaning and derivations of the commoner anatomical terms is included. When you begin your study of anatomy, you will also encounter a number of specific **anatomical terms** used to describe the position of different structures and their relationship to each other; these are described and illustrated in Section 1.3. Study of anatomical specimens will help you understand and memorize structures much more easily than any amount of reading or studying of illustrations and will give you the true scale of things. Anatomical specimens take many forms; it may be yourself or a partner (living anatomy), a **cadaver** in a dissecting room on which you can carry out your own dissection, a **prosection** (a prepared dissection), or anatomical models. If you are fortunate enough to have access to a dissecting room, cadaver, or prosections, make full use of the opportunity you have been given. Human beings, like all other organisms, vary in all aspects of their structure and function. All structures of the body vary in size, shape, and arrangement and you will encounter such variations in every facet of your clinical career. No two anatomical specimens, living or dead, are identical; you will frequently find that the specimens you are examining differ considerably from the textbook description. Using anatomical material to study the subject shows variation that idealized diagrams or selected photographs in textbooks cannot. The descriptions given within this book are those that are the most usual or typical, but common variations that may be clinically relevant are described.

1.3 Descriptive anatomical terms

1.3.1 The anatomical position

For consistency and a basic reference point, the body is always referred to as if it were in the **anatomical position** which is illustrated in Figure 1.1.

Examine the illustration and note that:

- The individuals are standing erect;
- Their face and eyes are directed forward;
- Their hands are by their sides with palms directed forward;
- Their heels are together, the feet pointing forward so that the great toes are adjacent.

Anatomical descriptions are always written from this reference position. Much more significantly, your patients are *always* described as if they were in the anatomical position. If you remember this basic rule,

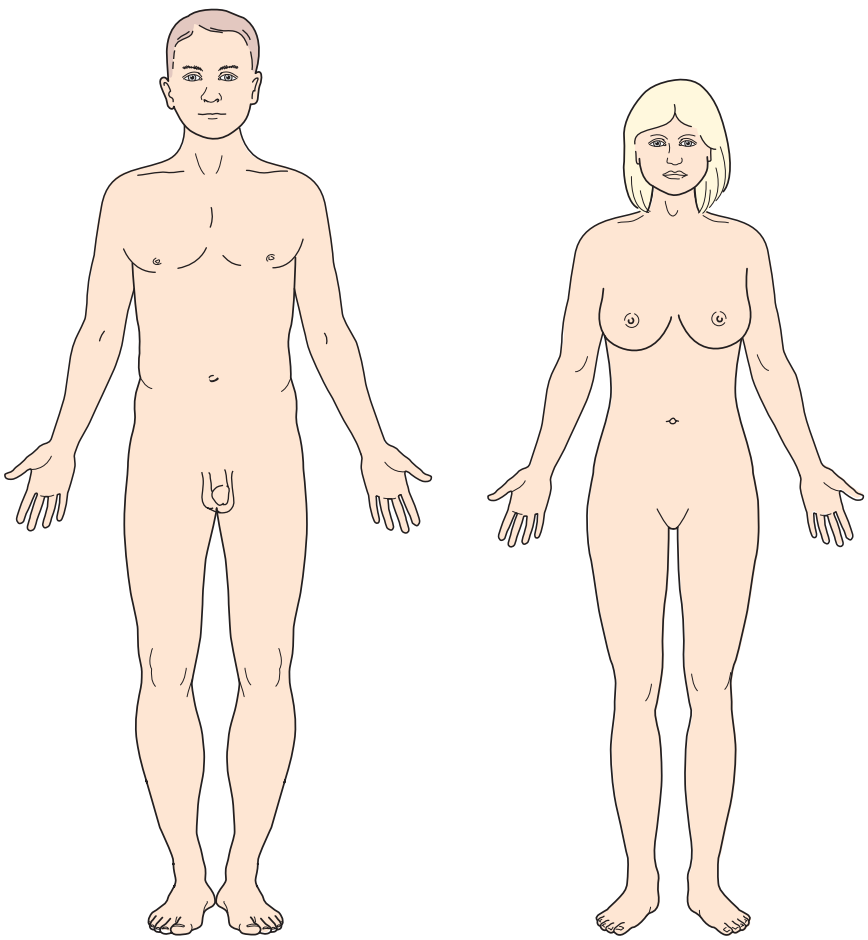

Fig. 1.1 The anatomical position.

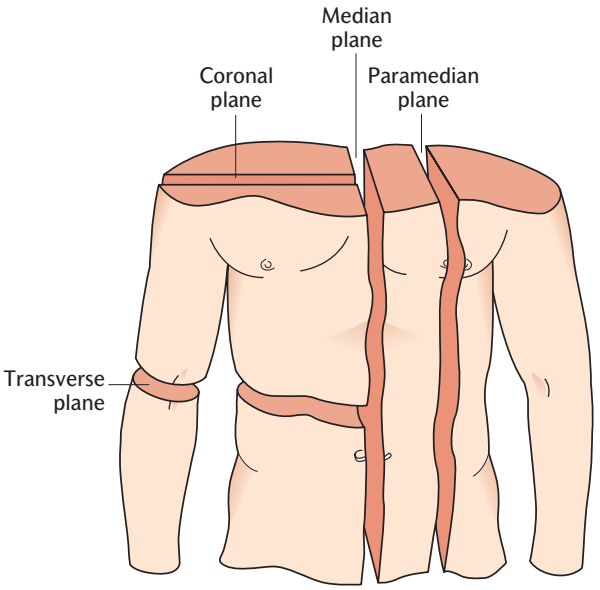

Fig. 1.2 Planes of section of the body.

you will never extract the wrong tooth by taking one from the opposite side of the body than the one intended.

1.3.2 Anatomical planes

Figure 1.2 illustrates the body standing in the anatomical position once again, but this time, the body is divided by three planes at right angles to one another. These planes are the reference points that anatomical descriptive terms are referred to.

The **median** or **sagittal plane** is the vertical plane which divides the body into left and right halves down the **midline**. It is named after the sagittal suture in the skull; the term 'sagittal' is in turn derived from the supposed resemblance of the suture in the skull of a newborn to an arrow. As you can see from Figure 1.2, any plane parallel to the median or sagittal plane is **paramedian** or **parasagittal**.

The **coronal plane** is any vertical plane at right angles to the median plane. It is named from the coronal suture passing through the crown of the skull and divides the body into front and back portions.

A **transverse** or **horizontal plane** is any plane at right angles to both median and coronal planes.

1.3.3 Anatomical descriptive terms

The following *pairs* of descriptive terms are related to the anatomical planes.

1. **Medial**—closer to the midline of the body;
   Lateral—further from the midline of the body.

If you are in the anatomical position, your arms are lateral to your chest and your chest is medial to your arms.

2. **Anterior**—nearer the front surface of the body;
   Posterior—nearer the rear surface of the body.

Your nose is anterior to your ears and conversely, your ears are posterior to your nose. **Ventral** and **dorsal** are used as synonyms for anterior and posterior. These terms are used in comparative anatomical descriptions of four-legged animals when the anatomical position cannot be applied. These terms have become incorporated into the names of structures you will encounter later in the book.

3. **Superior**—nearer the crown of the head;
   Inferior—nearer the soles of the feet.

Your head is superior to your chest and your legs are inferior to your chest.

4. **Proximal**—nearer the median plane;
   Distal—further from the median plane.

These terms are used to indicate the relative positions of structures along a long structure such as a nerve or blood vessel. A branch near to the origin of the vessel would be proximal to a branch further down the vessel. These two terms are also used extensively in description of the limbs; in Figure 1.1, your wrist is distal to your elbow, but your shoulder is proximal to your elbow.

5. **Superficial**—near to the skin surface;
   Deep—below the skin surface.

Note that all the terms defined above are *paired*. These terms are often incorporated into the names of structures as well as being used to describe their position. If you come across a structure with one of a pair of the terms described above in its name, you can be certain that there will be another structure with opposite term in its name. Two examples will show this. The *medial* pterygoid muscle and the *lateral* pterygoid muscle are two important muscles that move the jaw; the *superficial* temporal artery is just below the skin on the side of the head (and can even be seen in many bald individuals) whereas the *deep* temporal artery is hidden beneath a layer of muscles.

Terms of movement

There are many terms used to describe movements at joints in the body, but you will only encounter a few of them.

1. To **abduct** is to draw away from the midline median plane.
   To **adduct** is to move towards the midline.
2. To **protrude** or protract is to move forwards.
   To **retrude** or **retract** is to move backwards.

Other terms

Ipsilateral means on the same side of the body. **Contralateral** means on the opposite side.

Interior, internal, inside and **external, exterior, outside** are mostly used to describe position in relation to body cavities like the thorax or hollow organs like the gut.

Invaginations and **evaginations** are inward and outward bulges in the wall of a cavity and are often used to describe movement of structures during development so you will meet these terms again in Chapter 8 and other chapters.

2 The locomotor system

Chapter contents

The locomotor system comprises the skeleton, composed principally of bone and cartilage, the joints between them, and the muscles which move bones at joints.

2.1 The skeleton

The skeleton forms a supporting framework for the body and provides the levers to which the muscles are attached to produce movement of parts of the body in relation to each other or movement of the body as a whole in relation to its environment. The skeleton also plays a crucial role in the protection of internal organs.

The skeleton is shown in outline in Figure 2.1A. The skull, vertebral column, and ribs together constitute the **axial skeleton**. This forms, as its name implies, the axis of the body. The **skull** houses and protects the brain and the eyes and ears; the anatomy of the skull is absolutely fundamental to the understanding of the structure of the head and is covered in detail in Section 4.

The **vertebral column** surrounds and protects the spinal cord which is enclosed in the spinal canal formed by a large central canal in each vertebra. The vertebral column is formed from 33 individual bones although some of these become fused together. The vertebral column and its component bones are shown from the side in Figure 2.1B.

There are seven **cervical vertebrae** in the neck, twelve **thoracic vertebrae** in the posterior wall of the thorax, five **lumbar vertebrae** in the small of the back, five fused **sacral vertebrae** in the pelvis, and four **coccygeal vertebrae**—the vestigial remnants of a tail. Intervertebral discs separate individual vertebrae from each other and act as a cushion between the adjacent bones (see Figure 10.2); the discs are absent from the fused sacral vertebrae.

The cervical vertebrae are small and very mobile, allowing an extensive range of neck movements and hence changes in head position. The first two cervical vertebrae, the atlas and axis, have unusual shapes and specialized joints that allow nodding and shaking movements of the head on the neck. The thoracic vertebrae are relatively immobile. These carry the ribs which project forwards to join the sternum anteriorly; this

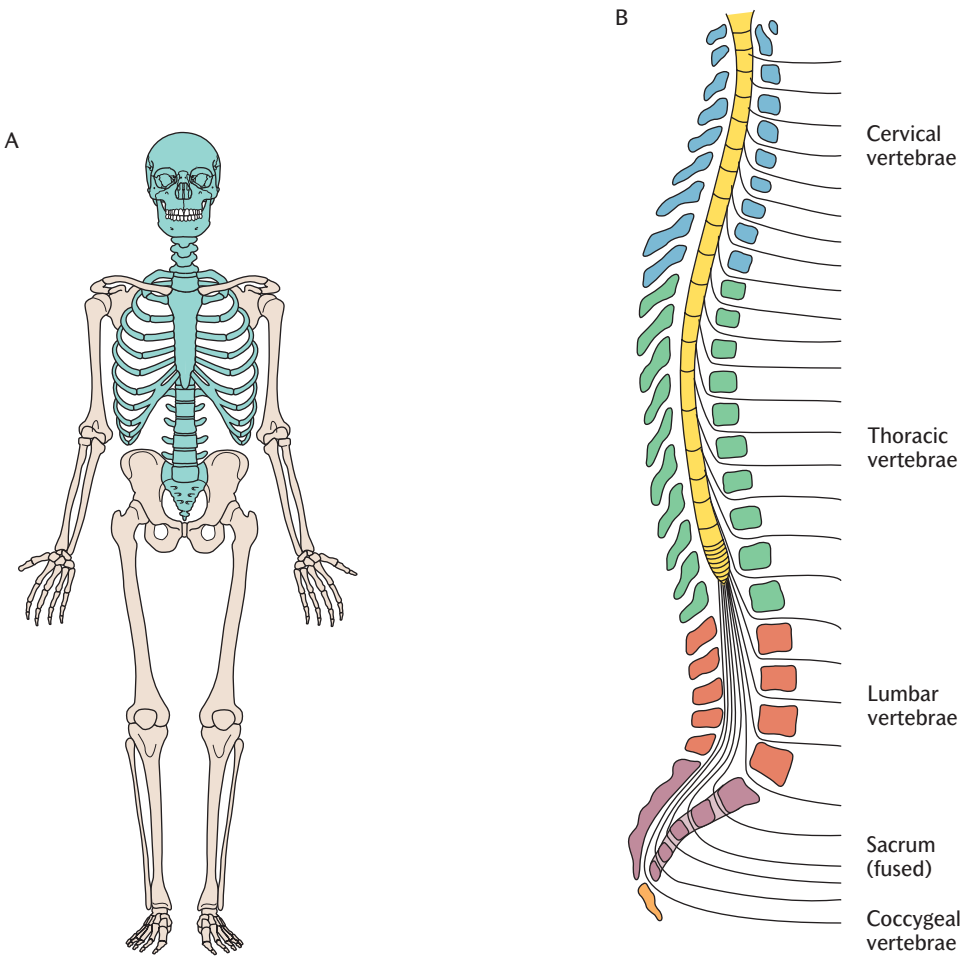

Fig. 2.1 A) The skeleton. The axial skeleton is shown in blue. B) The vertebral column viewed laterally.

combination of thoracic vertebral column, ribs, and sternum form the **thoracic cage** that protects the thoracic organs, the heart, and lungs and is intimately involved in ventilation (breathing). The lumbar vertebrae are large and robust as they carry the weight of the upper body. They are mobile to some degree, especially in sagittal plane, allowing you to bend your upper body back and forth on the hips. The fused sacral vertebrae form very strong joints with the pelvis, providing strong rigid attachments for the lower limbs. The coccygeal vertebrae are a pain should you fall on them.

The arms (forelimbs) and legs (hind limbs) are not directly connected to the axial skeleton; they are connected through the shoulder girdle and pelvic girdle, respectively. The girdles and limbs constitute the **appendicular skeleton**. The pelvic girdle is a very strong structure and is immobile, the lower limbs pivoting at the hip joints. The shoulder girdle comprises the **scapula** (shoulder blade) and **clavicle** (collar bone) on each side. Unlike the pelvic girdle, the shoulder girdle is only attached to the axial skeleton by a joint at the medial end of the clavicle, but it does have extensive muscle attachments. This enables the forearms and shoulder girdle to move much more freely than the legs and pelvic girdle. Some muscles of the neck attach to the shoulder girdle so the scapula and clavicle will be met again later in the book.

2.2 Bone and bones

When studying bone specimens prepared for anatomical examination, they are hard, dry, and very obviously dead. Many people think that this is what bone is like inside the body too. Nothing could be further from the truth. We have all experienced a bone fracture or know someone who has. The orthopaedic surgeon will bring the parts of the broken bone together and support them with a plaster cast. After a few weeks, the bone will have repaired itself and is able to function normally to support the person's weight, for example, so the cast will be removed. This shows that bone is very much alive and very adaptable. A bone fracture is an extreme example of change in bone, but even intact bones are changing all the time to meet the functional demands placed upon them. This is a process known as **remodelling** and preserves the mechanical efficiency of bones.

Bone is potentially heavy, but is beautifully designed so that maximum strength can be achieved for minimum weight. Unnecessary bone is removed and additional bone is added as required. In a paralysed limb, the bone becomes thinner and weaker; in an athlete or an overweight person, it may become stronger and heavier. Look at the bones available to you for study and you will quickly find a damaged bone. The outside of the bone is thick and dense and is called **compact** bone. Look inside and you will see a meshwork of bone with spaces in between; this is **cancellous** or spongy bone made up of a meshwork of individual **trabeculae** as shown in Figure 2.2.

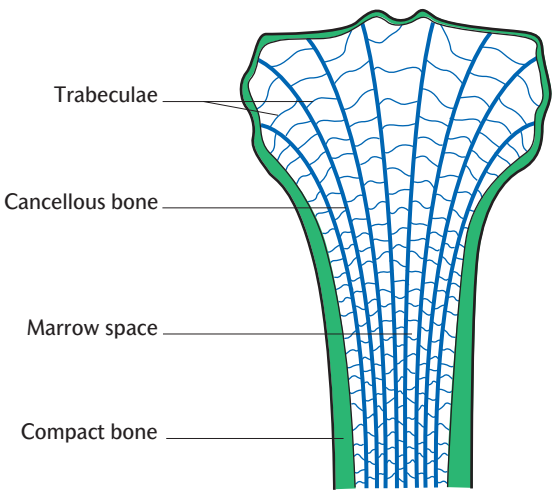

Fig. 2.2 The structure of bone.

If you look very closely at a damaged bone, it may be possible to see that the trabeculae making up the cancellous bone are not arranged at random, but are aligned very accurately along the lines of stress that the bone is subject to. Look more carefully at Figure 2.2. The cancellous bone trabeculae in the shaft are arranged at right angles to each other along the lines of stress arising from the weight bearing function of the bones. In the areas of bone forming the joint, stresses will be applied in different directions according to the movement of your body; the trabeculae are arranged radially so that some are always aligned along lines of stress.

2.2.1 Bone remodelling

After a fracture, the broken ends are united by a temporary framework (or **callus**). However the callus is not weight bearing which is why a support cast is required while the bone is repaired and remodelled back to a mechanically efficient structure of compact bone externally and cancellous bone internally. Remodelling continues for some time after the patient begins to use the bone again. The reason that the callus of a healing bone is not weight bearing is because it is formed from **woven bone**, so called because it has a network of randomly orientated disorganized trabeculae. Woven bone is remodelled to form compact bone externally and cancellous bone internally. Woven bone is also the type of bone that is formed when bone formation is initiated during development and growth.

How is remodelling and repair brought about? Any biological tissue needs cells to form, maintain and repair it, and a blood supply to bring in the nutrients required for these processes. Bone is a member of a large group of tissues called **connective tissues** that all have the same basic components:

- **Cells** that make;
- **Extracellular matrix (ECM)**, a jelly-like material;
- Long **fibres** with high tensile strength.

The proportions of ECM and fibres differ in individual connective tissues to give each one specific properties. The major fibre type found in the body is **collagen**, a triple helix of long chain molecules that give it a high tensile strength; it is 'biological rope'. In bone, the ECM is reinforced by the addition of inorganic crystals of calcium **hydroxyapatite**, a property bone shares with three tissues that make

up teeth—enamel, dentine, and cementum. The collagen fibres give bone its great tensile strength while the hydroxyapatite crystals provide its compressive strength. Bones are further strengthened by the muscles attached to them, contracting in such a way as to offset the applied force.

There are three types of cell associated with bone. **Osteocytes** are embedded in the rigidly mineralized matrix which makes bone incapable of growing by **interstitial growth**; the addition of material internally. Once bone has started to form, it can only grow by **appositional growth** of new material on its external and internal surfaces. Bone deposition is brought about by **osteoblasts**, some of which become entrapped as the new bone develops where they remain as osteocytes. It is usually necessary to remove bone from some surfaces as it is added to others to preserve the proportions of the bone and to stop it getting too heavy. Multinucleated giant cells called osteoclasts remove bone, a process known as **resorption**. Osteoblasts and osteoclasts are found in the **periosteum** and **endosteum** lining the external and internal surfaces of bones, respectively (see Box 2.1). When the load on bone changes, minute electrical currents are set up as the hydroxyapatite crystals are distorted; these currents stimulate cellular activity in osteocytes which in turn release signalling molecules that activate osteoblasts or osteoclasts.

Box 2.1 The clinical importance of periosteum

Periosteum is clinically important during operations on bone. It must be carefully reflected off the bone surface and then carefully replaced. Periosteum is the source of osteoblasts essential for repair of bone. It is also the main route for nutrition of bone; blood vessels passing over the bone give branches to the periosteum that then penetrate into the bone to supply it; if periosteum is not preserved, the bone will die by a process of aseptic necrosis, producing a weak spot in the skeleton.

2.2.2 Functions of bone

From the description already given, some of the functions of bone can be anticipated, but there are other functions of bone that are not so obvious.

Bone has the following functions.

- It forms a supporting framework for the body and forms the levers on which muscles act.
- It protects internal organs.
- It acts as a calcium and phosphorus store; 99% of the body's calcium is stored in bone from where it is easily mobilized. Calcium is essential for muscle and nerve function and calcium levels in the blood must be maintained within very precise limits. If dietary calcium proves insufficient to maintain blood calcium levels, calcium is released from bone during remodelling by osteoclasts.
- The spaces between cancellous bone trabeculae form **marrow cavities** containing bone marrow.

Box 2.2 Bone marrow testing and donation

When bone marrow needs to be tested in adults, it is drawn from the sternum as this bone is the most accessible of those that are still actively haemopoietic. When a larger quantity of marrow is required for bone marrow transplants, it is removed from the hip bones; they have a larger reservoir of marrow than the sternum, but are equally accessible.

Marrow is the site of **haemopoiesis**—the formation of red and white blood cells. During prenatal life, most bone marrow is active **red marrow**, but during the growth period, the areas of haemopoiesis become progressively restricted. In the adult, red marrow is found only in the bones of the skull, the vertebrae, sternum, ribs, shoulder girdle, pelvis, and the proximal ends of some long bones. Elsewhere, the marrow becomes converted to inactive fatty **yellow marrow** (see Box 2.2).

2.2.3 Development and origin of bone

During fetal and post-natal life, bone development and growth occurs by two methods. **Endochondral** or **cartilage-replacing** bone is formed by osteoblasts on a cartilaginous model of the bone. In this type of bone formation, growth occurs in cartilage (see Section 2.3) and the structure is consolidated into bone. **Intramembranous** or **dermal** bone is formed directly by osteoblasts in fibrous connective tissue without a preceding cartilage model. All the bones below the skull (the **postcranial skeleton**) are formed by endochondral ossification, except the clavicle. The vault of the skull and most of the facial skeleton are formed by intramembranous ossification, but the base of the skull and the bones surrounding the nose and internal ear are formed by endochondral ossification. Some skull bones form parts of the skull base and parts of the vault; they form by fusion of separate elements that develop by one of the two methods of bone formation (see Chapter 33). The clavicle has a similar mixed origin. Box 2.3 outlines the evolution of the two different bone types.

Box 2.3 The evolution of bone

The two types of bone formation have a long evolutionary history. A skeleton based on calcium rather than silicon appeared in the Cambrian geological period (between 545 and 510 million years ago), presumably because of a change in the chemistry of the ocean or the physiology of the creatures which lived in it. The first vertebrates had an exoskeleton consisting of bony plates within the skin. It is presumed that the same creatures had endoskeletons, possibly of cartilage, which were not preserved. In later vertebrates, cartilage-replacing bone developed in the endoskeleton and the exoskeleton was reduced considerably. The cartilage-replacing bones of modern vertebrates are believed to have been derived from the endoskeleton and their dermal bones from the exoskeleton of their evolutionary ancestors.

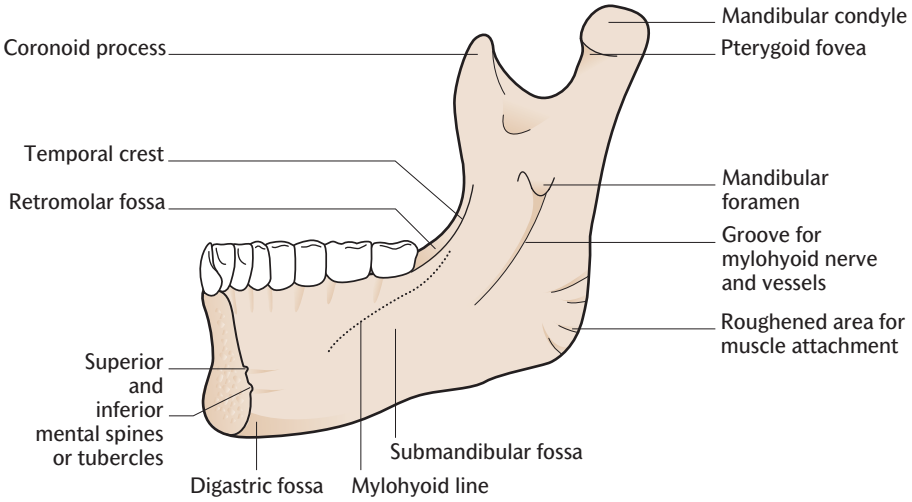

Fig. 2.3 Bony markings on dried bones using the mandible as an illustration.

2.2.4 Markings on dry bones

The living dynamic changing nature of bones has been emphasized above, but most students of dentistry will study dry bones. This is certainly less messy than studying fresh bone and the character of the surfaces of a dried bone gives us considerable information about the structures which were related to the bones during life. Muscles must be attached to bones for them to work efficiently as must ligaments supporting joints. Muscles are not attached directly, but through the fibrous tissue surrounding the muscle. This may be by a **tendon**, a cord-like structure, an **aponeurosis**, a broad sheet, or **fascia**, a dense sheet covering a group of muscles. Wherever any of these fibrous structures have been attached to bone, they will leave a mark on the bone.

Examples of some of the various structures that can be seen on dry bones are shown in Figure 2.3 and features mentioned in the following descriptions that appear in that picture are underlined. Many markings are seen as roughened areas or specific elevations. Linear elevations are termed, according to increasing size, **lines**, **ridges**, or **crests**; rounded elevations are called **tubercles** or **tuberosities**; knuckle-shaped smooth articular areas are condyles; sharp protrusions are called **processes** or spines. Note that fibrous tissue markings are absent from bones of the young, are first seen at puberty, and increase in definition with age; this can be of forensic use when trying to age bones. A depression in a bone surface is a **fovea** or **fossa**; it may be the site of a muscle attachment (the digastric fossa in Figure 2.3) or simply a shallow area between other prominent features (the retromolar fossa in Figure 2.3). Muscle attachments may also be marked by a roughened area on the bone.

An elongated **groove** may be produced by adjacent structures such as nerves or blood vessels, creating an impression in bone. Smooth areas are called **facets**. These have usually been covered in life with articular cartilage that covers joint surfaces, thus indicating where joints are formed. Knuckle-shaped articular surfaces are called **heads** or **condyles**. A **foramen** (pl = foramina) is a hole in a bone through which nerves or blood vessels or both pass. An elongated foramen is termed a **canal** or **meatus**. A fissure is a long crack-like aperture where usually several nerves and blood vessels pass through the bone. A **notch** or **incisure** is a depression in the margin of a bone.

2.3 Cartilage

Cartilage is another connective tissue that makes an important contribution to the skeleton. Unlike bone, cartilage contains no hydroxyapatite so is not rigid. The ECM of cartilage produces a firm solid structure that gives slightly under load. Cartilage is found at ends of bones as **articular cartilage** where it lines joint surfaces. Cartilage also extends some bones to provide additional flexibility to certain areas; the best example is the extension of ribs by costal cartilages which produces more efficient and adaptable respiratory movement (see Section 10.1.2). Some parts of the skeleton are formed by cartilage such as the tracheal rings and laryngeal skeleton in the respiratory tract; cartilage provides efficient support to prevent tubes collapsing as air pressure changes during ventilation, but allows some flexibility to accommodate volume and pressure changes.

Unlike bone, cartilage can grow **interstitially** by adding material around the formative cells deep within its substance as well as by **apposition** of new material on its surfaces. It is thus an important skeletal material during fetal and post-natal life when rapid growth of complex shapes is taking place. Cartilage forms the precursors of the bones of the post-natal skeleton, except the clavicle; it also forms the precursors of the base of the skull.

Microscopically, cartilage may be divided into three types according to the type and number of fibres in the matrix. **Hyaline cartilage** is the most common, forming all the cartilage referred to above. **Fibrocartilage** replaces hyaline cartilage in areas subject to great stress such as intervertebral discs and contains more collagen fibres than hyaline cartilage. **Elastic cartilage** contains elastin fibres which have elastic properties. The skeleton of the external ear and tip of the nose are formed from elastic cartilage; if you press your nose against a window, your nose will spring back into shape when you move on.

2.4 Joints

A joint is a junction between two or more bones, but the sites of union of bones can have very different properties. We normally associate joints with movement between different bones of the skeleton such as the knee, elbow, shoulder, or hip, but not all joints permit movement. Mobile joints are known as **synovial joints**, but the amount of movement permitted varies over a wide range. Non-mobile joints usually develop as **growth sites** during development and growth of the skeleton and they persist in different forms once growth is completed.

2.4.1 Synovial joints

Joints that permit a wide range of free movement are called **synovial joints**. Their major features are shown in Figure 2.4A which should be followed as the description is read.

Characteristically, the bones are separated by a **synovial cavity** filled with a very small volume of synovial fluid. A thin fibrous **capsule** arranged like a cuff around the joint retains the synovial fluid and unites the bones. The capsule is often thickened locally to form **ligaments**. The bones that contribute to a synovial joint articulate with each other at the **articular surfaces**; these are usually reciprocally curved and covered with a layer of hyaline **articular cartilage** which has a low coefficient of friction. **Synovial fluid**, essentially a dialysate of blood plasma, is secreted into and resorbed from the synovial cavity by the **synovial membrane**, a vascular sheet of vascular connective tissue which lines the capsule and covers non-articulating areas of bone within the joint cavity.

Goodness of fit or **congruence** between the articular surfaces forming a synovial joint determines its stability and range of movement. If the surfaces are congruent and fit closely, the joint is stable, but may only have a limited range of movement. If the fit of the bones is not so close, the joint loses some stability, but increases its range of movement. The outlines of the bones forming the shoulder and hip joints are shown in Figure 2.4B and C. Think about or, even better, try out the range of movement at each of those two joints. The range is greater in the shoulder than the hip joint because the bones do not fit so well. However, the shoulder joint is the most frequently dislocated major joint because the poor fit of bones renders it relatively unstable. The hip joint is relatively mobile, although not as much as the shoulder joint, but is incredibly stable.

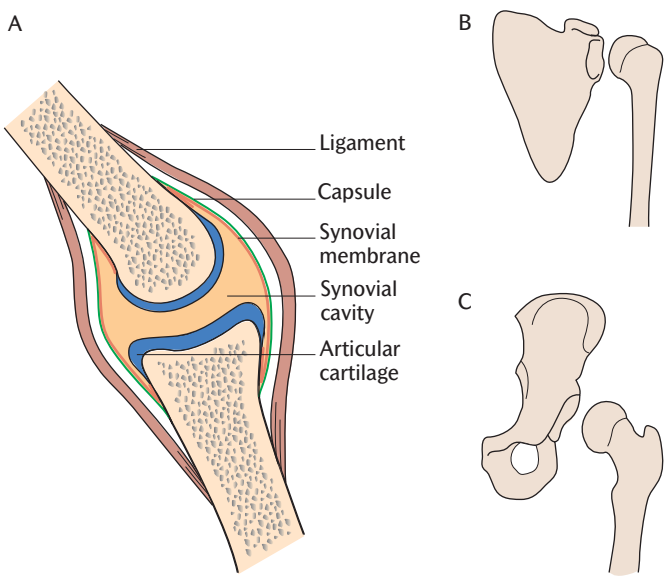

Fig. 2.4 A) A section through a synovial joint. For clarity, the distance between the articular cartilages has been exaggerated; B) Outlines of the shoulder joint; C) Hip joint to show how congruency of the articular surfaces influences the mobility and stability of synovial joints. The distance between the joint surfaces has been exaggerated for clarity.

Box 2.4 Osteoarthrosis

Osteoarthrosis is a common disorder of synovial joints in the elderly, especially those joints that bear much weight and stress such as hip and knee joints. In this condition, the articular cartilage is destroyed and movement at the joint becomes restricted and painful. Its cause is unknown. Irritation of the synovial membrane is usually followed by rapid production of large quantities of synovial fluid, causing painful swelling of the joint.

Various structures are found within synovial joints to improve the fit of the articular surfaces, hence the stability of the joint. The temporomandibular joint (TMJ) between the base of the skull and mandible is the only synovial joint of major concern to dentists so we will only deal with the features relative to that joint. As shown in Figure 24.1A, the joint cavity of the TMJ is completely separated into upper and lower compartments by a fibrous **articular disc**, but they do not move independently in the TMJ. Muscle tendons may pass through a joint space, enclosed in their own lubricating sheath; the articular disc of the TMJ is an extension of the tendon of the lateral pterygoid muscle. We will encounter these structures again when the structure and function of TMJ are examined in the context of jaw movements in Section 24.2 (see also Box 2.4).

Limitation of movement at synovial joints is necessary to avoid damage to the joint and adjacent structures. The capsules and ligaments around synovial joints contain stretch receptors which feed data into the nervous system about their degree of tension and relative position. This type of sensory information is called **proprioception**, but we are generally unconscious of it. The tension or tone in the muscles around a joint offers passive resistance to stretch and reflex contraction occurs in response to stimulation of the stretch receptors of the ligaments and capsule. The ligaments and joint capsules also contain pain receptors which are stimulated by excessive movement of the joints to act as an additional alarm signal of potential damage.

Ligaments are well-defined bands of fibrous tissue connecting bones. Most are positioned to resist or limit the movement of a joint in a certain direction as well as their function as sensory receptors. **Collateral** ligaments are local thickenings of the joint capsule whereas **accessory** ligaments are completely isolated from the capsule. In the past, the term 'ligament' was loosely applied to sheets of connective tissue unrelated to joints such as remnants of embryonic structures and tendinous muscle attachments. In subsequent chapters, you will come across some structures that are called ligaments which are not true ligaments

Box 2.5 Joint dislocation

A joint is said to be dislocated when it moves beyond its normal range so that the articular surfaces are no longer in their normal relationship. Some joints such as the shoulder, finger, and temporomandibular joints are particularly susceptible to dislocation because of poor fit of articular surfaces, poor support by ligaments, and weak supporting musculature. Dislocations are reduced by performing manoeuvres designed to relocate the joint without causing damage to surrounding structures.

although the original nomenclature has been retained. Dislocation of joints is outlined in Box 2.5.

2.4.2 Immobile joints

Sutures are examples of joints with no movement. You will meet them frequently in the description of the skull in Section 4 because sutures occur between the bones of the cranial vault and face. The bones forming sutures are separated by active bone-forming tissue and the edges of the bone are smooth during growth. As the amount of growth declines, the adjacent edges of the bones become serrated and interdigitate with their neighbours to form a strong immobile joint. Eventually, the sutures start to fuse and are usually obliterated in the skull of elderly people. At birth, the sutures are very wide and permit some movement which is important in allowing the head to mould as it passes through the vagina during birth.

Synchondroses are another form of relatively immobile joints in which the adjacent bones are connected by cartilage. They are found between the bones of the developing cranial base where they are the principal sites of growth (see Section 33.3.1). New cartilage cells are generated in a central zone and lay down cartilage by interstitial and appositional growth in both directions. After a certain amount of cartilaginous growth, the cartilage is converted into bone. Once growth is established, the rate of cartilage growth and its conversion into bone are equal so the thickness of cartilage forming the growth plate stays constant. Like sutures, synchondroses usually fuse after growth has ceased, which is why there are few remnants in the base of mature skulls.

2.5 Muscles

Muscle is a specialized contractile connective tissue; muscle cells contain actin and myosin which are contractile proteins that slide over each other to produce contraction. Muscle is divided into three types, according to its microscopical appearance determined by the arrangement of contractile fibres within muscle cells.

2.5.1 Types of muscle

Muscles that can be identified as individual structures and would be recognized as muscle by a lay person are made of **striated muscle** (also known as **skeletal** or **voluntary** muscle). It is so called because the actin and myosin fibres are regularly arranged, producing stripes across muscle fibres when viewed under a microscope. Striated muscles are attached directly or indirectly to the skeleton and are responsible for moving bones relative to each other at synovial joints and moving the whole body in relationship to its environment, hence one of the alternative names as skeletal muscle. It is often termed voluntary muscle because it is controlled by the somatic nervous system which is under conscious control (see Section 3.1). Some actions carried out by striated muscle, such as ventilation of the lungs, are far too important to be left to chance and are controlled by internal mechanisms so they make breathing occur automatically most of the time. The automatic control mechanisms can still be voluntarily overridden. A good example is when we speak—the normal automatic rhythmic breathing is changed dramatically by our voluntary actions.

Cardiac muscle

Cardiac muscle is only found in the heart; it is also striated, but has adaptations visible under a microscope that enable it to contract synchronously (see Section 4.1.1).

Smooth muscle

Smooth muscle (also known as **visceral** or **involuntary** muscle) is largely invisible to the naked eye. It is found in the walls of tubular structures forming internal organs (viscera) and blood vessels, hence the alternative name of visceral muscle. Smooth muscle is controlled by the autonomic nervous system which is responsible for the internal control of the bodily environment (**homeostasis**) and is largely independent of conscious control, hence the alternative name of involuntary muscle. Smooth muscle contracts relatively slowly, but is able to remain contracted over long periods without fatigue. Smooth muscle is usually arranged as cylindrical sheets in the wall of hollow tubular viscera. In many viscera, smooth muscle occurs as two concentric cylinders with differing fibre orientation, creating circular and longitudinal muscle layers. Smooth muscle also forms **sphincters** which can close off a tube and therefore regulate passage of whatever the tube may be transporting.

Striated muscle

Striated or skeletal muscles are attached to bone, although not always directly. Traditionally, their attachments to two or more bones have been distinguished as origins and insertions. Conventionally, the more proximal end of the muscle or the end that moves least was designated as the origin and the other end the insertion. Although some muscles only have a one-way action, many muscles may act from insertion to origin too. It is therefore much simpler to designate the connections of muscles to bones simply as **attachments**, thereby avoiding potential confusion; this usage is adopted in this book. Distinct regions can often be recognized within the muscle. The bulk of a muscle is known as the **belly** and is made up of contractile fibres which have a high metabolic rate and are consequently well vascularized to ensure a good blood supply. The belly is enclosed by connective tissue known as **epimysium** or **fascia**. The covering fascia extends beyond the belly to form the muscle attachment to bone.

The attachment comprises collagenous connective tissue which has high tensile strength and is excellent in resisting friction, but is non-contractile, inelastic, and has a poor blood supply. Muscle attachments may be of several types. A **fleshy** attachment covers a large area of

Box 2.6 Sprains and other soft tissue joint injuries

Tendons and ligaments heal slowly when damaged because of their poor blood supply. A torn tendon is a serious injury and requires prompt treatment because immediately after the injury, the severed ends move away from each other and are resistant to being rejoined. Likewise, a sprained ankle (torn ligaments) may take longer to heal than a broken ankle (bone fracture) because of the differences in blood supply to ligament and bone.

bone so that the pull on the bone is diffuse. Fleshy attachments tend to be marked by roughened areas on dry bones. A **tendinous** attachment is much smaller in cross-sectional area and thus exerts a more localized pull; tendinous attachments often raise crests or ridges where they attach to bones. The fibres forming tendons intertwine so that a single area in the fleshy muscle body is represented everywhere in the tendinous insertion and as the angle of a joint changes, different parts of the tendon take the pull; this arrangement ensures that full muscle power is available at all times. Attachments may also form flat sheets termed **aponeuroses**. Tendons and aponeuroses appear white because of their high fibre content and low vascularity. Fibrous muscle attachments blend with the periosteum of the bone, but also insert into the bone itself as **Sharpey's fibres**. See Box 2.6 for the effects of soft tissue injury in joints.

Form and function in striated muscles

There is a wide functional variation in the size and shape of muscles and in the arrangement of their constituent fibres. When muscle fibres contract, they can shorten to about one half of their resting length. The potential degree of shortening of a whole muscle belly is therefore determined by the **resting length** of its individual muscle fibres which may be up to 30 cm long in some limb muscles. On the other hand, the potential power exerted of a muscle belly is dependent upon the number and diameter of the muscle fibres it contains per unit of cross-sectional area. The diameter of individual muscle fibres ranges from 10 to 100 µm. Precision muscles controlling the fine movements, such as those of the hand or eye, have many fine fibres per unit area whereas power muscles, such as those of the lower limb or buttock, have fewer larger fibres in each unit of cross-sectional area.

A **motor unit** is a group of muscle fibres innervated by a single nerve process. In precision muscles, each motor unit is small with one process controlling about 10 muscle fibres; in contrast, power muscles have large motor units with ratios of 1:1000 or more. Precision muscles can therefore produce fine gradations in contraction whereas power muscles can generate plenty of power, but with little precision.

Muscles do not suddenly change from relaxation to contraction. At any given time, some motor units will be contracting while others will be relaxing and some will be at rest or recovering. The various activities of different motor units result in continuous activity which can be varied according to the functional demands put on the muscle. Many of our muscles rarely relax completely, but maintain a low level of activity (**muscle tone**) by constantly cycling between contraction and relaxation of individual motor units. It is muscle tone that counteracts the effect of gravity on our mandible, thus stopping our mouths from dropping open all the time. When people are deeply asleep, the muscles may relax so completely that muscle tone ceases and their mouths drop open.

We may have created the impression in the description of skeletal muscle that muscle fibres simply run in parallel from end to end of the muscle belly. This arrangement is comparatively rare and the arrangement of fibres and the connective tissue in which they are enclosed varies from muscle to muscle. The more common arrangements of muscle fibres are illustrated in Figure 2.5.

A simple parallel arrangement of muscle fibres shown in Figure 2.5A is relatively uncommon, but is found in the infrahyoid muscles in the anterior neck (see Section 23.1.3); they have a long range of contraction, but not a lot of power because of the relatively small number of fibres. The fibre mass is increased in muscles where a large number of fibres converge into a tendon so their power increases, as shown in Figure 2.5B. Sometimes muscles have two bellies joined by an intermediate tendon, as shown in Figure 2.5C; the digastric muscle below the chin is the best example. Figure 2.5D and E illustrate other ways in which the mass of muscle fibres can be increased by inserting the fibres into the sides a tendon as seen in the temporalis muscle, one of the muscles of mastication moving the mandible (see Section 24.3.1). As many more fibres can be packed in by this arrangement, the muscles are powerful, but have a limited range of contraction because the individual muscle fibres are shorter. Most muscles have an internal arrangement where there are several small tendons of variable length throughout the muscle (Figure 2.5E) to optimize strength within a limited area. This is not obvious when examining most muscles of the head and neck

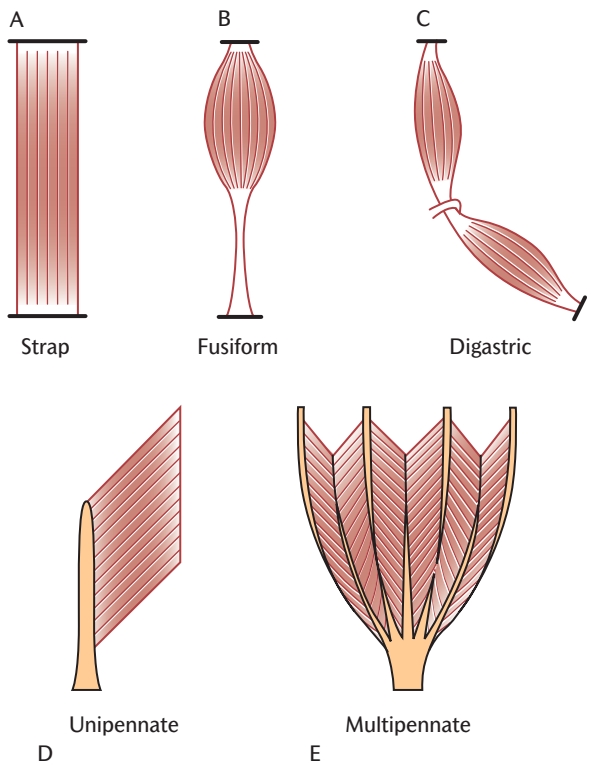

Fig. 2.5 Variations in the arrangement of muscle fibres and connective tissue in striated muscles.

on anatomical specimens and can only be seen by closer microscopic examination of the muscles.

Muscles contract and as they do so, the attachments of the muscle are brought closer together. It is therefore usually straightforward to work out what the action of that muscle must be by observing the attachments of the muscle to bones and the position of the attachments in relation to any joint the muscle may pass over. This is convenient and is the way that most muscles will be treated initially when specific regions are considered later in the book. This approach is a great oversimplification because muscles rarely work in isolation. Our brains think in terms of the intended movement first, then only exert control over individual muscles once the sequence of events has been worked out. Most movements which we perform are therefore a result of the concerted actions of groups of muscles. These groups may be physically close together and act over the same joint or they may be separate and help to control different phases of a complex of movements involving several joints.

Most movements are thus produced by contracting the correct number of motor units in a given muscle to produce the intended movement at a specific joint. The muscle producing this action is the **agonist** or prime mover. If the joint is going to move freely, any muscle that would produce the opposite action, the **antagonist**, must relax to enable the movement to take place. If the agonist and antagonist act together, there will be no movement as their actions will cancel out. However, this action will fix the joint and thus stabilize it and the muscles are now acting as **fixators**. The agonist often generates unwanted movement in a direction not required because of the complexity of joint shapes and of the mechanical effects of the precise position of muscle attachments. This unwanted effect is opposed by **synergists**, muscles working with other muscles during the course of a movement. Any single muscle may play different roles in different movements or sequences; it may be an agonist, antagonist, fixator, or synergist at different times.

Innervation of striated muscles

As we will describe more fully in Chapter 3, certain nerves are classified as either motor or sensory. Put simply, motor nerves convey information from the brain to stimulate muscles to contract whereas sensory nerves convey information about the internal and external environment to the brain. Anatomically identifiable nerves are made up of several individual components. You might therefore anticipate that all the components of nerves supplying a muscle were motor nerves. In fact, only about 60% of the components are motor nerves and the remaining 40% are sensory. What are sensory nerves doing innervating structures designed for motor activity?

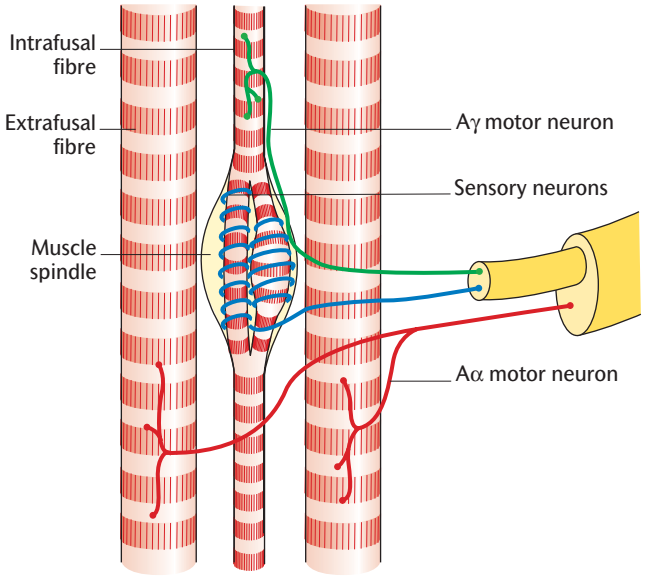

Fig. 2.6 The motor and sensory innervation of striated muscles.

Your brain needs to know precisely what the state of each and every muscle in your body is at any given time if motor movements are to be carried out successfully. The sensory nerves convey this information from muscles to your brain. Study Figure 2.6 as you read this paragraph.

Extrafusal fibres are the contractile muscle fibres that make up the bulk of a muscle; they are supplied by large motor nerves called **alpha motor neurons**. A small number of fine **intrafusal fibres** are scattered within the muscle belly; they lie parallel to the extrafusal fibres and contain complex sensory receptors known as **muscle spindles**. As the extrafusal fibres contract and relax, the intrafusal fibres and the muscle spindles within them will react and convey information about the state of the muscle to the brain. The intrafusal fibres are innervated independently of the extrafusal fibres by small nerves known as **gamma motor neurons**. This enables the length of intrafusal fibres to be reset without affecting the overall action of the muscle. In reality, the

Table 2.1 Muscle fibre types

	Type I	Type IIA	Type IIB
Location	Postural muscles	Powerful movement	
Contraction	Slow	Fast	Fast
Power	Low	Intermediate	High
Metabolism	Oxidative	Oxidative/ glycolytic	Anaerobic/ glycolytic
Fatigue	Fatigue-resistant	Fatigue-resistant	Fatigue easily

Box 2.7 Changes in muscles with use and disuse

Muscles which are heavily used tend to increase in size and power. This is caused by hypertrophy (increase in size) of the individual muscle fibres, not by an increase in their number. Muscles which are not used, for example, in bed-ridden patients or otherwise immobile patients, will atrophy (shrinkage), but the atrophy is potentially reversible once the muscles come back into use.

If a muscle loses its nerve supply through nerve injury or disease, it becomes paralysed and the fibres will be completely relaxed (flaccid); this is flaccid paralysis. If the nerve supply does not regenerate, the muscle will undergo irreversible atrophy.

structure and innervation of muscle spindles is much more complex than the outline given above, which is sufficient to understand the basic principles involved; the detail is more physiological and is not covered.

The mechanism of independent nerve supply of extrafusal and intrafusal fibres enables the condition of the muscle to be continuously monitored and readjusted as loads on the muscle change. You have, by your side, a bottle of soft drink as you read this. Taking the top off the bottle should be easy and you will apply the requisite amount of muscle force that experience has shown you is enough to remove the cap. However, this bottle cap sticks so you need to apply more force by recruiting more motor units. The muscle spindles will tell you this and your brain will do the rest by activating the new recruits through alpha motor neurons. Gamma motor neurons will reset the muscle spindles to the new force applied. Once the cap is loosened, you will require less force to continue removing the cap so muscle spindles will once again feed back this information to the brain. Selected motor units will not be stimulated any more and the muscles spindles will be reset once again. The sensory monitoring of muscle action is another aspect of **proprioception**, previously mentioned in the context of sensation from synovial joints above.

Muscle fibre types

There are several types of extrafusal muscle fibres that differ in their biochemical properties. These differences determine how fast muscle fibres react to nerve stimulation and how long the contraction can be sustained before the muscle fibre fatigues. This depends particularly on whether the muscles use oxygen to derive their energy (oxidative metabolism) or use blood glucose or convert glycogen stored in the muscle into glucose (glycolytic metabolism). Different muscles have different fibre make-up to enable them to perform different functions, but many muscles contain a mixture of types. The different type of muscle fibres are shown in Table 2.1. Changes to muscle due to change in use are outlined in Box 2.7.

3 The central nervous system

Chapter contents

3.1 Introduction

The nervous system is an integrating system which acts rapidly by transmitting signals as electrical impulses over often considerable distances to coordinate bodily activities. The brain and spinal cord make up the **central nervous system (CNS)**; incoming information travels in **ascending** (sensory) tracts that link the spinal cord to the brain and outgoing information passes down **descending** (motor) **tracts** linking the brain to the spinal cord. The CNS integrates responses to incoming information and sends the information to effector tissues (usually striated or smooth muscles or glands). Incoming and outgoing information is carried to and from the periphery to the CNS via 12 pairs of cranial nerves connected to the brain and 31 pairs of spinal nerves connected to the spinal cord; they constitute the **peripheral nervous system (PNS)**.

Sensory (afferent) information from the external environment is obtained through the organs of special sense in the eyes, ears, nose and tongue, and skin and mucosa lining bodily cavities: we are aware of these stimuli. Information from internal sources is equally important and vital for maintaining homeostasis, but we are usually unconscious of it; for example, blood chemistry must be monitored as must the degree of stretch of internal organs. Proprioception, knowledge of the state of muscles and joint position, as described in Section 2.4.1, is another important source of internal information as are our cognitive processes. **Motor** (efferent) stimuli are conveyed from the CNS to effector tissues through the cranial and spinal nerves of the PNS.

The peripheral nervous system is further subdivided into the somatic and autonomic nervous systems. The **somatic nervous system** receives and transmits external sensory stimuli to the CNS and conveys motor responses to striated muscles. The **autonomic nervous system** conveys motor stimuli to smooth muscle and glands and is responsible for homeostasis. The autonomic nervous system has three subdivisions, the **sympathetic**, **parasympathetic**, and **enteric** systems; the first two act on many systems in an antagonistic manner where one system stimulates activity and the other suppresses it. The enteric system plays a role in the control of gut motility.

3.2 The cellular components of the nervous system

Neurons are the basic cellular units of the nervous system. As the principal function of the nervous system is conduction of electrical signals over considerable distances, neurons are highly specialized for this function. Neurons have:

- A specific shape with **long cellular extensions**;
- Highly specialized membranes to **control ionic movements** to allow electrical activity to spread along the cellular extensions;
- A very specialized **internal transport system** to distribute cellular metabolites along the processes.

The general shape of neurons is shown in Figure 3.1. Note first of all, the relatively large **cell body** near the top of the picture; this contains the nucleus and the intracellular organelles necessary for synthetic functions so is similar to any other cell. What make neurons special are the long processes that emanate from the cell body. **Dendrites** are short multiple processes that branch extensively from and transmit impulses *towards* the cell body. Compare the dendrites in Figure 3.1 with the other process, the **axon**, which transmits impulses *away* from the cell body. Axons are generally much thicker than dendrites and there is usually a single axon arising from the cell body; it may branch, but often at some distance from the cell body or as it nears its target. Axons may be extremely long. The cell bodies of neurons conveying information to muscles in the sole of your feet originate in the lower regions of the spinal cord in your lower back. Think of your inside leg measurement from when you last bought a pair of jeans, then add another 9 inches or about 20 cm; that is how long axons can be!

3.2.1 Nerve action potentials

We need to consider some cellular physiology to understand how neurons can transmit signals. All cells in the body possess ion channels in their membranes that allow ions to pass through so that the distribution of positively and negatively charged ions balances out between the inside of the cell and the surrounding extracellular fluid. The most common positively charged ions are sodium (Na^+) and potassium (K^+), and chloride (Cl^-) is the most abundant negatively charged ion in bodily fluids. If positive and negative ions are equally distributed between the cell and

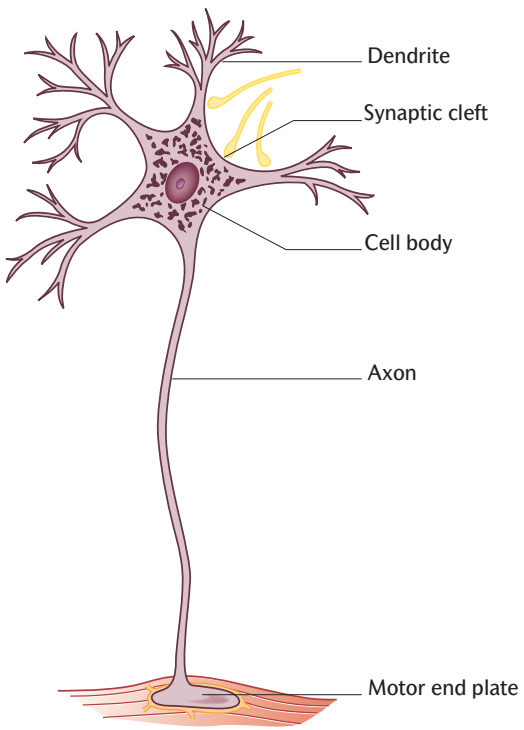

Fig. 3.1 A schematic diagram of a typical neuron.

the surrounding fluid, positive and negative charges balance out so there is no electrical voltage difference across the membrane. If neurons are going to be able to conduct electrical impulses, the distribution of ions must be uneven so that there is an electrical potential difference across the membrane (the **membrane potential**). This is a state of polarization. Figure 3.2A shows the distribution of ions in a neuron at rest. The resting membrane potential across neuronal cell membranes is about –70 mV; this is achieved by forcibly pumping Na^+ ions out of the cell. Sodium channels are closed to prevent Na^+ ions getting back into the cell. The removal and exclusion of Na^+ ions leaves a surplus of Cl^- ions inside making the inside of the cell negative with respect to the outside.

Figure 3.2B shows what happens when a neuron is stimulated. Na^+ ion channels open and allow the excess Na^+ ions to rush into the cell, changing the potential across the membrane from negative to positive. If enough Na^+ ions get in to change the potential difference between the inside and outside of the cell to around +15 mV, the **threshold potential** is exceeded and the electrical activity will become self-generating along the dendrites and axons. This is called an **action potential** or **depolarization** (or 'firing' or nerve conduction). If the threshold potential is exceeded, the neuron will fire; if it is not, it will not generate an action potential. Neurons are basically on–off switches; they can only be on or off. This is known by physiologists as the '**all or none**' rule of nerve conduction.

How does the movement of ions start and lead to an action potential? Ion channels in cell membranes can be opened or closed by three basic mechanisms. The most common **ligand-gated channels** are opened by a chemical signal (for example, a hormone or some other intercellular messenger) binding to a receptor on the cell membrane that opens the channel. Receptors are usually very specific for particular ligands. **Mechanically gated channels** are opened by mechanical changes such as pressure or tension; this is how a touch on your skin is converted into electrical activity in neurons. Finally, and most important in the context of generation of action potentials, ion channels may be opened by changes in voltage across the cell membrane; these are **voltage-gated channels**. The sequence of events is shown in Figure 3.2B. Thus a touch on your skin will cause mechanically gated sodium channels to open and cause depolarization of the neuron at a particular point. As the membrane potential changes in this area, it will cause voltage-gated channels in adjacent areas to open and the action will be propagated along the neuron by opening of further channels. We can now see the chain of events that generate an action potential, usually in dendrites or the cell body, in a single neuron which will propagate along the axon. We must now consider what happens when the action potential reaches the end of the axon.

3.2.2 Synapses

Axons terminate by forming specialized intercellular junctions with dendrites and/or the cell body of another neuron or with effector tissues such as muscles or glands. The junctions between neurons are called **synapses** and those between neurons and muscle are **neuromuscular junctions**. Electrical activity cannot simply jump from one neuron to another or from neuron to effector. This is absolutely vital in preventing the electrical activity spreading at random and generating total chaos in the nervous system. We have already seen that neurons will either generate an action potential or they will not—the 'all or none' rule. It follows that if a neuron is conducting an impulse, there is no physiological way to stop the neuron conducting. Along the chain of neurons that are required to get information from one part of the brain to another or from sensory receptor to effector, the *only* places where information may be modified are at synapses.

Figure 3.3 is a diagrammatic representation of a synapse. The first thing to notice is the **synaptic cleft**, a space between the axon of one

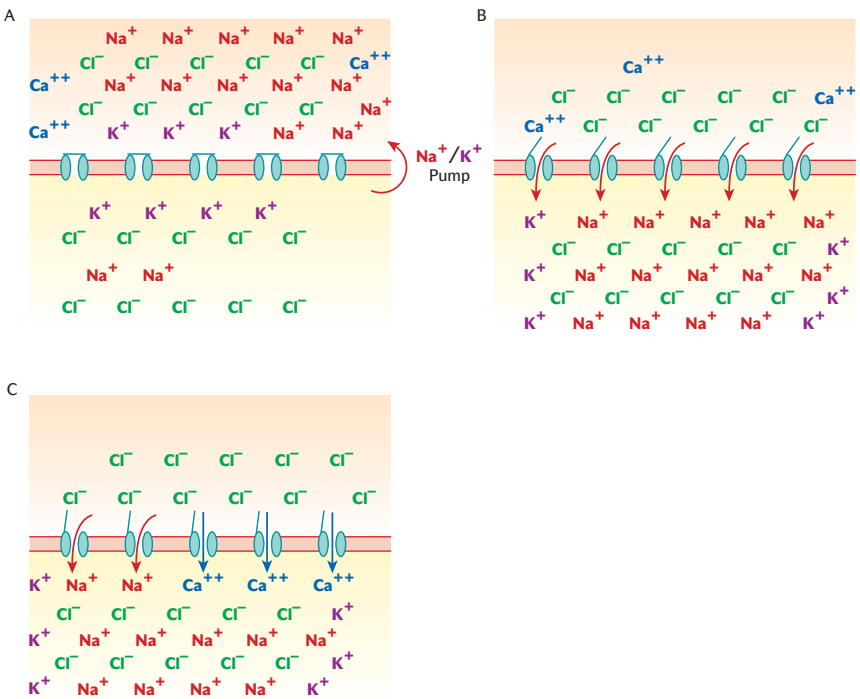

Fig. 3.2 Ionic changes across neuronal cell membranes. A) At rest; B) During depolarization; C) At the synaptic bouton.

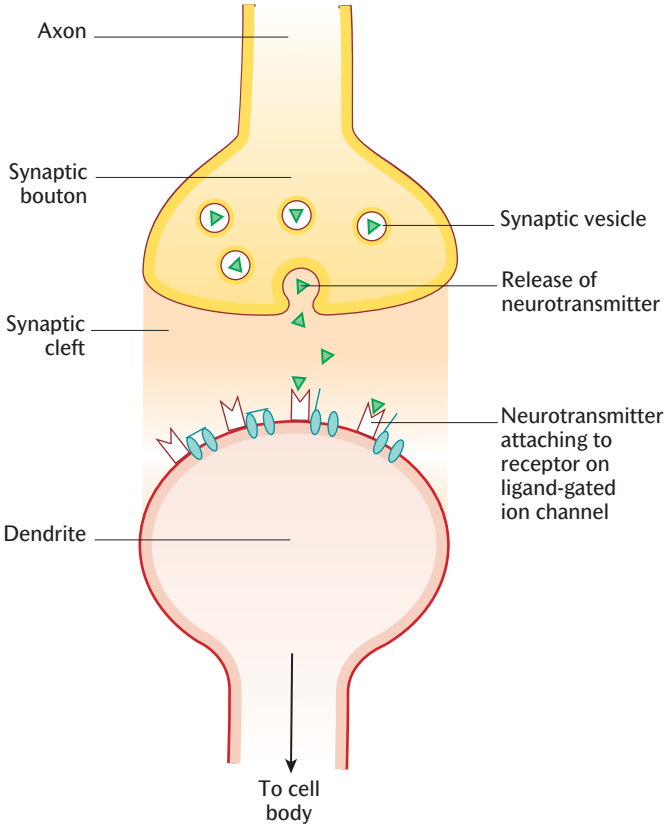

Fig. 3.3 A schematic diagram of a neuronal synapse.

neuron (the **presynaptic neuron**) and the dendrites and cell body of another (the **post-synaptic neuron**). This space is about 400 nm wide, minute, but sufficiently wide to prevent electrical connection. Observe in Figure 3.3 that the axons of the presynaptic neuron are expanded into synaptic boutons occupied by **synaptic vesicles** that contain **neurotransmitters** which are chemical messengers that allow communication between the two neurons. When an action potential reaches this zone of the neuron, the ion channels change to voltage-gated calcium channels. As the wave of depolarization opens these channels, calcium flows into the neuron and facilitates the fusion of the synaptic vesicles with the cell membrane, allowing release of the neurotransmitter into the synaptic cleft. Notice the structures drawn on the post-synaptic neuron in Figure 3.3. The neurotransmitter will cross the synaptic cleft and attach to membrane receptors on the post-synaptic neuron, opening ligand-gated ion channels as they do so. If the ion channels thus opened allow Na^+ ions in, then a wave of depolarization will be set in train as described above. If, on the other hand, the channels were Cl^- channels, then chloride would enter the post-synaptic neuron, making its membrane potential more negative and therefore, more difficult to depolarize (see Figure 3.2). Thus some neurotransmitters will be stimulatory whereas others will be inhibitory, depending on the type of ion channels they open. Any post-synaptic neuron has synaptic connections with several presynaptic neurons so the final balance between inhibitory and stimulatory signals received determines whether a neuron fires or not.

3.2.3 Nerve myelination

Neurons, as we have already seen, may have extremely long processes. They will become less efficient at conducting impulses over long distances unless they are insulated just like electrical cables on domestic appliances. Another advantage of insulation is that it minimizes the risk of cross-talk between neurons travelling side by side. The main insulating material used in the nervous system is a lipid called **myelin** that is wrapped around the axons. Some neurons are covered in this way and are therefore described as **myelinated neurons** whereas others do not have these layers and therefore are **unmyelinated neurons**. Myelin is not produced by neurons themselves, but by the non-excitable supporting cells of the nervous system called **neuroglia** or **glial cells**. Glial cells are, however, not simply insulating cells. They are also involved in the regulation of energy and metabolites reaching neurons. Neurons are very active cells and have large energy requirements. Something like 25% of the energy produced by your body is being used by your brain to process and control whatever your body is doing. The percentage may go even higher if you are reading this while sitting in a chair or lounging on your bed which requires little muscular effort.

Neuroglial cells outnumber neurons in the CNS by about ten to one. They comprise:

- Astrocytes;
- Oligodendrocytes;
- Microglia;
- Ependymal cells.

Figure 3.4 is the same diagram as Figure 3.1, but with glial cells added to the neuron. **Oligodendrocytes** manufacture the myelin sheaths for nerve axons within the CNS, a function taken over by the Schwann cells in the PNS. In Figure 3.4, examine the way in which the myelin-rich plasma membrane of oligodendrocytes wraps around the axon to provide several covering layers, which determines the thickness of the myelin sheath. The more layers there are, the better the axon is insulated. One oligodendrocyte can provide the myelin sheaths for up to 50 axons. **Astrocytes** are present in large numbers throughout the CNS. They have numerous cytoplasmic processes, giving the cells a star-like appearance, hence their name. Look at the structures adjacent to the astrocyte processes on Figure 3.4. Many of these processes terminate on small blood vessels, usually capillaries, while others end close to the surface of neurons; astrocytes are thus able to transport material from blood vessels to neurons. **Microglia** are small cells scattered throughout the CNS. They have a number of short processes and can actively ingest materials and debris, a process called **phagocytosis** that they share with some other cell types. They are part of the macrophage system for non-specific defence by removing dead or invasive material. **Ependymal cells** form a thin layer lining the ventricles of the brain and the central canal of the brainstem and spinal cord that contain cerebrospinal fluid (see Section 15.4.2).

The dotted lines across the axon in Figure 3.4 indicate the point where the axon leaves the CNS and enters the PNS. Note the change in the myelinating cells at this junction from oligodendrocytes to **Schwann cells**. Schwann cells are the only significant glial cells in the peripheral nervous system. They provide myelin sheaths for the peripheral nerve axons in exactly the same way as oligodendrocytes do in the CNS. Even

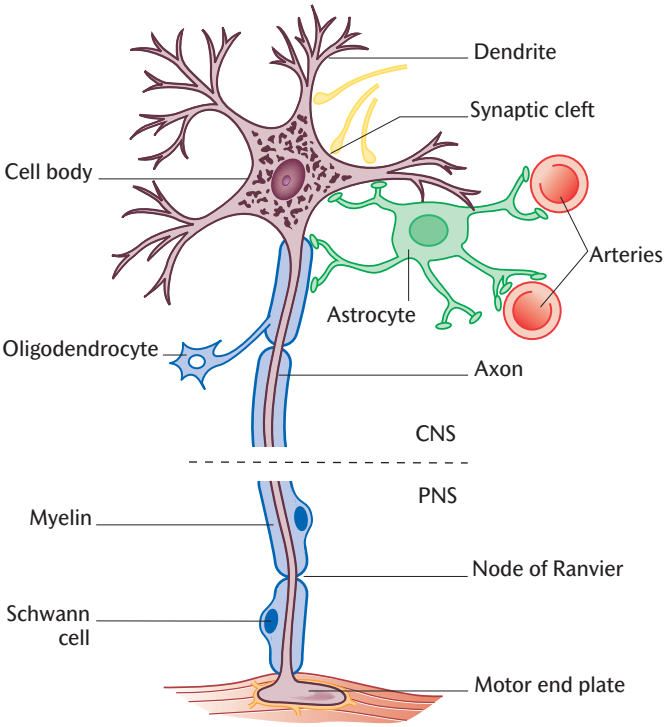

Fig. 3.4 Neuroglial cells and their relationship to neurons.

unmyelinated axons are not naked despite lacking myelin sheaths; they are surrounded by Schwann cell or oligodendrocyte cytoplasm that serves to insulate the axons from one another. Any axon longer than a few micrometres in either the CNS or PNS requires several oligodendrocytes or Schwann cells to cover it. The contributory glial cells form an **axon sheath** around the neuron. As illustrated in Figure 3.4, there is a gap in the myelin sheath called a **node of Ranvier** where the territory of one glial cell ends and another begins. You might perhaps have already wondered how tiny ions are going to get in and out of ion channels on the neuronal cell membrane if they are covered in several thick layers of myelinated glial cell membrane. The answer is that they cannot. However, they can gain access to the neuronal cell membrane at the nodes and this is where all the ion channels are concentrated on myelinated axons. The depolarization jumps along the axon from node to node. This known as **saltatory conduction** and is more rapid and efficient than continuous conduction. The effects of loss of myelin is outlined in Box 3.1.

3.2.4 From neuron to nerve

All neurons have the same general structure described above, but they vary greatly in their detailed size and structure. The diameters of neuronal cell bodies range from as little as 5 µm to more than 100 µm. Axons may be less than a micrometre in diameter or as thick as 20 µm; those larger than 1 µm are usually myelinated. Axons may be less than a millimetre long or approach a metre in length. The number and pattern of branching of the dendrites are particularly variable. Axon branching can also vary considerably, especially within the CNS where significant **collateral branches** are given off to intermediate targets.

Variations in the overall shape of neurons are related to their function. The principal components of long tracts connecting different parts of the CNS are formed from long axons arising from large cell bodies. Long processes also constitute peripheral nerves that can be seen during dissections or are represented on anatomical models. Small neurons, on the other hand, are usually involved in forming extensive local circuits which play a major part in complex neural functions.

Recognizable anatomical nerves forming parts of the PNS that you may dissect vary from thin structures, the diameter of a cotton strand, to the thickest nerve in the body, the sciatic nerve, running through the buttocks to the leg, which is about the thickness of your thumb. Each nerve contains anything from a few tens to several thousand processes. Each optic nerve supplying the eye contains about 1,000,000 processes. The individual processes forming peripheral nerves are bundled together by a sheath of connective tissue called **epineurium** (See Box 3.2).

It is the convention in anatomy textbooks, including this one, to colour nerves yellow in diagrams. This is not an arbitrary choice, but one intended to convey nerves in as life-like a manner as possible. Nerves appear whitish-yellow in life and in cadaveric specimens because myelin is whitish-yellow, thus giving nerves their characteristic hue. Nerves are greyer in tone if only a few neurons in a nerve bundle are myelinated.

Processes form the major part of peripheral nerves. The cell bodies from which these processes extend are not scattered at random, but grouped together into structures called **ganglia** which produce a swelling on the nerve because the cell bodies are larger than the processes. Their exact anatomical location will be described in Section 3.3.

In the CNS, cell bodies, their associated dendrites, and synapses are similarly grouped together, but such a collection is known as a **nucleus**. These components of neurons lack myelin sheaths so nuclei have a

Box 3.1 Demyelinating diseases

Several diseases are due to demyelination of axons which interferes with the efficient propagation of action potentials. **Multiple sclerosis** is a chronic degenerative disease; it may be linked to abnormalities of the immune system, which results in the bodily defence systems attacking their own components (**autoimmunity**). There are extensive patches (plaques) of demyelination in the white matter of the spinal cord and in the optic nerves carrying visual sensation from the eyes, but the axons remain intact. Propagation of action potentials becomes slower and may become impeded altogether because the axons lose their insulation. Initially, the patient may experience weakness and fatigue and abnormal sensations such as 'pins and needles' in the limbs. Visual disturbances occur as the disease develops.

A similar loss of myelin occurs from peripheral nerves in **Guillain–Barré** syndrome. Cranial nerve transmission is severely affected, leading to paralysis of eye movement, facial expression, and muscles involved in swallowing and in the larynx. The effects on the larynx may be so severe that mechanical ventilation is required to maintain breathing.

Box 3.2 Repair and regeneration of peripheral nerves

If a peripheral nerve is cut, the epineurium will retract, pulling the two ends of the nerve apart. In all cases, regeneration is possible from the proximal section of the nerve if the processes are still attached to their cell bodies in the dorsal root ganglia or ventral horns (see Section 3.3). The distal parts of the processes isolated from their cell bodies will degenerate, leaving behind empty myelin sheaths enclosed by Schwann cells.

If the two ends of the nerve are located, they can be rejoined by microstitching through the epineurium. During reconnection of the nerves, there is no way of ensuring that the axon sheaths in the two components of the nerve are in register. Regenerating processes will grow along the sheaths vacated by the degenerate peripheral segments but, unfortunately, will occupy any empty axon sheath irrespective of whether it is the correct one. Thus, the chances of successful connection to their correct targets are tiny since restoration of function is only successful if the regenerating processes reach the right kind of end organs. An axon of a motor neuron aiming for a muscle will not be able to function if it is re-routed to the skin for instance. Regeneration is thus always less than perfect.

If a peripheral nerve is crushed rather than severed, the distal part will still degenerate; the axon sheaths will be flattened initially, but will recover their shape quite quickly. As the proximal part regenerates, it will have an intact sheath so will establish the correct connections. A crush injury will produce temporary paralysis and loss of sensation, but recovery is usually very good, although it may not be 100%.

greyish appearance and hence are known as **grey matter**. Grey matter is the site of synapses, therefore the place where information passing along chains of neurons can be modified. As well as forming nuclei at various places in the brain, grey matter also forms the rim of the brain, the **cerebral cortex**, where various higher functions are processed. Areas of grey matter are connected by axons. These interconnecting tracts appear white due to the presence of the myelin sheaths enclosing the axons, hence they are called **white matter**.

3.3 The peripheral nervous system

The **peripheral nervous system (PNS)** joins peripheral sensory organs and effector tissues such as muscle to the **central nervous system (CNS)**, the brain, and spinal cord. The PNS comprises 12 pairs of cranial nerves and 31 pairs of spinal nerves arising from the brain and spinal cord respectively.

Box 3.3 Spinal nerves: the evolutionary legacy of body segmentation

Our remote evolutionary ancestors were probably a type of segmented worm. Each segment, a repeat of the one ahead and the one behind, was a fairly self-contained slice of the body. Sensation from the skin and control of muscles in each segment is carried out by a pair of **segmental nerves**. The segmental nerves were linked and their activities coordinated as a primitive spinal cord developed.

In segmented animals which are extant today, the muscle in each segment is termed a **myotome**, the skeleton a **sclerotome**, and the skin a **dermatome**. During embryological development of complex animals, these basic components can be recognized in segment-like subunits (see Section 8.35). Despite the modifications to body pattern consequent upon the development of the limbs and tail, the legacy of segmentation still persists in all modern vertebrates. For example, the thorax, as you will see in Section 2, is still arranged segmentally, with each segment containing the same components—a vertebrae, a pair of ribs, segmental muscles, and a pair of spinal nerves—although there are detailed anatomical differences in each segment. This clearly reflects the segmentation of our evolutionary ancestors. Segmentation is also apparent in the distribution of spinal nerves; they only supply muscles that arise from the sclerotome of the same segment and skin derived from the segmental dermatome.

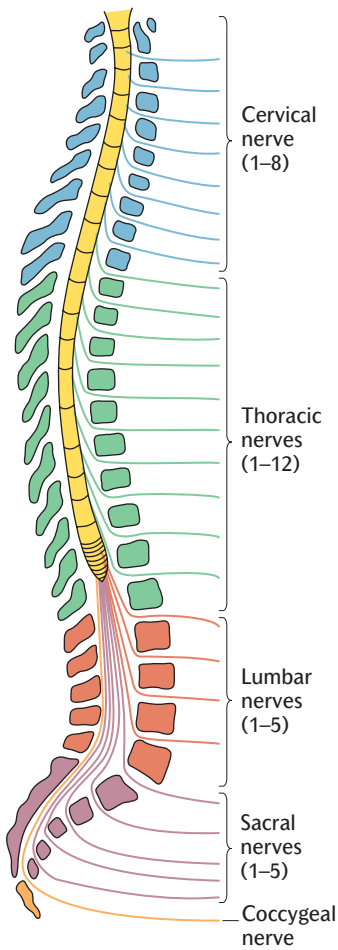

Fig. 3.5 A cross section of the spinal cord and the connections of spinal nerves.

The human spinal cord is enclosed in the **spinal canal** formed by a large central canal in each vertebra. There are seven cervical vertebrae in the neck, twelve thoracic vertebrae in the chest, five lumbar vertebrae in the small of the back, five fused sacral vertebrae in the pelvis, and four coccygeal vertebrae. Look at Figure 3.5 and you can see the spinal nerves leave through foramina formed on each side between adjacent vertebrae; each nerve is lettered and numbered according to the vertebra above. For example the nerve exiting below the fifth thoracic vertebra is T5. There is actually an extraspinal nerve, designated C1, emerging above the first cervical vertebra so the numbering of cervical nerves is related to the vertebra below, with C8 emerging below the seventh cervical vertebra. There are thus eight pairs of **cervical spinal nerves** (C1–8), twelve pairs of **thoracic nerves** (T1–12), five pairs of **lumbar nerves** (L1–5), five pairs of **sacral nerves** (S1–5), and one coccygeal nerve, making 31 pairs in all. See Box 3.3.

The spinal nerves are **mixed nerves** containing **sensory** (or afferent) processes coming in and **motor** (or efferent) axons going out. These two types of processes are mixed together and are indistinguishable from each other. However, they are anatomically separated when they make their connections with the spinal cord. Look at the cross section through the spinal cord illustrated in Figure 3.6. You will see that there is a central H-shaped mass of **grey matter** made up of accumulations of cell bodies, surrounded by **white matter** made up largely of myelinated nerves. This general arrangement of grey and white matter is consistent along the length of the cord. If we concentrate on the grey matter, the upward extensions are the **dorsal horns** and the downward extensions are the **ventral horns**. In reality, the cross section would be orientated horizontally so the dorsal horns would be nearer your back (dorsum) and the ventral horns nearer your belly (ventrum), hence their designation. Now follow the mixed peripheral nerve in towards the spinal cord from the left hand side of Figure 3.6. You will see that the sensory (coloured blue) and motor processes (red) segregate from each other close to the spinal cord. The bundles of sensory processes are destined to enter the dorsal horns and are therefore called the **dorsal roots**. Motor axons begin their journey to the muscles from the ventral horns of the spinal cord in the **ventral roots**. Note the swelling on the dorsal root shown in Figure 3.6; this is where the cell bodies of sensory neurons are grouped together as a ganglion on each spinal nerve dorsal root; these ganglia are called **dorsal root ganglia**.

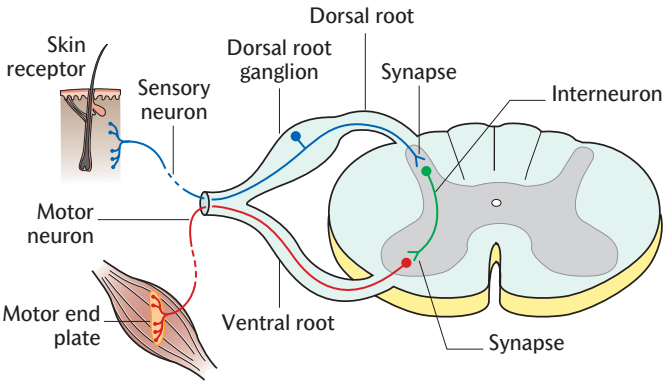

Fig. 3.6 The relationship of spinal nerves to the vertebral column.

The **cranial nerves** do not follow such a reproducible pattern as spinal nerves. Some cranial nerves are mixed nerves like spinal nerves, but some are purely sensory and others purely motor. The cranial nerves will be described in detail in Chapter 18.

3.4 The brain

The brain is the expanded head end of the spinal cord. The major divisions of the brain are shown in Figure 3.7. The two **cerebral hemispheres** are the most obvious and by far the largest parts of the brain. You can see in Figure 3.7 that the surfaces of the hemispheres are highly folded into a series of **gyri** separated by deep clefts called **sulci**. Folding provides a high surface to volume ratio, enabling a lot of tissue to be packed into a relatively small space. The right and left cerebral hemispheres are incompletely separated by a midline central fissure. However, they are joined by a thick band of transversely running axons called the **corpus callosum** which can be seen in Figure 3.7B. The corpus callosum literally lets your left hand know what your right hand is doing. In Figure 3.7A, the **brainstem** emerges from the underside of the cerebral hemispheres to become continuous with the spinal cord below.

The brainstem has several components. The upward continuation of the spinal cord is the **medulla (oblongata)** and its internal structure is similar to that of the spinal cord. The **pons** is the thicker part above the medulla; the axons forming the bulge cross from one side of the brainstem to the other, forming a bridge (Latin: pons = bridge). A short section about 1.5 cm long links the pons to the cerebral hemispheres; this is the **midbrain**. If you compare Figure 3.7A and B, you will see that the midbrain is hidden by the cerebral hemispheres when viewed from the lateral aspect; it is only clearly visible from the medial aspect in a hemisection. As the brain develops, the neural tube, the precursor of the CNS, expands into three sections known as the forebrain, midbrain, and hindbrain (see Section 19.3.1). The forebrain becomes the cerebral hemispheres, the hindbrain becomes the medulla and pons, and the midbrain is the midbrain. Another part of the mature brain develops from the hindbrain. This is the **cerebellum**, a cauliflower-like structure attached posterior to the brainstem and lying inferior to the posterior parts of the cerebral hemispheres. Like the cerebral hemispheres, the surface of the cerebellum is highly folded with very narrow gyri separated by parallel sulci.

The basic anatomy of the brain has been described from above downwards, but in functional terms, the activities of the brain become more complex as we progress upwards from the spinal cord to the cerebrum.

3.4.1 CNS functions

The spinal cord can only function in relatively simple postural reflexes between sensory afferent neurons carrying proprioceptive information from muscles to motor neurons to muscles. In Figure 3.6, the green neuron linking sensory and motor neurons in the grey matter represent **interneurons** which complete a simple reflex arc.

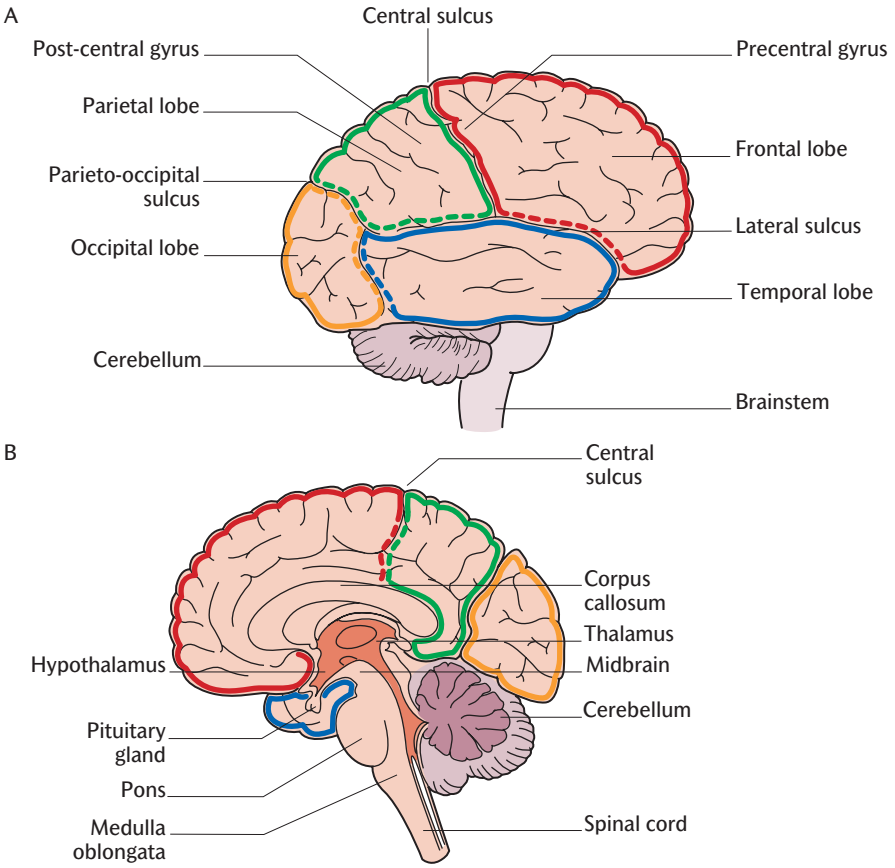

Fig. 3.7 The brain. A) Viewed from the lateral aspect; B) Viewed from the medial aspect.

There are three components to a reflex arc:

- An **afferent** (or sensory) **limb** conveying the stimulus to the CNS;
- A central coordinating centre;
- An **efferent** (or motor) **limb** conveying the response to the effector tissue.

In the well-known 'knee jerk' reflex, the sensory nerves synapse directly on to the motor neurons, but this is very unusual.

The white matter of the spinal cord comprises longitudinally running ascending and descending tracts. Some a**scending sensory tracts** are formed from the axons of post-synaptic neurons located in the dorsal horns that receive information from incoming sensory neurons; others are formed from sensory processes that bypass the grey matter of the spinal cord and enter the ascending tracts directly. They convey sensory information to the brain. **Descending motor tracts** are formed from the axons of neurons whose cell bodies are in the brain. They carry motor information to the motor components of the spinal nerves.

3.4.2 The brainstem

Ascending and descending tracts continue into the medulla oblongata, the lowest part of the brainstem, which is illustrated in Figure 3.8. The medulla is also the level at which the lower six cranial nerves originate. More fundamentally, the medulla contains networks of neurons that form the **respiratory** and **cardiovascular centres.** These centres receive unconscious sensory inputs from various sensory receptors within the cardiovascular and respiratory systems, monitoring blood pressure, blood chemistry, and degree of lung expansion, among other things. Reflex motor outputs from these centres back to the heart and respiratory system regulate their functions in response to altered functional demands placed on these systems by changes in bodily activity. The potential risk to these centres is outlined in Box 3.4.

Box 3.4 Raised intracranial pressure and 'coning'

The brain is enclosed by the skull and the spinal cord is housed in the spinal canal formed by the central vertebral foramen passing through each vertebra. The medulla oblongata and spinal cord become continuous at a large midline foramen, the **foramen magnum**, in the base of the skull (see Figure 20.5). Essentially, this is an enclosed system.

Trauma to the brain that causes haemorrhage or **oedema** (swelling) of the brain produces **raised intracranial pressure** within the closed cranial cavity. Unfortunately, the brain has nowhere to expand and the raised pressure will force the medulla down into the foramen magnum where it will be compressed. The functions of the respiratory and cardiovascular centres are severely compromised as is the function of the two systems they control. This displacement of the medulla is known as **'coning'** in medical parlance. It is a severe medical emergency requiring specialist intensive care to control and reduce the intracranial pressure as swiftly as possible.

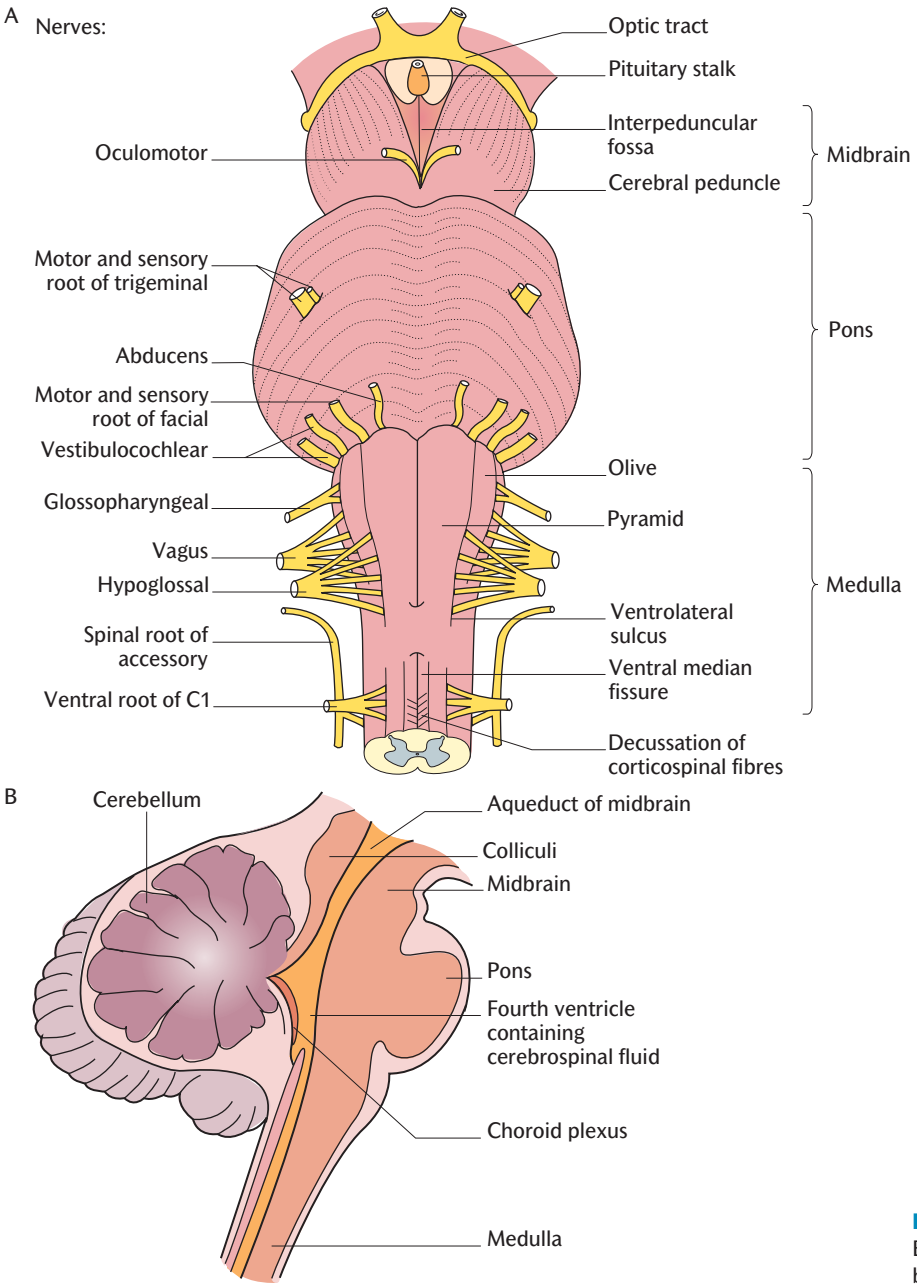

Fig. 3.8 A) The brainstem viewed anteriorly; B) A sagittal section of the cerebellum and brainstem.

Ascending and descending tracts continue longitudinally through the pons, the upward continuation of the medulla shown Figure 3.8. The fifth and sixth cranial nerves emerge from the pons. The pons has important connections to the cerebellum from descending motor pathways. From the anterior aspect shown on Figure 3.8, the midbrain can be seen to divide into two distinct components, the **cerebral peduncles**; each one enters the corresponding cerebral hemisphere, carrying ascending tracts to and descending tracts from the forebrain. The midbrain is also the level of origin of the third and fourth cranial nerves. The **red nuclei**, so called because of their pinkish tinge in a fresh cut brain, nestle within the descending tracts and are important synaptic relays where information from the cerebellum is fed into the motor pathways. The posterior part of the midbrain is not divided like the more anterior cerebral peduncles. This area is known as the **tectum** (Latin: = roof) and has four bulges projecting posteriorly. The **superior colliculi**, the upper pair of bulges, are important areas for coordinating eye, head, and body movements to track objects moving across the visual fields. The **inferior colliculi,** the lower pair, have a similar role in coordinating responses to auditory signals.

3.4.3 The cerebellum

The **cerebellum** receives proprioceptive information from muscles and joints and computes the state of these structures at any given time. When you wish to move, the brain sends signals via motor pathways to the appropriate muscles, but you need to know the state of those muscles

so that appropriate force can be applied. Follow the circuit illustrated in Figure 3.9, linking motor pathways and cerebellum. As motor tracts pass through the pons, they give off collateral axons to pontine nuclei that relay the information to the cerebellum. The information about these muscles computed by the cerebellum from proprioceptive information is then fed back into the motor pathways via the red nuclei in the midbrain.

3.4.4 The cerebral hemispheres

As the cerebral hemispheres constitute such a large volume of the brain, they are subdivided into lobes for descriptive purposes. These roughly correspond to the bones of the skull that overlie them—the frontal, parietal, occipital, and temporal bones (see Figure 20.3). In Figure 3.7, each major lobe is indicated in a different colour. Starting anteriorly, the **frontal lobes** form the front part of each hemisphere; they are covered by the frontal bones and are separated from the **parietal lobes** by the **central sulcus**. The occipital lobes form the posterior poles of each hemisphere. There is no clear demarcation between the parietal and occipital lobes on the lateral side of each hemisphere. The **temporal lobes** are tucked in inferior to the parietal and occipital lobes and are separated from the parietal lobes by the deep **lateral fissure**.

Specific areas that deal with specific functions have been identified in each hemisphere. These are known as primary areas and require a little more anatomical descriptive detail to fix their positions. Figure 3.10 superimposes the primary areas on to the lobes. The **primary motor area** (or cortex) is located in the frontal lobe in the **precentral gyrus**, just anterior to the central sulcus. This area controls movement of voluntary muscles. The **post-central gyrus** posterior to the central sulcus is the **primary somatosensory area** where sensory information from peripheral receptors terminates. The **primary visual area** is on the extreme posterior pole of the occipital lobe, about as far from the eye as it is possible to get and receives visual information. The **primary auditory cortex** is in the superior margin of the temporal lobe where the gyri run transversely, the **transverse temporal gyri**.

The primary somatosensory and motor areas have a **somatotopic** organization. The cortex receiving or emitting signals retains the same general arrangement as the body, except that, as shown in Figure 3.10, it is upside down so that legs are superior followed by trunk, arms, and head. However, as you should be well aware, even before studying anatomy, some areas of your body are extremely sensitive—fingers and lips, for example—and some areas can make extremely detailed fine precision movement such as the fingers and tongue. To make sure that two closely placed sensory stimuli can be discriminated from each other, one sensory neuron supplies only a very few sensory receptors

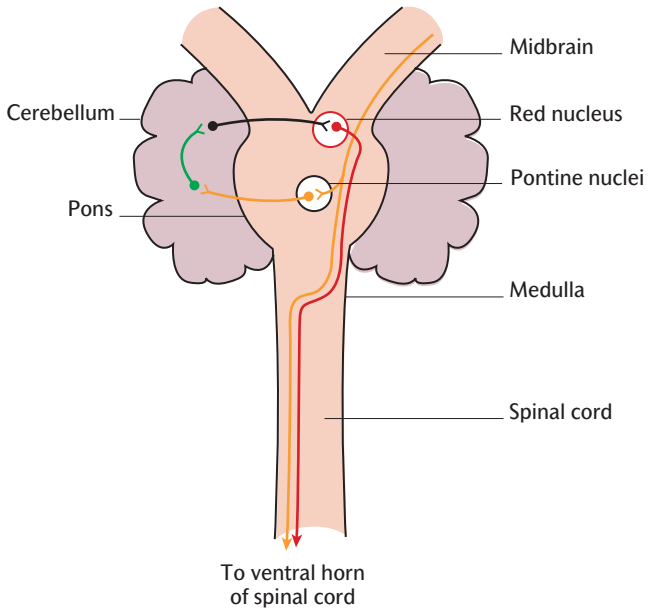

Fig. 3.9 The connections between the cerebellum and descending motor pathways.

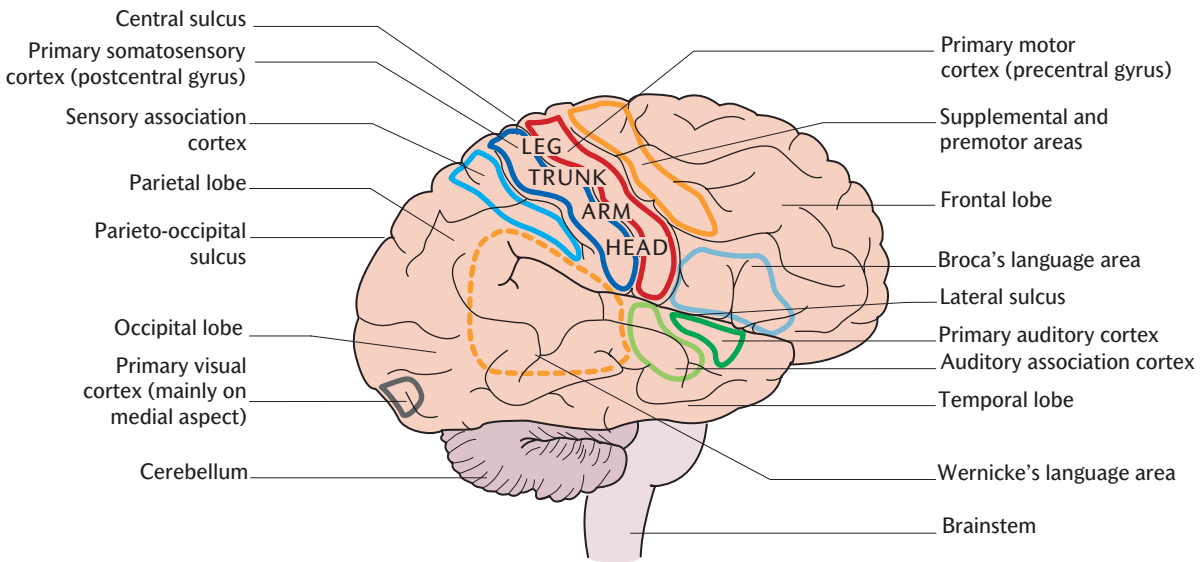

Fig. 3.10 The right cerebral hemisphere viewed laterally, showing the lobes and the primary functional areas.

covering a very small area (or receptive field) of skin or mucosa. Furthermore, the information should not be diluted as it passes from neuron to neuron as it travels from periphery to the somatosensory cortex and should be kept distinct from information coming from an adjacent receptor field. This means that there are a far greater number of neurons entering the relevant part of the sensory cortex from sensitive areas than from those which are not so sensitive. Likewise, if a muscle is going to be precisely controlled, it needs small motor units as we saw in Section 2.5.1 and therefore, requires a dense supply of motor neurons. Dense inputs or outputs occupy far more cortex than less dense ones so the area of cortex used to control our tongue or fingers is huge. In fact, the amount of motor cortex devoted to the control of each finger is as large as the area of cortex given over to controlling the postural muscles of the trunk.

The areas of the cerebral hemispheres between the primary areas are known as **association areas** or **association cortex** and their name indicates what they do. They associate information from different sources and enable you to interpret it correctly. For example, if someone treads on your toe, you will feel pain. However, if the perpetrator apologizes to you and enquires if you are all right, you are likely to interpret things differently than if they snarl 'Out of my way!' and rush on; the somatosensory signal of pain is the same, but the visual and auditory signals that accompany the pain are different.

For the nervous system to perform its integrative function, it must be integrated itself by linking its various components. This is achieved, as already alluded to in previous descriptions, by bundles of axons passing between one area of grey matter and another. These bundles are called **tracts** or **fascicles** in anatomical terminology or, more simply, **pathways** when thought of in functional terms; several different tracts contribute to motor pathways, for example.

3.4.5 Sensory pathways

Sensory pathways are simpler than motor pathways in their general plan. Essentially, a three-neuron chain is required to convey somatosensory information from a peripheral receptor to the somatosensory cortex. The schematic pathway is illustrated in Figure 3.11 and should be followed on the diagram as the description is read. The first, **primary afferent neuron**, carries information from the receptor to the central nervous system—either the spinal cord in spinal nerves or the brainstem in cranial nerves. They synapse in grey matter with the second neurons, the **thalamic projection neurons**, that carry the information to the **thalamus.** The thalamus consists of paired nuclei in the base of the cerebrum just above the midbrain and is shown on Figure 3.7B. Note that thalamic projection neurons cross (**decussate**) to the opposite side in the spinal cord or brainstem. The majority of pathways in the CNS decussate at some point in their course, but it is still somewhat of a mystery why this occurs. The thalamus is a large 'telephone exchange' where messages can be relayed to various parts of the brain. The thalamic projection neurons synapse with the third neurons in the chain, **the thalamocortical neurons**, that carry information to the relevant part of the somatotopically arranged somatosensory cortex. As well as passing the information on to the sensory cortex, the thalamus also sends signals to other parts of the cerebrum to alert them that there is incoming information.

This brief outline of sensory pathways is somewhat simplified at this stage and will be dealt with in more detail in Section 16.2. Note three things about sensory pathways at this stage.

- There are several interneurons located between the major neurons at each synaptic site to allow for convergence or dissemination of information and greater opportunity to inhibit or modify information.
- Not all sensory pathways follow the exact route shown in Figure 3.11, but they still have the basic three-neuron pattern.
- The sensory pathways conveying special senses have their own dedicated pathways and do not obey the same basic three-neuron rule.

3.4.6 Motor pathways

Motor pathways are very complex when compared to sensory pathways. Sensory pathways have a relatively simple job to do in conveying information from one place to another. It is only when the CNS has received and analysed information from various sources that it can formulate an appropriate response. If this requires bodily movement, then motor pathways are involved. Essentially, the final outcome of activity in motor pathways is to move a number of muscles in the correct sequence using the correct force to execute the movement desired. On the face of it, this sounds quite simple until we consider the information required to make specific muscles act correctly.

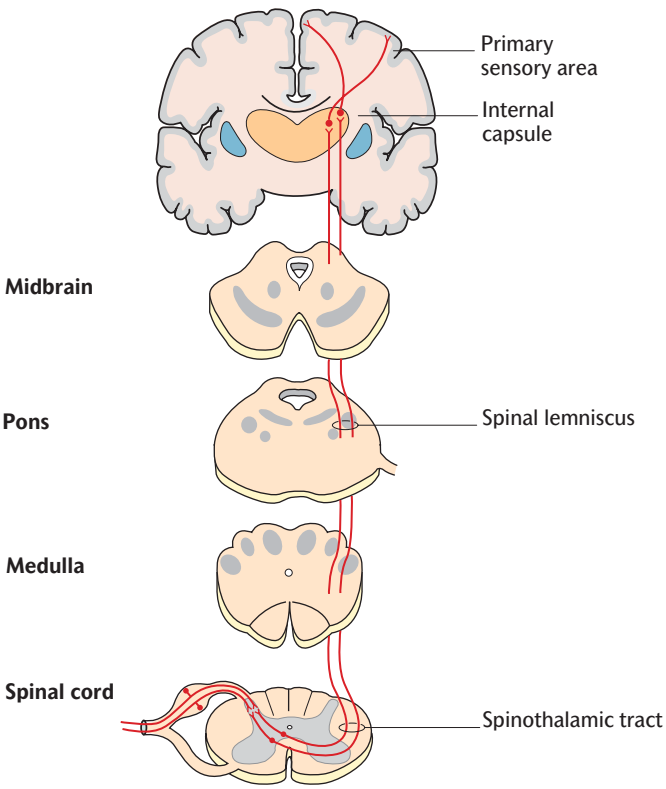

Fig. 3.11 The general scheme of sensory pathways.

As I am writing this, I am sitting at my desk, typing on my computer. I have my legs under the desk and now I am thinking about it, I notice that my legs are crossed. Suddenly, the fire alarm sounds and I must leave my room as quickly as possible. What have I got to do? You may think the answer is simple—stand up, move to the door, open the door, leave the room. Given my position with legs crossed under the desk, I cannot just stand up. I might knock everything over, but more seriously, my legs might be trapped under the desk. Furthermore, if I do not uncross my legs, I will almost certainly fall over—all delaying my escape. Before I even move a muscle, my brain will go through several steps at lightning speed. It will formulate a response to the fire alarm, 'Get out now'. It will put together a programme of events I need to achieve this goal. It will be somewhat like the list above with the first one being 'Stand up—but avoid your desk as you do so'. It will work out the groups of muscles required and the sequence of muscle action needed to stand up. Only then will the information be passed to the primary motor cortex which will recruit the muscles required via the motor pathways.

I will not think about uncrossing my legs because my brain already knows that my legs are crossed from proprioceptive information computed by my cerebellum. It also knows that if I am going to push my chair back and stand up, I need both feet on the floor. My legs will uncross almost by magic but, of course, it is not magic but preplanning by the brain. From experience, I know how far to push my chair back so that my knees will clear the desk when I do stand up and I will use that learned pattern to push back my chair the required distance. We tend to forget that mechanisms within our inner ears monitor balance and head position all the time. We have all had some experience of being dizzy and know how difficult it is to perform even the simplest action if we do not know where we are in relation to our normal reference points.

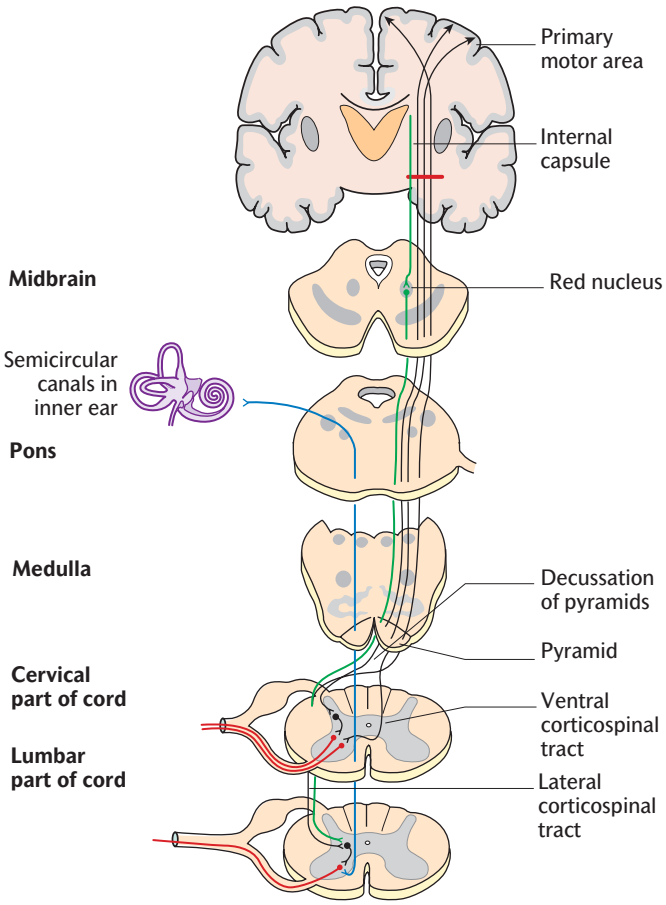

Fig. 3.12 The general scheme of motor pathways.

Box 3.5 Clinical application: upper and lower motor neuron damage

As can be seen in Figure 3.13, if action potentials along the lower motor neurons are interrupted at point B by a **lower motor neuron lesion**, no information from any source can pass to the muscle. The muscles supplied by that nerve will go floppy, a condition known as **flaccid paralysis.** As indicated in Box 3.2, the chances of successful nerve regeneration following cutting of a peripheral nerve are low. If regeneration does not occur, the affected muscles will **atrophy** (waste away) after about six months, which may be noticeable if the muscle is superficial or bulky. If denervation persists after about a year, the muscle will be replaced by fibrous tissue; as this tissue forms, it contracts, producing an effect similar to that of having the muscle contract all the time but; in this case, the muscle can never be relaxed. This causes a distortion called a **contracture**. Contractures may cause functional impairment or disfiguration or both.

In contrast, if upper motor neurons are damaged at, for example, point A in Figure 3.13, no information can get from the brain to the lower motor neurons in the spinal cord and hence to the muscle. We do not get flaccid paralysis because proprioceptive information from the muscle itself can still act on the lower motor neurons by reflex arcs in the spinal cord (Figure 3.6). Thus the muscle will not be totally paralysed and will still contract. However, the reflex proprioceptive activity cannot be modified by information from the brain so the muscles overcontract, a condition called **spastic paralysis**. The effects on spinal nerves of an upper motor neuron lesion within the brain are *always* expressed on the **contralateral** side to the injury because upper motor neurons decussate at the pyramidal decussation.

Note that neurons in the CNS, including upper motor neurons, cannot regenerate if injured so that damage is permanent. You may think that this is very peculiar when most of the body has some regenerative capacity. The reason is that during the development of the nervous system, different tracts develop at different times. To prevent neurons from one tract growing into an adjacent tract and probably making the wrong synaptic connections, the glial cells of the more mature tracts secrete inhibitory substances that turn growing axons away. Unfortunately, this mechanism is not switched off when development is complete so, although axon regeneration takes place, they are inhibited from making synaptic connections by the surrounding glial cells.

Severing of the spinal cord will produce anaesthesia of the skin and paralysis with wasting of the muscles below the level of damage.

Information about balance and head position is required if we are to execute a chosen movement successfully. If you were reading this book sprawled on your bed, you could still get up and leave the room if the fire alarm was activated because you would compensate for your head being in a different orientation from the anatomical position. Proprioceptive mechanisms will continuously feed in information to the motor pathways via the routes through the cerebellum described earlier and illustrated in Figure 3.9.

The basic arrangement of motor pathways is illustrated in Figure 3.12. Note that there are several colour-coded pathways sharing a similar route, but all converging on to motor neuron cell bodies lying in the ventral horn of the spinal cord or their equivalent in the brainstem for cranial nerves. One pathway travels directly from the motor cortex to the spinal cord without synapsing. Note once again that this motor pathway crosses to the opposite side at the **pyramidal decussation** in the lowest part of the medulla. As the motor pathways pass through the medulla, they form bulges on either side of the midline on the anterior surface of the medulla known as the pyramids. These pathways are the **direct corticospinal pathways**. [Note that the nomenclature used to describe nerve pathways is consistent; the first part of the name describes the origin of the tract and the second part the destination. In this case, they originate from the cortex (= cortico) and terminate in the spinal cord (= spinal), giving corticospinal when the two elements are combined.] The **indirect corticospinal pathways** follow the same route as the direct pathways, but as you can see in Figure 3.12, they have an intermediate synapse in the red nuclei of the midbrain as described earlier (Section 3.4.1) where information from the cerebellum is fed into the motor pathways. These tracts are sometimes referred to as the corticorubrospinal tracts, 'rubro' being derived from the Latin word for red. If a tract has three components to its name, this tells you that it has an intermediate point for connections or synapses. **Vestibulospinal tracts** convey information about balance and head position from the inner ear to the motor neurons, the vestibule being the part of the inner ear where these parameters are monitored. The sum of the information passing down the motor pathways determines the effect of motor neurons passing through the peripheral nerves will have on the muscles.

Clinically, pathways that relay motor information within the CNS are referred to collectively as **upper motor neurons.** The motor axons in peripheral nerves transferring information from the CNS to the muscles are distinguished as **lower motor neurons**. Damage to upper motor neurons by disease or trauma produces a different outcome from damage to lower motor neurons by similar causes, as described in Box 3.5 and shown in Figure 3.13.

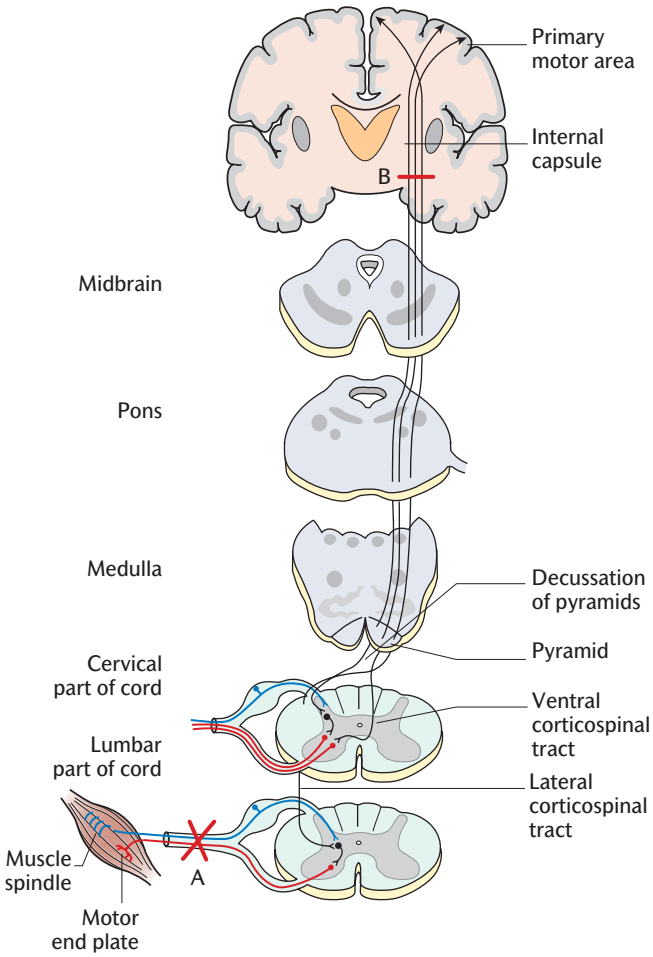

Fig. 3.13 The effects of nerve damage to lower motor neurons (A) and upper motor neurons (B).

3.5 The autonomic nervous system

So far, we have dealt with the part of the nervous system concerned with external stimuli and responses to them. A major part of the nervous system, the **autonomic nervous system (ANS)**, is concerned with the control and maintenance of the internal environment (**homeostasis**). This consists of efferent pathways from the CNS to smooth muscle and glands in the viscera (internal organs) and other structures in the skin involved in thermoregulation. There are some specific areas and centres and associated pathways within the brain and spinal cord controlling autonomic function. The most notable of these is the **hypothalamus**, a small complex of nuclei located anteroinferiorly to the thalamus shown on Figure 3.7B. As well as a role in autonomic function, the hypothalamus controls secretion from the pituitary gland which in turn regulates the function of several hormone-producing endocrine glands.

The ANS is divided into two parts—the **sympathetic system** and the **parasympathetic system**—on anatomical, pharmacological, and functional grounds. The sympathetic system leaves the CNS, with the ventral roots of the spinal nerves leaving from first thoracic (T1) to the second or third lumbar segments (L2, L3) of the spinal cord (the **thoracolumbar outflow**). The parasympathetic system leaves the CNS in some of the cranial nerves and in the sacral spinal nerves (the **craniosacral outflow**). There are also afferent neurons supplying

the viscera, but these are usually designated as viscerosensory nerves and considered as part of the peripheral sensory nervous system so that the term autonomic is usually restricted to just the motor neurons defined above.

If you stop and think for a moment about the previous paragraph, there is apparently a very uneven distribution—sympathetic neurons in the thoracic and upper lumbar regions but no parasympathetic nerves and conversely, parasympathetic nerves in the head and pelvic areas but no sympathetic nerves. This is true of their emergence from the CNS, but not of their subsequent distribution which covers the 'missing' areas in each case. The large area between the head and sacral region is covered by the tenth cranial nerves, the **vagus nerves**, which begin in the medulla oblongata, but travel considerable distances into the lower abdomen, supplying parasympathetic innervation to the thoracic and abdominal viscera. Sympathetic neurons leave the thoracic and lumbar spinal nerves they exit the CNS with to link together as the **sympathetic trunks** on either side of the vertebral column. Each trunk extends up into the neck as the **cervical sympathetic trunk** and down into the pelvis as the pelvic sympathetic trunk to ensure a sympathetic supply to the neck and head and pelvis and legs, respectively.

As you can see on Figure 3.14, in both autonomic divisions, it takes two efferent neurons to carry information from the CNS to the target organ; they synapse at an intermediate autonomic ganglion. Refer to Figure 3.14 as you read the following description. In the parasympathetic parts,

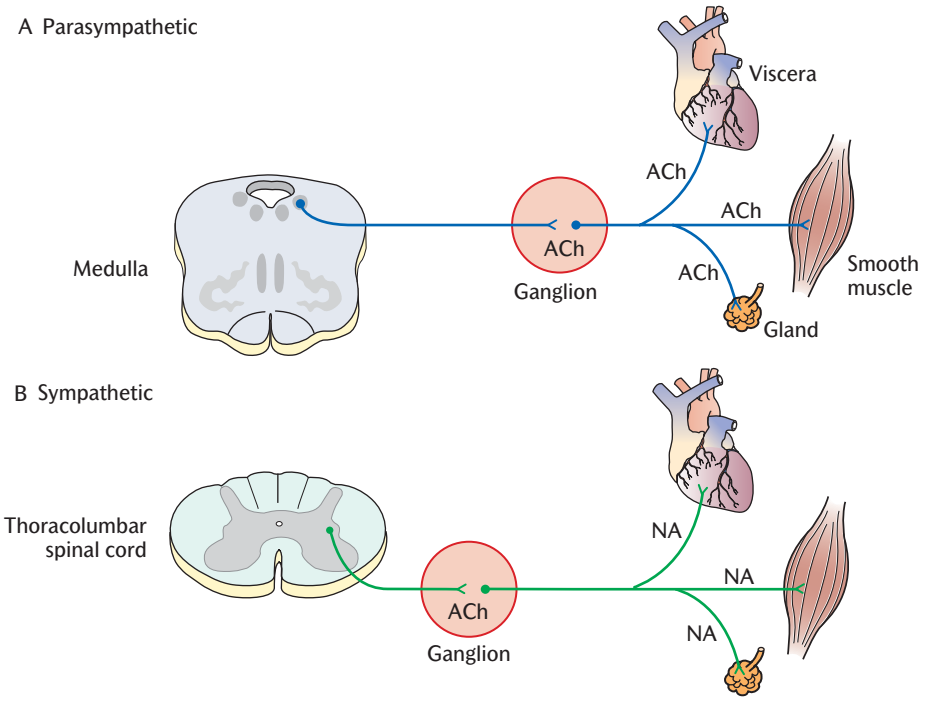

Fig. 3.14 A comparison of the arrangement of: A) Parasympathetic; B) Sympathetic autonomic nerves.

Box 3.6 Drug side effects and the ANS

The functions of the ANS may be modified by drug actions. Many of these are of immense therapeutic value. Blood pressure may be lowered and controlled by the administration of drugs which affect the sympathetic nervous system and lead to vasodilatation of the peripheral blood vessels. These 'beta blockers' block the noradrenalin receptors (β adrenergic receptors) on the target tissues. These drugs, while having a beneficial effect on one aspect of autonomic function, may also interfere with other activities of the ANS, producing undesired side effects. Other totally unrelated drugs can also have a **sympatheticomimetic** effect by either blocking the parasympathetic nervous system or, as the name suggests, mimicking the effect of the sympathetic nervous system. The outcome is the same irrespective of mechanism of action.

From a dental point of view, the most important side effect of sympatheticomimetics is the inhibition of saliva flow, producing a dry mouth or **xerostomia**. We have all experienced a dry mouth when we have been nervous, before an interview, for example. Clinically, xerostomia is when salivary production is below 50% of normal output. With low salivary flow, people find it difficult to chew, swallow, and talk. The oral hygiene functions of salivary are also lost, resulting in rapidly advancing caries, periodontal disease, and opportunistic oral fungal infections. There are about 500 drugs with sympatheticomimetic side effects in the current British Pharmacopoeias and it is a serious problem for people on medication from beta blockers, decongestants for respiratory problems, and some anti-depressants, to name but a few categories of sympatheticomimetic drugs.

illustrated in Figure 3.13A, the preganglionic neurons leave from specific cranial nerve nuclei in the brainstem or sacral spinal nerves. The preganglionic neurons are usually quite long because the ganglia in the parasympathetic system are usually situated in or near the target organs or tissues being supplied. Compare Figure 3.13A and B and you will see that in the sympathetic system, the cell bodies of the **preganglionic** neurons are located in the spinal cord in the intermediolateral horns between the dorsal and ventral horns. Their relatively short axons synapse with the second **post-ganglionic** neurons in sympathetic ganglia formed by the cell bodies of the post-ganglionic neurons. They form a chain of ganglia either side of the vertebral column known as the **sympathetic trunks**.

As Figure 3.14 indicates, pharmacologically, **acetylcholine** (ACh) is the neurotransmitter used at autonomic ganglia and at the effector organ in the parasympathetic system. **Noradrenalin** (NA) is the neurotransmitter used between post-ganglionic neurons and effector organs in the sympathetic nervous system.

Under normal circumstances, the sympathetic and parasympathetic systems act in concert to maintain an appropriate level of visceral activity and maintain homeostasis although their effects are often antagonistic to each other. For example, sympathetic stimulation of the heart will increase heart rate whereas parasympathetic stimulation of the heart will decrease it. By appropriate stimulation of the heart through the two systems, the heart rate can be adjusted to meet the functional demands placed on it by changes in bodily activity.

Wholesale stimulation of the sympathetic nervous system prepares the body for an emergency. It accelerates the heart, constricts the peripheral blood vessels, and raises the blood pressure. It also inhibits activity in the digestive tract, including drying up saliva in the mouth. The result of these activities are a redistribution of the blood so that it leaves the skin and intestines and becomes available to the brain, heart, and skeletal muscles ready for you to run away or fight your way out of the threatening situation. The extreme sympathetic stimulation is known as the **'flight or fight reaction'**. Once the threat has passed, the parasympathetic nervous system reverses these trends to conserve energy and restore normal function. It slows the heart and increases activity in the intestines.

Many drugs have side effects that can interfere with normal functions of the ANS as outlined in Box 3.6.

4

The circulatory system

Chapter contents

The circulatory system has two interrelated, but distinct parts, the **cardiovascular system** which circulates blood around the body and the **lymphatic system** which returns excess fluid from the tissues to the cardiovascular system.

4.1 The cardiovascular system

The function of the cardiovascular system is to oxygenate blood in the lungs and distribute the oxygenated blood to the tissues of the body. At the same time, carbon dioxide that accumulates as a result of metabolism of oxygen within the tissues is removed from the tissues and transported to the lungs where it is released from the blood and exhaled. The cardiovascular system comprises the heart, a muscular pump, and blood vessels. Arteries convey blood to thin-walled capillaries where gaseous exchange takes place and veins return blood to the heart.

The cardiovascular system is often described as two parallel systems; **the pulmonary circulation** moves blood through the lungs and **the systemic circulation** circulates blood through the body. Trace the circulation of blood through the two systems in Figure 4.1 by following the arrows from the side of the heart coloured red. It follows a figure-of-eight (8) pattern with the two systems interlinked at the heart, the upper loop representing the pulmonary circulation and the lower loop the systemic circulation.

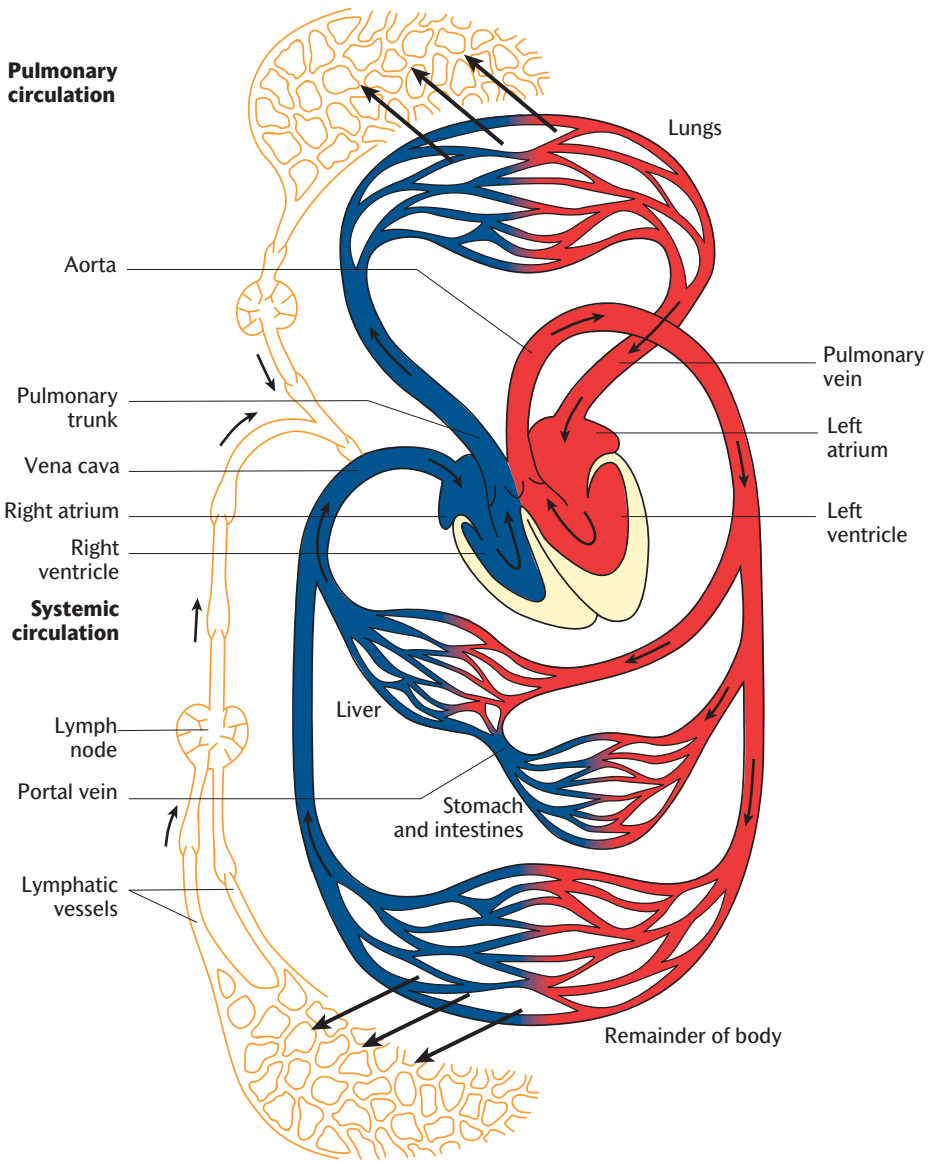

Fig. 4.1 A diagrammatic view of the cardiovascular and lymphatic systems. (Note that arteries and oxygenated arterial blood are demarcated in red and veins and deoxygenated blood are indicated by blue in this and other figures showing blood and blood vessels.)

The heart is a muscular pump driving blood at considerable pressure through arteries that get progressively smaller in both circulations until capillaries are reached. Arteries are sometimes dismissed as mere plumbing, but they play a vital role in regulating the blood flow through organs and tissues (see Section 4.1.2). Capillary walls are only one cell thick, allowing for the efficient diffusion of gases and small nutrient molecules to and from tissues. Waste gases and metabolites are also returned to the circulatory system through capillaries and these unite to form veins carrying blood under comparatively low pressure back to the heart.

4.1.1 The heart

The heart comprises two muscular pumps arranged in parallel and beating in unison. As you can see in Figure 4.1, these two pumps are designated as the right and left sides of the heart. Each pump consists of two chambers, a thin-walled **atrium** that receives blood from one or other circulation and a thick-walled **ventricle** that ejects blood into the circulations.

The walls of the heart chambers are made of a specialized type of muscle tissue known as **cardiac muscle**. Cardiac muscle fibres are branched so that impulses can travel from one fibre to another through specialized intercellular junctions called **intercalated discs** that allow the passage of small ions which cause muscle contraction. The contractile proteins that make up cardiac muscle are fatigue-free so that your heart can continue beating at an average of around 70 beats per minute throughout life without rest. The atria and ventricles contract in series (one after the other), but the right and left atria beat synchronously followed by the right and left ventricles contracting together. The coordinated contraction of the chambers of the heart is managed by the **conducting system.** The sequence of events can be followed in Figure 4.2. Waves of depolarization spread from the **sinoatrial node** (also known as the **SA node** or **pacemaker**) across the atria through cardiac muscle fibres, ensuring both atria beat together. The wave of muscle contraction quickly reaches the junction between the atria and ventricles, but can progress no further; there is a non-conducting band of fibrous tissue supporting the heart valves that is also made of connective tissue that prevents electrical connection between the upper and lower chambers. As shown in Figure 4.2, the **atrioventricular node** (**AV node**) sits just above this band and receives the contractile stimuli that have spread across the atria. In response, the AV node depolarizes and the current passes down a bundle of **conducting fibres** made from modified cardiac muscle cells that lack contractile proteins, the only electrical conduit between the atria and ventricles. The current passes down the wall separating the ventricles to their base, then upwards across the ventricles. The delay between impulses leaving the SA node causing atrial contraction and reaching the AV node and stimulating ventricular contraction ensures that the ventricles are filled completely by atrial contraction before they empty. The conducting system receives inputs from the autonomic nervous system to regulate heart rate and cardiac output according to functional demands. It is described in more detail in Section 12.4.4.

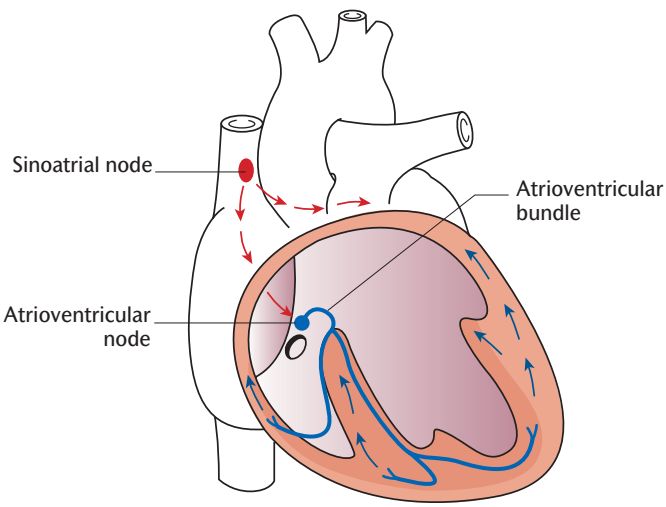

Fig. 4.2 The conducting system of the heart.

Blood leaving the ventricles enters the great vessels, the **pulmonary trunk** leading to the lungs and the **aorta** feeding the systemic circulation. The entrances to both these vessels contain pocket valves to prevent backflow when the ventricles relax, as shown in Figure 4.3; they are known as **semilunar valves** because each valve flap is supposedly shaped like a half moon.

The right side of the heart receives deoxygenated blood (shown in blue in Figure 4.1) from the body and transmits it to the lungs; the left side receives oxygenated blood (shown in red) from the lungs and transmits it to the body. The blood, therefore, passes alternately round the smaller **pulmonary circulation** and the larger **systemic circulation**. Trace the circulation of blood in these two systems again in Figure 4.1, noting the names of key vessels in these circulations. For the **pulmonary circulation**, begin at the right atrium of the heart; blood circulates from **right atrium → right ventricle → pulmonary trunk → pulmonary arteries → pulmonary capillaries → pulmonary veins → left atrium.** Blood passing through the pulmonary circulation is returned to the left atrium in the **systemic circulation.** In Figure 4.1, starting where oxygenated blood is returned to the heart by the pulmonary circulation, blood travels from **left atrium → left ventricle → aorta → arteries → capillaries → veins → venae cavae (→ right atrium).**

Both atria are thin-walled because they are only pumping blood a very short distance to the wide ventricles; the muscle in the atria only has to overcome the resistance to open the **atrioventicular valves** that close again to prevent backflow of blood from ventricle to atrium. Each ventricle has a similar capacity of 60–70 ml, but the resistance to blood flow in the pulmonary circulation is less than that in the systemic circulation because the lungs are large, open, sponge-like structures whereas the systemic tissues and organs are comparatively dense. The wall of the left ventricle is, therefore, considerably thicker than that of the right to generate more force of contraction to overcome the greater resistance.

The effects of occlusion of blood vessels are described in Box 4.1

4.1.2 The systemic circulation

The main systemic artery, the **aorta**, is about 30 mm in diameter (about the thickness of your thumb). It gives off numerous branches which, in their turn, branch repeatedly. The branches become progressively

Box 4.1 Cardiovascular disease

The commonest contributing factor to death in developed countries today is **atherosclerosis**, a degenerative change in vessel walls which may lead to their partial or complete occlusion. This disease may attack any artery, but is particularly prevalent in the **coronary** and **cerebral arteries** supplying the heart and brain respectively.

The resulting diminished blood supply to the heart muscle causes severe cramp-like pains called **angina** (**pectoris**) or death of a segment of the heart wall (**myocardial infarction)** if the blockage is complete. These are commonly known as a 'heart attack' or 'coronary' (see Box 12.3). The causes of deposition of atherotic plaques are not fully understood, but there is compelling evidence that too much animal fat in the diet, resulting in high blood cholesterol, obesity, and smoking are causative factors. Heart attacks are usually associated with those over 50, but can and do occur earlier in life; indeed fatty streaks are often present in the arteries of the newborn.

Blockage of cerebral arteries causes a **cerebrovascular accident (CVA),** more commonly known as a 'stroke' if rapid (see Box 15.10) and may contribute to dementia if gradual.

smaller in diameter as they become more numerous. The smallest vessels which may be just visible to the naked eye are **arterioles** (about 0.3 mm diameter); they break up into **capillaries** which are 0.5–1 mm long and only just wide enough to allow red blood cells to pass through in single file (the diameter of red blood cells is 7 μm). The capillaries reunite to form **venules** which unite to form progressively larger **veins**.

The blood in arteries is under considerable pressure. Arteries have smooth muscle and elastic connective tissue in their walls. The very large arteries near the heart (often called the great vessels) have a very high proportion of elastic tissue; this allows the vessel to distend as each pulse of blood is pumped out of the heart and then recoil, helping to smooth out the pressure in the great vessels. Smaller arteries further from the heart have a greater proportion of smooth muscle. The diameter of these **muscular arteries** is accurately monitored by sensors in their walls and controlled by the autonomic nervous system so as to regulate blood flow according to temperature, body activity, and other physiological variables. Blood flow at a more intimate level is controlled by resistance vessels called muscular arterioles, ending in **precapillary sphincters** formed from the smooth muscle in their walls; these provide the chief resistance to systemic blood flow.

The blood then enters a network of **capillaries.** Capillaries usually have complete walls only one cell thick. Other capillaries are **fenestrated** with 'windows' about 100 μm in diameter in their walls that allow the passage of quite large molecules; fenestrated capillaries are found in the kidneys, the intestinal walls, and in connective tissue underlying epithelia. **Sinusoids** are specialized capillaries with large holes in their walls and a tortuous course which slows down the flow of blood, allowing maximum opportunity for the exchange of substances between blood and tissues. Sinusoids are found in the liver and tissues in the lymphatic system.

Capillaries unite to form post-capillary venules that drain into a network of low-pressure, high-volume veins which return blood to the heart. Veins are larger in diameter than the corresponding arteries and have a lower rate of flow. Veins are often surrounded by a dead space occupied by loose connective tissue into which they can expand when full of blood. Venous pressure is so low that even gravity can interfere with the flow of blood back to the heart. In areas where the venous return is against gravity, reverse flow is presented by the presence of venous **valves**. These simple pocket valves illustrated in Figure 4.3 allow flow towards the heart, but fill with blood and occlude the lumen of the vessel when reverse flow starts. They operate just like the semilunar heart valves mentioned in Section 4.1.1. Valves are plentiful in the veins of the limbs, but are infrequent in the head and neck. Venous return is aided by the action produced by the contraction and relaxation of the muscles around the vessels, **muscular pumps**. This is why soldiers standing for long periods on ceremonial duty are trained to flex their leg muscles to maintain venous return and passengers on long-haul flights are encouraged to move their calves while sitting for long periods to avoid deep venous thrombosis described in Box 4.2. The effects of raised blood pressure on the circulation are outlined in Box 4.3.

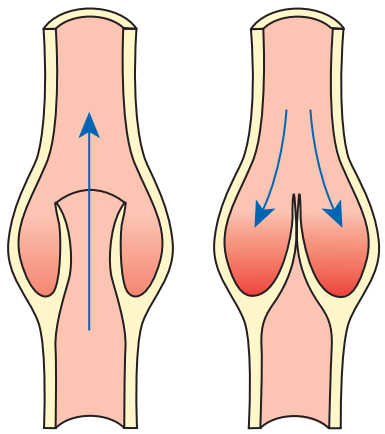

Fig. 4.3 A diagram of semilunar valves found in the heart and veins.

Box 4.2 Venous thrombosis

Veins may be blocked by **thrombi** (clots). In superficial veins, they cause phlebitis, inflammation of the vessel walls. **Deep venous thrombosis** is potentially more serious because of the risk of the clot breaking up and being transported elsewhere, notably to the lungs, brain, or heart, where they may block vital blood vessels. These thrombi are most common in the large venous sinuses of the muscles in the calf and may occur in bedridden patients. Anticoagulants are often given after operations to decrease the risk of thrombosis occurring.

Varicose veins are a common, painful, and disfiguring condition in which the vessels become irregularly dilated, tortuous, and lengthened. The condition is commonest in the lower limb due to incompetence of venous valves, allowing reflux and stasis of blood. This stasis may lead to ulceration.

4.1.3 Anastomoses

Smaller arteries often unite to form a branching network before entering capillaries. These networks are called **anastomoses** and can occur between branches of equal or unequal size. Anastomoses allow an alternative source of blood from two or more arteries to enter a capillary bed so that if a single artery is occluded by pressure, disease, or damage, the blood supply is not interrupted. A **collateral circulation** provides an alternative route for arterial blood, often by following small vessels by very indirect routes. They are especially common around joints where postural changes might temporarily occlude vessels. If a major artery is slowly blocked by a disease process such as atherosclerosis, a collateral circulation may provide a maintenance blood supply sufficient to prevent tissue damage. Over a relatively short period, the vessels forming the collateral circulation will increase in diameter until they are carrying the same amount of blood as the original route. However, a sudden blockage may cause **avascular necrosis** if the collateral circulation is inadequate or non-existent. For example, the **coronary arteries** supplying the muscle of the heart wall and the conducting system of the heart have **potential anastomoses** between them which are capable of opening up to some degree if the blockage of a major vessel takes place slowly but not if it occurs suddenly. Given that cardiac muscle only carries sufficient metabolic reserves to maintain muscle function for about 15 minutes, avascular necrosis will ensue fairly rapidly following sudden occlusion of a coronary artery (a heart attack or cardiac arrest). **End arteries** have no anastomotic connections between feeder arteries; a capillary bed is supplied by a single artery so if that supply is cut off, the tissue nourished by that capillary bed will die. The central artery of the retina, arteries supplying the brain, and the arteries of the kidneys are all end arteries.

Vascular shunts

In many areas, blood may be routed directly from arterioles to veins without passing through the capillary bed. As mentioned in Section 4.1.2, arterioles have **precapillary sphincters** that can narrow or even close down the vessel. At times of low demand by the tissue supplied by the capillary bed in question, most of the precapillary sphincters will be closed so that only a maintenance supply of blood passes through the capillary bed. When demand is high, most sphincters will be opened.

In other cases, direct **arteriovenous anastomoses** may be present between smaller arteries and veins. When open, much of the blood flows directly from artery to vein thus bypassing the capillary bed. Arteriovenous anastomoses in the skin play an important part in temperature regulation. Blood is diverted away from the skin in cold conditions by closing precapillary sphincters to force blood through the arteriovenous anastomoses. When it is hot, blood is directed through the skin capillaries to allow heat exchange between blood and the external environment. This is why your skin flushes when you are hot, but turns white or even blue when you are cold.

4.1.4 The pulmonary circulation

The general arrangement and structure of the pulmonary circulation are similar in essence to those of the systemic circulation. The principal difference is terminal arteries opening directly into the capillary bed without intervening arterioles. Functionally, this means that there is a limited capacity for controlling the distribution of blood flow within the lungs.

Box 4.3 Hypertension

Hypertension or raised blood pressure is one of the commonest diseases affecting the circulatory system. In **essential hypertension,** cardiac output is normal but peripheral resistance within the tissues is increased. Features of untreated hypertension can include enlargement of the left ventricle which has more work to do to overcome the increased resistance, increase in size and thickening of the walls of blood vessels, headaches from thickened blood vessels in the brain, and eventual damage to kidney tissue which is fatal if untreated. The risk of stroke and heart attack is increased in patients with hypertension.

Blood pressure can be controlled by drugs which interfere with sympathetic nerve function which regulates blood flow by narrowing arteries (**vasoconstriction**) in most areas of the body. Blood vessels dilate due to the internal pressure, thus lowering the peripheral resistance if sympathetic activity is inhibited.

4.2 The lymphatic system

The tissue fluid which bathes the cells and fibres of the body is a clear fluid formed as an exudate of blood plasma in capillary beds. The cells obtain their nutrients from the tissue fluid and excrete their waste products into it. Lymph commonly contains phagocytic cells capable of ingesting bacteria and other foreign bodies; the cells enter tissue spaces from the bloodstream and return via lymph. There is a constant turnover of metabolites between the tissue fluid and the blood in the capillaries. Most of the tissue fluid returns to capillaries and venules, but 10–20% remain in the tissues. As fluid is constantly leaving the circulatory system and entering the tissues, the tissues would rapidly become **water-logged** (**oedema**) if the excess fluid was not drained away. As illustrated in Figure 4.1, the excess fluid is collected as **lymph** by lymph capillaries which coexist with blood capillaries in the capillary beds. Lymph capillaries unite into larger lymph vessels which are small diameter, thin-walled vessels with valves at frequent intervals. Lymph vessels tend to follow the same course and drain the same territories as veins. In common with venous return, lymph flow is normally maintained by muscular pumps exerting intermittent pressure on the valved lymphatic vessels during movement. Negative or positive pressure generated in the thorax and abdomen during breathing may also play a part.

As shown in Figure 4.1, lymphatic vessels eventually converge into major lymphatic vessels in the thorax, which in turn empty into the major veins entering the heart thus returning lymph to the cardiovascular system. Even major lymphatic vessels are still surprisingly small and difficult to locate.

4.2.1 Lymph nodes

Lymph nodes are small, bean-shaped bodies situated along the course of lymphatic vessels, especially at junctions such as the armpits or groin. Lymph passes through at least one set of lymph nodes as it flows from the tissue to re-enter the bloodstream. Figure 4.4 shows how lymph draining the tissues enters around the periphery of the node and is filtered through masses of densely packed **lymphocytes**, a type of white blood cell, represented by the shaded areas. Lymph leaves from the hilus**,** a slight depression on the opposite side of the node; the hilus is also where blood vessels enter and leave. The node is surrounded by a capsule which sends radial bands of fibrous tissue into its substance to support the aggregates of lymphocytes.

Antigens, foreign material carried in the lymph, are processed in a complex series of interactions between phagocytic cells and subtypes of lymphocytes, which results in one of two types of **immune response**. Lymphocytes may respond by dividing and producing antibodies which bind specifically to a particular antigen to neutralize it, thus rendering it harmless—this is **humoral immunity**. Humoral immune responses are made when bacterial infections are detected. In **cell-mediated immunity**, killer lymphocytes are produced by cell division in the lymph node; as their name suggests, these lymphocytes are capable of destroying specific antigenic tissues. They enter the circulation where they will eventually home in on the foreign tissue and destroy it. Cell-mediated immunity will occur when an organ transplant is performed unless the immune responses are prevented by immunosuppressive drugs. Box 4.4 outlines the role of the lymphatic system in the spread of infection and cancer.

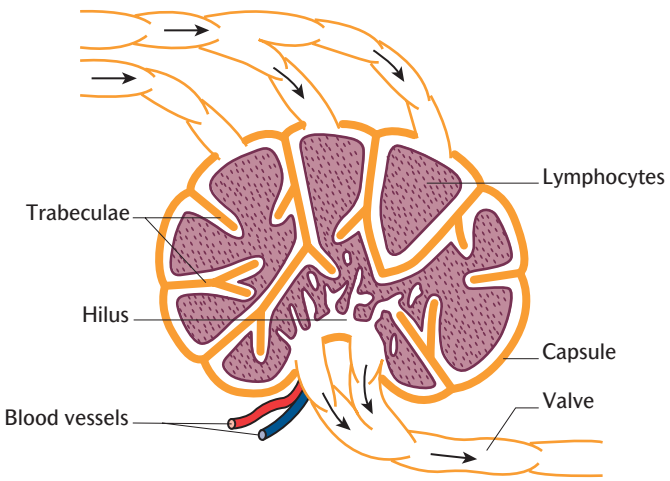

Fig. 4.4 A diagram of a lymph node.

Box 4.4 The role of the lymphatic system in infection and cancer

The lymphatic system is primarily a beneficial defence system, generating non-specific reactions (phagocytosis) and specific immune response to defend and protect the body from invasion by foreign organisms. However, lymphatic vessels can also act as a distributor of disease throughout the body.

An infection which overwhelms local tissue defences may spread down lymphatic channels. If the infection is superficial, the vessels may become reddened and visible as they become inflamed (lymphangitis). When the infection reaches lymph nodes, they will become swollen as lymphocytes multiply to make an immune response (**lymphadenitis**) and may be palpable if the nodes are superficial. A knowledge of which tissues and organs drain into a particular group of lymph nodes, the **regional lymphatic drainage**, is useful for detecting infections that may not be visible such as those in the pulp cavity of teeth (see Sections 23.2.8 and 25.4).

Malignant cancerous cells lose the mechanisms that keep cells attached to each other. As a result, cells may migrate from the primary cancer to invade other tissues and organs. Invasive cells may settle at sites distant from the primary tumour and form **metastases** (secondary tumours). A very common route for cancerous invasion is through lymphatic vessels; metastases are often located in lymph nodes. When cancer is detected, the lymphatic drainage should also be carefully examined by palpation where possible and imaging methods to assess whether any invasion or metastasis has occurred. Treatment of cancer aims at removing the primary malignancy and also real or potential secondary growths. This may be achieved by surgery, radiotherapy, chemotherapy, or a combination of treatments. The lymphatic system itself may be the primary site of malignant disease (Hodgkin's disease).

5 The respiratory system

Chapter contents

5.1 Introduction

Oxygen derived from the air is essential for providing energy to drive the metabolic processes in cells and tissues. Air is drawn into and expelled from the body through the respiratory system by the process of **ventilation**. Within the respiratory system, gaseous exchange takes place between air and blood in the lungs. This is **respiration** in its true sense; oxygen enters the blood and carbon dioxide leaves it. The activities of the respiratory system must be regulated to ensure adequate oxygen supplies and clearance of carbon dioxide to meet the functional demands of the body. The respiratory and cardiovascular systems work in concert to maintain homeostasis and share several control mechanisms. The respiratory system also provides the driving force for production of speech and modifying sounds during speech.

Anatomically, the respiratory system consists of a series of air passages that terminate in the lungs where gaseous exchange takes place across the thin walls of individual alveoli within them. The air passages are supported by bone or cartilage to prevent them from collapsing when air pressure is reduced. A schematic diagram of the respiratory tract is shown in Figure 5.1. In succession, the nose, pharynx, larynx, trachea, and bronchial tree constitute the **conducting portion** of air passages and the lung alveoli form the **respiratory portion** where gaseous exchange takes place. Clinically, the air passages as far as the larynx are known as the **upper respiratory tract (URT)** and the passages below the larynx and the lungs are the **lower respiratory tract (LRT).**

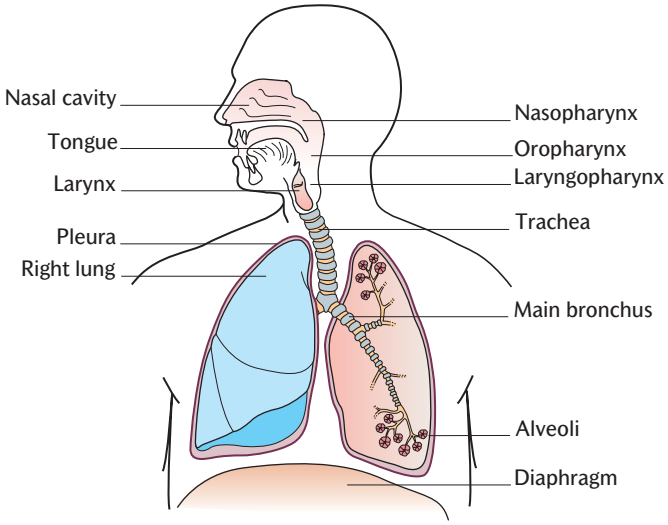

Fig. 5.1 A diagrammatic representation of the respiratory system.

5.2 The upper respiratory tract

Air is drawn into the body through the nose. The nose is more than a simple air passage; it has important functions in cleaning, warming, and moistening air. Air is filtered by hairs at the entrance to the nose, warmed by heat exchange with the abundant blood vessels in the mucosa of the nasal cavities, and humidified by fluid evaporating from mucus secreted by the lining mucosa. Figure 5.2A shows how bone in the lateral walls of the nasal cavities is folded to increase the surface area available and thus increase their efficiency of heating and humidification. The mucosa lining the respiratory portion has an outer covering known as **respiratory epithelium** although its full description, pseudostratified ciliated columnar epithelium with goblet cells, is more informative. Its structure is shown diagrammatically in Figure 5.2B. 'Pseudostratified' means that there appear to be several layers (strata) of cells when in fact, there is only a single layer of columnar cells; 'ciliated' indicates that the cells are covered by **cilia**, tiny hair-like structures, on their outer surface and **goblet cells** which are mucus-secreting cells embedded between the ciliated cells. Aggregates of mucus cells also form mucous glands in the connective tissue below the epithelium to add to the volume of mucus. As well as its function in humidifying the air, mucus traps tiny particles that may be harmful to the LRT. The cilia are in constant wave motion to move the mucus across the epithelium. In the nasal cavities, the cilia are beating back towards the pharynx. In the air passages below the pharynx, the cilia are beating in an upward direction. Thus mucus containing potentially harmful material is moved towards the pharynx from where it can be swallowed and rendered harmless.

Notice in Figure 5.2A, the large holes in the bones forming the lateral walls of the nasal cavity. These are two of the **paranasal air sinuses** that occupy the middle of some of the skull bones surrounding the nasal cavity. The air sinuses connect with the nasal cavity and are lined with respiratory epithelium. They probably function to provide an additional

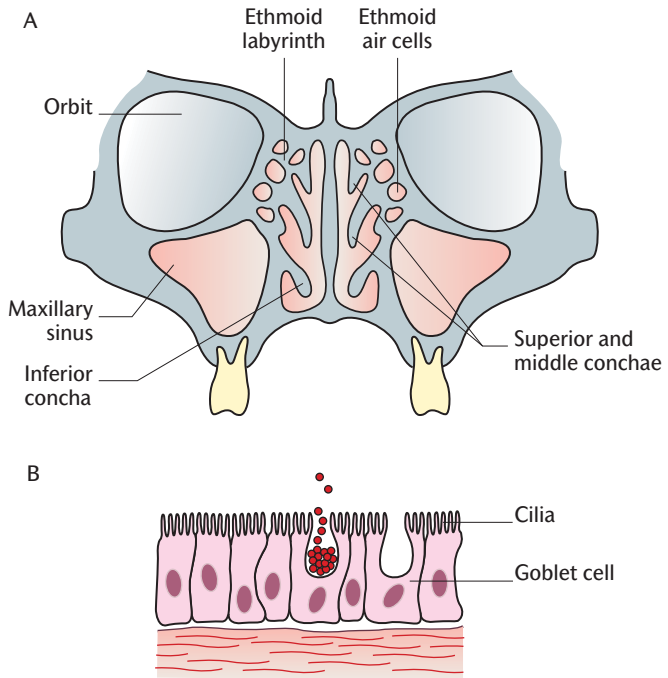

Fig. 5.2 A) A diagrammatic coronal section through the nasal cavity and paranasal air sinuses; B) Respiratory epithelium.

reservoir of mucus enclosed in chambers that are not in the direct air stream and therefore, less susceptible to dehydration. The paranasal sinuses are described more fully in Section 27.4.

Air passes from the nose into the **pharynx,** a muscular tube. The pharynx is a common passageway shared by the respiratory tract and the gastrointestinal tract. Follow the course of the pharynx in Figure 5.1 and you will see that it begins posterior to the nasal cavities, continues down behind the mouth; its final portion lies posterior to the entrance to the larynx. Air passes from the nose to the larynx, passing posterior to the mouth as it does so. Food enters the mouth and is passed back into the pharynx as it is swallowed and passed into the oesophagus lying posterior to the larynx. Air and food thus share the area of the pharynx known as the **oropharynx** posterior to the mouth. We are, of course, breathing all the time but only eating and drinking for short periods. There are several protective mechanisms to ensure that food and drink go down the oesophagus to the stomach and not into the LRT, including temporary suspension of ventilation during swallowing. Mechanisms protective to the LRT are covered more comprehensively in Section 29.2.

From the lower part of the pharynx, air enters the **larynx** which guards the entrance to the LRT and lies in the anterior part of the neck. The larynx is contained in a series of cartilages that articulate with each other through synovial joints, allowing precise movement. The lining mucosa of the larynx has two pairs of folds and the narrowest part of the larynx lies between the lower pair, the **vocal folds**. The default position of the vocal folds is open to allow passage of air through the larynx, but they can be moved together to act as a sphincter to close off and protect the LRT during swallowing. The precision movements of the laryngeal cartilages enable the vocal folds to be tensed so that they vibrate as air passes over them, producing sound. Tension, length, and thickness of the vocal folds can be minutely regulated to change the pitch of the sound. **Phonation,** the mechanism of voice production during speech, is discussed more fully in Section 29.2.1.

5.3 The lower respiratory tract

Follow the description of the LRT shown in Figure 5.3 as you read the description. The larynx opens below into the **trachea,** a tube reinforced by C-shaped rings of cartilage to prevent it from collapsing during changes in air pressure during ventilation. The oesophagus lies immediately posterior to the trachea and anterior to the vertebral column. It lies behind the open part of the C-shaped cartilages of the trachea to allow it to distend as food is swallowed. The trachea passes from the lower part of the neck into the upper thoracic cavity where it divides into right and left **main bronchi.** These enter the lung where they undergo repeated branching. The final and smallest branches, the **bronchioles,** lose their cartilaginous support. Their walls contain smooth muscle so that their diameter can be regulated to control the amount of air entering the extremely thin-walled sacs, the **alveoli**, that make up the structural components of the lungs and the respiratory portion of the respiratory tract.

The millions of alveoli that form each lung provide an enormous surface area for gaseous exchange. It has been estimated that if the alveolar lining of each lung was flattened out, it would cover the area of a tennis court. Figure 5.4 shows that alveolar walls are only one cell thick. Moreover, alveolar cells have comparatively little cytoplasm so they are extremely thin. The alveolar cells are surrounded by a rich capillary network fed by the pulmonary circulation. The blood in capillaries is separated from the alveolar air

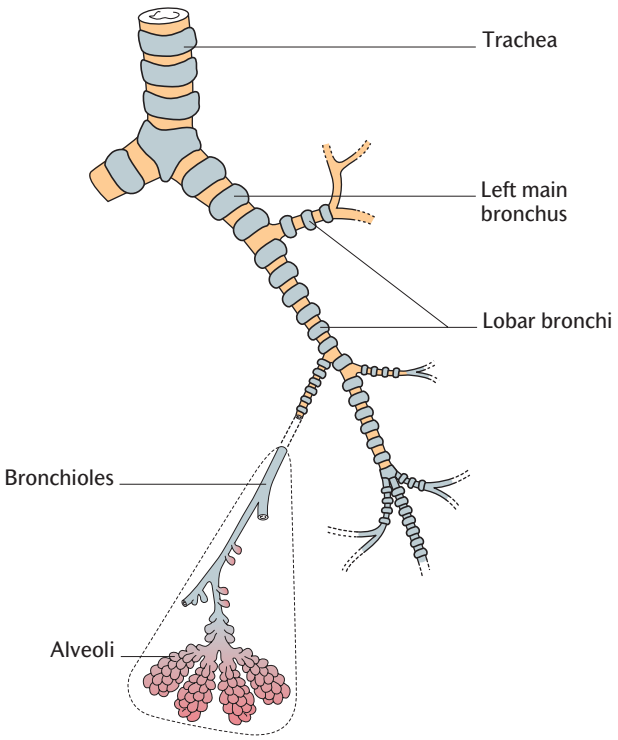

Fig. 5.3 The lower respiratory tract.

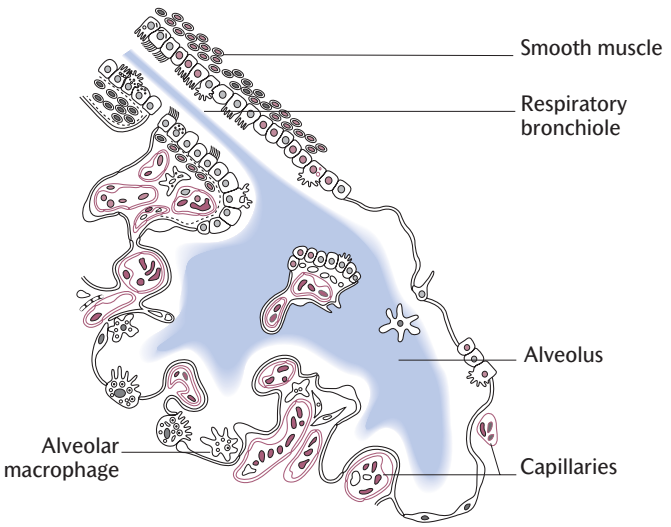

Fig. 5.4 The structure of pulmonary alveoli.

by only the cell membranes of the alveolar cells and the endothelial cells forming capillary walls; together, they are about 0.5 µm in thickness which permits the rapid and efficient diffusion of oxygen and carbon dioxide.

Despite the cleaning of air by the hairs in the nose and the mucus secreted by the respiratory epithelium, some of the smallest pollutant particles eventually reach the alveoli where they would quickly damage the delicate alveolar walls if not removed. These particles are engulfed by **macrophages** which then migrate through the lymphatic system to the lung surface or adjacent lymph nodes. It is this material, particularly carbon particulates, that is responsible for the grey or black 'marbling' effect on lungs in human cadavers. The lungs of city dwellers are usually more marked than those living in rural areas and the lungs of smokers or those working in heavy industries such as mining or engineering are usually blackened.

5.3.1 Lungs

The left and right **lungs** occupy a roughly cone-shaped space on either side of the heart and great vessels which occupy the **mediastinum,** the middle part of the thorax. The bronchi, bronchioles, alveoli, blood vessels, together with a small amount of connective tissue and nerves and lymphatic vessels, form the substance of the left and right lungs. Fresh animal lungs, which can sometimes be seen in butchers' shops as animal food, are collapsed heaps, but the lungs in cadavers are a spongy consistency due to the preservative fluids maintaining the structure of the lungs. Each lung has an oval area on its medial aspect called the **hilus** where the main bronchi and pulmonary blood vessels enter and leave. The right lung has three lobes separated by fissures whereas the left lung has only two lobes, the reduction in size of the left lung being due to the presence of the heart. The lungs are more fully described in Section 11.3.

5.3.2 Pleura

Each lung is enclosed in two layers of **pleura**. In Figure 5.5, the two layers are distinguished by different colours. The outer layer, shown in red, is called the **parietal pleura** and lines the thoracic wall and

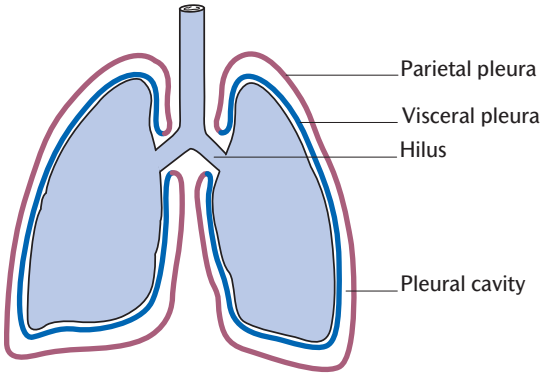

Fig. 5.5 A diagram of the pleura.

upper surface of the diaphragm to which it is attached and will, therefore, move with it when the thoracic dimensions are changed during ventilation by actions of the intercostal muscles and diaphragm. The inner layer, shown in blue, is the **visceral pleura** and is adherent to the surface of each lung. The two layers are continuous with each other around the hilus which is itself not covered by pleura. The narrow potential space between the two layers of pleura is called the **pleural cavity** and contains a very small quantity of **pleural fluid**. This fluid is a lubricant so that the two layers can slide easily across each other during ventilation and also acts as an adhesive agent sticking the two pleural layers together. When the parietal pleura move with the thoracic wall, the visceral pleura and, therefore, the lungs will follow their movement because of the adhesion of the two layers. You may wonder why the pleura are necessary. Would it not be simpler just to attach the lungs directly to the thoracic wall? The answer is that point-to-point attachment would be relatively inefficient as the spaces occupied by the lungs within the thoracic cavity are somewhat irregular. The presence of pleura allows the lungs to expand to their maximum when maximal respiratory effort is required.

5.4 Breathing

Ventilation, commonly known as breathing, occurs as the dimensions of the thoracic cavity are altered which changes air pressure within the lungs. Essentially, to breathe in (**inspiration**), the size of the chest cavity is increased by the **intercostal muscles** between the ribs moving the ribcage up and out at the same time as the **diaphragm**, a muscular sheet between the thoracic and abdominal cavities, contracts and flattens. As these movements increase the chest volume in all three dimensions, the pressure is reduced so air is drawn in, aided by the pressure of the atmosphere outside. Breathing out (**expiration**) occurs when the intercostal muscles and diaphragm relax and the lungs deflate to their original dimensions and thus requires no muscular effort when breathing quietly.

Control of breathing is normally **automatic** but may be overridden to a certain extent (such as holding the breath, deep breathing exercises, or speech). The rate and depth of ventilation is controlled by several factors, the most important being the concentration of oxygen and carbon dioxide in the blood. If the concentration of oxygen in the blood falls or that of carbon dioxide rises, the respiratory rate and depth will increase. Blood oxygen levels are monitored by chemical receptors (**chemoreceptors**) in the aorta and the carotid arteries in the neck that supply the brain. This information is sent to the respiratory centres in the medulla oblongata. Increased carbon dioxide has a direct influence on the respiratory centres. Ventilation and its modification during exercise or exertion and speech are described further in Section 11.4.

6 The gastrointestinal system

Chapter contents

6.1 Introduction

The mouth and pharynx are the first parts of the digestive system; they are the principal areas of the gastrointestinal system of interest to dental students and practitioners and are fully described in Chapters 25 and 28. The anatomy of the remainder of the system is described briefly to provide a working knowledge for applications in other aspects of undergraduate dental courses such as nutrition.

In essence, the digestive tract is a long convoluted tube illustrated in Figure 6.1. It extends from the **mouth**, via the **pharynx**, **oesophagus**, **stomach**, **small intestine**, and **large intestine** to the **anal canal**. It is formed along most of its length by longitudinal and circular layers of smooth muscle. It is lined throughout by epithelium which shows marked structural differences from region to region to match different functional requirements of secretion of digestive enzymes, absorption of nutrients, and excretion of waste products.

The **liver** and **pancreas** are large organs essential to the function of the gastrointestinal system.

Food is ingested through the mouth and then prepared for swallowing by being broken up and mixed with saliva by the chewing action of the teeth. Saliva has a major lubricant and minor digestive function. The food is formed into a pellet or **bolus** and is then **swallowed** by being moved back by the tongue into the pharynx. Once food is in the pharynx, swallowing becomes a reflex mechanism designed to coordinate contraction of muscles to push the food through the pharynx and oesophagus to the stomach as well as ensuring food and drink do not enter the lower respiratory tract. Swallowing is complex, involving several sets of muscles in the head and neck and is described in more detail in Section 29.1. The passage of food along the remainder of the digestive tract is achieved by regular contractions (**peristalsis**) of the smooth muscle layers in its walls.

6.2 The oesophagus

The oesophagus is a muscular tube about 25 cm in length. It begins in the neck as a continuation of the pharynx and lies posterior to the trachea as it enters the thorax. It passes through the thorax, lying slightly to the left against the vertebral column before passing through the muscular part of the diaphragm and entering the stomach below. The diaphragmatic muscle acts as an external sphincter which prevents gastric contents from regurgitating into the oesophagus.

If **gastric reflux** occurs regularly, the strong stomach acid causes the pain known as 'heartburn' and can, in fact, cause physical burns in the lining of the oesophagus.

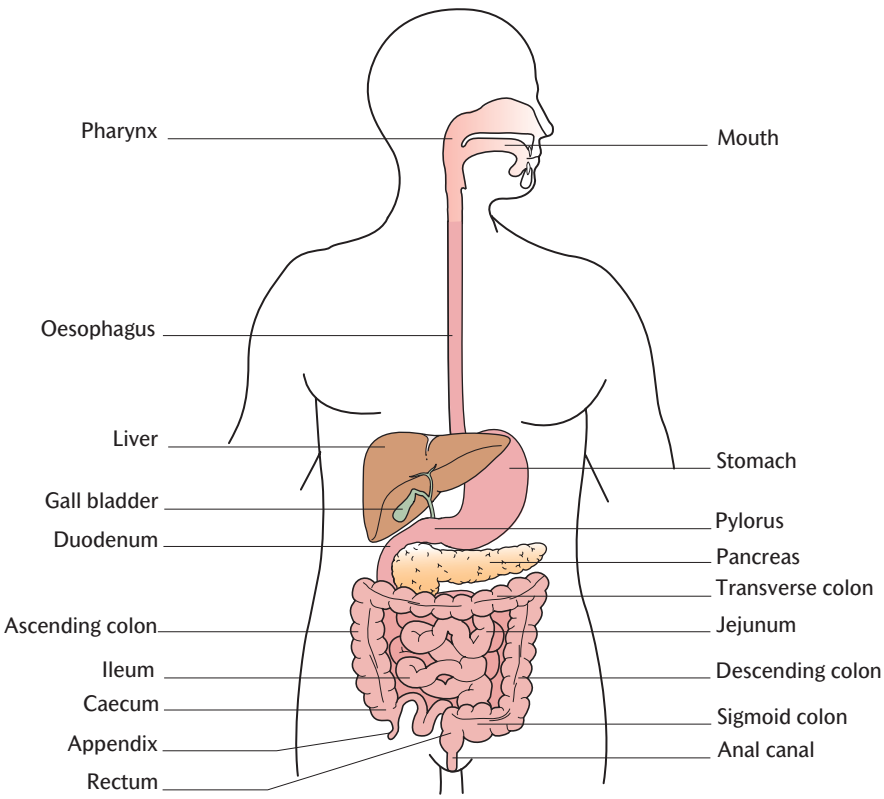

Fig. 6.1 The gastrointestinal system.

6.3 The stomach and small intestine

The stomach is a dilated muscular bag immediately below the diaphragm within the abdominal cavity. It secretes hydrochloric acid and digestive enzymes as well as mucus to prevent damage from its own acid products. Activity of the stomach wall and the action of enzymes break food down into a creamy consistency. Some absorption of nutrients takes place in the stomach. The stomach opens through the **pyloric sphincter** into the **duodenum,** the first short part of the 6 m of tubing that comprise the small intestine; the remainder is formed by the **jejunum** and **ileum.** Because of its great length, the small intestine is highly folded as you can see on Figure 6.1 and occupies a large part of the abdominal cavity.

As food passes through the stomach and small intestine, it is acted upon by a large variety of enzymes and other digestive substances secreted by the glands of the lining mucosa. The **pancreas**, an accessory gland of the digestive tract, produces large amounts of enzymes that enter the duodenum through the pancreatic duct. The pancreas also contains islets of cells producing the hormones insulin and glucagon. The pancreas is thus an **exocrine gland**, releasing its secretions through a duct system and an **endocrine gland** where the secretory product is released directly into the blood. Defective insulin production causes diabetes mellitus.

Many digestive enzymes are secreted in the form of inactive precursors which are harmless to the cells which secrete them; they are activated in the lumen of the digestive tract. The intestinal wall is itself protected from digestion by the copious secretion of mucus. The mucosal lining of the small intestine is folded into minute finger-like processes, the **villi**, and the luminal cell membranes of its individual cells are folded into microscopic **microvilli**, thus producing an enormous surface area for absorption. The products of digestion in the form of sugars, amino acids, fats, and glycerol are absorbed into the bloodstream from the small intestine. Fats, however, cannot be digested until emulsified with water. The emulsifying agent is **bile** which is secreted by the liver, stored in the **gall bladder**, and passes into the duodenum through the **bile duct** (see Section 6.5).

Digestion is usually complete by the time food has reached the halfway point of the small intestine. The rest of the jejunum and ileum is concerned with absorption rather than digestion.

6.4 The large intestine

The small intestine becomes continuous with the large intestine on the lower right side of the abdominal cavity as you can see in Figure 6.1. The first part of the large intestine is a short, blind-ending sac called the **caecum** which has the **appendix** attached to its posterior wall. From the caecum, the **ascending colon** passes up the right side of the abdominal cavity towards the liver where it turns to cross beneath the stomach as the **transverse colon** and then turns down as the **descending colon** on the left side. The descending colon is continuous with the **sigmoid colon** which, in turn, is continuous with the **rectum** that opens into the **anal canal**. The colon is the major site for resorption of water and ions secreted with digestive juices; about 70% of the material entering the colon is resorbed. Resorption is linked with the ability of the cells of the colon to transfer sodium from the intestinal lumen into the blood. Undigested residue consolidates into the faeces for excretion. The effects of malfunction of the large intestine are described in Box 6.1

Box 6.1 Diarrhoea

The volume of water which can be resorbed by the colon depends on the rate at which its contents pass through. If the transit rate is rapid, in response to certain foods and bacteria which stimulate peristalsis, water resorption is low and **diarrhoea** ensues. Apart from its inherent unpleasantness, diarrhoea can cause severe dehydration and consequent disturbance to ionic balance as excess water and electrolytes are excreted instead of being resorbed and can prove fatal. As well as treating the causative agent of the diarrhoea, rehydration therapy with isotonic fluids must also be instituted to restore ionic balance.

6.5 The liver

As you can see in Figure 6.1, the liver is a large, solid organ which occupies the upper part of the abdominal cavity. It lies immediately adjacent to the lower surface of the diaphragm and is protected by the lower ribs.

Digestive products absorbed by the small intestine enter capillaries and then veins that coalesce into the **hepatic portal vein** (see Figure 4.1). This enters the liver alongside the **hepatic artery.** The hepatic portal vein breaks up within the substance of the liver into a second capillary bed where digestive products are taken up and processed by liver cells. The hepatic portal vein thus begins and ends in capillary beds. The liver consists of a large number of lobules, each of which receives blood from both the portal vein and the hepatic artery.

The liver has numerous functions, including the metabolic processing and storage of digestive products absorbed from the gut, manufacture of plasma proteins, synthesis of bile salts for emulsification of fats, conversion of the breakdown products of haemoglobin into bile pigments, and detoxification of substances (for example, alcohol) circulating in blood.

The bile salts and bile pigments pass into the bile canaliculi, small vessels lying close to the liver cells, which unite to form the **hepatic duct** system that leaves the liver and opens into the duodenum by the **bile duct.** From the hepatic duct, the **cystic duct** enters the **gall bladder** which hangs from the underside of the liver. When the connection between bile duct and intestine is closed, bile backs up and is stored in the gall bladder in preparation for the next meal. The problems associated with the impairment of bile secretion are described in Box 6.2.

Box 6.2 Jaundice

If bile cannot enter the hepatic duct system or small intestine, it will remain in the liver where it is absorbed into the blood. The bile pigments discolour the blood and this colour will show through the skin or whites of the eyes, giving a yellow tint known as **jaundice**. Jaundice is usually an indication of liver dysfunction although jaundice can have other unrelated causes.

6.6 Nerve supply of the digestive tract

The digestive tract receives a rich autonomic innervation. The parasympathetic supply as far as the left flexure of the large intestine is through the **vagus** (tenth cranial) **nerves;** below this level, it is through the parasympathetic nerves travelling with sacral spinal nerves. The sympathetic supply is through branches of the sympathetic trunks. Parasympathetic stimulation of the digestive tract increases motility and peristaltic activity and secretion of digestive enzymes. Sympathetic stimulation has the opposite effects of decreasing motility and inhibiting secretion and also causing vasoconstriction to divert blood away from the intestines to muscles. Sympathetic stimulation is responsible for the feeling commonly expressed as 'butterflies' in the stomach when you are nervous.

7 Skin and fascia

Chapter contents

7.1 Skin

Skin is a specialized boundary tissue which forms the entire external surface of the body and is continuous with mucosa lining the respiratory, gastrointestinal, and urinogenital tracts at their respective openings. Skin is the largest organ in the body but is often overlooked in this respect.

Skin has many functions, some of which are not immediately obvious.

- It minimizes damage from mechanical, thermal, osmotic, chemical, and sunlight insults.
- It forms a barrier against microorganisms.
- It has a major function in thermoregulation.
- It is a sensory surface equipped with touch, pressure, temperature, and pain receptors.
- It has good frictional properties useful in locomotion and handling objects.
- It is waterproof.
- It is the site of vitamin D synthesis.
- It also plays a role in non-verbal communication when we blush, alter our facial expression, or use tactile communication such as touching or kissing.

Skin has two distinct parts when seen under a microscope, the superficial epidermis and the deeper dermis.

7.1.1 Epidermis

The **epidermis** is a surface epithelium in which the outer cells are keratinized. Keratinization is the deposition of tough mats of keratin which are intracellular fibrous proteins that make the cells tough; keratinization also kills the superficial cells so the outer layers of your skin are dead. The epidermis varies in thickness. The thickest and most heavily keratinized areas are on the soles of the feet and palms of the hands whereas the epidermis on the face and back of the hand is much thinner and less heavily keratinized. Habitual activity, such as holding a pen, digging with a shovel or using scissors, may produce localized thickenings of thick skin by increasing the thickness of keratin to produce calluses. Cells below the keratin layer have a special coating that forms a permeability barrier, preventing water moving between cells, thus preventing water loss from the body and water-logging when exposed to water. Epithelium does not contain blood vessels, which is why you do not bleed when you lightly knock your skin. To bleed, you need to expose the blood vessels that lie in the dermis and supply the overlying epidermis by diffusion of nutrients through fenestrated capillaries.

7.1.2 Dermis

The dermis is made up of fibrous connective tissue, containing numerous blood and lymphatic vessels and nerves. It is also variable in thickness. In the dermis, the bundles of collagen fibres are mostly arranged in parallel rows whose direction is seen as **skin creases** on the surface. These may be **tension lines** which are small, irregular furrows dividing the skin into a series of lozenges, most easily seen on the back of the hand, or **flexure lines** which are associated with regular movements and are conspicuous in skin overlying joints, especially on the palms and soles, where they indicate the points of attachment of the skin to the underlying deep fascia (see Section 7.2). An incision made in the direction of a skin crease will cut few collagenous fibre bundles and heal with a minimum of scarring; a cut across the skin creases tends to leave a scar. Distinct parallel arrays ridges and grooves, called **papillary ridges** or friction ridges, are confined to the finger tips and feet. Friction ridges produce a high friction surface to aid in gripping objects or the ground, hence the term 'friction ridges'. Papillary ridges on the fingers are the basis of fingerprinting (dermatoglyphics) used forensically to identify individuals.

7.1.3 Epidermal appendages

The skin contains nails, hair follicles, sebaceous glands and sweat glands.

Nails are keratinized plates on the dorsum of fingers and toes to give extra protection against mechanical shock. **Hairs** grow from hair follicles formed from downgrowths of the epidermis into the dermis. Distribution of hair over the body surface is dependent on age, sex, and race. Each follicle is connected to the dermis by a slip of smooth muscle, the **arrector pili** muscle, innervated by the sympathetic nervous system. Contraction of the muscle makes the hair 'stand on end' in response to cold or fright. Skin disturbed by these responses is called **gooseflesh** or goosebumps. Hair erection in response to cold traps air next to the skin and prevents loss of heat.

Sebaceous glands lie adjacent to hair follicles and secrete oily **sebum** onto hair shafts, maintaining the flexibility of the hair and helping to waterproof the surrounding skin. **Sweat glands** are found in skin all over the body except the vermilion border of the lips (see Section 25.1) and the skin beneath the nails. Sweat is a hypotonic watery fluid containing about 0.5% electrolytes, the chief ones being sodium and chloride. If we are exposed to a warm atmosphere or generate excess internal heat by exercise, for example, we sweat over the whole body surface. This is usually accompanied by flushing of the skin produced by dilatation of blood vessels in the dermis. The sweat is then evaporated by heat extracted from the blood in the dilated vessels, thus cooling the body down. We may break out into a 'cold sweat'—sweating without flushing—when frightened or anxious. Such sweating occurs chiefly on palms, soles, and in the axillae. Stimulation of the sweat glands is produced as a result of sympathetic nervous innervation.

Skin colour is determined by at least five pigments, melanin (brown), melanoid (similar to melanin, but differently distributed), carotene (reddish), haemoglobin (purple), and oxyhaemoglobin (pink). In pale-skinned individuals, pigmentation is variable within certain limits and exposure to sun and wind darkens or tans the skin. Melanin is produced by cells called melanocytes in the deepest layer of the epidermis; these are dendritic cells having long cytoplasmic processes that insinuate between the other epidermal cells. Melanin helps to protect the deeper layers of the skin from the carcinogenic effects of ultraviolet radiation. This is probably the reason why the populations originating from regions closer to the equator where the sunlight and heat is intense are often dark-skinned.

7.2 Fascia

Fascia is an anatomical term used in the dissecting room for a sheet of fibrous connective tissue large enough to be seen with the naked eye. Historically, the number of named fasciae recognized by anatomists rose abruptly when formalin was introduced as a dissecting room fixative; formalin cross-links proteins and therefore, denatures connective tissue particularly well. This subsection is important to those of you who have the opportunity to dissect human cadavers or to study prosections (prepared dissections). Nevertheless, those who do not have those opportunities should read this material as the term 'fascia' will crop up in several places in later sections of the book.

We have already met **connective tissue** in the context of bone, cartilage and muscle, and their associated tendons in Chapter 2. As you will discover in Section 8.3.3, a layer of cells called mesoderm forms during early development of the embryo. Mesodermal cells subsequently differentiate into specific types of cells that form muscle, cartilage, and bone and other specific connective tissues but also less specialized connective tissue that fills spaces and accumulates between groups of muscles, internal organs, and other large structures.

The arrangement of this connecting and packing tissue is varied. It forms a visible, shiny, silvery layer or sheath on the surface of muscles. Packing tissue between organs often contains considerable amounts of fat (**areolar tissue**) that allows movement or provides a protective layer. Major blood vessels often follow a common course with a large artery accompanied by the large vein draining the corresponding area. Nerves often accompany blood vessels and they are often bound together to form a **neurovascular bundle**. The larger blood vessels are often enclosed by a dense connective tissue sheath whose functions are not clear. The common carotid artery and internal jugular vein in the neck are enclosed by the **carotid sheath** (see Section 23.1.5). They may help venous drainage by holding large veins close to pulsating arteries.

7.2.1 Superficial fascia

Superficial fascia, or **subcutaneous tissue**, is found between skin and underlying structures. This layer, also known as the **hypodermis**, is also considered part of the skin and is the site of the largest accumulations of body fat. Body fat varies in thickness and regional distribution according to age and sex (see Figure 1.1) as well as diet. Fat is more abundant and more generally distributed in adult females, producing rounded contours. In males, it is less abundant, especially towards the extremities. Fat becomes more noticeable in both sexes in middle age where it tends to be thickest and most conspicuous on the anterior abdominal wall.

In areas where relative movement between skin and underlying structures is undesirable, such as the scalp, sole of foot, or palm of hand, dense bands of connective tissue unite the surface layers and deeper structures with relatively little subcutaneous fat intervening.

Local subcutaneous infections, such as abscesses and boils, are relatively painless in areas where superficial fascia is abundant as they have room to swell; they become very painful where the superficial fascia is meagre, restricting swelling and causing distortion of the surrounding tissues.

7.2.2 Deep fascia

Deep fascia has many parallel collagen fibres forming dense sheets of connective tissue. Layers of deep fascia are frequently found between groups of muscles, often separating different muscle groups with different functions. They can act like an extension to the bone to increase the area of attachment of the muscles and this arrangement is seen in the neck and limbs.

8

Embryonic development—the first few weeks

Chapter contents

8.1 Introduction

Embryology is a fascinating subject and is the foundation of the development, growth, and maturation of all the cells, organs, and tissues of the body. Strictly, **embryology** is the study of the early processes of development beginning at fertilization and following the processes that turn a single cell into a multicellular organism. It is all about generation of the building blocks required to make a human body. **Developmental anatomy** is the study of how these building blocks are turned into specific cells, tissues, and organs as well as the general growth of the body. As you will soon appreciate in the following paragraphs, all organs and systems do not develop at the same rate so there is a degree of overlap between embryology and developmental anatomy. For example, the heart and circulatory system must develop and be functioning very early in development to ensure adequate supplies of nutrients to the developing fetal tissues. Teeth, on the other hand, are not going to be used until about six months after birth at the earliest; while the heart is already beating away, each developing tooth is merely a tiny group of cells bearing little resemblance to a fully formed tooth.

Human gestation is considered to take nine months; more accurately, it usually lasts for 38 to 39 weeks from fertilization to birth. Clinically, it is divided into three **trimesters** of three months each. In this chapter, we will focus on events in the first few weeks. During the first two and a half weeks after fertilization, the very basic building blocks are formed from the single fertilized cell; this is the **pre-embryonic period**. The **embryonic period** covers the next five and half weeks during which these basic building blocks develop into the cells, tissues, and organs. As already indicated, some of these may be in a very rudimentary state at the end of the embryonic period. The remaining 30 or so weeks is the **fetal period** when the tissues and organs of the body grow and develop and the fetus grows considerably. We are not fully mature organisms at birth and have another 20 years a-growing.

This chapter is an account of the development of the embryo from fertilization to about the middle of the embryonic period when all the rudiments and building blocks of significance to dental students are formed. The subsequent development of specific systems—the circulatory, respiratory, and nervous systems—and structures forming the head and neck are dealt with in the corresponding sections describing their anatomy.

8.1.1 The importance of understanding development

Understanding the very early stages of development provides a general knowledge of how you developed and will equip you with a vocabulary to understand the terms and processes that takes place during the development of those systems relevant to dental practice.

Knowledge of the way in which different structures develop often clarifies the mature anatomy, especially the overall patterns that govern the way things are arranged in the body.

Embryology can provide a logical explanation of anatomy where there appears to be none. The embryology or developmental anatomy of given structures can often explain apparent anomalies or illogicalities. Why is the diaphragm innervated by nerves that originate in the neck? Why do nerves innervating the larynx go all the way down into the thorax and come all the way back up into the neck when it would be much simpler to direct the nerves straight across the neck to the larynx? The mysteries of the strange courses of the phrenic nerves and recurrent laryngeal nerves will be explained in context in Chapters 10 and 13 respectively.

As you read the descriptions in this and other chapters covering development, you will see that embryology and development is an awe-inspiring process. You will wonder how anybody ever arrives in this world with 20 tiny fingers and 20 tiny toes on those little limbs, those eyes, and those rosy cheeks wherewith life begins. Alas, many individuals do not. Something has gone wrong during their embryological development to cause the absence or deformity of tissues or organs. Such developmental abnormalities may be life-threatening at worst; even at best, many will cause functional and/or cosmetic defects that render life difficult.

As practising dentists, you will see abnormalities of the face, jaws, and teeth which you may be able to correct yourself or refer to one of your colleagues specializing in their treatment. You will be expected to understand how such conditions arose (if known) as many patients will be at least curious and may be very concerned about the consequences and possible risks of inheritance by their children. You will also come across records of abnormalities in other systems during the taking of patients' medical histories. Some conditions will affect the way in which you treat your patients and must be taken into account during your treatment planning.

The most dangerous period for developmental abnormalities to arise is the pre-embryonic period. The embryonic period is only marginally less dangerous and still fraught with hazard. The fetus is not entirely safe until delivered at the end of gestation. The risks should be borne in mind when treating pregnant patients. The use of many drugs and chemicals is very strongly contraindicated during pregnancy as they can cause developmental defects.

8.2 Fundamental processes underlying development

Embryology is essentially the development of all the different types of cells of the body from a single cell. It follows, therefore, that **cell division** or **mitosis** is a major process in development. However, cell division also needs to be *limited* in certain areas at certain stages of development, otherwise each cell type would be equally as numerous; your teeth would be the same size as your brain. The rate of mitosis in one tissue also needs to be coordinated with cell division in other tissues so that structures develop in harmony. Cell death is another important process in embryological development. It may seem strange to introduce *death* into a chapter about the beginning of life; cell death during development is not random but is **programmed cell death** or **apoptosis** in which cells are programmed to commit suicide at specific times

and places. Two examples of the role played by apoptosis are the formation of hollow tubes from a solid cord of cells or the removal of tissues that would restrict the movement and development of other tissues.

Movement of cells is another absolutely vital process in development and takes place at several levels. Cells have to change **shape** to bring about a specific configuration so that their functions are maximally efficient. Coordinated changes of shape in all cells across a sheet of tissue can alter the shape of the tissue from a flat sheet into a curve or even a tube. If cells simply accumulated as a result of multiple cell divisions, we would end up as a large ball of cells without any recognizable form. Cells have to move as individuals or populations along defined pathways to produce definitive shapes and structures; this is **cell migration.** Often, one population of cells may be required to migrate while another remains static.

Ultimately, millions of cells are going to arise from one single fertilized ovum. It may be surprising that only about 200 different basic cell types, recognizable by their different shapes and functions, make up the human body although some cell types are further subdivided by function or chemical differences that cannot be seen directly with microscopes. Tissues are aggregates of cells, usually comprising several different cell types. Cells must, therefore, use the repertoires outlined in the previous paragraphs to divide and migrate to the correct locations at the right time. Ultimately, tissues are combined to form specific organs and systems by further division, migration, and combination.

You will already be aware that most cells in the body have a specific function; when cells are fully **differentiated** to carry out their specific function, most cannot change their function at all and certainly not radically. Fat cells cannot be used as neurons and muscle cells cannot be converted into bone. What creates this difference between cells? All cells within the body contain the same genetic information, with the exception of red blood cells that lack nuclei and the gametes (ova and spermatozoa) that only contain half the genetic information. So why is a neuron different from an adipocyte (fat cell)? It is all due to **gene expression**—the specific genes that are expressed within each cell type that make each one unique from another cell by determining the types of proteins that a particular cell will produce. The proteins, in turn, determine the shape and function of cells, how they attach to each other and other cell types, what type of receptors they carry and, therefore, what messengers they can respond to, and many other functions. Many genes are active in all cells to direct basic cell functions such as energy production and repair; these are **housekeeping genes.** During development, many genes direct the formation of proteins that switch on or switch off other genes; these are **gene-regulatory proteins.** Essentially, as development proceeds, the majority of genes within a cell are *switched off* so that only a very limited amount of the total genome in each cell is operating to produce the specific function required. The orderly switching on and off of genes during development ensures that cell division, death, migration, and differentiation take place in the correct sequence, at the correct time, and in the correct place to produce the complex functioning organism that is the human body.

Another important concept to understand is that once cells embark down a pathway of **differentiation**, there is usually no going back to an earlier less differentiated stage. Cells, therefore, become much more restricted in what they can become as development proceeds until eventually they are fully differentiated into a specific cell type. There are cells that retain some of their embryonic characteristics and can differentiate into several cell lines. These cells are useful for generating new cells to replace cells lost by normal wear and tear, trauma, or disease; they are known as **stem cells** (see Box 8.3).

As cells of the embryo differentiate, they initially form specific **germ layers** from which certain cell types, but not others, can differentiate. Four germ layers develop during the pre-embryonic and early embryonic period, each giving rise to a specific set of cell types. Cells originating from two or more different germ layers are usually required to form tissues and organs. The development of the right number of cells of each kind, their aggregation, and differentiation must be coordinated precisely. Signalling between different germ layers is described as an **interaction.** Often one germ layer determines what another germ layer does by causing a change in shape, movement, or function. This process is **induction**; the determinant germ layer produces signalling molecules that act on another recipient germ layer. The inducing cells may not show any obvious change as they act on other cells, but may then exhibit marked changes in their own behaviour. This change is often induced by the cells they originally induced and when germ layers take it in turns to induce each other, this is a **reciprocal interaction.**

Now the fundamental processes involved in embryological development and growth have been outlined, we will apply them to examine how development takes place in the first few weeks of life.

A whole raft of arcane terminology is used in many textbooks to describe embryos and the nervous system. As you will see, the front and back and head and 'tail' end of an embryo can be distinguished from a very early stage; everything can, therefore, be referred to the anatomical position (Section 1.3.1), obviating the need for obscure terminology.

8.3 The first three weeks of development

8.3.1 The first week

The major events in the first week of life are **fertilization** followed by rapid cell division to produce more cells. Towards the end of the first week, two distinct groups of cells can be recognized, those that will go on to form the **embryo** and those that are involved in implanting the embryo in the uterine wall and forming the **placenta**.

The sequence of events in the first week of life is shown diagrammatically in Figure 8.1. This diagram represents the **ovarian** (Fallopian) **tube** connecting to the uterus in the top right hand corner; the ovary from which the ova (eggs) are released would be off the bottom left of the picture. Events begin at the bottom left hand corner and

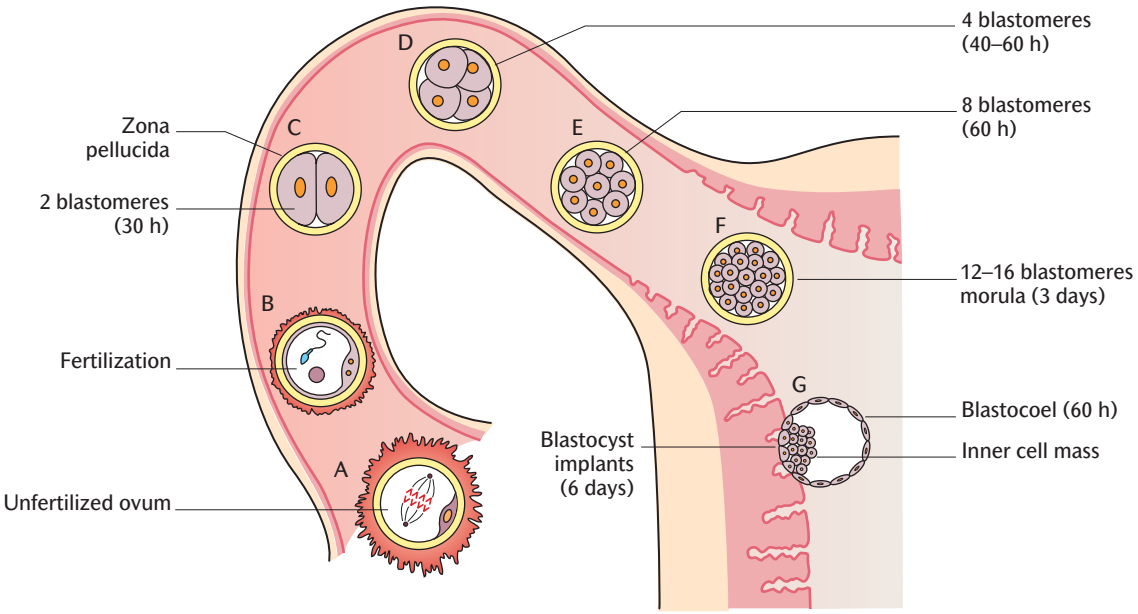

Fig. 8.1 Events of the first week: fertilization and cleavage.

follow in sequence to the top left and across and down to the bottom right.

Fertilization usually occurs between 12–24 hours after **ovulation**, the release of an ovum (egg) from the ovary (Figure 8.1A). Fertilization occurs in the distal third of the oviduct when an ovum is fertilized by a spermatozoon. The **gametes** (ovum and spermatozoon) each contain only 23 chromosomes—the haploid number. When the egg is fertilized as shown in Figure 8.1B, the chromosomes from each gamete meet in the nucleus of the **zygote**, producing 46 chromosomes (the diploid number), 22 pairs of autosomal chromosomes, and one pair of sex chromosomes. The sex of the zygote depends on whether the sex chromosome of the fertilizing sperm is an X or a Y; the sex chromosome carried on the ovum is always an X. At fertilization, the zygote commences a series of mitotic cell divisions (**cleavage divisions).** These divisions will finally result in the formation of a new individual comprising millions of cells, all derived from one single cell. The zygote will give rise to all other cells in the body. Put another way, it has the potential to produce all cells of the body and is, therefore, **totipotential**.

As shown in Figure 8.1C to F, the original single-celled zygote divides into two, four, eight, and 16 cells. Each cell is known as a **blastomere**. The time after fertilization at which each stage is reached is shown in Figure 8.1 the two-cell stage is attained about 30 hours post-fertilization, the 4-cell stage after 40–50 hours, and the 12–16 cell stage at about three days. By the time the 16-cell stage has been reached, the ball of blastomeres, now called a **morula,** has reached the uterus. If you have not noticed already in Figure 8.1, the 16-cell stage morula (F) is the same size as the original ovum (A). This is a depiction of the reality; the 16 blastomeres occupy no more space than did the original egg. As each cleavage division occurs, each new nucleus takes its share of the original cytoplasm of the ovum. Only new nuclei and cell membranes are added at each division. Note in Figure 8.1A to E that the dividing ovum is surrounded by the **zona pellucida;** it prevents the entry of subsequent sperm once one has penetrated the ovum and fertilized it.

If the blastomeres making up the 16-cell morula were separated from each other at this stage, each one has the potential to form a separate embryo; each blastomere is still totipotential. These totipotential cells are known more generally as **embryonic stem cells** and are the subject of intense ethical debate about their use in medical research.

Further examination of the final steps of the journey of the fertilized ovum to the uterus in Figure 8.1G shows that the morula continues to divide but the cells now begin to segregate. Those forming the inner cell mass in the centre are differentiated from those on the surface of the ball. The inner cell mass forms the embryo whereas the outer cells will form the **trophoblast** from which the placenta will develop. The **blastocoel** shown in Figure 8.1H is created as uterine fluid passes between the outer cells; the inner cell mass gravitates to one end of this cavity. The resulting structure is now known as a **blastocyst** which contains 100–150 cells. The zona pellucida has disappeared.

Towards the end of the first week, at about 5½–6 days after fertilization, the blastocyst becomes attached to the uterine wall as shown in the last picture in the sequence of Figure 8.1. Attachment of the blastocyst is the first event in the process known as **implantation** during which the placenta forms. The placenta is the organ where exchange of nutrients and waste products takes place between the maternal and fetal circulation. Figure 8.2A shows the effects of proteolytic secretions from the trophoblast; they erode the lining of the uterus and cells from the outer cell mass invade the space created. The blastocyst thus becomes implanted (embedded) in the uterine wall.

As you can imagine, even in this apparently simple sequence of mitoses during the first week, there is a lot of scope for things to go wrong. These are described in Box 8.1.

Box 8.1 Early embryonic abnormalities

Accidents of fertilization are common but their causes are poorly understood. It has been estimated that between one and two out of three zygotes develop an abnormal number of chromosomes or major mutations within the normal number of chromosomes as early cleavage divisions take place. Most of these abnormal embryos spontaneously abort in the very early stages of pregnancy, often without the mother being aware that an abortion has occurred, the loss of the embryo appearing as a late period. Because so many abnormal embryos spontaneously abort, only some six per 100,000 individual live births have detectable chromosome abnormalities. Probably the best known of these is **Down syndrome** in which there are three instead of the usual pair of the 21st chromosomes (trisomy 21).

Multiple pregnancies arise in two ways. **Dizygotic twins** or more result from the fertilization of separate eggs by separate sperm and are no more alike than brothers and sisters. Identical or **monozygotic twins**, triplets, or quadruplets are the result of the splitting of one zygote into two or more individual blastomeres that each develops into a separate embryo. These embryos are of the same sex and genetically identical. About 70% of twins are dizygotic (seven per 1000 births) and 30% (three per 1000) are identical.

Ectopic pregnancy is produced when a blastocyst implants outside the normal area of the uterine mucosa. Sites where this may occur are the oviducts, ovary, cervix of the uterus, and the abdominal or pelvic cavities. Such pregnancies almost invariably fail to go to term and may produce serious haemorrhage as they burrow into the surfaces of abdominal viscera.

8.3.2 The second week

In the second week of development, the major event is the **implantation** of the embryo into the uterine wall and establishment of the placenta. Relatively little change occurs within the embryo although some important changes occur externally as a prelude to establishment of the embryonic germ layers.

Placentation

As implantation takes place, the distinction between the trophoblast (outer cell mass) and the inner cell mass becomes more obvious. The trophoblast continues its erosion of the uterine wall so that by the eighth day post-fertilization, the blastocyst is firmly embedded in the uterine wall. As shown in Figure 8.2B, the trophoblast differentiates into two parts as it works its way deeper into the uterine wall. The leading portion adjacent to the uterine tissue becomes the **syncitiotrophoblast** in which the individual cells lose their cell membranes to form a multinucleate syncitium; the cells adjacent to the embryo remain as distinct cells forming the **cytotrophoblast.** At this stage, the embryo proper is growing much more slowly than the tissues surrounding it. By the ninth day of development, the blastocyst is deeply embedded in the uterine wall due to further enzymatic activity of the syncytiotrophoblast. As shown in picture C of the sequence in Figure 8.2, the syncytiotrophoblast now surrounds the blastocyst completely and large spaces develop within it known as **lacunae.** Figure 8.2D shows how, by the tenth day, the lacunae have made contact with maternal sinusoidal blood vessels in the uterine wall. The embryonic vacuoles and maternal sinusoidal vessels provide a large contact area between the two circulatory systems for exchange of nutrients and waste products, thus forming the basis of the placental circulation (see Box 8.2).

Formation of the bilaminar embryo

While the placental circulation is being established, certain changes take place in and around the **embryonic disk** itself. If you compare Figure 8.2A and B, you can see how the inner cell mass shown in (A) differentiates into two layers shown in (B) as implantation proceeds. A layer of columnar cells, the **epiblast** or future ectoderm**,** faces the cytotrophoblast and a layer of small, flattened cells, the **hypoblast,** faces the blastocoel cavity. The embryo briefly exists in this two-layered form as the **bilaminar embryo**.

In Figure 8.2C, examine the changes around the embryonic disc itself. A loose agglomeration of cells of uncertain origin appears and spreads along the inner surface of the cytotrophoblast and surrounds the embryonic disc. These cells form the **extraembryonic mesoderm**. At the same time, as shown in Figure 8.2C, the hypoblast extends to form a cavity called the yolk sac. The **extraembryonic mesoderm** between the cytotrophoblast and the yolk sac forms a loose tissue with large vacuoles that later unite to become the **extraembryonic coelom** that plays a crucial role in the nutrition of the developing embryo until a vascular system is established (see Section 8.3.5). As the coelom forms, a more solid area of extraembryonic mesoderm remains which connects the embryo to the trophoblast; this is the **connecting stalk** which is the first sign of the umbilical cord.

8.3.3 The third week—gastrulation

The third week produces significant changes in the embryo. Two more germ layers, intraembryonic **mesoderm** and **endoderm,** are added to the epiblast in a series of processes known as **gastrulation.** Towards the end of the third week post-fertilization, the central nervous system begins to form during **neurulation** and a fourth germ layer known as **ectomesenchyme** is produced as it does so. All the basic building

Box 8.2 Pregnancy testing

Fertilization usually occurs about 14 days before the next menstrual period is due. Considerable development has already occurred by this time; the embryo has reached the bilaminar stage and gastrulation is beginning. The usual signal that a female is pregnant is when the next period fails to occur.

In many countries, pregnancy testing kits are available for self-diagnosis if pregnancy is suspected. These detect the presence of a hormone known as **human chorionic gonadotrophin (HCG)** in the urine. HCG is produced from the trophoblast as implantation and placentation take place in the second week of pregnancy. The role of this hormone is to signal the presence of an implanted embryo and to suppress the usual cyclical release of hormones that control the maturation and release of ova at ovulation.

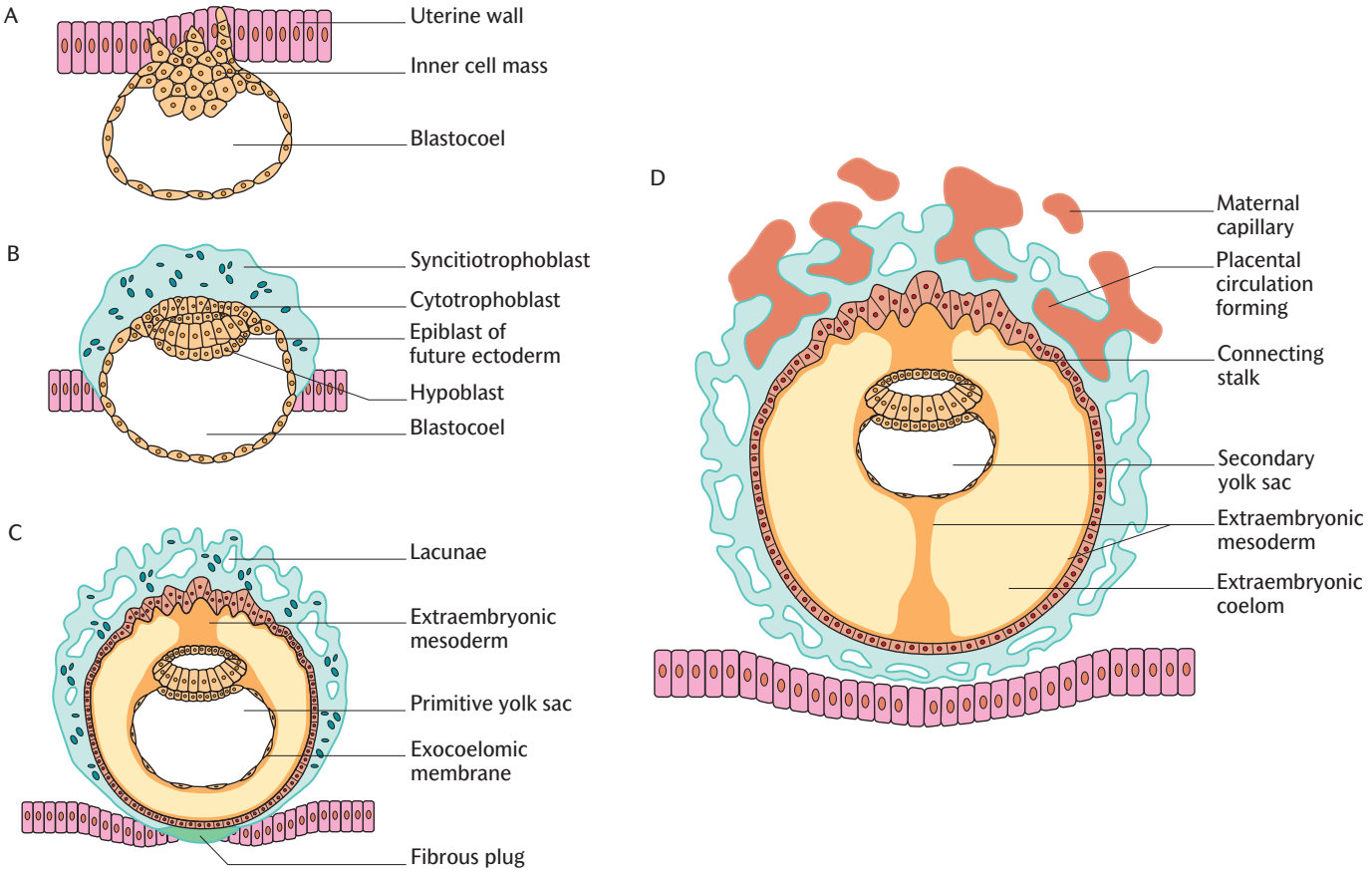

Fig. 8.2 Events of the second week: implantation of the embryo into the uterine wall and development of the bilaminar embryo.

blocks are then in place to differentiate into the specific cell types that will aggregate into tissues and organs.

Examine Figure 8.3 and orientate yourself with respect to the diagram before reading the description that follows. Figure 8.3A shows the embryonic disc viewed from *above* with the surrounding membranes cut open; the yolk sac surrounded by the extraembryonic mesoderm hangs down below the embryonic disc. Figure 8.3A indicates the situation at the beginning of the third week and Figure 8.3B is a few days later.

Identify the embryonic disc coloured blue in Figure 8.3A and note the thickening at one end of the embryonic disc into the **prochordal plate**. The prochordal plate indicates the future head end of the embryo and shows that head-to-tail polarity is achieved although the embryo lacks an overt head and tail at this stage. The cells know where they are destined even before there is a destination to go to. The tail end is at the opposite end of the embryonic disc and is where the connecting stalk is attached. The embryonic disc also has bilateral (left–right) symmetry about a midline axis through the prochordal plate. This axis is determined by a complex series of signalling from the hypoblast to the epiblast although there are no immediate visible signs of this induction.

It is at the future tail end that the next major change takes place, resulting in the formation of the next embryonic layer, the **intraembryonic mesoderm.** This process can only take place when both head–tail and left–right polarity have been established beforehand.

Formation of intraembryonic mesoderm

The arrows shown in Figure 8.3A indicate how at the tail end of the embryo, epiblastic cells divide, round up, and migrate towards the midline where they pile up to form a multilayered structure along the midline. Cells start to sink below the surface of the embryo to form a midline groove in the epiblast, the **primitive streak.** Once the primitive streak has been established, the hypoblast inhibits the formation of any similar structures, thus defining a single left–right axis. When epiblastic cells reach the midline, the cells break away from the plate of epiblastic cells and turn downwards into the plane between ectoderm and hypoblast. The leading epiblastic cells migrating through the primitive streak on days 14–15 invade as far as the hypoblast and displace its cells; the new layer of cells is known as the **endoderm** which is the first true germ layer.

The next waves of migrating epiblastic cells from day 16 onwards spread outwards between the ectoderm and endoderm to form a new embryonic layer, the **intraembryonic mesoderm.** The movement of mesodermal cells is limited initially to the inferior one-third of the embryonic disc along the primitive streak which, as shown in Figure 8.3B by green arrows, is clearly visible in the 15- to 16-day-old embryo. The superior end of the streak is known as the **primitive** (or Hensen's) **node.** As shown by the blue arrows in Figure 8.3B**,** the same process of cell migration takes place at the primitive node. However, the cells invading through the node stay together to form a blind-ending midline

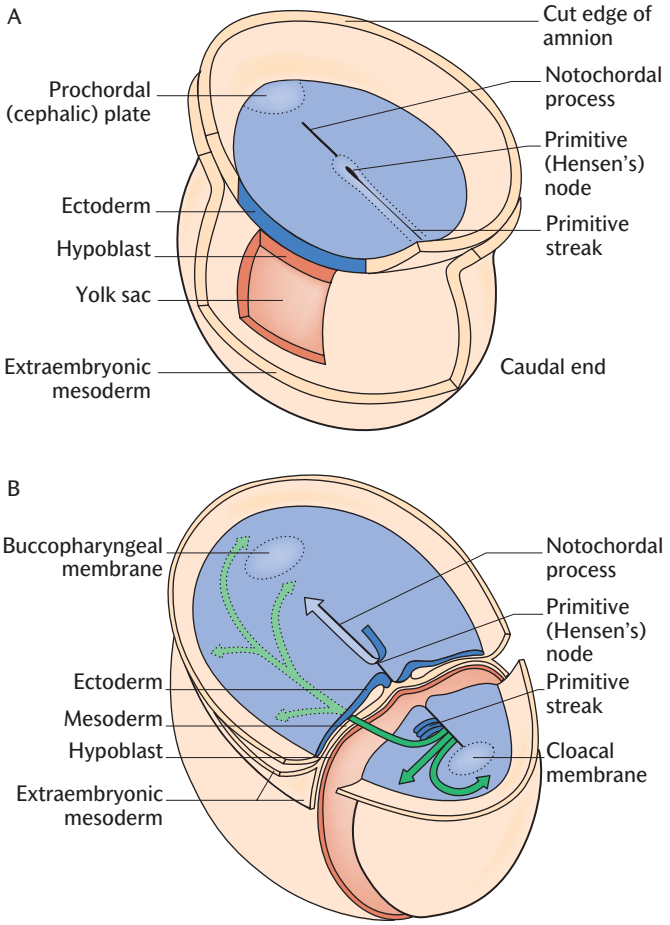

Fig. 8.3 Events of the third week: gastrulation. A) Formation of the primitive streak and node; B) Formation of intraembryonic mesoderm.

tube, the **notochord** under the midline. As more cells are added, the notochord gradually enlarges, reaching the prochordal plate on day 17. The epiblast now becomes known as **ectoderm** and constitutes another specific germ layer.

The spreading intraembryonic mesoderm shown by arrows in Figure 8.3B forms a layer separating ectoderm and endoderm; when it reaches the edges of the embryonic disc, it meets and fuses with the extraembryonic mesoderm. However, ectoderm and endoderm remain in contact in three areas as shown in Figure 8.3B:

- The prochordal plate which later forms the **buccopharyngeal membrane**;
- The cloacal membrane at the inferior end;
- The midline axis occupied by the notochordal process.

The embryo now comprises three distinct germ layers—the **ectoderm**, **mesoderm**, and **endoderm**—and is now a **trilaminar embryo**. All three germ layers are derived from the epiblast, but the cells of each germ layer become more specialized as they differentiate. Each germ layer of the trilaminar embryo can still produce many, but not all, of the cells required to form the body; they are no longer totipotential, but **multipotential**.

During gastrulation, the almost circular embryonic disc has now become pear-shaped, mainly as a result of selective cell migration allied to some growth. The disc is widest at the head end and narrows towards the tail end. The primitive node and streak remain the same size so appear to move towards the tail end during this change in shape. They still continue to add new cells to the mesoderm until the end of the fourth week of development.

Another important thing that occurs during the process of gastrulation is the organization and determination of the fate of cells along the longitudinal axis of the embryo from head to tail.

Homeobox genes

As embryonic ectodermal cells move through Hensen's node and the primitive streak to become mesenchymal cells during gastrulation, they are exposed to doses of retinoic acid (RA). A remarkable family of genes called **homeobox genes** are activated by RA as they pass through the node and streak. The genes closest to the 3' end of the chromosome carrying them respond to low doses of RA whereas those nearest to the 5' end are activated by higher doses. The first cells to pass through the primitive streak receive a low dose, but end up towards the future head end of the embryo; later migrating cells do not migrate so far. Homeobox genes are expressed in a specific sequence along the longitudinal axis of the embryo with the 3' genes more anterior than the 5' genes because of their exposure to different doses of RA. Homeobox genes play a significant role in the development of the central nervous system and the head and neck, and their effects will be described in Chapters 19 and 21.

The fate of the embryonic germ layers

Ectoderm will eventually provide the cells that form the epidermis, the outer layer of the skin, and associated hair follicles, sweat, and sebaceous glands, not to mention tooth enamel. *Endoderm* will form the cells lining the major tubular systems of the body, the respiratory and gastrointestinal tracts. The *mesoderm* will form a wide variety of tissues known collectively as connective tissues. These include bone, cartilage, muscle, fibrous tissue forming tendons and ligaments and tissue supporting skin epidermal cells, and the cells lining the gut. Mesoderm also forms the cardiovascular system and blood. Each of the three germ layers can only produce cells destined for the specific functions listed above for each layer; at this stage, they can produce any of the possible types so are still multipotential.

If you add up all the derivatives of the three germ layers that have been formed by around 18 days post-fertilization, you will notice a rather glaring omission from the list—the nervous system. The nervous system begins its development soon after the formation of the notochord and in the process of the development of the central nervous system, a fourth germ layer is formed that plays a significant role in the development of the head and neck.

8.3.4 Neurulation—development of the central nervous system

Figure 8.4 is a series of cross sections of an embryo at about 17 days post-fertilization, shortly after the notochord has developed. The arrows indicate how the notochord induces the ectoderm overlying the midline of the embryo to become the **neuroectoderm** although no difference from adjacent ectoderm can be seen initially. The neuroectoderm will

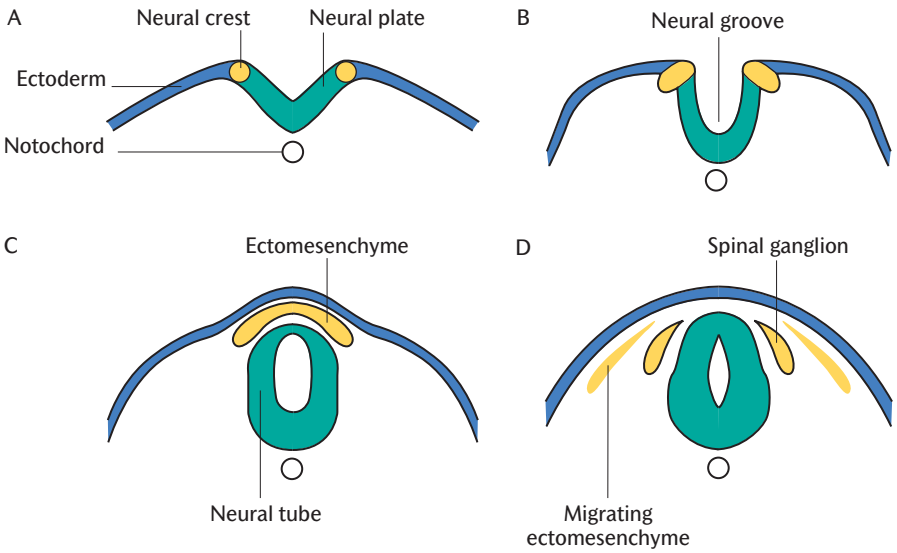

Fig. 8.4 Neurulation and formation of ectomesenchyme.

ultimately form the central nervous system and the motor components of peripheral nerves.

The notochord then induces the neuroectoderm to change shape into the shallow v-shaped **neural plate** overlying the notochord and the mesoderm immediately adjacent to it. The plate quickly becomes much wider at the head end, the region of the future brain, but remains narrower posteriorly where it will form the spinal cord. Compare Figure 8.4A and B and you will see that at the beginning of the fourth week of development, the neural plate deepens to form the **neural groove.** Cells in the neural plate change their shape from columnar to wedge-shaped to bring about the change in conformation of the neural plate. The cells on the crest of the neural groove at the junction of neural plate and ectoderm become distinguishable as a new tissue, the **neural crest**.

The next stage of development of the nervous system is shown in Figure 8.4C. As the neural groove cells continue to narrow at one end and become more wedge-shaped, the edges of the neural plate are brought together and eventually fuse in the midline to form the **neural tube.** The neural tube remains open at its superior and inferior ends for a few days, then closes completely in the fourth week. The completed neural tube is the precursor of the central nervous system; the cylindrical inferior portion will become the future spinal cord and a broader tube superiorly will develop into the brain.

The fate of neural crest cells

What happens to the neural crest as the neural tube forms? The answer is shown diagrammatically in Figure 8.4C and D and described here. Figure 8.4C shows that the neural crest tissue is not incorporated into the neural tube, but is pinched off to form a plate between the dorsal part of the neural tube and overlying ectoderm. Figure 8.4D indicates how almost immediately, some of these cells coalesce on each side of the spinal cord to form the dorsal root ganglia of spinal and cranial nerves whereas other cells migrate into the mesoderm. The cells that derive from the neural crest are referred to as **ectomesenchyme** as they are derived from *ecto*derm, but the cells behave as if they were from mesoderm (known as *mesenchyme* later in its development).

Ectomesenchyme migrates widely at all levels of the embryo, but far more neural crest cells are produced in the region of the developing brain area of the neural tube; much more ectomesenchyme is, therefore, found in the head region compared with the spinal cord level. Some derivatives of ectomesenchyme are found universally, some are found *only in the head and neck*; a few others are limited to the trunk.

- **Spinal and cranial nerve sensory ganglia** and the **sensory** components of the corresponding nerves, **sympathetic** and **parasympathetic ganglia** and **Schwann cells** providing the myelin sheaths of peripheral nerves, are formed from head to tail.
- **Melanocytes**, the pigment cells of the skin, are also derived from ectomesenchyme along the whole head-to-tail axis.
- In the head and neck, ectomesenchyme forms the dermis under the epidermis of the skin, **skeleton** of the facial skeleton and neck, but not the cervical vertebrae which are formed from the cervical somites (see p. 57). Most of the **skull**, the **meninges** surrounding the central nervous system, and **tendons** and **fascia,** the connective tissue components of the muscles of the head region, are also derived from ectomesenchyme. In short, ectomesenchyme does in the head and neck what mesoderm does elsewhere in the body. In the head and neck, the only tissue formed directly from mesoderm is muscle.
- **The adrenal medulla**, lying above the kidneys in the abdomen, is also derived from ectomesenchyme.

8.3.5 Further development of the germ layers

During the formation of the neural tube and ectomesenchyme, things are far from static in the other germ layers. To appreciate what else has been going on, we need to step back a few days.

By the middle of the third week, the embryo consists of three layers of cells. The ectoderm is continuous with the wall of the amnion above, the endoderm with the wall of the yolk sac below. The mesoderm is continuous with the extraembryonic mesoderm peripherally and separates the ectoderm and endoderm, except along the midline where it

is interrupted by the notochord centrally, the buccopharyngeal membrane, and cloacal membrane as described on p. 55. The neural tube is forming and ectomesenchyme is shortly to be added to the three extant germ layers. The ultimate fate of the cells emanating from each germ layer has already been indicated in general terms, but we need to examine some aspects of the further development of the germ layers, particularly the mesoderm, to appreciate how they give rise to more specific derivatives.

Mesoderm

The intraembryonic mesoderm initially forms a thin sheet of tissue on either side of the midline as it migrates from the overlying ectoderm. By day 17, the mesoderm on either side of the notochord proliferates and thickens to form a mass of tissue, the **paraxial mesoderm.** The mesoderm lateral to the paraxial mesoderm is the **intermediate mesoderm** and **lateral plate mesoderm** occupies the most lateral area.

The destination and subsequent fate of mesodermal cells is determined by their migration route through the primitive streak and node. The first formed intraembryonic mesoderm migrates through the most caudal part of the primitive streak and becomes lateral plate mesoderm. Cells migrating through the same area a little later become intermediate mesoderm. Epiblastic cells passing through near to the primitive node and in the superior areas of the streak become paraxial mesoderm. The light green arrows in Figure 8.3B show how some of the mesoderm migrates superiorly to extend around the buccopharyngeal membrane. This area of mesoderm is known as **cardiogenic mesoderm** and is the site where heart development begins.

The formation of somites from paraxial mesoderm

By the end of the third week of development, the paraxial mesoderm has proliferated to form two solid rods of tissue, one on each side of the central notochord. Follow up the mesoderm from the inferior end of the embryo in Figure 8.5A; about one-third of the way superiorly, you can see whorls of cells called **somitomeres** appearing at intervals along the paraxial mesoderm. In reality, somitomeres first appear adjacent to the superior end of the notochord. As shown in Figure 8.5A, the cell whorls clump together, a process known as **condensation,** to form bilaterally paired cuboidal masses, the **somites,** either side of the notochord and developing neural tube**;** the condensing somites are clearly visible as bilateral lumps at the superior end of the embryo under the overlying ectoderm. This process is referred to as **segmentation** and recapitulates the repeated segmental body pattern of our remote evolutionary ancestors outlined in Box 3.3.

Segmentation progresses from head to tail and the number of pairs of somites is often used as a measure of the age of the embryo. By the end of the fifth week of development, there are 42–45 pairs of somites. These are classified according to the areas of the body they give rise to into four **occipital** (or metotic), eight **cervical**, 12 **thoracic**, five **lumbar**, five **sacral**, and 8–10 coccygeal somites. You may realize that the numbers of cervical, thoracic, lumbar, and sacral spinal nerves are identical to the number of somites in the same area (see Section 3.3); this is no coincidence, but another manifestation of the same basic segmental patterning. The 'extra' somites in this scheme are the four pairs of occipital somites that eventually contribute tissue to the skull and tongue (see Section 21.6) and the coccygeal somites, the majority of which disappear as development proceeds.

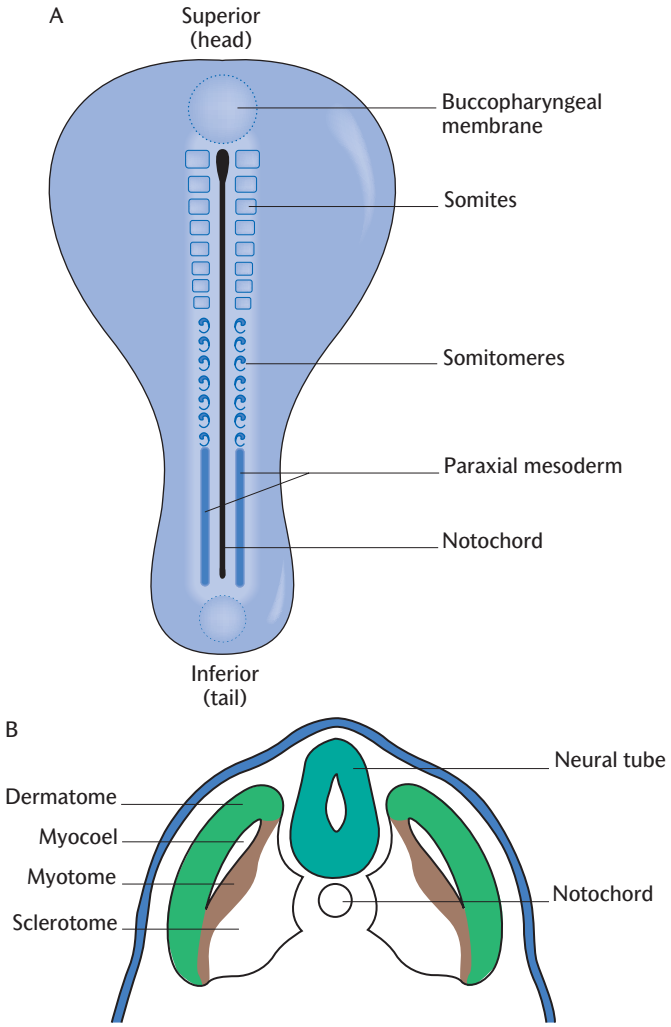

Fig. 8.5 Differentiation of somites. A) Somite formation seen from above; B) Somite formation in cross section.

Peripheral nerve development is related to somite development. There appears to be no overt segmentation of the spinal cord and motor nerves grow out from the developing spinal cord as a continuous set of rootlets. Sensory ganglia are formed from condensations of ectomesenchyme as it migrates laterally from its position above the neural tube. The migration route of the neural tissue is constrained by the somites; the superior part of each somite allows nerves to pass through but the inferior part inhibits their passage. The nerves are, therefore, channelled along specific routes to form distinct spinal nerves related to the superior end of each somite. The motor nerves aggregate with the peripheral components of the sensory nerves to form mixed spinal nerves beyond the ganglia.

Further differentiation of somites

As segmentation progresses from head to tail, the first formed somites begin to differentiate further during the fourth week of development. As can be seen in Figure 8.5B, each somite has a vertical, slit-like cavity, the myocoel, which is soon obliterated by cell division as the somite grows. The tissue lateral to the myocoel is the **dermatome**

and will eventually spread out beneath the ectoderm to become the dermis of the skin. The somite tissue medial to the myocoel has two divisions. The medial division nearest the notochord is the **sclerotome;** this differentiates into connective tissue or **mesenchyme** and migrates to form a mass of tissue around the notochord and developing neural tube. The mesenchyme derived from the sclerotome will eventually form the vertebral column, ribs, and sternum. The lateral division becomes the **myotome** which differentiates into muscles of the vertebral column, the intercostal muscles between the ribs, and the limb muscles in the lower cervical and upper thoracic region, and lumbar and sacral regions.

The intermediate mesoderm lateral to the developing somites gives rise to the urinary system, the adrenal cortex, and much of the reproductive system. The lateral mesoderm of the intraembryonic mesoderm is continuous with the extraembryonic mesoderm surrounding the amniotic cavity and yolk sac; its further development involves the formation of a new cavity, the **intraembryonic coelom.**

The somites are confined to the region of the embryo between the buccopharyngeal and cloacal membranes. However, as seen in Figure 8.3B, lateral plate mesoderm extends into the areas anterior and posterior to these two membranes. A series of vacuoles appear in the lateral plate mesoderm and coalesce to form the intraembryonic coelom. As shown by the pink-shaded area in Figure 8.6, the intraembryonic coelom develops into a U-shaped cavity with its base in front of the buccopharyngeal membrane, linking its two arms that pass backwards each side of the paraxial mesoderm. The anterior part of the intraembryonic coelom is the primitive pericardial cavity where cardiogenic mesoderm will form the heart and the lateral limbs are the primitive pleural and peritoneal cavities that enclose the lungs and abdominal viscera, respectively. Lateral to the arms of the U, the mesoderm breaks down so that intraembryonic and extraembryonic coeloms are continuous over a certain distance. These cavities act as a primitive circulatory system to distribute fluids and their nutrients around and into the embryo (see Section 13.2.1). Eventually, these cavities will be replaced by true blood vessels.

Box 8.3 Embryonic stem cells

Blastomeres are totipotential stem cells that can form a whole embryo. As the germ layers form, the potential fate of the cells becomes more restricted to form certain types of cell; the repertoire of cells that can develop from a given set of embryonic cells become more restricted as development proceeds. The most obvious example of progressive restriction is when the paraxial mesoderm segregates into dermatome, myotome, and sclerotome. Myotome cells can only become muscle cells whereas sclerotome cells can give rise to cells that will form bone, cartilage, and other elements of the skeletal system.

In most tissues and organs, some cells remain in an embryonic state as **stem cells** and can give rise to new cells to replace cells damaged by disease, trauma, or general wear and tear. The cells forming the basal layer of skin epithelium are a good example of 'permanent' stem cells; as cells are lost from the surface of the skin in the course of daily activities, they are replaced from below. A basal cell will divide and one of the daughter cells will mature into a skin cell whereas the other cell will remain in the basal layer as a stem cell to divide again and again as circumstances demand.

One of the objectives of developmental biology research is to be able to identify and isolate these undifferentiated stem cells from different tissues. By manipulation of these cells in the laboratory by providing the correct nutrients and appropriate signalling molecules, the stem cells can be directed down a specific line of differentiation to produce new cells. The new population of cells could then be reintroduced into the body to replace cells lost through trauma and disease. Although progress in this field is rapid, we are still a long way from being able to use this technology as a cure for various diseases.

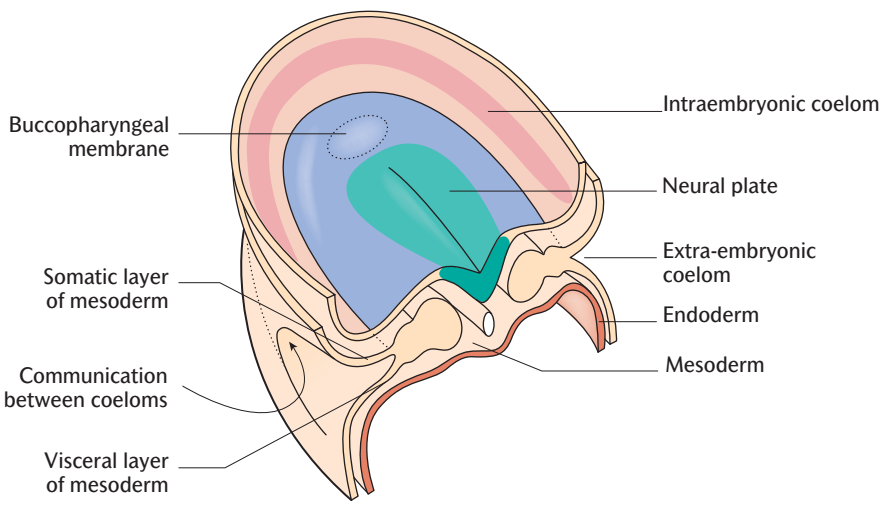

Fig. 8.6 Formation of the intraembryonic coelom.

8.4 Folding of the embryonic plate and its consequences

As already indicated, the endoderm forms the cells that line the respiratory and gastrointestinal tracts. The flat embryonic disc has to become folded if the endoderm is going to be in a suitable location to become these linings.

In earlier stages of development, the embryo is a flat plate of cells differentiating into the different germ layers. The growth of the ectoderm during formation of the neural tube and of mesoderm during segmentation has been described; this growth has profound consequences for the relative positions of the embryonic germ layers and specific structures formed from them. Most of the growth takes place on the dorsal surface initially and the principal effect is that the embryo bulges upwards into the amniotic cavity. The resulting folding or flexion of the embryo is not confined to the transverse axis but occurs longitudinally too.

We will follow a series of diagrams illustrating the folding of the embryo in longitudinal and transverse sections to observe the consequences of folding on the relative position of different organs and tissues.

8.4.1 Longitudinal folding

Figure 8.7 is a series of sequential sagittal sections of the embryo, starting with the flat embryonic disc in Figure 8.7A. The following features can be seen in sequence, moving from *left to right* on the diagram which is from head to tail on the embryo:

- Converging layers of ectoderm and endoderm;
- A region of lateral plate mesoderm between the ectoderm and endoderm known as the **septum transversum**;
- The primitive **pericardial cavity** in the intraembryonic coelom;
- The **buccopharyngeal membrane**;
- A sandwich with **neural plate** above, **notochord** between, and **endoderm** below, forming most of the length of the embryo;
- The cloacal membrane;
- Diverging layers of ectoderm and endoderm.

You can also see as you study the sequence of diagrams comprising Figure 8.7 that the neural plate expands considerably at the head end to produce the rudiments of the brain, displacing the pericardial cavity inferiorly into the chest region. As the neural plate expands anteriorly and posteriorly to form the central nervous system, the longitudinal curvature of the embryo increases and the relative position of the other tissues changes.

As shown in Figure 8.7B, the buccopharyngeal and cloacal membranes become folded under the superior and inferior ends of the embryo, respectively, and in the process, they rotate so that their originally ventral surfaces come to face dorsally. As the curvature continues to increase, as seen in Figure 8.7C, part of the yolk sac becomes incorporated into the embryo to become the gastrointestinal tract. Figure 8.7D shows how the communication between the gut and the yolk sac becomes constricted as the folding becomes more pronounced until it is eventually reduced to the narrow **vitelline duct** which will form the umbilical cord.

Note also the radical alteration to the position of the pericardial cavity as folding takes place. It moves from its position anterior to the embryo shown in Figure 8.7A to lie beneath the superior part of the gut (the foregut) in the future chest region shown in Figure 8.7D. The septum transversum also moves to lie between the pericardial cavity and the enclosed yolk sac; eventually, the diaphragm and the liver will form in the septum transversum. In Figure 8.7C, the buccopharyngeal and cloacal membranes, the only areas where the

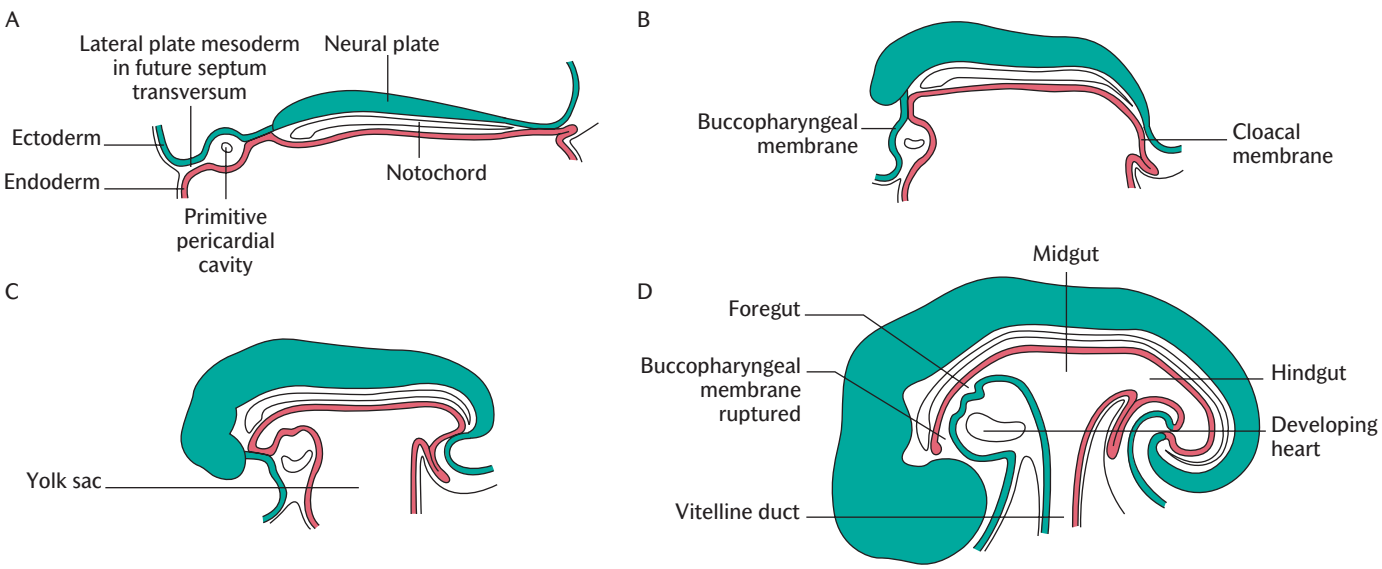

Fig. 8.7 Longitudinal folding of the embryo.

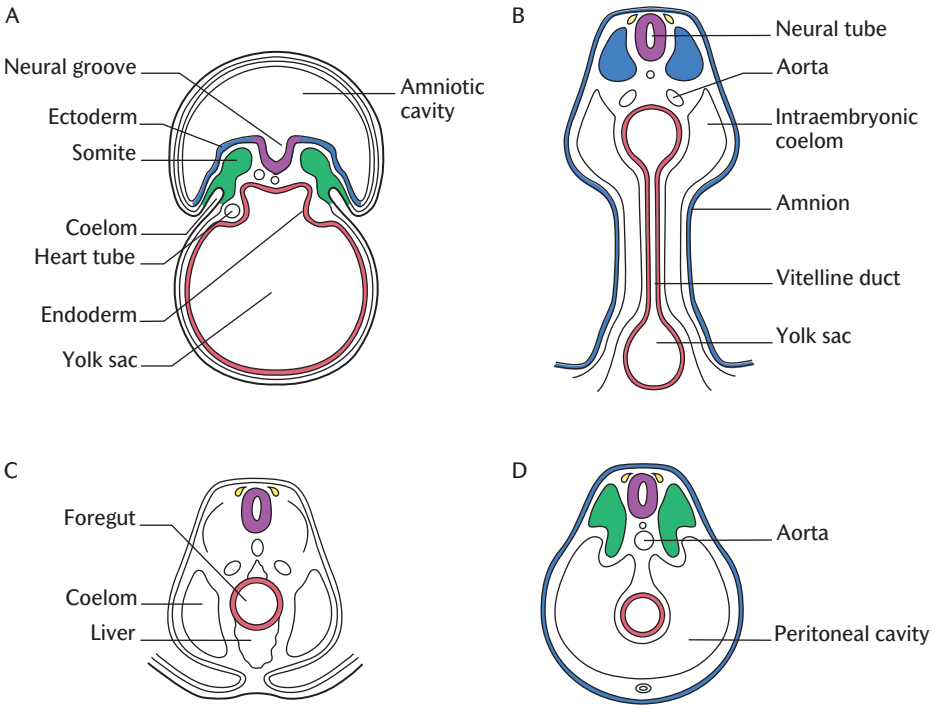

Fig. 8.8 Transverse folding of the embryo.

ectoderm and endoderm are not separated by the mesoderm, come to lie at the beginning and end of the forming gastrointestinal tract. In Figure 8.7D, you can see that both membranes break down so that the gastrointestinal tract is now open at both ends. The superior end is the position of the future oral cavity and the inferior end is the position of the anus.

8.4.2 Transverse folding

A series of transverse sections through the flat embryonic disc are shown in Figure 8.8. Identify the amniotic cavity, then the following layers, starting from the base of the cavity and progressing downwards:

- The **ectoderm**, thickened centrally to form the neural plate;
- The **notochord** centrally flanked by blocks of **mesoderm, somites**; note how the lateral plate mesoderm splits laterally to form two layers continuous with the extraembryonic mesoderm separated by the intraembryonic **coelom**;
- The **endoderm**.

The greatest growth is in the neural plate. By comparing Figure 8.8A and B, you can see that increases in size of the neural plate cause the lateral margins of the embryonic disc to rotate downwards about an axis approximately through the notochord. This movement incorporates part of the yolk sac into the embryo as the gastrointestinal tract, as already seen during longitudinal folding described in Section 8.4.1.

The lateral margins of the folding disc eventually come into contact as transverse folding proceeds as shown in Figure 8.8C; the part of the yolk sac enclosed by the lateral folding of the embryo forms a tube to become the gastrointestinal tract. The intraembryonic coelom will form the peritoneal cavity in which the gastrointestinal tract and its

Box 8.4 Teratogenic effects of drugs and disease

As we have seen, the complex processes of development require precise coordination. It can take very little to disrupt these processes and the results can be serious. The causes of many developmental defects are unknown, but the causative agents of some conditions have been identified. Such agents defects are called **teratogens** (from the Greek = monster-forming). Two examples are given; although they actually affect events outside the first three weeks, they are graphic illustrations of teratogens, many of which probably act in the time window of this chapter.

Morning sickness often afflicts pregnant women and is usually most prevalent in the sixth to twelfth weeks of pregnancy; the limbs are developing in the early part of this time. In the late 1960s, a drug, thalidomide, was developed to relieve the symptoms of morning sickness. This drug proved to have disastrous side effects and interfered with the processes that control the formation of the limbs. Many babies born where thalidomide was prescribed were born with malformations of arms and legs as a result.

All females in developed countries are now immunized against **rubella (German measles)** at around the time of puberty. German measles in itself tends only to produce a rash and high fever and a general feeling of being unwell and lasts, at most, only a week or two. If a pregnant female contracts rubella up to the twelfth week of pregnancy, infection by the rubella virus may produce **congenital rubella syndrome**. There are three common malformations which may occur singly or in any combination. The inner ear is affected in about 60% of cases, resulting in hearing loss, the eyes may be small or have cataracts (40%), and heart development is affected in about 50% of cases; patent ductus arteriosus is a frequent manifestation of heart malformations (see Section 13.2.4).

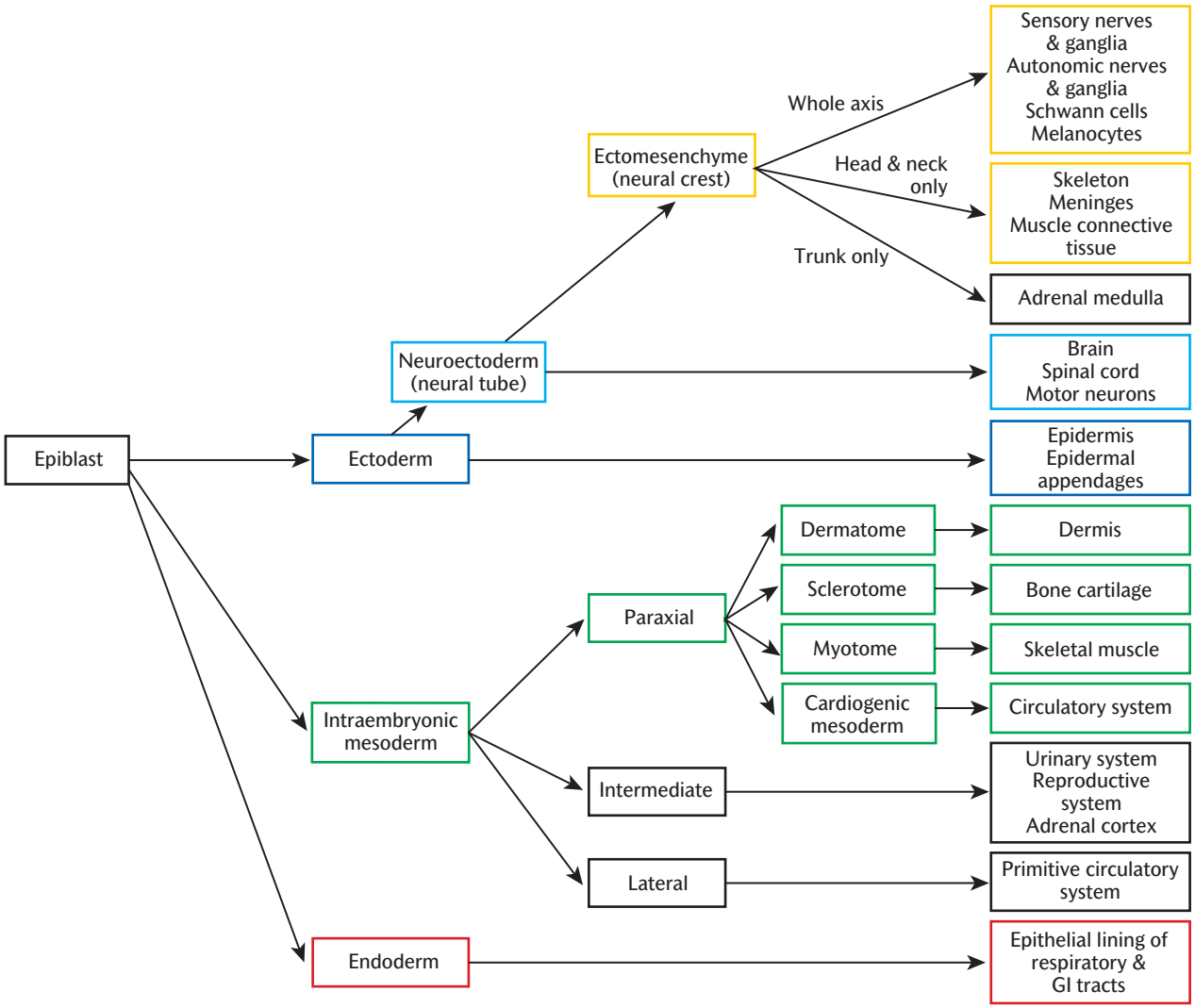

Fig. 8.9 The origins of the embryonic germ layers and their derivatives.

associated organs will develop. The ectoderm also grows round to form the outer covering of the entire embryo.

This is a brief overview of the complex changes that occur to generate a whole organism from a single cell. The formation of the embryonic germ layers and their derivatives are summarized diagrammatically in Figure 8.9. Box 8.4 gives examples of the ways in which disease and therapeutic drugs can affect the complex sequence of events in the early stages. The details of how specific tissues and organs are formed by the interaction of the embryonic germ layers will be described in the context of their anatomy in Chapters 13, 19, 21, 32, and 33.

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Section 2
The thorax

Section contents

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9 The surface anatomy of the thorax

Chapter contents

9.1 Introduction

The thorax is the region of the body commonly known as the chest between the neck and the abdomen. The **thoracic cavity** is the hollow in the thorax that is occupied by the thoracic viscera, the **heart** and its associated vessels in the midline, and the **lungs** laterally. The thoracic viscera are enclosed by the bony and muscular **thoracic cage**. The bony components of the cage are the 12 **thoracic vertebrae** posteriorly, the 12 pairs of **ribs** and their anterior cartilaginous extensions, the costal cartilages that meet the **sternum** anteriorly. The **intercostal muscles** fill the intercostal spaces between the ribs and are involved in ventilation. Another muscle involved in ventilation is the **diaphragm**, a sheet of muscle that separates the thoracic from the abdominal cavity. If you are not familiar with the basic outline and arrangements of the circulatory and respiratory systems, refer back to Chapters 4 and 5 before reading this section.

A good way to appreciate where these structures lie in relation to each other is to examine their **surface anatomy,** the position of internal organs related to features that can be observed or palpated (felt) on the surface of the body. Relating surface anatomy to deeper structures is a clinical skill essential not only to the study of the thorax, but also of structures in the head and neck important in dental practice.

9.2. Reference landmarks

In the clinical examination of the living subject, the position of the internal thoracic organs is defined with reference to a set of vertical and horizontal lines running through the surface of bony landmarks. The significant vertical lines are shown in Figure 9.1 as the:

1. Mid-sternal line—in the median plane anteriorly;
2. Mid-clavicular line—through the midpoint of the clavicle;
3. Mid-axillary line—midway between the anterior and posterior axillary folds, formed from skin overlying muscles. If you raise your arm while looking into a mirror, the two folds are obvious; they can also be palpated very easily even with clothes on.
4. Median posterior line—in the median plane anteriorly.

The horizontal position can be defined with reference to the ribs or, less easily, the vertebrae.

9.3 The anterior thoracic surface

Examine Figure 9.1 and yourself, if convenient to do so, at the same time as reading the description. First, run your finger down the anterior midline of your neck. As you do so, you will feel the cartilaginous rings reinforcing the trachea until it disappears behind the sternum. You should now feel the **suprasternal notch** on the superior margin of the sternum. If you move your fingers laterally from the notch, you will feel the heads of the **clavicles** on each side as they articulate with the upper part of the sternum. As you can see in Figure 9.1, the first rib lies deep to the clavicle and consequently, is difficult to palpate. It is more practical, therefore, to start rib counting from the second rib. If you return your finger to the midline of the suprasternal notch and run it vertically downwards in the midline, you will encounter the **sternal angle**, a ridge made by the joint between the manubrium and the body of the sternum about 2.5 cm below the sternal notch. If you now slide your finger laterally, it will pass on to the skin over the second rib and its associated costal cartilage. The second intercostal space is below the second rib and costal cartilage. Once the level of the **second rib** is established, it is simple to count down the successive ribs and intercostal spaces.

Return to the midline and continue tracing the sternum down. At the lower border of the sternum, you may be able to feel its short sharp downward process, the xiphoid process. Once you get to this level, you will have to move your hands laterally to be able to feel the lower ribs and intercostal spaces. As you can see in Figure 9.1, the **costal margin,** the lower margin of the thoracic cage, curves downwards and laterally. The costal margin is formed by the costal cartilages of the seventh, eighth, ninth, and tenth ribs. There are usually 12 pairs of ribs in all, but you will probably not be able to feel the eleventh and twelfth rib unless you are thin as they are short and have free ends.

Figure 9.2 shows the results of projecting a series of imaginary line from various points on the sternum backwards through the thoracic cavity. A horizontal line from the sternal angle intersects the intervertebral disc between the fourth and fifth thoracic vertebrae (T4 and T5). This is the **sternal plane** and is another important landmark as you will see

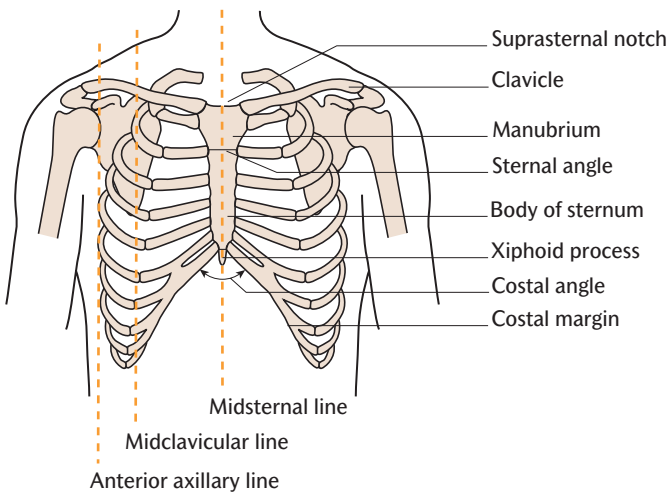

Fig. 9.1 Thoracic landmarks on the anterior thoracic surface.

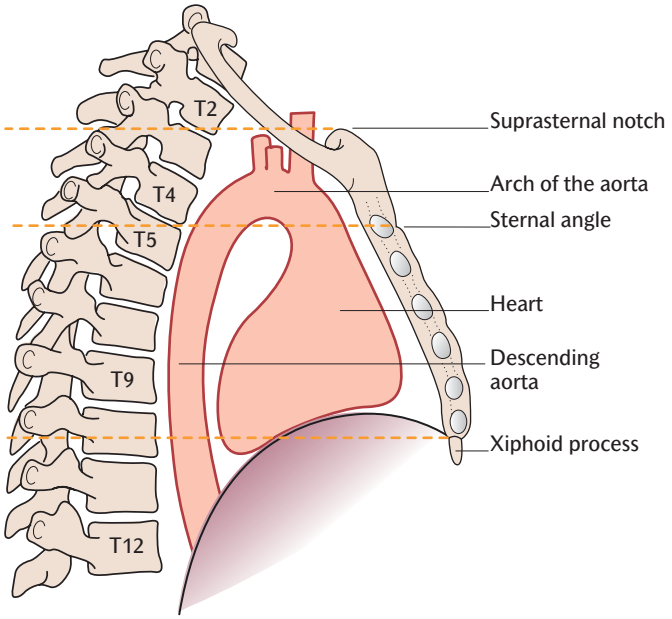

Fig. 9.2 Thoracic landmarks viewed lateral.

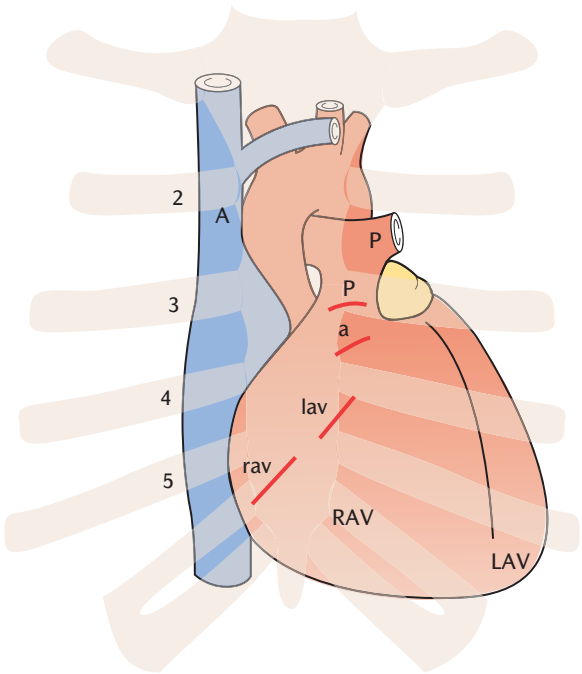

Fig. 9.3 Surface markings of the heart in relation to the ribs and sternum in anterior view. The anatomical position of the aortic, pulmonary, left, and right atrioventricular valves is shown by a, p, lav, and rav, respectively. The site where valve sounds are heard loudest with a stethoscope is shown in UPPER CASE letters.

below. The sternal notch is on the same level as T2 and the xiphoid process on the same level as T9.

9.3.1 The heart

The heart occupies a midline position in the thoracic cavity between the sternum anteriorly and the thoracic vertebrae posteriorly. Figure 9.3 shows the outline of the heart viewed from the front with the superimposed sternum and ribs indicated by grey shading. The heart is best imagined as quadrangular in outline with the superior border being narrower than the lower border. The easiest landmark to locate is the lower left angle which is in the fifth left intercostal space in the midclavicular line. This position, labelled LAV in Figure 9.3, also marks the place where the **apex beat** of the heart can be listened to (auscultated) with a stethoscope. The sound is produced by closure of the left atrioventricular valve as the left ventricle contracts. Locate the apex beat on your own chest by placing your hand over the heart. Note where the lowest and outermost beat is and then count ribs to establish that your apex beat is in the right place. If you cannot feel the apex beat, try leaning forward a little.

The apex beat is also the best place to auscultate the general heart sound, usually rendered as 'lub dup'. The apex is the lowest and outermost point of definite cardiac pulsation. Its position varies to some degree with posture, build, and height, and can alter significantly in pathological conditions where the heart is enlarged or displaced (see Box 9.1).

The location of the rest of the heart can be visualized or marked out on the chest wall once the position of the apex beat is located. Trace the points in Figure 9.3 as you read the description. The lower right angle of the heart is where the fifth right costal cartilage joins the sternum. The upper right angle is more or less in the mid-sternal line at the level of the third costal cartilages. The upper left angle is slightly lateral to the left margin of the sternum behind the third left costal cartilage.

Box 9.1 Heart sounds

The sounds produced by closure of the heart valves are diagnostically important in many heart diseases. The actual anatomical locations of the heart valves are marked by bars in Figure 9.3. Note that they lie quite close together. When auscultating the heart with a stethoscope, it is difficult to separate the sounds of individual valves if the stethoscope were to be placed directly over the position of the valve in question. Clinically, the sounds of individual valves are auscultated where the loudest sound from each valve can be best heard. These usually correspond to where sound is conducted through a heart chamber or blood vessel. Their positions are marked in Figure 9.3 in upper case letters.

9.3.2 Lungs and pleura

The lungs covered by the pleura occupy the rest of the thoracic cavity. Blue lines in Figure 9.4 indicate the division of each lung into lobes. The right lung has three lobes; superior, middle, and inferior. The superior and middle lobes are separated by the **horizontal fissure** and the middle and inferior lobes by the **oblique fissure**. The left lung has only two lobes as it is smaller than the right lung due to the presence of the heart; the superior and inferior lobes are separated by the **oblique fissure**. You can follow the oblique fissures from posterior to anterior by starting in Figure 9.4B from the spine of the third thoracic vertebra (T3); continue tracing the oblique fissure on to Figure 9.4A where you can

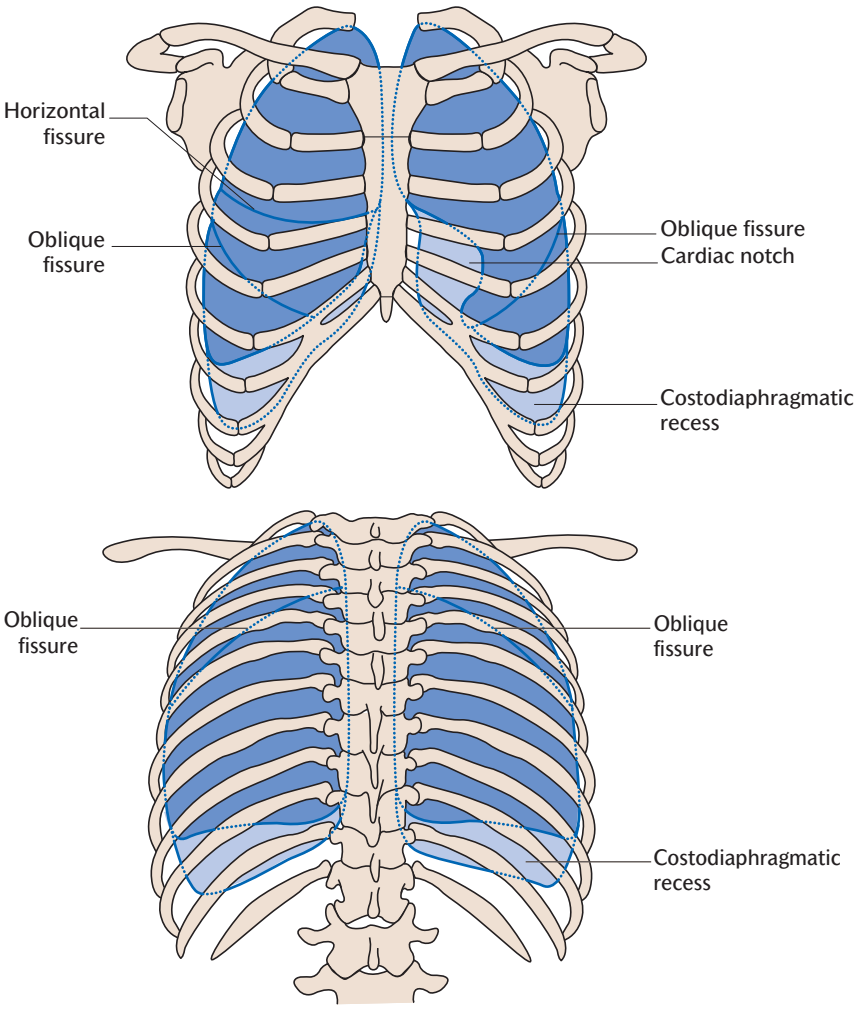

Fig. 9.4 Surface markings of the lungs (dark blue) and pleura (light blue) in: A) Anterior view; B) Posterior view.

see that it runs to the sixth rib. As marked in Figure 9.4A, the **horizontal fissure** of the right lung corresponds to a line drawn horizontally along the fourth costal cartilage and rib to meet the oblique fissure at the mid-axillary line.

Figure 9.4 also shows the **surface markings** (or **surface landmarks)** of the lungs and pleura (their projection on to the anterior and posterior chest walls). The position of the lower borders of the lungs varies as you breathe in and out so their projection is usually a compromise, giving their position in mid-inspiration. The inferior extent of the parietal pleura is shown in light blue in Figure 9.4. As you can see, they extend lower than the lungs. The apparent gap between the lower borders of the lungs and pleura is a potential space, the **costodiaphragmatic recess** (see Section 10.4.2). This space is roughly two ribs wide at its greatest extent.

Observe also in the anterior view in Figure 9.4A that the lungs and their covering pleura extend above the margin of the first rib into the lower neck. As the first rib cannot be palpated, the surface landmarks of these upward extensions are referred to the clavicle instead. The apex of each lung projects about 2 cm above the medial third of the clavicle. The use of these surface landmarks in lung examination is covered in Box 9.2.

Box 9.2 Auscultation of the lungs

Auscultation of the lungs to listen to the sound of air entering and leaving is an important diagnostic tool in the investigation of several respiratory diseases. Each lung is examined thoroughly by placing the stethoscope over each lobe of each lung in turn. If you have ever had a chest examination, you probably thought that your doctor was placing the stethoscope at random. They were not; they were making use of the surface landmarks and experience to examine the lungs thoroughly. Note from Figure 9.4A and B that the upper and middle lobes can be auscultated most easily from the front whereas the lower lobe is most accessible from the back.

10

The thoracic wall and diaphragm

Chapter contents

10.1 The thoracic cage

The thoracic wall is made up of skeletal elements that form the thoracic cage (or more commonly, but less accurately, the rib cage) and muscles that move the components of the thoracic cage relative to each other for ventilation and postural movement. The thoracic cage is made up posteriorly by the thoracic part of the vertebral column, laterally and anteriorly by the ribs and costal cartilages, and by the sternum in the anterior mid-sternal area.

10.1.1 The thoracic vertebral column

The thoracic vertebral column is made up of 12 **thoracic vertebrae** and their intervertebral discs. The thoracic vertebrae are not arranged in a straight line, but are concave anteriorly as shown in Figure 9.2.

All vertebrae have the following general configuration as shown in Figure 10.1A:

- A heart-shaped **body** with two backward projections, the **pedicles**, either side of the **vertebral foramen**. The foramen forms the spinal canal with the foramina of other vertebrae. Note in Figure 10.1C that the pedicles are slightly shallow above and strongly grooved below to form **intervertebral foramina** with adjacent vertebrae for the passage of spinal nerves;
- Two stout **transverse processes** running laterally and slightly posteriorly;
- Two flat plates called **laminae** which join to form a long **spinous process**—you can feel the tips of the spinous processes very easily under the skin in the midline of your back;
- Superior and inferior **articular processes** at the junction of the pedicles and laminae. In thoracic vertebrae, the superior facets are set vertically with the facets on the superior processes facing posterolaterally and those on the inferior processes anteromedially; the relative movement of the vertebrae is thus mainly rotary, but there is very little actual movement in the thoracic part of the vertebral column.

The thoracic vertebrae are modified from this basic pattern to articulate with the ribs through several more articular facets as shown in Figure 10.1 A, B, and C. They carry on each side:

- Shown most clearly in Figure 10.1 C, a superior and inferior **demi-facet** (a half facet) on each side of the body for the **heads** of two ribs in the case of T2–T9 or a single complete facet for the head of one rib in the case of T1 and T10–T12;
- Shown in Figure 10.1 A and B, a facet near the tip of each transverse process for the **tubercle** of a rib (except T11 and T12).

Vertebrae are separated from each other by an **intervertebral disc** as shown in Figure 10.2C. Each intervertebral disc has an outer ring of fibrocartilage, the **annulus fibrosus**, which encloses the **nucleus pulposus**, an ovoid gelatinous mass. The intervertebral discs are a shock-absorbing mechanism; sudden downward forces will tend to compress the nucleus vertically, but the simultaneous lateral expansion will be resisted by the fibres of the annulus. The slight degree of movement possible between adjacent vertebral bodies occurs by a rolling

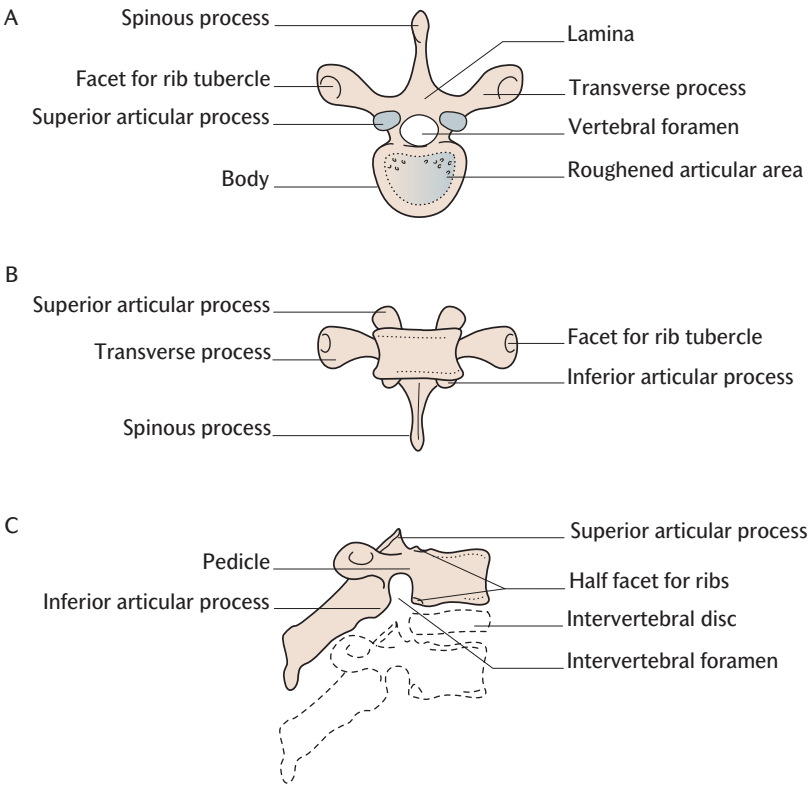

Fig. 10.1 A typical thoracic vertebra seen in: A) Superior; B) Anterior; C) Lateral views.

movement on the relatively incompressible nucleus with the annulus being stretched on one side and compressed on the other. The effects of overload on the intervertebral discs are described in Box 10.1.

Box 10.1 Slipped disc

Sometimes forces generated are so great that the nucleus of the intervertebral disc herniates through the annulus; this is a **slipped disc** and is most usually in the lumbar region of the vertebral column. The herniated nucleus may compress the spinal cord and inhibit nerve transmission; the body would be paralysed and anaesthetic below the level of compression. More commonly, the herniated nucleus extrudes laterally and impinges on the spinal nerve that exits at that level on that side, causing pain to radiate down the distribution of the nerve. With increasing age, the nucleus becomes thinner and less aqueous and is eventually converted to fibrocartilage; some degree of mobility in the vertebral column is lost as these age changes occur.

10.1.2 Ribs and costal cartilages

A typical rib has the following features shown from above and anteriorly in Figure 10.2A and B respectively. Most of the length of the rib is formed by the **shaft** which is convex externally. The superior border is rounded whereas the inferior border is sharp; these features are best seen in Figure 10.2B. The sharp lower border is the lateral edge of the **costal groove** on the medial surface, a shallow groove running along the lower border that houses the intercostal artery, vein, and nerve. The radius of curvature of the shaft decreases at the **angle,** seen best in Figure 10.2A. In Figure 10.2A, observe the **tubercle,** a posterior projecting elevation just medial to the angle. It has a small facet which articulates with the transverse process of a vertebra; this feature is absent from ribs 11 and 12. The stout **neck** connects the tubercle and the **head** which carries superior and inferior facets that articulate with the bodies of the vertebrae as shown in Figure 2.6B; note that ribs 1, 10, 11, and 12 have only one facet. The first rib is very atypical; it is described in Box 10.2 for those who require such detail.

Each rib is continued anteriorly by a costal cartilage that articulates either directly or indirectly with the sternum anteriorly.

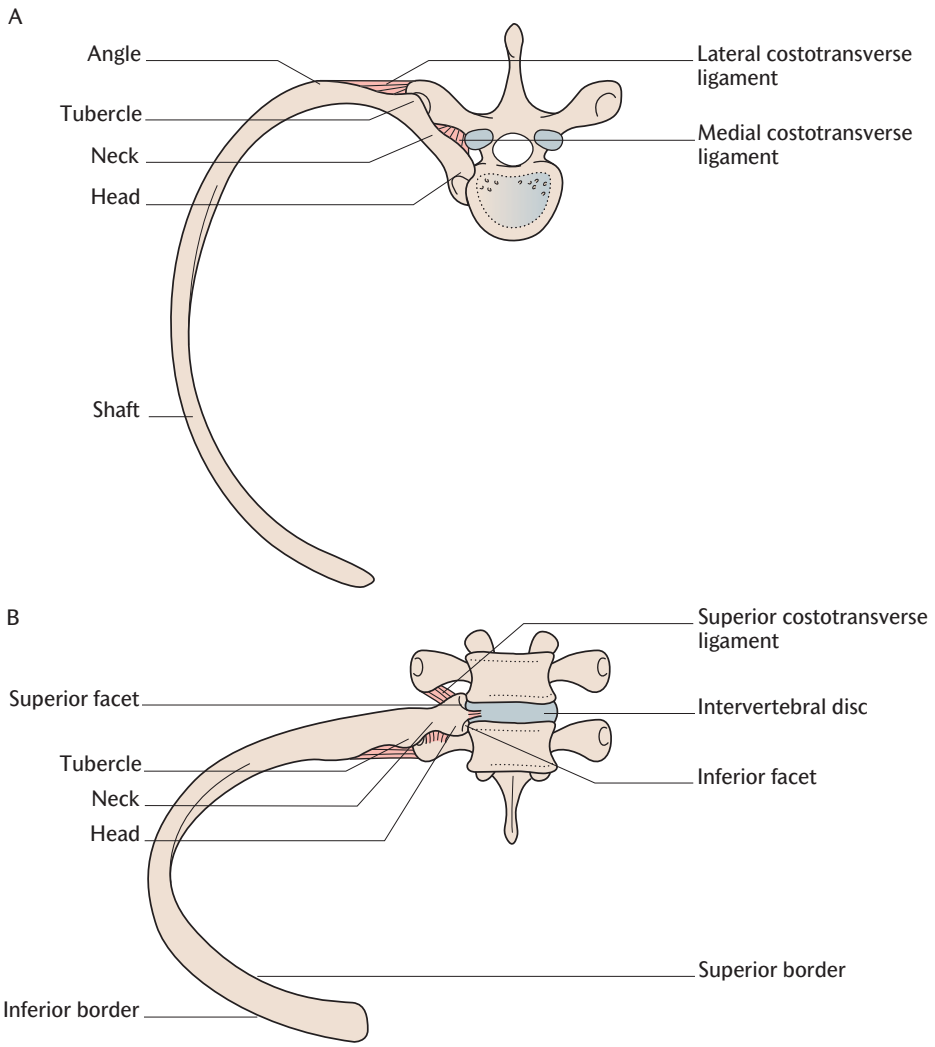

Fig. 10.2 The articulations between a rib and vertebrae. A) Superior view. B) Anterior views.

Box 10.2 The first rib

The **first rib** is atypical as you can see in Figure 10.3; it is flattened from above downwards and is shorter and more curved than any other rib. It shows prominent rough areas on its upper surface where the scalene muscles, arising from the cervical vertebrae, attach to it. The first rib is often grooved between the muscle attachments by the roots of the brachial plexus and the subclavian artery. A similar marking may be made by the subclavian vein in front of the attachment of scalenus anterior.

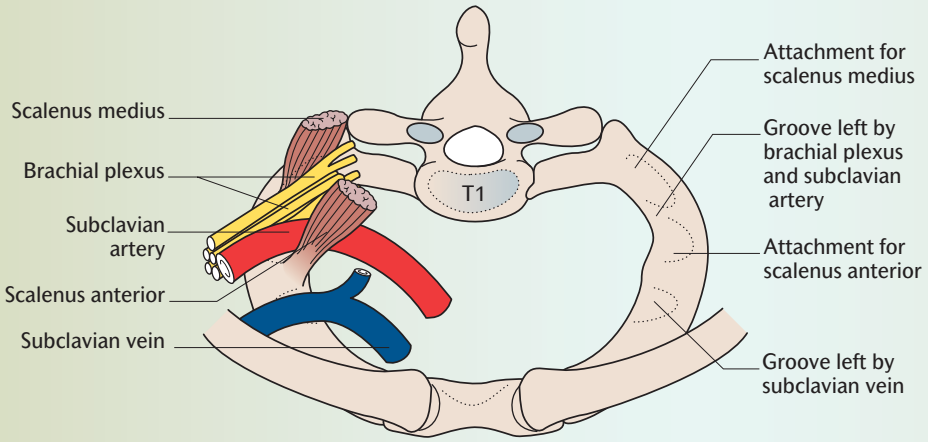

Fig. 10.3 The first rib and structures related to it.

Costal joints

As illustrated in Figure 10.2, each rib has several articulations.

- The major articulation of the head of each rib with the vertebrae is seen best in Figure 10.2B. The inferior facet on the head of each of ribs 2–9 articulates with the superior demi-facet on the body of the corresponding vertebra. The superior facet articulates with the inferior demi-facet of the vertebra above and with the intervertebral disc between them. The heads of ribs 1, 10 (usually), 11, and 12 have a rounded head with a single articular facet that articulates *only* with the corresponding vertebra. These joints are all synovial.
- As seen in Figure 10.2A, the tubercles of the first ten ribs form synovial joints with the facets of the transverse processes of the corresponding vertebrae. Strong costotransverse ligaments unite each rib and vertebra.

The double articulation of each rib with the bodies of two vertebrae and the transverse process of its corresponding vertebra and the relative positions of these attachments means that the ribs twist on their articulations rather than slide up and down (see Section 11.4)

10.1.3 The sternum

Identify the three parts of the sternum in Figure 10.4; the **manubrium**, **body**, and **xiphoid process.** We have already met these while studying the surface anatomy of the thorax in Chapter 9. These components develop separately, but fuse by late teen age.

The concave **suprasternal notch** is the upper border of the manubrium, deepened by the sternal ends of the clavicles articulating with the sternum at the synovial sternoclavicular joints. The costal cartilages of the first ribs articulate immediately below the sternoclavicular joints.

The body articulates with the manubrium by a fibrocartilaginous joint, allowing movement of about 2 degrees during maximum inspiration. This joint produces the sternal angle; the second costal cartilage articulates with the sternum at this level. The third to seventh costal cartilages articulate with the body of the sternum.

The xiphoid process extends downwards and slightly backwards from the body so that the anterior surface of the xiphoid is thus inset. The xiphoid varies in length and is made of cartilage initially. In older age, the cartilage usually calcifies.

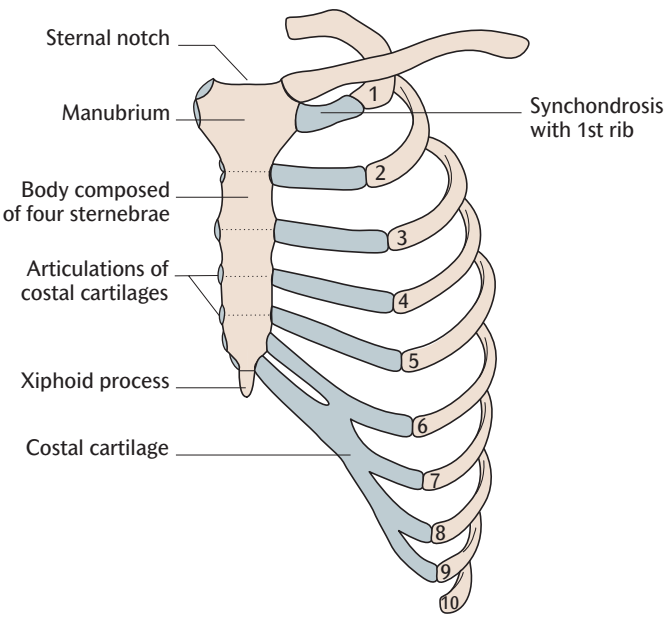

Fig. 10.4 The sternum and its articulation with the ribs.

The articulations of the costal cartilages with the sternum are also shown in Figure 10.4; some connect directly whereas others are attached indirectly. The first seven ribs articulate via their costal cartilages directly with the sternum and are known as **true ribs**. The cartilages of ribs 8, 9, and 10 articulate with the cartilages immediately above them and hence to the sternum by the seventh costal cartilage; these are **false ribs**. The two lower ribs are designated as **floating ribs** because the short costal cartilages of ribs 11 and 12 are free. The cartilages of the true ribs originally articulate with the sternum at synovial joints, but the cavities of these synovial joints are usually obliterated by fibrous union. The first costal cartilage is united with the manubrium by a synchondrosis. Rib fractures are outlined in Box 10.3.

10.1.4 General features of the thoracic cage

There are several features of the thoracic cage that are important to understand in the context of movement of the chest wall. You can confirm the points listed below on a mounted skeleton and some of the features can also be seen in Figure 10.4.

- Most ribs run downwards and anteriorly, but their costal cartilages run upwards and anteriorly; the first rib and its costal cartilage run downwards throughout.
- The sternal attachment of each rib lies lower than its vertebral attachment.
- The middle of each rib around the mid-clavicular line lies lower than either end.
- Ribs and their cartilage increase in length from the first to the eighth rib, then decrease again; the transverse diameter of the thorax is thus maximum at the eighth ribs.
- The inferior margin of the cage is formed by the twelfth thoracic vertebra posteriorly, the twelfth ribs and the tips of the eleventh ribs at the sides, and by the cartilages of the tenth to seventh ribs anteriorly. In the anterior midline, the margin is formed by the xiphoid process.

Box 10.3 Fractures of the ribs

The ribs are not fully ossified until adult life so the rib cage of children is very flexible and fractures are rare. In adults, rib fractures tend to occur near the angle. The upper two ribs protected by the clavicle and the lower two floating ribs are the least likely to be fractured.

10.2 Thoracic muscles and intercostal spaces

The thorax cage is more or less entirely covered in muscles. Many, such as the pectoral muscles running from the anterior thoracic wall to the humerus in the upper limb and the abdominal muscles running from the lower ribs down to the pelvis, can play a role in ventilation (see Section 11.4.2). The intercostal muscles of the thoracic wall are located beneath the postural muscle layer.

10.2.1. Intercostal muscles

The **intercostal spaces** between the ribs are occupied by intercostal muscles attaching to the ribs above and below each space. As Figure 10.5 illustrates, the muscles are arranged in three layers, but in practice, only the two outer layers are functionally significant.

The **external intercostal muscles** form the outer layer and their fibres run forwards and downwards. Each muscle extends anteriorly from the tubercle of the rib as far forward as the costochondral junction where it is replaced by a fibrous aponeurosis (see Figure 10.6).

The internal intercostal muscles make up the middle layer and their fibres run backwards and downwards. These muscles extend from the sternum anteriorly to the angles of the ribs where they are replaced by aponeurotic tissue as shown in Figure 10.6. The fibres of the external and internal intercostal muscles run at right angles to each other.

The **intercostalis intimi** muscles form the inner layer; this muscle layer is usually incomplete. Muscle is only present in the lower intercostal spaces and in the middle part of the ribs as shown in Figure 10.6. Their fibres are orientated in the same direction as the internal intercostal muscles and function with them.

The intercostal muscles play a major role in ventilation. Both external and internal muscles elevate the ribs, but the internal intercostals can also depress the ribs, depending on the action of other fixator muscles (see Section 11.4.1).

Intercostal neurovascular bundles

The main vascular and nervous supply to the tissues of intercostal space and adjacent tissues runs as a neurovascular bundle just below the costal groove of the rib forming the upper boundary of each intercostal space. The nerves and blood vessels are protected by the sharp inferior margin of the rib. The bundle runs deep to the internal intercostal muscles.

The **intercostal arteries** supply the skin and subcutaneous tissues over the side of the chest wall and give branches to the intercostal muscles and underlying pleura. Perforating branches pierce the chest wall to supply the pectoral muscles and the breast. In the lactating female, these vessels may be of considerable size. Corresponding veins run with the arteries.

The **intercostal nerves** are the ventral primary rami of the upper 11 thoracic spinal nerves. Their course and distribution are illustrated in Figure 10.6; trace them as you read the description. Each pair of spinal nerves emerges from the intervertebral foramina formed between two adjacent vertebrae (see Figure 10.1). On each side, the nerve immediately splits into two branches, the ventral and dorsal primary rami. The **dorsal primary rami** supply the muscles acting on the vertebral column and the skin immediately overlying the vertebrae posteriorly. The **ventral primary rami** pass anteriorly to form the intercostal nerves in the intercostal spaces running with the intercostal vessels. A lateral cutaneous branch emerging near the mid-axillary line supplies sensory innervation

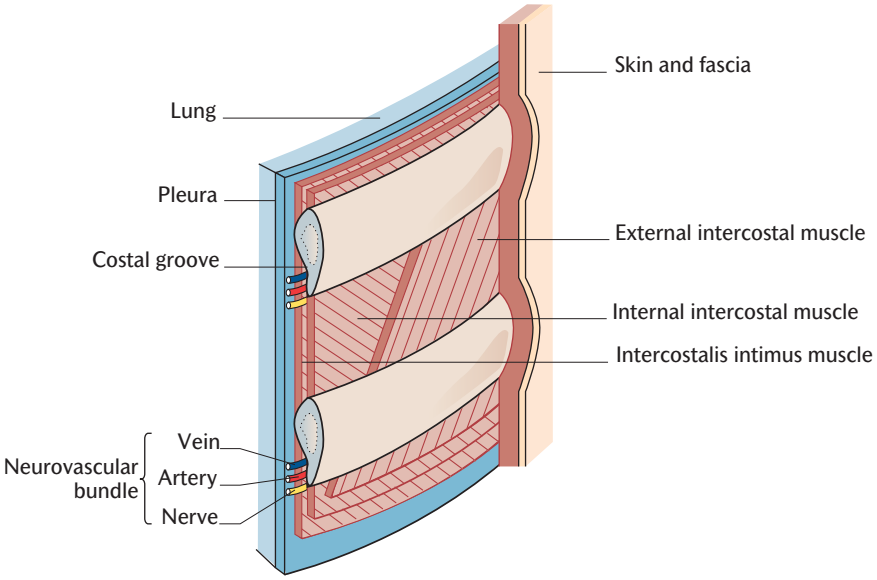

Fig. 10.5 The intercostal muscles.

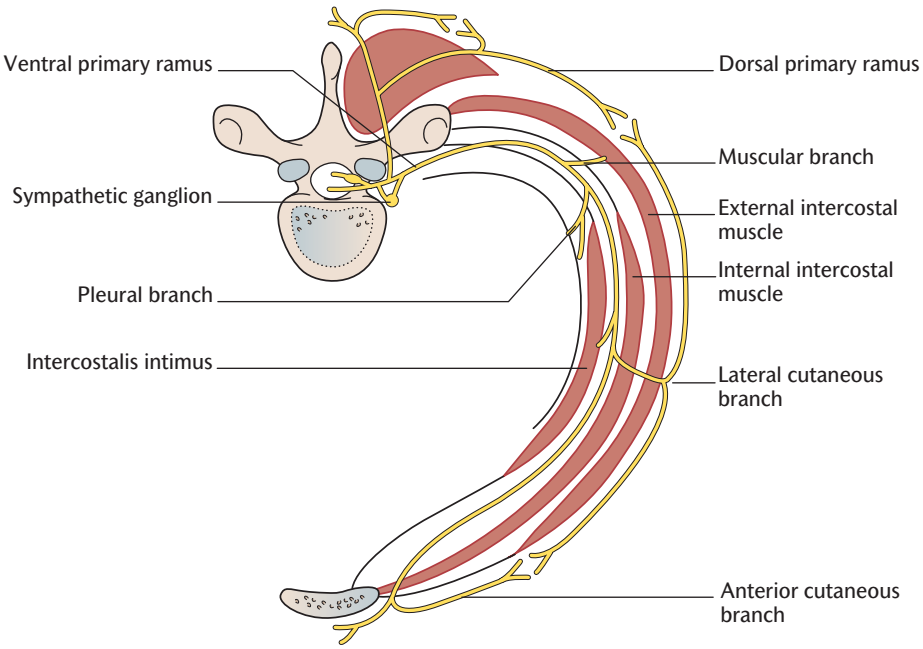

Fig. 10.6 An intercostal nerve and its branches.

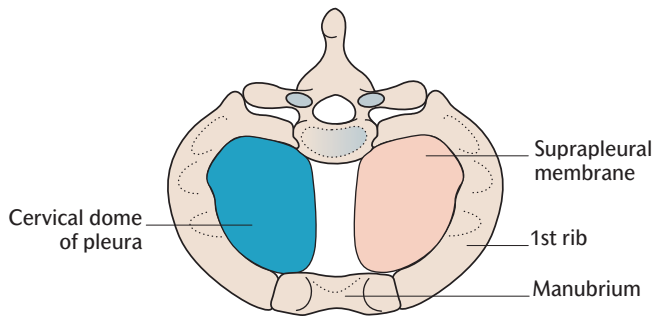

Fig. 10.7 The thoracic inlet.

to the skin over the side of the chest and an anterior cutaneous branch near the edge of the sternum supplies the skin on the front of the chest. Several motor branches supply the intercostal muscles. There are also pleural branches carrying sensory information from the parietal pleura.

As can be appreciated from Figure 10.4, the lower intercostal spaces are much shorter than those between the true ribs above. However, the intercostal nerves do not stop where the intercostal space is closed by the costal cartilages, but carry on across the abdominal wall to supply it. The lower six pairs of intercostal nerves carry motor axons to the abdominal muscles and sensory processes from the skin of the abdominal wall and peritoneum lining the abdominal cavity. Clinically-related aspects of the intercostal nerves are outlined in Box 10.4.

Box 10.4 Pleural fluid sampling and shingles

Blood, pus, or air may be aspirated from the pleural cavity via a needle inserted between the ribs. If the needle is kept close to the upper border of a rib, the main neurovascular bundle is safe; the needle will displace the smaller branches of the nerve and vessels rather than puncture them.

Infection of the dorsal root ganglia by the **Herpes zoster** (**chickenpox**) virus leads to a burning pain, followed by a rash over the skin supplied by the intercostal nerves arising from the infected ganglia, a condition known as **shingles.**

10.2.3 The thoracic inlet

The thoracic inlet is the junction of the neck and thorax. As shown in Figure 10.7, the boundaries of the inlet are the superior border of the manubrium, the deep borders of the first ribs, and the body of T1 posteriorly.

Laterally, the thoracic inlet is closed on each side by a dome of dense fascia, the **suprapleural membrane** covering the **cervical dome** of the pleura. As already pointed out in Section 9.3.2 and illustrated in Figure 9.4A, the lungs and their covering pleura rise above the level of the first rib and about 2½ cm above the medial third of the clavicle. A penetrating wound or the displaced fragments of a fractured clavicle could rupture the pleura, producing a pneumothorax (see Box 11.2) or even damage the lung itself. Structures that pass through the inlet between the thorax and neck lie towards the midline between the lungs and their coverings.

10.2.4 The diaphragm

The thoracic cavity is separated from the abdominal cavity by a musculotendinous sheet, the **diaphragm**. The aorta, inferior vena cava, oesophagus, and vagus nerves are transmitted through apertures in or behind the diaphragm.

As shown in Figure 10.8A, the diaphragm has a peripheral muscular part surrounding a **central tendon**. The striated muscle fibres are attached at one end to the central tendon and at the other end to the lower margins of the thoracic cage, the xiphoid process anteriorly, and the lower six costal cartilages laterally. Figure 10.8 also illustrates the posterior attachments of the diaphragm. It is attached posteriorly by muscular slips called crurae to the upper lumbar vertebrae and by

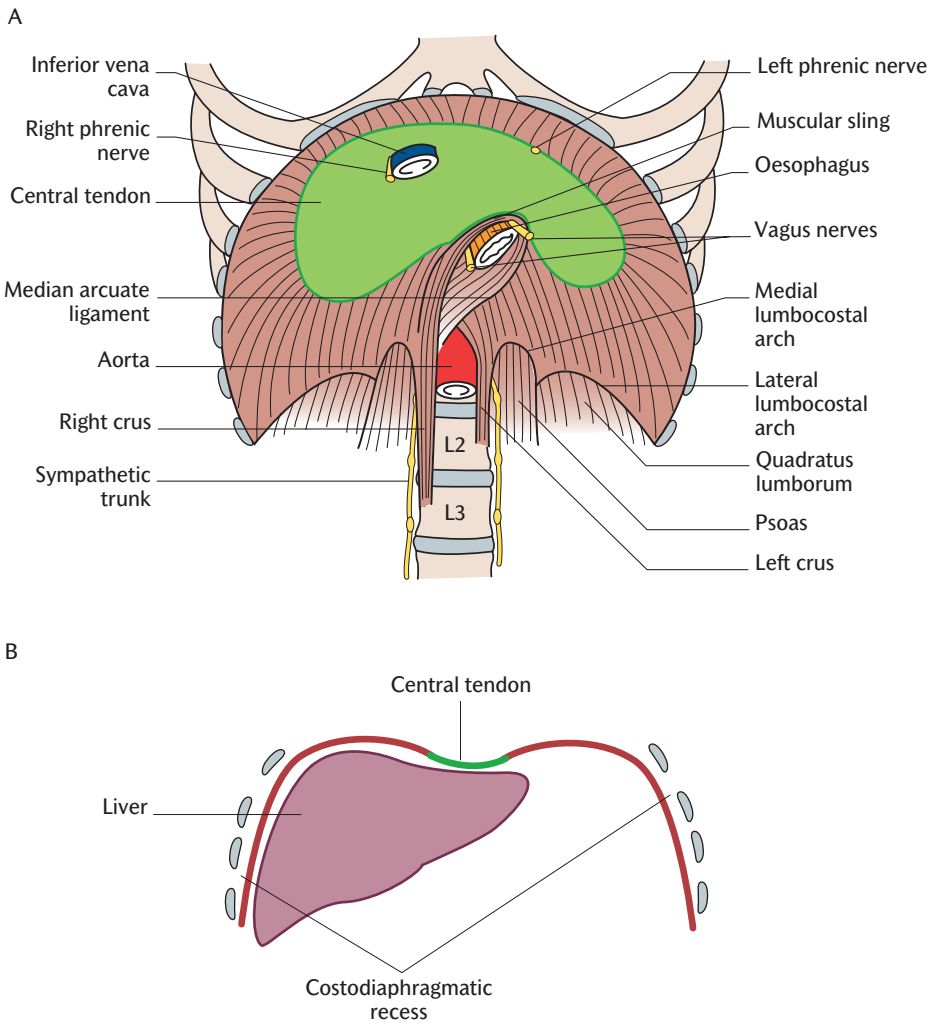

Fig. 10.8 A) An inferior view of the diaphragm. B) An anterior view of the curvature of the diaphragm.

arcuate ligaments which are attached to the fascia covering two muscles of the posterior abdominal wall, the psoas and quadratus lumborum muscles.

From Figure 10.8A, it should be obvious that the anterior attachments of the diaphragm are much higher than the posterior attachments. The diaphragm, therefore, has a curved shape if viewed from the side (see Figure 9.2). As shown in Figure 10.8B, when viewed from the front, the diaphragm is a double dome. The right side of the dome tends to be higher than the left because it overlies the liver, a solid organ. Both domes sit slightly higher than the relatively flat **central tendon** which lies at the level of the xiphisternum. As Figure 10.8B indicates, the lateral attachments are much lower than the attachments to the central tendon, producing a marked curvature of the muscular part of the diaphragm on each side. This produces an acute angle where the diaphragm meets ribs, forming a narrow gutter, the **costodiaphragmatic recess**.

Functions of the diaphragm

The diaphragm is an excellent example of the action of one muscle group affecting the action of others. The diaphragm does not operate alone but works in concert with the intercostal muscles to alter the dimensions of the thoracic cavity during ventilation. If we consider the movement of the diaphragm in isolation, contraction of the muscular components during inspiration will lower the central tendon towards the lower margin of the thoracic cage. Basically, the shape of the diaphragm will change from a dome to a flatter profile, thus expanding the vertical dimensions of the thoracic cavity. At the same time, as the diaphragm is contracting, the intercostal muscles are also contracting, moving the rib cage upwards and outwards. Rib movement elevates the position of the attachments of the diaphragm towards the central tendon, thus tending to flatten the diaphragm. These actions are described in the context of ventilation in Section 11.4.

The most obvious function of the diaphragm is as a muscle of ventilation, but it is also used to aid micturition, defecation, and parturition. If you take a deep breath and then close the vocal folds and breathe out, the air will be unable to pass through the larynx and pressure will build up in the thoracic cavity. This will prevent the diaphragm from rising, thus raising the intraabdominal pressure. Simultaneous contraction of the muscles of the abdominal wall will further increase the intraabdominal pressure, pushing on the bladder, rectum, or uterus to expel their contents as the specific action requires.

Diaphragmatic openings

The diaphragm has three major apertures through which vessels and nerves pass from thorax to abdomen or vice versa. They are shown in Figure 10.8A.

- The **aortic opening** transmitting the aorta actually lies behind the diaphragm between the median arcuate ligament and the twelfth thoracic vertebra in front. The descending thoracic aorta becomes the abdominal aorta as it passes through the diaphragm. The sympathetic trunks also pass behind the medial arcuate ligaments on each side of the aorta.
- The **oesophageal opening** is at the level of T10 and transmits the oesophagus and the vagus nerves. Figure 10.8A illustrates how a sling of diaphragmatic muscle surrounds the oesophageal opening; this forms a sphincter which plays an important part in preventing reflux of stomach contents into the oesophagus.
- The **caval opening** transmitting the inferior vena cava is at the level of T8. It passes through the central tendon to the right of the midline which maintains the patency of the opening; its diameter is, therefore, not reduced by diaphragmatic contraction so venous return to the heart is not impeded.

The consequences of weakness of the sphincter are explained in Box 10.5.

Box 10.5 Diaphragmatic hernia

In some patients, usually of middle age, the oesophageal opening may become weakened and widened, producing a **hiatus hernia.** In the commonest type, the short abdominal part of the oesophagus and upper part of the stomach rise through the hiatus when the patient lies down or bends over. Unpleasant reflux of acid stomach contents into the oesophagus may follow, causing heartburn. In the longer term, repeated exposure to acid can cause ulceration of the oesophageal mucosa. In persistent cases, the acid reflux can even affect the mucosa of the pharynx and larynx and cause erosion of dental enamel from the teeth.

Box 10.6 The mystery of the innervation of the diaphragm

Why is the diaphragm, positioned between the thorax and abdomen, innervated by nerves arising in the cervical spinal cord and not the thoracic or lumbar as would seem logical? The reason is due to the embryological origins of the diaphragm. Its embryology is complex, but the important points are that the central tendon develops from the septum transversum. This structure comes to lie at the level of cervical segments as the embryo develops a longitudinal curve (see Section 8.4 and Figure 8.7). The septum transversum is displaced inferiorly as the respiratory tract develops as an outpouching of the foregut that expands out to form the bronchial tree and lungs (see Section 13.3). The potential central tendon carries with it muscles derived from the cervical myotomes which will form the muscular parts of the diaphragm; these drag their nerve supply, the phrenic nerves, with them as they migrate downwards.

Nerve supply to the diaphragm

The phrenic nerves carry the motor nerve supply to the dome of the diaphragm. They arise from the ventral rami of cervical spinal nerves 3–5. ('*C3, 4, and 5 keep the diaphragm alive*' is a rhyme used by generations of students to recall this information.) Their course is described in more detail in Section 12.6.2. The phrenic nerves also carry the sensory supply from the parietal pleura covering the upper surfaces of the diaphragm, the peritoneum covering its lower aspect, the mediastinal pleura adjacent to the heart, and the pericardium surrounding the heart (see Section 12.6.2). The reason why the diaphragm receives its innervation from cervical spinal nerves is explained in Box 10.6.

11

The lower respiratory tract and its role in ventilation

Chapter contents

11.1 The pleural cavities

The two lungs occupy the right and left **pleural cavities** in the thoracic cavity. They are separated by the central **mediastinum** containing the heart and the great vessels, the trachea, and other structures passing through the thorax to the abdomen.

The left and right pleural cavities are entirely separate from each other. Figure 11.1 shows how they are formed as the developing lung buds invade part of the coelomic cavity by pushing a layer of the wall before it. This is analogous to pushing your finger into a partially inflated balloon. Your finger is the equivalent of the lung bud, the wall of the balloon it is pushing into is the inner layer of pleura, and the far wall of balloon is the outer layer of pleura. The pleura are thus divided into outer and inner layers.

As outlined in Figure 11.1D, the outer **parietal layer** lines the inner surface of the thoracic wall, the thoracic surface of the diaphragm, and the medial wall of the mediastinum. The inner **visceral layer** closely covers all surfaces of the lungs. The **pleural cuff** is formed where the two layers become continuous at the junctional region, surrounding the **root** (hilus) of the lung. A fold in the pleural cuff called the **pulmonary ligament** allows some space for movement of the lungs relative to the mediastinum during ventilation.

The **pleural cavity** separates the two layers of pleura, but is normally a *potential space* over most of the lung surface. The visceral and parietal pleurae are in virtual contact, separated by only a thin layer of **pleural fluid**. Pleural fluid has two important functions.

- It lubricates movement of the lungs and their attached visceral pleurae against the parietal pleurae.
- It also adheres the two pleural layers to each other, thus maintaining the inflation of the lungs.

The parietal pleura is often described as having **costal, diaphragmatic**, and **mediastinal** surfaces; this is useful when considering their nerve supply. The costal pleura is supplied segmentally by intercostal nerves, the mediastinal pleura by the phrenic nerves, and the diaphragmatic pleura centrally by the phrenic and peripherally by the lower five intercostal nerves. The visceral pleura is insensitive to pain and touch as it only receives an autonomic vasomotor supply. The effects of lung disease and infection on the pleura are described in Box 11.1.

The effects of failure of adhesion between the pleural layers is described in Box 11.2.

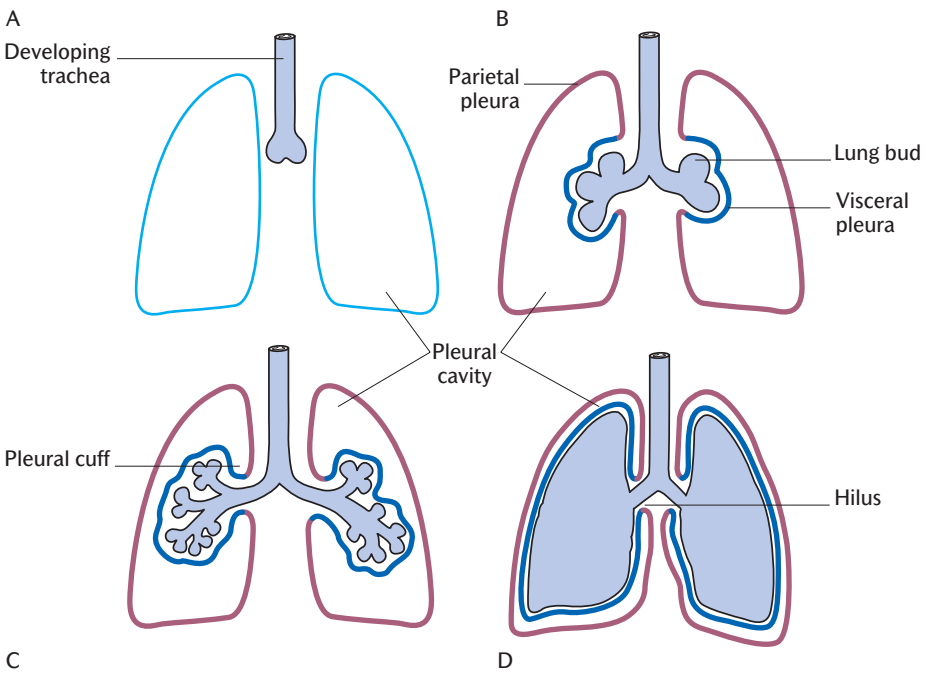

Fig. 11.1 Development of the lungs and formation of the pleural cavities.

Box 11.1 Lung infection and pleurisy

Disease states of the lung (e.g. pneumonia) are often painless until they reach the surface of the lung and the pleurae become involved. Local infection of the pleural cavity may lead to a breakdown of lubrication between pleural layers. Lack of lubrication can produce a painful pleural rub, audible through a stethoscope, and adhesions between the two pleural layers due to the formation of fibrous scar tissue. The pain is often referred to the cutaneous distribution of the segmental nerves supplying the pleural area affected. The pain experienced may be referred to the abdomen via the lower five intercostal nerves or to the shoulder via the phrenic nerve.

General infection of the pleura (**pleurisy**) is also painful. Interestingly, pleural pain may be abolished by applying local anaesthetic to the appropriate skin area.

Box 11.2 Pneumothorax

The pleural cavity may become infiltrated by air, excess pleural fluid, blood, or pus; the effect will be the same irrespective of the infiltrating agent. The pleural fluid will either be no longer in a thin enough layer to maintain adhesion or will be displaced by a non-adherent agent. The two pleural layers will pull apart and the lung will collapse.

1. A **pneumothorax** is produced when air enters the pleural cavity, usually via a diseased lung, much less commonly by the ends of broken ribs and even less frequently by stab wounds through the chest wall. Spontaneous pneumothorax may occur in healthy lungs after great exertion. The most dangerous type of pneumothorax is where the ruptured lung has a valvular effect, allowing air to pass from the lung to pleural cavity, but not vice versa. This is a tension pneumothorax and leads to a progressive build-up of pressure in the pleural cavity and hence collapse of the lung. The effects include breathlessness and pain because the mediastinal structures are displaced towards the opposite side.
2. **Pleural effusion** is excess serous pleural fluid usually stimulated by lung disease. Secondary infection of the excess fluid is common.
3. Pus (empyema) and/or blood (**haemothorax**) may also infiltrate the pleural cavities from lung infections.

Fluid or air is usually aspirated from the pleural cavity by means of a pleural tap. A wide bore needle is inserted laterally through a lower intercostal space so as to pass into the pleural cavity beneath the lung, but above the diaphragm. The needle is placed above the rib forming the lower margin of the relevant intercostal space so that it is well away from the intercostal neurovascular bundle running beneath the rib above.

11.2 The trachea and bronchi

The trachea is the first part of the lower respiratory tract. Figure 11.2 shows its course. The trachea starts at the lower border of the larynx denoted by the cricoid cartilage in the neck and runs down in the midline to enter the thorax deep to the sternal notch. After a relatively short course in the thorax, it bifurcates into the left and right main bronchi *at the level of the sternal angle*.

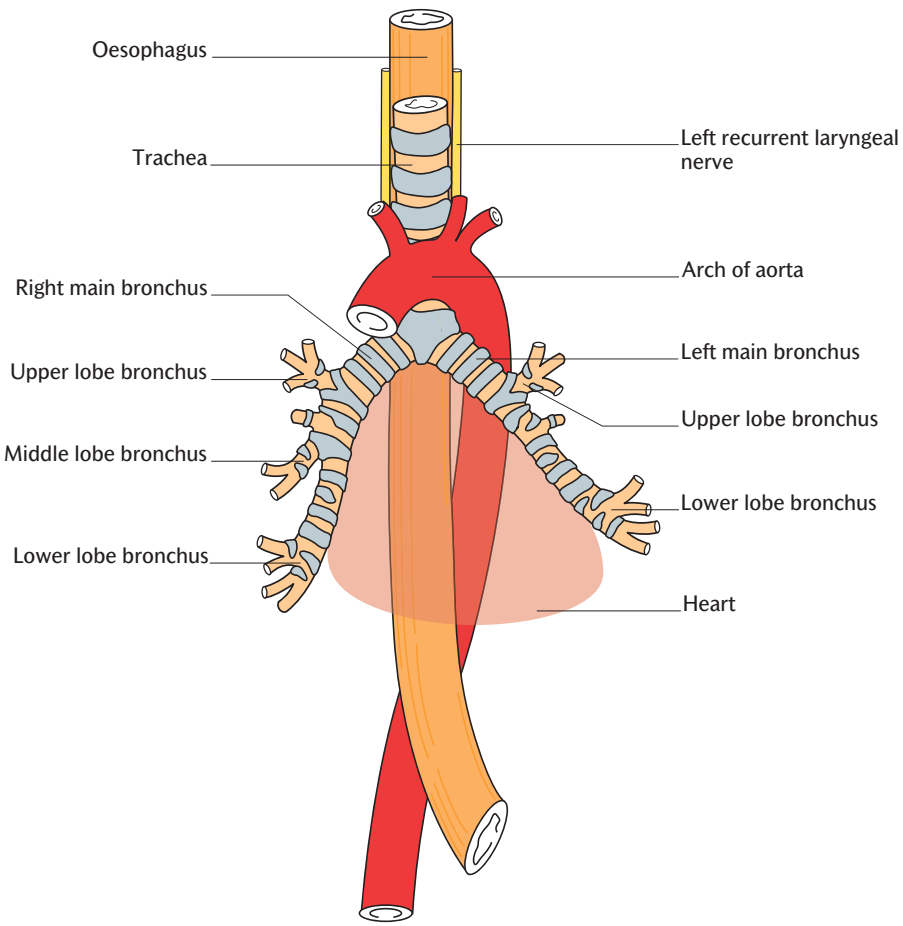

Fig. 11.2 The trachea and oesophagus and structures related to them.

The trachea is about 2.5 cm in diameter in adults. It is easier to remember that the internal diameter of the trachea is about the same diameter as the *patient's* little finger, especially if you need to create an emergency airway (see Box 28.9). The trachea is reinforced by C-shaped rings of cartilage; the open side of the C faces posteriorly to allow the oesophagus, which lies immediately behind the trachea, to dilate during swallowing. The wall between the rings is made up of fibrous tissue and smooth muscle. As expected for a component of the respiratory tract, the lumen is lined by respiratory epithelium (Figure 5.2B).

As you can see in Figure 11.2, the two main bronchi are not identical in size or position at the tracheal bifurcation. The right main bronchus is shorter, wider, and more vertical than the left bronchus. The left bronchus is supplying two lung lobes as opposed to three on the right and the left lung is displaced laterally by the heart, thus making more of an angle between the left bronchus and the trachea. The left main bronchus is about twice as long as the right. Box 11.3 describes how the anatomy of the main bronchi influences the likely destination of inhaled foreign bodies. Box 11. 4 outlines the added complications in unconscious patients.

The right and left main bronchi divide within the substance of the lung into lobar **bronchi** supplying each lobe. The structure of the walls of the bronchi is similar to that of the trachea although the cartilages become increasingly irregular below the bifurcation. The lobar bronchi divide again into segmental bronchi, each supplying a **bronchopulmonary segment**. Each lung is divided functionally into a number of these segments, each supplied by its own segmental bronchus and accompanying blood vessels. Each segment thus forms an individual functional unit; if disease is restricted to one or two adjacent segments, they may be removed by a thoracic surgeon with minimal loss of blood or leakage of air from adjacent lung tissue.

Box 11.3 Foreign bodies in the lower respiratory tract

Foreign bodies entering the larynx and trachea will stimulate a powerful cough reflex. The ridge formed by the bifurcation of the trachea is the last area to contain sensory nerve endings capable of eliciting the cough reflex and may thus be considered a last line of defence. In reality, small objects that pass the larynx are likely to continue down the trachea to its bifurcation. When foreign bodies do pass the bifurcation, they usually enter the right main bronchus because it is wider and in a more direct line with the trachea. If the patient is upright, the foreign object will usually enter the lower lobe bronchus. If they are lying down, as is common in many dental procedures, the object may enter the right middle lobe bronchus. An inhaled foreign body may block one of the bronchi, leading to the collapse of part of the lung or may cause a lung abscess if the object is infected.

Box 11.4 Reflexes in unconscious patients

The cough reflex is in abeyance in unconscious subjects. Blood or other foreign objects, e.g. tooth fragments, may enter the lungs in the victims of road accidents and other violence without stimulating the cough reflex. The vomiting reflex is virtually the last reflex to be suppressed even in deeply unconscious subjects; they may, therefore, vomit and then inhale it without eliciting a cough reflex with the potential to asphyxiate on their own vomit. To avoid this potentially fatal event, it is vital, therefore, to place unconscious subjects in the **recovery position** if it is safe to do so and their injuries allow. In the recovery position, the patient is laid prone with their head turned to one side propped by one of their arms; the knee on the side to which their head is turned is bent to ensure they do not roll over. The recovery position ensures that if the patient does vomit, the vomit will leave their mouth and not enter the lower respiratory tract where the consequences can be, and often are, fatal. When a patient is given a general anaesthetic for dental extraction, procedures must be adopted to ensure that tooth fragments and other foreign bodies are not inhaled; the throat is packed or an endotracheal tube is passed.

11.3 The lungs

Both lungs are covered by visceral pleurae which adhere tightly to their surfaces. As we have already seen in Figure 9.4, each lung is conical with a blunt apex projecting about 2.5 cm above the medial third of the clavicle, a concave base over the diaphragm, an extensive convex costal surface, and a concave mediastinal surface. In fixed post-mortem lungs that you may see in a dissecting room, evidence of the structures adjacent to each lung can be seen as grooves moulded around the ribs and aorta, for example. These markings are, of course, not present in the living where the lungs have a texture rather like sponge rubber.

The left lung is slightly smaller than the right because the heart decreases the size of the left pleural cavity. The left lung has a recess, the **cardiac notch**, in its medial side which accommodates the heart. As already described in Section 9.3.2, both lungs are divided by an **oblique fissure** into upper and lower lobes (see Figure 9.4). The upper part of the right lung is subdivided by a **horizontal fissure** into upper and middle lobes.

11.3.1 The roots of the lungs

The lung root (also known as the hilus) is the communication between each lung and the rest of the body. The roots of the right and left lungs are shown schematically in Figure 11.3. The main **bronchus** enters each lung root with the corresponding **pulmonary artery** carrying deoxygenated blood from the heart. Two **pulmonary veins** leave the root, carrying oxygenated blood back to the heart. A small **bronchial artery** accompanies or is embedded in the wall of each bronchus. The pattern of vessels and bronchi entering and leaving the hilus is very variable, depending on how close to the lung surface the incision is made to remove the lung to view its root. In addition, it is very difficult to distinguish pulmonary arteries and

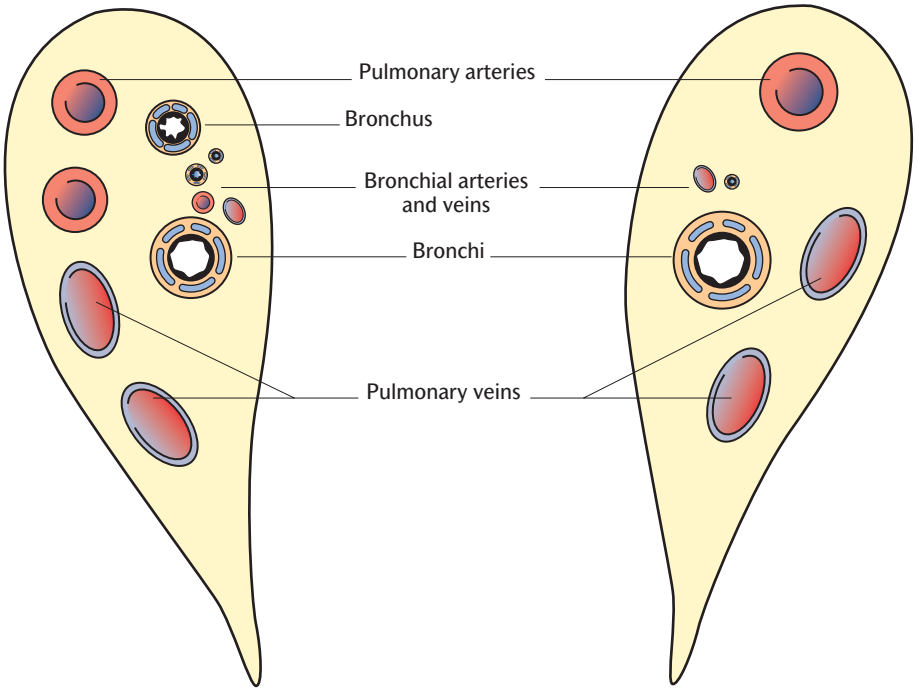

Fig. 11.3 The roots of the lungs.

veins from each other on anatomical specimens because their walls are of similar thickness. The bronchi can be distinguishing without difficulty as they are reinforced with cartilage which can be felt quite easily.

11.3.2 Pulmonary vessels and nerves

Blood supply

It is important to realize that there are two blood supplies to each lung. The most obvious vessels are the **pulmonary arteries** which are branches of the pulmonary trunk carrying deoxygenated blood from the right ventricle of the heart and **pulmonary veins** returning oxygenated blood into the left atrium. The alveoli receive oxygen and nutrients as gaseous exchange between blood and air occurs through their walls. The bronchi and bronchioles, connective tissue, and visceral pleura receive oxygenated blood from the much smaller **bronchial arteries** which arise from the descending thoracic aorta. Anastomoses between these systems become important when either is occluded by disease. The effects of blockage of pulmonary arteries are described in Box 11.5.

Lymphatic drainage

Lymphatic drainage follows the bronchi and pulmonary vessels to the lung root. There are many lymph nodes in the tracheal bifurcation; if you have the opportunity to dissect the thorax, you may be able to identify these nodes because they are usually stained black by atmospheric pollution even in non-smokers and country dwellers. The spread of lung cancer through the lymphatic drainage is described in Box 11.6.

Box 11.5 Pulmonary embolism

Pulmonary (thrombo)embolism is due to the transport of a blood clot to the lung through the right ventricle and pulmonary arteries; the clot usually originates from a distant site such as deep leg veins (see Box 4.2). A large clot can be fatal and a small one may cause damage to a bronchopulmonary segment by blocking its artery.

Box 11.6 Lung cancer

Carcinoma of the bronchus is one of the most common forms of cancer in developed countries. The predisposing factor is tobacco smoking and now lung cancer is increasing in incidence in developing countries as tobacco products become more available. Lung cancer is not easy to detect in the early stages; by the time the cancer is diagnosed, the prognosis is generally poor because it has often metastasized extensively. The lymphatic drainage of the lungs facilitates its spread to the pleura and to lymph nodes at the hilus of the lung and in the mediastinum. Metastases within these lymph nodes soon spread to more distant organs, especially the vertebral column and brain.

Nerve supply

The lungs receive an autonomic nerve supply through the **pulmonary plexus**, comprising branches from the sympathetic trunk and parasympathetic branches from the vagus nerves. Sympathetic activity produces bronchodilatation and parasympathetic bronchoconstriction. Sensory nerves from stretch receptors in the bronchi and alveolar walls are important in the reflex regulation of the degree of lung inflation. Over 50% of these receptors are in the bronchial tree within the lungs and the rest are located in the trachea and main bronchi outside the lungs. Despite their name of stretch receptors, they respond to pressure differences across the walls rather than physical stretching of the walls of the lower respiratory tract. As the pressure changes during ventilation, stretch receptors are stimulated and inhibit contraction of the muscles of inspiration in the respiratory centres in the medulla, allowing expiration through passive elastic recoil (see Section 11.4.1; Box 11.7).

Box 11.7 Asthma

Bronchial asthma is due to spasm of smooth muscle in the bronchial walls, which results in bronchoconstriction accompanied by thick mucous secretions. Asthma is a restrictive lung disease; patients have more difficulty emptying the lungs than filling them. It causes wheezing and difficulty breathing and victims often 'fight for breath'. It is a major problem in developed countries and has multiple causes, including allergies to inhaled or ingested substances.

11.4 Mechanisms of ventilation

Respiration is the term commonly used to cover breathing, but it should really be more accurately called **ventilation**. Respiration strictly relates to the exchange of oxygen and carbon dioxide between air and blood through the alveolar walls and blood and tissues through capillary walls throughout the body. Many of the fundamentals of ventilation are known but some of the detailed movement is still not completely understood.

Thoracic volume must be increased, leading to a drop in pressure within the lungs for air to move into the lungs from outside the body. The air is drawn in by the reduced pressure in the lungs, aided by the greater atmospheric pressure outside the body. The increase in volume during inspiration has two main components: an increase in vertical height of the thoracic cavity produced by movement of the **diaphragm** and an increase in diameter of the anteroposterior and lateral axes by movement of the ribs and sternum produced by the **intercostal muscles**. In babies and young children, respiration is entirely diaphragmatic as the ribs lie almost horizontally and have not yet acquired their familiar downcurved shape. Adult men are said to move their ribs less than women and therefore rely more on diaphragmatic movements.

Ventilation rates and depth are matched to the demands of the body, another example of homeostasis. **Quiet ventilation** takes place when we are relatively inactive such as sitting down reading. **Forced ventilation** occurs in response to exercise. Ventilation tends to be rhythmic; the inspiratory and expiratory phases of each breath take about the same length of time, irrespective of whether we are breathing slowly during quiet ventilation or much more rapidly during forced ventilation.

Ventilation is under the control of the somatic nervous system, not under the aegis of the autonomic nervous system, so is a voluntary activity. However, ventilation is too important to be left to chance so is controlled automatically in most circumstances. The main drivers of ventilation are the levels of oxygen and carbon dioxide in the blood and cerebrospinal fluid. These levels are monitored through **chemoreceptors** in the arterial circulation that send signals to respiratory and cardiovascular centres in the medulla of the brain. The respiratory centres will increase ventilation if there is not enough oxygen in the blood; likewise, ventilation will increase if too much carbon dioxide accumulates.

The automatic control of ventilation can be overridden. Holding your breath is the simplest example, but if you forget to breathe out, your body will override your actions and make you breathe again when carbon dioxide reaches dangerous levels. Breathing during speech is another example of overriding automatic rhythmical ventilation.

11.4.1 Quiet ventilation

Quiet inspiration

The muscular fibres of the diaphragm contract in **quiet inspiration**. Their action is to pull the central tendon downwards since they are firmly anchored peripherally; this movement increases the vertical dimension and decreases pressure within the thoracic cavity. The abdominal viscera are pushed down as the diaphragm contracts. At the same time, the first rib is fixed by the scalene muscles in the neck and the upper intercostal muscles are active. Comparatively little movement of the upper ribs occurs, but the sternum is displaced anteriorly, increasing the anteroposterior diameter of the thorax. The sternum is moved forwards and upwards during inspiration because of the obliquity of the ribs attached to it. The lower ribs pivot at their posterior and anterior attachments and swing out, increasing the lateral dimensions of the thorax (See Box 11.8 and Figure 11.4). The middle ribs are able to move laterally because of the flexibility of the long inclined costal cartilages which connect them to the sternum. The costal angle between the costal cartilages of the lower ribs on each side increases appreciably during deep inspiration (see Figure 9.1). In quiet ventilation, about 75% of the increase in thoracic volume is produced by movement of the diaphragm and only 25% by rib movement.

The movements of the chest wall during ventilation are followed by the lungs which, therefore, expand drawing air in or contract to force air out. The pleural layers play an essential part in this process. The parietal pleura will follow the movements of the thoracic wall as they are directly attached to it. The visceral pleura will follow the movement of the parietal pleura because of the adhesion of the two layers brought about by the thin layer of pleural fluid between them. Pleural fluid has the same effect on the pleura as a thin film of fluid between a glass and a wet table; the glass may be slid easily along the table, but can only

be raised away from the table by using considerable force. The pleural fluid enables the pleural layers to slide over each other wherever they are in contact and also to resist the opposing forces generated by the movement of the chest wall and the elasticity of the lungs. The lungs will inflate because the visceral pleura are directly attached to the lungs.

Quiet expiration

Quiet expiration is brought about by **passive elastic recoil** of the lungs and chest wall. The term 'passive' indicates that no muscular effort is required. When sufficient air has been drawn into the lungs to meet the functional demands, the intercostal muscles and diaphragm relax. There is some elastic tissue in the lungs that will recoil back to its original dimensions. There is also a certain amount of torsion set up in the joints between the ribs and vertebrae and in the costal cartilages as they move during inspiration. As the muscles relax, these tissues will also recoil to their starting point. They do not recoil like an elastic band; the recoil movement is slow as there is resistance within the lung tissue and the air being expired meets resistance as it leaves the lungs through the comparatively narrow tubes of the lower and upper respiratory tracts.

Box 11.8 Rib movements

The movements of the joints between the ribs and vertebral column determine the different movements of segments of the rib cage during ventilation. Figure 11.4 illustrates the differences between the joints at different levels in the thorax. Firstly, you must appreciate that movement takes place *simultaneously* at the joints between the heads of the ribs and vertebrae and at the costotransverse joints between the tubercle of the rib and transverse processes of the vertebrae. The degree of movement at both joints is small because of the strong ligaments uniting the ribs and vertebrae. Although the articulations between ribs and vertebral bodies all appear similar on cursory examination, Figure 11.4 shows that the joints between the tubercles and transverse processes form a series, each differing a little from those above and below. In the upper six costotransverse articulations, the articular surface on the rib is convex and that on the vertebra is concave. The intercostal muscles acting on the upper margin of the shaft of one of these ribs raise it, the rib rotating about a long axis running through its neck from the joint at the head to the costotransverse joint. In the seventh to tenth joints, the articulating surfaces are flat and angled so that the surfaces on the rib tubercles face downwards and medially as well as backwards. Hence, no rotation about the axis of the rib neck is possible. The joint surfaces can only slide over each other a little, a movement which displaces the rib principally outwards and only slightly upwards.

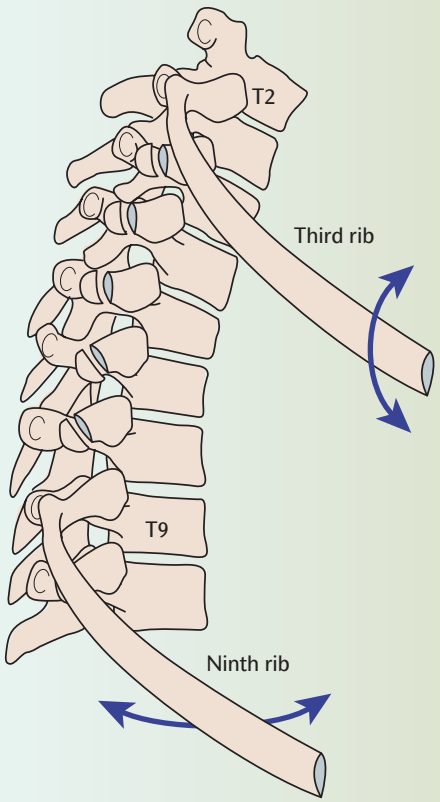

Fig. 11.4 Movements of the ribs during ventilation.

11.4.2 Forced ventilation

In deep forced ventilation, the capacity of the thorax during the inspiratory phase is increased by a much larger amount by greater movements of the ribs and diaphragm. They may have to move enough to allow inhalation of the maximum amount of air possible for that individual—the **vital capacity**. Accessory muscles may be recruited to assist the respiratory muscles. An accessory muscle is defined as one that normally carries out a particular action, but can be recruited to aid another action. For example, the pectoralis muscles running between the upper ribs and upper arm normally move the arm; if the arm is fixed, the pectoralis muscles can then act to pull on the thoracic cage and thus aid inspiration.

Forced inspiration

In **forced inspiration**, the first rib is raised a little by the contraction of scalene muscles that run from the cervical vertebrae to the first rib; their attachments to the first rib are shown on Figure 10.3. Contraction of both layers of the intercostal muscles moves the second rib in relation to the first, the third in relation to the second, and so on, the effect being maximal at about the seventh or eighth rib. Continued contraction of the diaphragm after the point where it can descend no further causes the lower ribs to move upwards, but principally outwards because of the constraints imposed by the shape of the costovertebral joints. The erector spinae muscles may contract to straighten the thoracic spine.

The sternum is pulled up by sternocleidomastoid muscle running between the mastoid process on the base of the skull and the sternum and clavicle (see Figure 20.8). When ventilation at or near vital capacity is required to restore homeostasis, the pectoral muscles running from the humerus, the bone of the upper arms, to the thorax contract to help elevate the ribs even more; the subject will usually fix their arms by placing their hands on their hips to prevent arm movement or grasping a fixed object.

Forced expiration

In **forced expiration**, the normal elastic recoil of ribs and anterior abdominal wall is reinforced considerably by contraction of the muscles of the abdominal wall acting as accessory muscles of respiration. The abdominal muscles are attached to the lower parts of the thoracic cage above and the pelvic bones below. Their effect is twofold. Firstly, they pull down the lower ribs, thus decreasing the lateral and anteroposterior dimensions of the thorax. Secondly, they compress the abdominal contents, forcing them under the diaphragm which is displaced upwards, thus decreasing the vertical dimensions of the thorax. In general, forced ventilation is still rhythmical, but each cycle is deeper and faster than in quiet ventilation.

During inspiration, the bronchial tree expands so that the lung roots move downwards, outwards, and forwards; during deep inspiration, the tracheal bifurcation may descend by about 5 cm. In quiet ventilation, the central tendon of the diaphragm moves up and down by only about 1 cm whereas in deep forced ventilation, it may move through a considerably greater range. The average volume of air exchanged in quiet respiration is 500–600 ml and the normal ventilation rate is about 12 to 15 per minute for a healthy adult; by contrast, the normal respiratory rate of a newborn baby is about 40 per minute. During exercise or stress, the respiratory rate will increase and inspiratory volume may approach vital capacity which is about 4–5 litres, depending on age, sex, body build, and overall fitness of the individual.

Ventilation must be forced externally during artificial respiration, a skill described in Box 11.9 which all members of the dental team must practice regularly.

11.4.3 Speech ventilation

During speech ventilation, the normal rhythmical pattern of breathing is heavily modified. Air is drawn in at a suitable pause in speech so inspiration is usually more rapid and deeper than during quiet ventilation; sufficient air is taken in to supply enough air to match the length of the utterance that is about to be delivered. For quiet conversational speech, the tidal volume may increase to about 1–1.5 litres. When one of your lecturers is trying to project their voice across a lecture theatre, they may be inhaling 2–2.5 litres with each breath so they are working five times harder than you just to breathe. This is achieved by using the diaphragm and external intercostal muscles more than in quiet ventilation; the internal intercostals are also recruited for deeper breaths.

Speech is produced on the exhaled (egressive) air stream. This must be strictly controlled to maintain the air flow for the length of time required to produce the desired utterance. Rather than simply relaxing the intercostal muscles and diaphragm as in quiet ventilation, they are relaxed in graded fashion so that air is squeezed out of the lungs progressively instead of in one go. When an utterance is long, it is often necessary to prolong the exhalation by using the abdominal muscles to push more air out of the lungs.

Box 11.9 Artificial respiration

Artificial respiration can be carried out in many forms. **Mouth-to-mouth resuscitation** is a technique which all dentists must know and refresh *annually* according to UK guidelines for clinical practice. The patient is laid supine, with their neck fully extended so that the head is as far back as it can go. This ensures that the airway is patent. If the patient wears dentures, you should remove them. You may need to move the tongue forwards by moving the mandible forward to clear the airway if necessary. You hold the patient's jaw open with one hand and pinch their nostrils shut with the other. After taking a deep breath, you blow into the patient's mouth, observing the patient's chest as you do so. It should rise when you blow into the patient's mouth; if it fails to do so and resistance is met as you blow, the airway is blocked and must be cleared before proceeding. Stop blowing and take another deep breath while passive expiration occurs. The cycle should be repeated for as long as necessary. Mouth-to-mouth resuscitation is usually carried out in conjunction with external cardiac massage as part of **cardiopulmonary resuscitation (CPR)**. Guidelines on the ratio of breaths to cardiac compression differ from country to country and change with clinical experience so they are not given here.

The lungs will not work if filled with water. If the patient has inhaled a large amount of water, it must be removed first. The patient is laid prone with their head to one side and their chest is compressed by placing the weight of your upper body on your hands positioned over their lower ribs. This will force water out of the lungs. When water has been removed, mouth-to-mouth resuscitation may begin.

12

The heart, pericardium, and mediastinum

Chapter contents

12.1 Introduction

The heart, the arteries and veins leaving and entering the heart which are usually referred to as the great vessels, the trachea and bronchi, the oesophagus, and the vagus and phrenic nerves and sympathetic chains occupy the **mediastinum**, the area in the middle of the thoracic cavity between the two pleural sacs. The anteroposterior dimension of the thorax is narrowest in the mediastinum because of the presence of the thoracic vertebrae posteriorly. Laterally, the pleural sacs enclosing the lungs extend much further back alongside the vertebrae in the areas known as the paravertebral gutters.

The great vessels enter and leave the superior aspect of the heart. The large veins draining the head, neck, and arms lie most superficially; they unite to form the superior vena cava that enters the right atrium of the heart. These veins overlie the two large arteries exiting the heart, the aorta, and pulmonary trunk. The aorta has a short ascending part, then forms the aortic arch passing backwards and to the left before continuing down the posterior wall of the thorax as the descending thoracic aorta. The subclavian and common carotid arteries, supplying blood to the arms and head and neck, respectively, arise from the aortic arch. The oesophagus is the deepest structure lying on the vertebrae and the trachea and main bronchi lie superficial to it. The sympathetic chains lie lateral to the vertebral bodies and the vagus and phrenic nerves are in intermediate positions. All these structures will be described in more detail in the rest of this chapter.

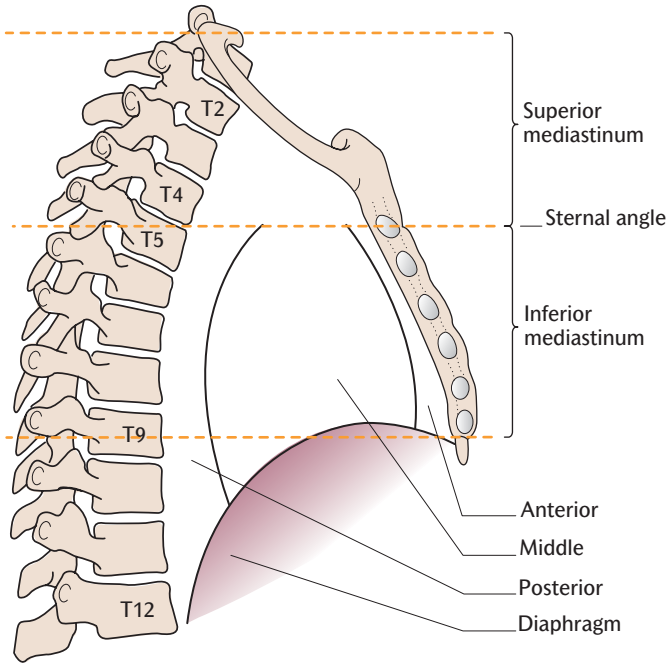

Fig. 12.1 Divisions of the mediastinum.

12.2 The mediastinum

The mediastinum is divided, for descriptive convenience, into the superior and inferior mediastinum. Figure 12.1 shows the imaginary line of division joining the sternal angle and the intervertebral disc below T4 that demarcates the boundaries of the superior and inferior of the mediastinum. The superior mediastinum occupies the space between the thoracic inlet above and the imaginary horizontal plane. The inferior mediastinum lies below that line and extends as far as the diaphragm. The lateral borders of both subdivisions of the mediastinum are the parietal pleura covering the medial aspect of the lungs, the mediastinal pleura.

The inferior mediastinum is further divided into three parts as shown in Figure 12.1:

- The anterior part between the sternum and fibrous pericardium;
- The large middle mediastinum containing the heart enclosed in the pericardium and the roots of the great vessels;
- The posterior part between to the fibrous pericardium and thoracic vertebrae.

12.2.1 Contents of mediastinal divisions

Figure 12.2 shows the important contents of the **superior mediastinum**. From anterior to posterior they are: the superior vena cava and brachiocephalic veins, phrenic nerves, branches of the aortic arch,

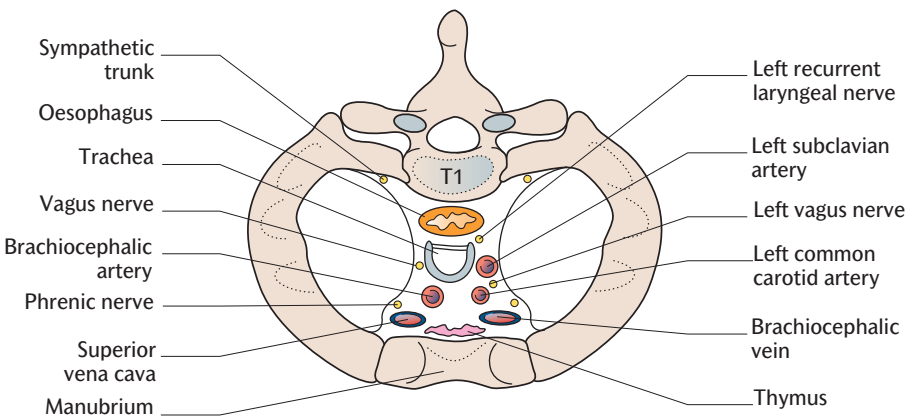

Fig. 12.2 Structures in the superior mediastinum at the level of the first rib.

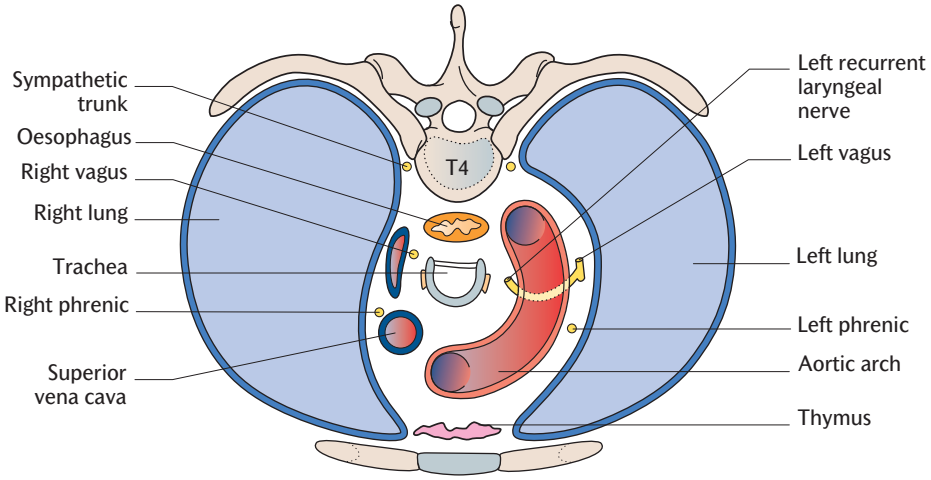

Fig. 12.3 Structures at the junction of the superior and inferior mediastinum at the level of T4.

vagus and left recurrent laryngeal nerve, trachea, oesophagus, and sympathetic trunks.

The important contents of the inferior mediastinum are shown in Figure 12.3. The anterior mediastinum contains the thymus gland. The **middle mediastinum** contains the pericardium and heart inferiorly and the great vessels entering and leaving the heart superiorly. The vagus and phrenic nerves lie in an intermediate position. The trachea dividing into the right and left bronchi lies more posteriorly. The major structures in the **posterior mediastinum** are the oesophagus, descending thoracic aorta, and sympathetic chains. Note that the aorta, trachea, oesophagus, and vagus nerves all pass through more than one mediastinal division.

Box 12.1 Pericarditis

Inflammation of the pericardial sac (**pericarditis**) produces severe substernal pain. Pericarditis may follow bacterial or viral infection or may also be a sequel to rheumatic fever. **Rheumatic fever** usually follows about three weeks after throat infections by streptococcal bacteria and produces rheumatism-like symptoms of joint ache and pain, hence its name. Although rheumatic fever is now uncommon in developed countries, it is still prevalent in developing world where 5- to 15-year-olds are most frequently affected.

Pericarditis is frequently accompanied by the production of excess serosal fluid (pericardial effusion) which may be copious enough to compress the pulmonary veins and interfere with the action of the heart. Acute pericarditis often produces a pericardial rub similar to the pleural rub described in Box 11.1. A wound penetrating the pericardium commonly injures the heart wall, causing bleeding into the pericardium, again compressing the heart and interfering with its efficient function.

Fluid may be drained from the pericardial sac via a wide bore needle inserted through the fifth or sixth intercostal space near the sternum.

12.3 The pericardium

The heart is enclosed in a tough fibrous sac, the **fibrous pericardium.** The fibrous pericardium is lined by the **serosal pericardium** that has two layers. The parietal layer lines the fibrous pericardium and the visceral layer (or epicardium) covers the heart and roots of the great vessels. The serosal pericardium is thus arranged similarly to the two pleural layers and function in a similar fashion. There is a potential space between the two layers of the serosal pericardium, containing a thin layer of lubricant serosal fluid that allows the heart to move within the fibrous pericardium as it contracts and relaxes.

The pericardium conforms to the external surface of the heart, although allowing free movement of the muscular walls of the ventricles and atria. The fibrous pericardium blends with the connective tissue outer layers of the walls of the great vessels, not very far from their roots, and is firmly attached to the central tendon of the diaphragm and the anterior chest wall by fibrous ligaments which help to anchor the heart. The heart is also indirectly anchored by its vessels, especially by the pulmonary veins entering the lungs. Box 12.1 outlines the effects of disease on the pericardium.

12.4 The heart

The heart consists of four chambers, the right and left atria and the right and left ventricles. The chambers are arranged as two chambers in series on each side, the atria above the ventricles, as shown in Figure 4.1. Essentially, the heart consists of two parallel pumps, the right atrium and ventricle receiving deoxygenated blood from the systemic circulation and pumping the deoxygenated blood via the pulmonary trunk to the lungs where gaseous exchange takes place and blood is oxygenated. Oxygenated blood returns to the left atrium of the heart, passing to the left ventricle where it is pumped via the aorta through the systemic circulation to supply the tissues with oxygen and other nutrients. Valves guard the orifices between the atria and ventricles and the ventricles and the pulmonary trunk and aorta to prevent backflow of blood as pressure changes occur in the chambers and vessels.

The heart fills and empties during each cardiac cycle. The normal heart rate is about 70 beats per minute in a resting adult, up to 130 times per minute in a newborn child; the heart rate is even faster in a fetus when contractions start as early as the third week of development (see Section 13.2).

Blood returns continuously to both sides of the heart from the circulatory system when the ventricles relax (**diastole**). The atrioventricular valves open as the atria contract and blood flows into the ventricles. When the ventricles are nearly full, the next cycle of cardiac contraction (**systole**) begins. The ventricles contract in a wave which passes upwards from the apex of the heart towards the aortic and pulmonary openings. The resultant increase in pressure closes the atrioventricular valves and prevents regurgitation of blood into the atria. Because these valves are closed, returning blood accumulates in the large veins and atria. Once the intraventricular pressure exceeds that in the large arteries, the semilunar valves at the exits into the aorta and pulmonary trunk are forced open and blood is ejected from the heart. As soon as the contraction ceases, the intraventricular pressure drops below that of the great vessels and the semilunar valves close. Further relaxation lowers ventricular pressure to below that of the atria, now refilled from the general and pulmonary circulation and the atrioventricular valves open as the cycle begins again.

12.4.1 The general arrangement of the heart chambers and great vessels

The four chambers of the heart are conventionally referred to as the right and left atria and ventricles. However, this nomenclature does not reflect their true position in the heart as it lies in the chest. As shown in Figure 12.4 and 12.5, the right side of the heart is, in fact, *anterior* to the left side. The right side of the heart is formed by the **right atrium**, with the **superior and inferior venae cavae** returning deoxygenated blood to the right atrium from the head, neck and arms, and the lower body, respectively. As can be seen in Figures 12.4 and 12.5, they enter the right atrium from the aspects that their names suggest. Figure 9.3 shows the position of the **right atrioventricular (tricuspid) valve** separating the right atrium from the **right ventricle**. It is almost vertically behind the lower end of the sternum such that the right ventricle lies to the left of the right atrium. The right ventricle actually lies *centrally* when the heart is viewed from the front. Blood leaves the right ventricle superiorly through the **pulmonary valve** and flows into the pulmonary circulation through the **pulmonary trunk** which splits into right and left pulmonary arteries after a short course; they supply the corresponding lungs.

After gaseous exchange in the lungs, oxygenated blood returns from the lungs via the four **pulmonary veins** to the **left atrium** situated on the posterior side of the heart. These vessels can be seen in Figure 12.5. Internally, the left and right atria are separated by the atrial septum. Blood passes from the left atrium through the **left atrioventricular (mitral) valve** to the **left ventricle** which lies posterior to the right ventricle. The two ventricles are separated internally from each other by the **interventricular septum**. Blood leaves the left ventricle via the **aortic valve** to enter the systemic circulation via the aorta which runs at first upwards and then curves backwards over the bifurcation of the pulmonary trunk. Note the relative positions of the two major arteries in Figure 12.4—the aorta lies to the *right* of the pulmonary trunk. The cusps of the valves between the atria and ventricles and the ventricles

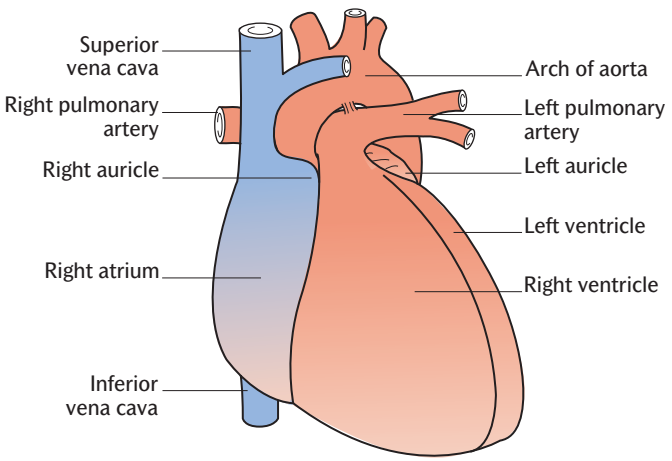

Fig. 12.4 Anterior view of the heart.

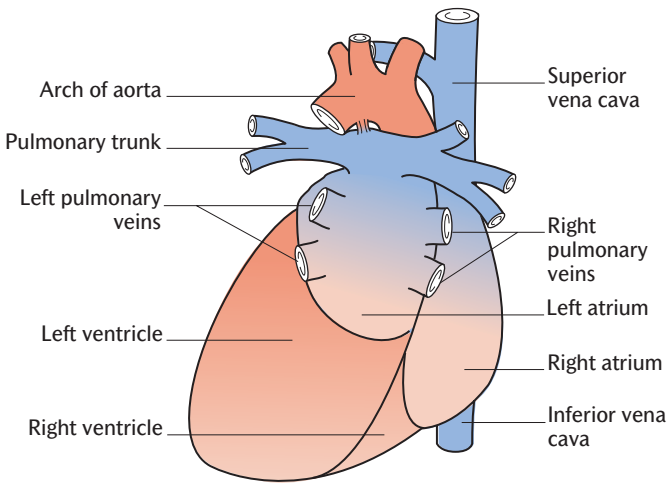

Fig. 12.5 Posterior view of the heart.

and pulmonary trunk and aorta are arranged so that the flow of blood can only take place in the desired direction.

The heart is usually described as having three surfaces and three borders. Study Figure 12.4 and 12.5 to determine the chambers that contribute to each surface. The **sternocostal surface** (Figure 12.4) is formed by the right atrium plus the right ventricle plus the apex of the left ventricle, the **diaphragmatic surface** (Figure 12.5) by the right and left ventricles plus the part of the right atrium admitting the inferior vena cava, and the **posterior surface** or **base** (Figure 12.5) comprises the left atrium plus a small contribution from the right atrium.

The chambers contributing to the borders of the heart can also be appreciated from Figure 12.4. The **right border** is formed entirely by the right atrium, the **left border** entirely by the left ventricle, and the **inferior border** is formed from right to left by the right atrium, right ventricle, and apex of the left ventricle. Grooves on the external surface of the heart indicate the relative positions of the four heart chambers. The **atrioventricular groove** separates the atria from the ventricles and the **interventricular groove** separates the two ventricles.

12.4.2 Chambers of the heart

The **myocardium**, consisting mainly of cardiac muscle, forms the walls of the four chambers of the heart. The walls of the two atria are continuous with each other as are those of the two ventricles. However, there is *no* muscular continuity across the atrioventricular junction. The junction is occupied by a sheet of fibrous tissue that supports the cusps of the tricuspid and mitral valves. The presence of this band of fibrous tissue means that electrical activity *cannot* flow directly from atria to ventricles, except through the atrioventricular bundle (see Section 12.4.4). Fibrous tissue also surrounds the pulmonary and aortic orifices and extends into the upper part of the ventricular septum. These fibrous tissues form the **skeleton of the heart** and support the valves and the attachments for the cardiac muscle fibres.

The principal features of interest in the interior of the chambers of the heart are summarized in sequence.

The right atrium

The major features of the internal structure of this chamber are shown in Figure 12.6. The superior vena cava enters superiorly and the inferior vena cava enters inferiorly and has a rudimentary valve. Medially, there is a small opening of the coronary sinus that drains venous blood from much of the heart wall; this also has a rudimentary valve. The right atrium passes blood through the right atrioventricular (tricuspid) valve into the right ventricle.

The **fossa ovalis** forms a shallow depression in the interatrial wall. This indicates the position of the **foramen ovale** in the developing heart and its function is described in Section 13.2.4. The right atrium is divided vertically by the **crista terminalis,** a ridge on its lateral wall. Most of the atrial wall is smooth; it becomes ridged anterior to the crista terminalis by **musculi pectinati,** parallel muscle bundles running from the crista upwards towards the **right auricle,** a forward projection of the atrium around the base of the aorta as seen on Figure 12.4.

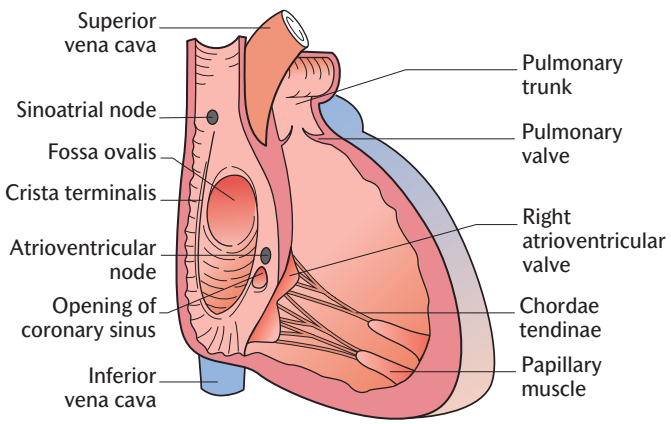

Fig. 12.6 An anterior view of the interior of the right atrium and ventricle.

The right ventricle

The inlet from the right atrium into the right ventricle is guarded by the **right atrioventricular (tricuspid) valve**. As shown in Figure 12.7, it has three cusps as its name implies. Each flap-like cusp is attached to the fibrous atrioventricular ring laterally. In Figure 12.6, you can see the tough strands of fibrous tissue, the **chordae tendinae,** attached to the free edge of each cusp and to **papillary muscles** in the ventricular wall. The papillary muscles contract when the ventricular walls contract, taking up slack in the chordae tendinae, thus preventing the eversion of the valve flaps when ventricular pressure exceeds that in the atrium, and thus preventing back flow of blood into the atrium. The atrial sides of each cusp in contact with flowing blood are smooth whereas the ventricular sides are roughened by the insertion of the chordate tendinae.

The outlet from the right ventricle is into the **pulmonary trunk** guarded by the **pulmonary valve.** As shown in Figure 12.7, the valve comprises three semilunar cusps attached to the atrial wall at their bases. Each cusp is shaped like a pocket with the concave side facing into the pulmonary trunk (see Figure 4.3). The cusps are pressed against the wall of the artery when the ventricle contracts and are filled with blood flowing back when pressure in the right ventricle falls below that in the pulmonary artery; they distend and thus occlude the pulmonary artery. The semilunar cusps have great intrinsic strength because of their shape and require no external bracing. As you can observe in Figure 12.6, the myocardium of the ventricles is thicker than that of the atria with numerous muscle bundles, the **trabeculae carneae,** forming irregular elevations on their internal surfaces. A muscular bundle, the moderator band, crosses the ventricle from septal to anterior wall, carrying the right branch of the **atrioventricular bundle** (see Section 12.4.4).

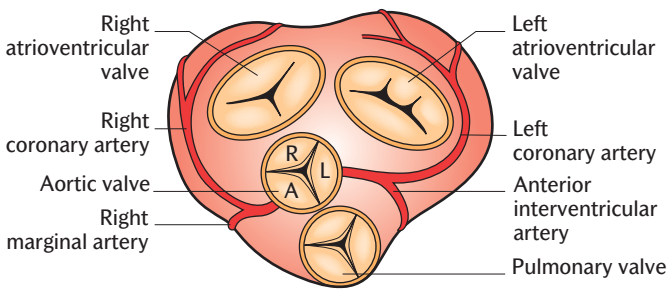

Fig. 12.7 Superior views of the cusps of the heart valves after removal of the atria. A = anterior, L = left, R = right.

The left atrium

Oxygenated blood returns from the lungs to the left atrium through the four pulmonary veins that lack valves. Blood moves into the left ventricle through the left atrioventricular (mitral) valve. Most of the atrial wall is smooth, but the walls of the finger-like auricle are roughened by muscular ridges.

The left ventricle

Compare the diagram of the two atrioventricular valves in Figure 12.7; the **left atrioventricular (mitral) valve** is similar in construction to the right valve, but only has two cusps. Their free edges are attached to the ventricular walls by chordae tendinae and papillary muscles. Resistance to blood flow in the systemic circulation is higher than that in the pulmonary circulation, therefore, the cardiac muscle in the left ventricle has to be much stronger to overcome the increased resistance. In a healthy heart, the left ventricular walls are three times thicker than those of the right ventricle. The left ventricle is circular in cross section and the bulge of the septal wall of the left ventricle makes the chamber of the right ventricle into a crescent. Trabeculae carneae are well developed in this chamber.

Blood is pumped from the left ventricle into the aorta through the **aortic orifice** guarded by the **aortic valve.** Figure 12.7 shows that the aortic valve has three semilunar cusps and is almost identical to that of the pulmonary valve, except in minor detail. The aortic wall above each cusp bulges into an **aortic sinus.** As illustrated in Figure 12.7, the **left** and **right coronary arteries** originate from the sinus above the left cusp and the anterior cusp, respectively (see also Box 12.2).

Box 12.2 Diseases of the heart valves

Damage to the heart valves may lead to narrowing (**stenosis**) of the opening in question. Scarring of the cusps may impede blood flow or stop the valve from closing properly, leading to **valvular incompetence**. The two conditions often coexist. The valvular deformities may be congenital or may be caused by disease, especially **rheumatic fever** (see Box 12.1), in which antibodies raised by the victim to the invading microorganisms may attack the tissues of the heart valves, especially the atrioventricular valves. Deformed valves reduce the efficiency of the heart as a pump and may also form a site at which microorganisms in the blood can be deposited and proliferate, which may lead to **bacterial endocarditis**—inflammation of the lining endothelial layer of the heart chambers.

The presence of valvular disease can be detected by listening to the heart with a stethoscope because both stenosis and incompetence produce an irregular blood flow with an accompanying murmur. The valves affected and the nature of the deformity can be determined from the site of maximum propagation of the murmur and its timing. For example, incompetence of the left atrioventricular valve will produce a murmur best heard at the apex beat during systole because the irregular blood flow takes place during contraction of the left ventricle. Stenosis of the left atrioventricular valve will result in a murmur, also best heard at the apex beat, but occurring during diastole.

12.4.3 Blood supply to the heart wall

Arterial supply

Study Figure 12.8A as you read the following account of the arterial supply of the heart.

The right and left coronary arteries arise from the aorta immediately above the aortic valve as shown in Figure 12.7. They fill during diastole because the vessels penetrating into the cardiac muscle are almost occluded as the muscle contracts during systole. In addition, the valves of the aortic valve partially obscure the entrance to the coronary arteries when they are flattened against the aortic wall as blood is ejected from the left ventricle. During systole, the aorta is distended by the high blood pressure. When the ventricles relax during diastole, the elastic tissue in the aortic wall recoils, pushing blood back towards the closed aortic valves; some of this blood now enters the coronary arteries.

The **right coronary artery** supplies the right atrium and ventricle. Its origin above the anterior flap of the aortic valve is shown in Figure 12.7 and you can follow its course and branching in Figure 12.8A. It runs forwards between the right auricle and pulmonary trunk, then descends in the right atrioventricular groove and continues on to the base of the heart along the groove where it anastomoses with the left coronary artery. One major branch, the **marginal branch,** runs along the right border of the heart towards the apex. A second major branch, the **posterior interventricular artery,** runs in the posterior interventricular groove towards the apex.

The **left coronary artery** is larger than the right. Figure 12.7 indicates its origin above the left flap of the aortic valve. Figure 12.8A illustrates how

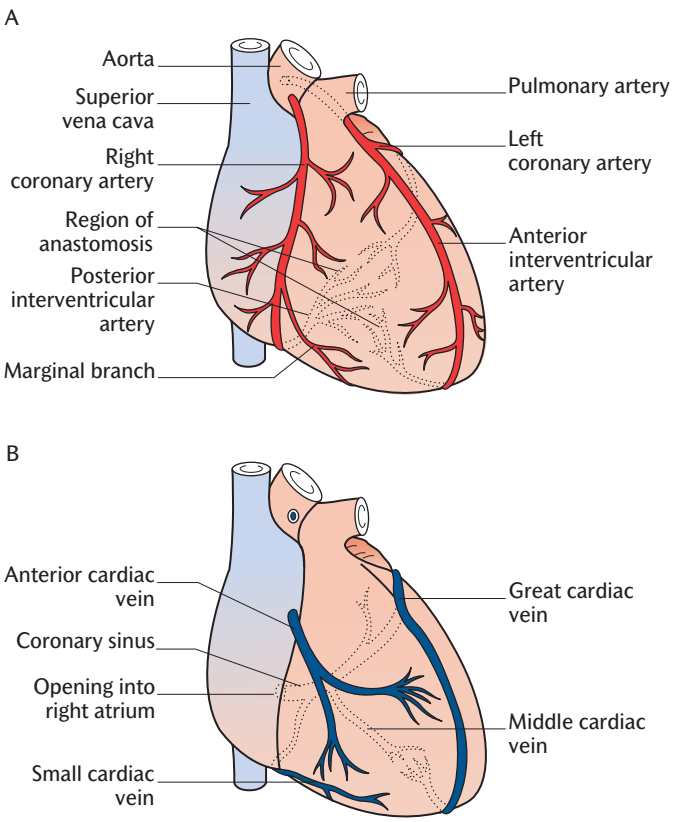

Fig. 12.8 The blood supply of the heart wall. A) Arteries; B) Veins.

it passes behind and to the left of the pulmonary trunk and enters the left atrioventricular groove which it follows until it anastomoses with the right coronary artery on the base of the heart. The **anterior interventricular artery** arises at the **anterior interventricular groove** and follows it to the apex. The anterior interventricular artery usually ascends a short distance in the posterior interventricular groove to anastomose with the posterior interventricular artery. The anterior and posterior interventricular branches supply blood to both ventricles. The left coronary artery also has a left marginal branch running down the left border of the heart to the apex.

Potential anastomoses exist between the various branches of the coronary arteries where they meet on the base of the heart and the apex. These are, however, inadequate to bypass a sudden blockage of a large artery. Some aspects of heart disease are outlined in Box 12.3.

Venous drainage

Figure 12.8B shows the venous drainage of the heart. Most of the coronary veins accompany the corresponding coronary arteries, but the names of veins are not important, except where specifically mentioned. Most of these veins join to form the **coronary sinus** in the posterior atrioventricular groove. About two-thirds of the venous blood from the heart wall drains into the right atrium by this route. The **anterior cardiac vein**, draining most of the anterior surface of the heart, empties directly into the right atrium. The remainder of venous blood drains by small veins in the chamber walls (venae cordis minimae) that empty directly into the heart chambers.

12.4.4 Nerves of the heart

The heart receives its sympathetic supply through cardiac branches arising from the cervical and upper thoracic ganglia of the sympathetic trunk. Its parasympathetic supply is from cardiac branches of the vagus nerves. These branches join together to form **the cardiac plexuses,** networks of fine nerves on the base of the heart. The plexuses supply the coronary vessels supplying the heart and the intrinsic conducting system of the heart.

The conducting system

The conducting system of the heart has already been outlined in Section 4.1.1, but is considered in more detail here. It is made up of specialized pacemaker cells and Purkinje fibres. **Pacemaker cells** are small cardiac muscle cells that contain few contractile proteins but can still conduct impulses. **Purkinje fibres** are elongated cardiac muscle cells lacking contractile proteins which can conduct impulses; they act rather like specialized nerve axons. The function of the intrinsic conducting system is to control the heart rate and to keep the contraction of atria and ventricles in step.

The conducting system is illustrated in Figure 12.9 and comprises the following components:

Box 12.3 Coronary heart disease revisited

Myocardial ischaemia is inadequate blood supply to the cardiac muscle, usually caused by narrowing of the coronary arteries through arteriosclerosis (see Box 4.1). Its major symptom is severe vice-like pain (**angina pectoris**) over the middle of the sternum that often radiates down the left arm or both arms, up the neck, and even the jaw. The pain, caused by accumulations of metabolites in the myocardium, is transmitted from sensory nerve endings in the myocardium via cardiac branches of the sympathetic trunk and thence to the spinal cord through the dorsal roots of the upper thoracic spinal nerves. The nervous system is not familiar with the interpretation of noxious stimuli from viscera so the pain tends to be associated with the areas of skin supplied by the nerves that also supply the internal organ; this is **referred pain**. In the case of the heart, pain is predominantly referred to the arms.

Although coronary arteries anastomose with each other, they behave like functional end arteries in most instances. The heart will continue to function for a while after a sudden blockage of a large branch, but metabolic reserves stored in cardiac muscle cells will be depleted within 10–15 minutes. Lack of blood supply will quickly lead to **myocardial infarction** as cardiac muscle becomes necrotic and begins to die. In other areas of the body, we could rest the affected part while anastomoses open up to provide an adequate blood supply; opening usually takes 24–48 hours. Unfortunately, we cannot stop or rest our hearts while the anastomoses between coronary arterial branches open up.

This sequence of events is referred to, in lay terms, as a 'heart attack' or a 'coronary'. It may be immediately fatal or may lead to death after a few days if the necrosed and thinned heart wall ruptures. As the conducting system of the heart receives its blood supply via the coronary arteries, a blockage may interfere with conduction, producing arrhythmia or heart block (see p. 93). Factors contributing to **atherosclerosis,** the accumulation of fatty plaques in the coronary arteries, include smoking, being overweight, a diet high in saturated fats, and high blood cholesterol.

There have been rapid advances in the detection of coronary arterial narrowing and prevention and treatment of heart attacks in recent years. These include drug intervention or surgery. Low doses of aspirin thin the blood, making it flow more easily and render it less prone to clotting; stronger medicaments such as warfarin have a similar effect, but require careful medical supervision to monitor dosages. Other drugs such as statins are used to control cholesterol levels and thus minimize plaque deposition.

Surgically, severely occluded sections of damaged vessels may be removed and replaced by suturing in a section of vein in its place (a **coronary bypass**); the saphenous vein that runs superficially up the inside of the leg is usually used. Alternatively, a somewhat less invasive procedure is now frequently used. Stents, which are small spring clips, can be introduced into the affected blood vessels by manipulating thin fibre optic cables introduced through superficial blood vessels to the position where the vessel is occluded. The whole procedure is carried out using radiographic visualization of the circulatory system and coronary circulation to determine correct placement of the stent. Once positioned, the stent is released and springs open to maintain the patency of the affected blood vessel.

- The **sinoatrial (SA) node** or **pacemaker** is formed from an aggregate of pacemaker cells. As shown in Figure 12.9, it is situated within the wall of the right atrium at the upper end of the crista terminalis just to the right of the point of entry of the superior vena cava. It usually extends through the whole thickness of the atrial wall. It is well supplied with sympathetic and parasympathetic nerves from the cardiac plexuses.
- The **atrioventricular (AV) node,** also made of pacemaker cells, is somewhat smaller than the SA node. It is located in the wall of the right atrium, immediately above the opening of the coronary sinus.
- The **atrioventricular bundle**, comprising Purkinje fibres, leaves the atrioventricular node and crosses the atrioventricular junction to enter the interventricular septum. This is the *only* electrical connection between the atria and ventricles. Once in the septum, the bundle divides into two branches, one for each ventricle. The right branch passes down the right side of the septum to the moderator band crossing the right ventricle. It forms a Purkinje plexus in the walls of the right ventricle. The left branch runs down beneath the left surface of the ventricular septum. It often branches before terminating to form Purkinje fibre plexuses among the papillary muscles of the left ventricle.

Action of the conducting system

The following sequence of actions control the heart rate and allow delays between contraction of the atria and ventricles to allow efficient filling of each chamber.

1. Cardiac muscle fibres contract rhythmically and synchronously *without* external stimulation. This occurs in the embryo before nerves innervate the developing heart. The muscles that develop first in the atria beat faster than those that develop later in the ventricles.
2. The intrinsic muscular rhythms are regulated by pacemakers. The normal pacemaker of the heart is the SA node which is regulated by inputs from the sympathetic and parasympathetic nerves of the cardiac plexuses. The contractile wave passes from the SA node through the branched atrial cardiac muscle fibres, causing them to contract sequentially. The atria thus contract from above downwards, driving blood through the tricuspid and mitral valves into the ventricles. The impulse eventually reaches the AV node.
3. The wave of contraction passes from the AV node through the AV septum and into the AV bundle. Conduction is carried to the ventricles via its two limbs and their terminal Purkinje plexuses. There is a delay of about 40 milliseconds between activity reaching the AV node and beginning of contraction of the ventricles, thus giving the ventricles time to fill completely before they contract. Contraction of the ventricles begins at the apex and proceeds upwards to the pulmonary trunk and aorta, thus driving blood into the pulmonary and systemic circulations.

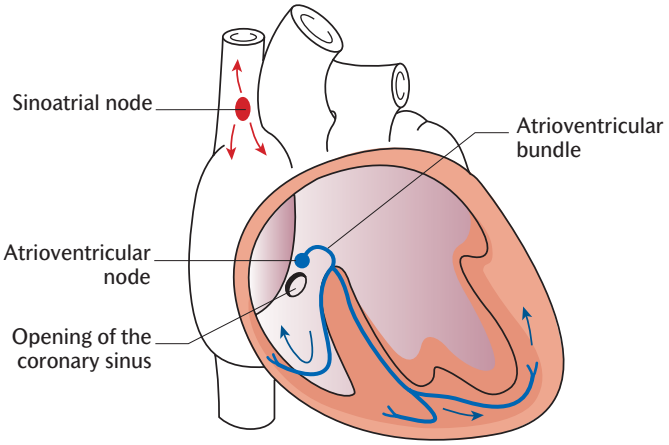

Fig. 12.9 The conducting system of the heart.

A complete heart block occurs when the conducting system between atria and ventricles is interrupted. The ventricles remain active, but beat more slowly than the atria; their rate drops to around 30 contractions a minute which is usually just enough to maintain adequate circulation. The majority of dental students and practitioners will not need to listen to the heart with a stethoscope, but those who undertake postgraduate training and practice in hospitals may need to. The procedures are outlined in Box 12.4.

Box 12.4 Auscultation of the heart

The heart sounds are usually characterized as 'lub dub' and can be heard over most of the chest wall in normal subjects. The first heart sound is produced when the AV valves close and the second sound when the semilunar valves close. There is usually no difficulty in determining which is the first and which is the second heart sound, but, if in doubt, time the heart at the apex beat.

As explained in Section 9.3, the sounds produced at each of the four valves tend to be heard best at specific locations at the **sites of maximum propagation** of sound along vessels or chambers. The starting point to listen to the general heart sound is the apex of the heart; its location and where to find it has been described in Section 9.3.1 and illustrated in Figure 9.3.

The sites for placement of a stethoscope to listen to specific heart valves are illustrated in Figure 9.3:

- The apex beat in the fifth left intercostal space in the midclavicular line for the **left atrioventricular (mitral) valve;**
- Lateral to the left sternal margin in the fifth space for the **right atrioventricular (tricuspid) valve**;
- Over the second *left* intercostal space just lateral to the sternal border for the **pulmonary valve** (remember, the pulmonary trunk lies to the left of the aorta);
- Over the second *right* intercostal space lateral to the sternal border for the **aortic valve**.

It must be emphasized that these are not the surface markings of the valves, but the sites at which clinical experience has shown their sounds are best heard.

12.5 The great vessels

12.5.1 The large veins of the thorax

Figure 12.10 shows the distribution of the large veins of the thoracic which should be studied as you read the following description.

The right and left **brachiocephalic veins** are formed at the root of the neck by the union of the **subclavian** and **internal jugular veins** of the same side just above the first rib. The brachiocephalic veins pass down behind the manubrium sterni and superficial to the large branches of the aorta to unite to form the **superior vena cava** a little above the level of the sternal angle. The left brachiocephalic vein is longer and runs more horizontally than the right vein. The superior vena cava passes downwards to the right atrium.

The **inferior vena cava,** draining venous blood from the body below the diaphragm, pierces the diaphragm and empties into the right atrium almost immediately.

Two **pulmonary veins** leave each lung and drain oxygenated blood into the left atrium.

The veins draining the thoracic cavity are a complex of unpaired **azygos veins;** their details are not important to dental students or practitioners.

12.5.2 The large arteries of the thorax

Figure 12.10 also shows the location of the large arteries of the thorax. Study the diagram as you read the following description.

The aorta

The **thoracic aorta** is conventionally divided into ascending, arch, and descending parts. The short **ascending aorta** leaves the left ventricle and runs upwards and forwards to the level of the sternal angle. Its only branches are the left and right coronary arteries.

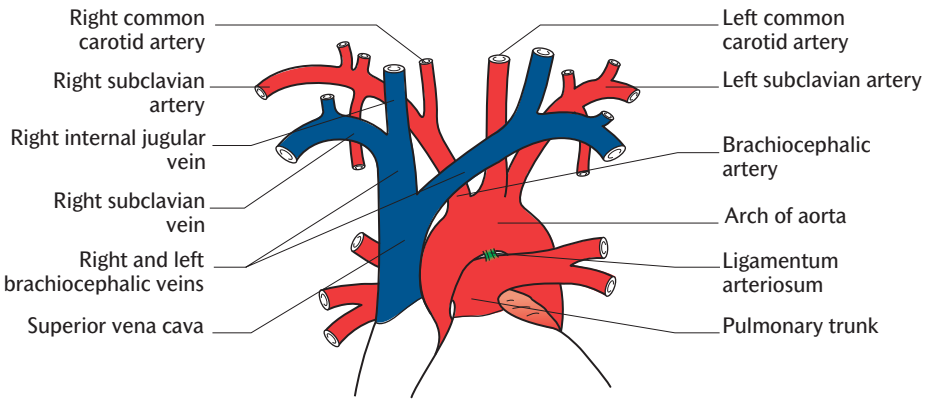

Fig. 12.10 Great arteries and veins of the thorax.

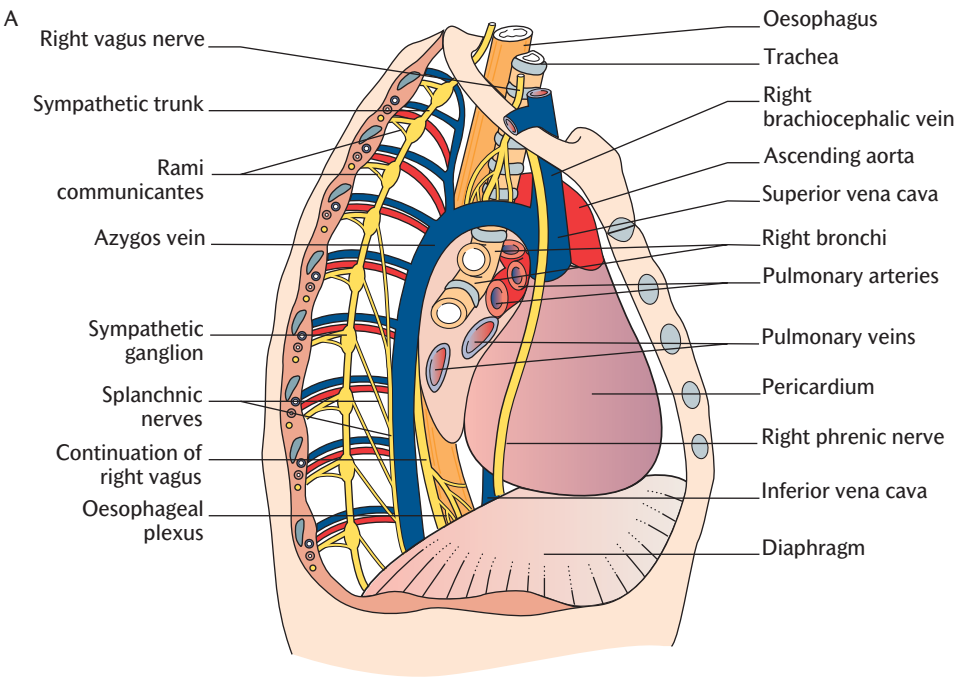

Fig. 12.11 The nerves of the thorax from: A) The right side; B) The left side.

The **arch of the aorta** lies behind the manubrium sterni and runs upwards, backwards, and to the left, in front of the trachea, then turns downwards and to the left, behind the left main bronchus. The **brachiocephalic artery** leaves the ascending part of the aortic arch and runs upwards to the right of the trachea where it divides behind the right sternoclavicular joint into the **right subclavian** and **right common carotid arteries** supplying the arm and head, respectively. On the left side, the common carotid and subclavian arteries arise separately from the upper surface of the arch; the **left common carotid artery** runs along the left side of the trachea to the neck and the **left subclavian artery** runs deep to the common carotid. Each subclavian artery gives off important branches that supply structures in the head and neck, including the brain; the significant components will be described in Sections 15.5 and 22.1.3.

The **descending (thoracic) aorta** runs downwards from the aortic arch and leaves the thorax through the aortic opening behind the diaphragm where it becomes the abdominal aorta. The descending aorta has many branches in the thorax. Pairs of **posterior intercostal arteries** supply the contents of the intercostal spaces, as shown in Figure 12.11, and the **subcostal arteries** which run below the twelfth ribs. Paired **pericardial**, **oesophageal**, and **bronchial** branches supply the corresponding structures. Great vessels may be affected by aneurysms as outlined in Box 12.5.

The pulmonary trunk

The **pulmonary trunk** is most clearly seen in Figure 12.4 and 12.5. It leaves the right ventricle and runs upwards, backwards, and to the left. It divides into the left and right pulmonary arteries, close to the concavity of the arch of the aorta. The right pulmonary artery runs behind the ascending aorta and superior vena cava to the root of the right lung. The left pulmonary artery runs in front of the descending aorta to the root of the left lung.

As illustrated in Figure 12.10, the fibrous **ligamentum arteriosum** runs between the bifurcation of the pulmonary trunk and the concave side of the aortic arch. It is a remnant of an embryonic structure in the circulatory system (see Section 13.2.4). The left recurrent laryngeal nerve loops around the lower border of the arch of the aorta behind the ligamentum.

Box 12.5 Aneurysms

Portions of the walls of the great vessels may be weakened by a variety of causes. When they do so, they partially give way under pressure of the enclosed blood and balloon outwards as **aneurysms**. Aneurysm of the arch of the aorta may compress any of the adjacent structures, including the trachea, oesophagus, recurrent laryngeal nerve, or sympathetic chain. An aorta damaged by atherosclerosis may develop a **dissecting aneurysm** with blood coursing between the layers of the aortic wall, often blocking off branches as it does so. Aneurysms, being due to weakness of the vessel walls, tend to rupture; the consequences are usually fatal if the aneurysm is in one of the great vessels.

B

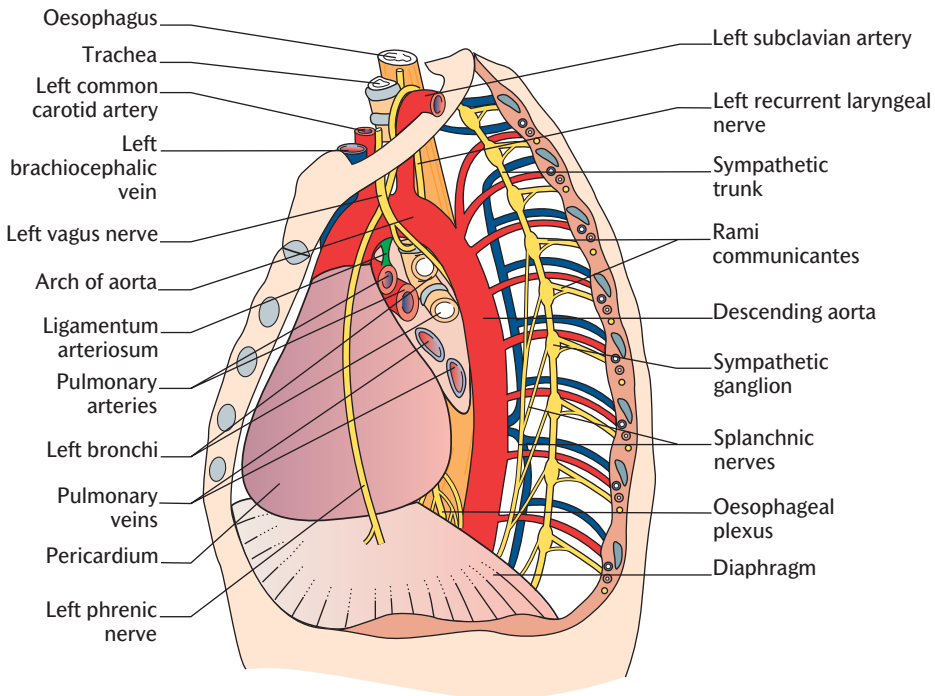

Fig. 12.11 (*continued*)

Box 12.6 Damage to and occlusion of the oesophagus

The oesophagus is normally flattened anteroposteriorly when swallowing is not taking place. During swallowing, it is indented where it is crossed by the arch of the aorta and the left main bronchus and as it passes through the diaphragmatic opening to the abdomen. These are the narrowest points of the oesophageal lumen; they are the likely resting places of large swallowed objects or sites of damage by corrosive fluids if swallowed.

The oesophagus is frequently narrowed by being compressed between the thoracic vertebrae posteriorly and a left atrium enlarged by heart disease anteriorly. This will interfere with swallowing, a condition known as **dysphagia**. This problem may also occur due to pressure from an aortic aneurysm or enlarged left main bronchus.

12.6 Other mediastinal structures

12.6.1 Oesophagus

The oesophagus runs from the laryngopharynx at the level of the lower border of the **cricoid cartilage** to the **cardiac orifice** of the stomach, a distance of approximately 25 cm. Its course and relationship to other structures is illustrated in Figure 11.2. Its upper end lies posteriorly in the midline of the neck. It then enters the thoracic inlet and transverses the posterior mediastinum before passing through the oesophageal opening of the diaphragm to the stomach. It has a muscular wall of striated muscle in its upper two-thirds and smooth muscle in its lower one-third and is lined by non-keratinized stratified squamous epithelium.

The trachea lies anterior to the oesophagus in the neck and superior mediastinum. The oesophagus is crossed in the thorax in succession by the arch of the aorta, the left main bronchus, and the pericardium covering the left atrium from above downwards. The lower cervical and thoracic vertebrae lie posteriorly for most of its course, but near the diaphragm, the descending aorta comes to lie posteriorly between the oesophagus and vertebrae.

The oesophagus receives its parasympathetic innervation from the recurrent laryngeal branches of the vagus nerves and sympathetic supply from the cervical and thoracic sympathetic ganglia.

12.6.2 Nerves of the thorax

Several nerves are present in the thorax. The intercostal nerves have already been described in Section 10.2. The other major nerves, the vagus nerves, phrenic nerves, and sympathetic trunks, have been mentioned several times in the preceding chapters; their anatomical appearance and general functions are now described. Their connections to other parts of the nervous system through neuronal pathways will be found in Section 3.

Sympathetic trunks

Figure 12.11 shows the position of the right and left sympathetic trunks on either side of the vertebral column in the posterior mediastinum. They cross the neck of the first rib, the heads of ribs 2–10, and the bodies of the eleventh and twelfth vertebrae. They then pass behind the diaphragm into the abdomen (see Figure 10.8). Twelve indistinct **ganglia**, one for each spinal nerve, swell each thoracic trunk. Often, the ganglion of the first nerve is incorporated with the inferior cervical ganglion to form the **stellate ganglion** (see Section 23.1.7). Preganglionic axons originating from the thoracic and upper three lumbar segments of the spinal cord exit through the ventral root of each spinal nerve and leave it as **white rami communicantes.** After synapsing in the sympathetic ganglia, the post-ganglionic axons are distributed to their target tissues via one of three routes:

- They may follow arteries, supplying the arteries themselves as they run along them on the way to their targets;
- They may rejoin the spinal nerve as **grey rami communicantes** and be distributed through them. Sympathetic preganglionic axons are myelinated whereas post-ganglionic axons are not, accounting for the colour difference between the white and grey rami;
- In the thorax, as shown in Figure 12.11, the lower seven ganglia have additional branches which are grouped into three splanchnic nerves that innervate the gastrointestinal tract. The first five thoracic ganglia send branches to the heart and great vessels, lungs, and oesophagus.

Box 12.7 Damage to nerves in the thorax and their consequences

The major thoracic nerves both pass close to the lung root. They may be subject to compression or even invasion by enlarged hilar lymph nodes draining the lungs, usually as a consequence of lung cancer. Compression of one phrenic nerve results in paralysis of the corresponding half of the diaphragm. The left recurrent laryngeal nerve is particularly vulnerable because of its close association with the left lung and its root; damage to the nerve will cause hoarseness of the voice which is often one of the first symptoms of lung cancer in the absence of laryngeal pathology. Damage to one of the sympathetic trunks will produce Horner's syndrome (see Section 23.1.7).

Vagus nerves

The right and left nerves are asymmetrical due to asymmetry of the great vessels in the thorax.

The **right vagus** crosses in front of the subclavian artery in the root of the neck where it gives off its recurrent laryngeal branch. As shown in Figure 12.11A, it enters the thorax behind the right brachiocephalic vein, then descends in contact with the trachea, and passes behind the root of the lung where it sends branches to the pulmonary plexus. It then passes on to the posterior surface of the oesophagus and passes with it into the abdomen.

The **left vagus** illustrated in Figure 12.11.B enters the thorax between left subclavian and common carotid arteries. As the nerve crosses the aortic arch, it gives off the left recurrent laryngeal nerve which passes below the arch behind the ligamentum arteriosum and then ascends in the groove between the trachea and oesophagus. The main nerve trunk runs behind the root of the left lung where it contributes to the pulmonary plexus. It then runs on the anterior surface of the oesophagus into the abdomen.

Both vagus nerves give cardiac branches in the cervical part of their course which then descend to the heart. The two nerves supply the lungs and oesophagus through the pulmonary and oesophageal plexuses as they pass through the thorax. They supply the abdominal viscera once in the abdominal cavity.

Phrenic nerves

The phrenic nerves are derived from the ventral rami of the third to fifth cervical nerves (C3–5) and form the motor nerve supply to the diaphragm which originates embryologically in the neck region (see Box 10.6). Each nerve passes downwards to enter the thoracic inlet between the subclavian artery and vein. As seen in Figure 2.12A and B, they descend between the mediastinum pleura and pericardium. When they reach the diaphragm, they pass through the caval opening to supply sensory innervations to the peritoneum on the inferior surfaces of the diaphragm. The phrenic nerves are also sensory to the mediastinal and diaphragmatic pleura and the fibrous and parietal layer of the serous pericardium. Thoracic nerves may be involved in diseases affecting the thorax and its contents; the consequences are outlined in Box 12.7.

13

Development of the heart, respiratory, and circulatory systems

Chapter contents

13.1 Introduction

The development of the circulatory and respiratory systems no longer occupies the time they once used to in dental curricula in the United Kingdom (UK). Apart from the intrinsic curiosity of knowing how two of the major body systems develop, the main reason that they were on the dental curricula was the same as any other aspect of developmental anatomy—the consequences of developmental abnormalities on the planning and execution of dental care. The diagnosis and treatment of developmental heart abnormalities has advanced so rapidly in recent years that people born with such conditions can now be treated so successfully that they can often live symptom-free for the rest of a normal lifespan. The adverse effects of heart defects on the functions of the circulatory and respiratory systems are less likely to be encountered and taken into account during dental treatment; people with problems that do impact on their general health are now most likely to be seen in specialist units. The same does not pertain in developing countries. This brief account is presented to provide an overview of the development of the two systems where knowledge of developmental defects and their consequences on the physiology of the circulatory and respiratory systems is required.

13.2 The circulatory system

The circulatory system must develop to a functional state before any other system in the body. The embryo does not need to grow very much before it reaches such a size that diffusion of nutrients from the surrounding fluids is no longer adequate to fulfil its nutritional requirements. The cardiovascular system must, therefore, be established and begin to function very early on in development.

Even while the embryo is in the trilaminar stage (see Section 8.3.3), a primitive circulatory system is established that connects the embryo to the maternal circulation through the connecting stalk. The heart begins to develop soon after this as a single tube but an atrium and a ventricle can soon be distinguished. These chambers and the veins and arteries entering and leaving the heart are subsequently divided into two so that the right heart and pulmonary circulation and the left heart and systemic circulation are thus established. The fetal circulation is different from the post-natal circulation because the fetus is supplied with oxygen and nutrients through the maternal circulation in the placenta rather than through the lungs; essentially, the lungs are bypassed by shunts. At birth, these shunts are closed and the familiar pattern of blood circulation is established.

13.2.1 The early vascular system

The vascular system begins to appear at the start of the third week of development. **Angioblasts** differentiate from the mesoderm in the cardiogenic area on the anterior margin of the embryonic disc and then from the extraembryonic mesoderm in the yolk sac wall. Angioblasts aggregate into isolated clusters or cords; the peripheral angioblasts flatten into endothelial cells, forming the blood vessel walls and the central cells float free to become red blood cells. The endothelial walls sprout to establish complex branching blood vessels that make numerous communications with each other. As already mentioned, the first vessels are in the yolk sac. They spread into the connecting stalk mesoderm and form a close association with maternal capillaries to form the placental and umbilical vessels (see Figure 8.2). Nutrients derived from the maternal circulation can thus be delivered to the developing embryo from a relatively early stage. Extraembryonic vessels become continuous with intraembryonic vessels around the fourth week of embryonic development.

13.2.2 The heart

In the third week post-fertilization, angioblasts form the primitive U-shaped heart in the lateral plate mesoderm anterior to the buccopharyngeal membrane. As shown in Figure 13.1A, the vascular system develops symmetrically so that even midline vessels are paired initially. Two heart tubes are formed originally; they quickly fuse to form a single tube by the end of the third week of development.

The next stage of heart development is brought about by the embryonic folding process already described in Section 8.4. As a result of the longitudinal folding, the heart tube rotates through 180 degrees and now lies inferior to the buccopharyngeal membrane (see Figure 8.7).

Figure 13.1 B and C show the single heart tube growing and bulging more and more into the pericardial cavity so that it begins to fold. In the very short period between 22 and 26 days, the folding heart enlarges considerably and differentiates into several distinct chambers and resembles the heart rather than a simple tube. The sequence of heart division can be

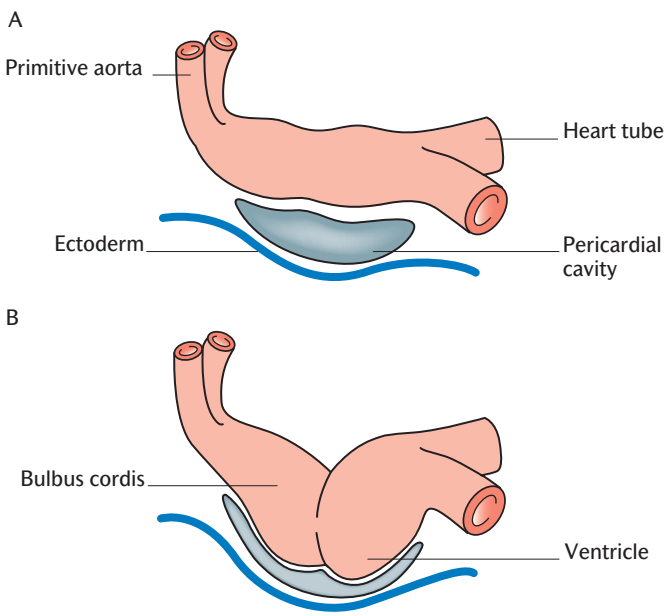

Fig. 13.1 The early stages of heart formation.

followed in Figure 13.2. Transverse grooves mark the divisions of the heart tube. The heart is first divided into the **bulbus cordis** and **ventricle** (Figure 13.2A), then a second transverse groove divides the ventricle into the **ventricle** and **atrium** which receives a short venous trunk, the **sinus venosus**.

As the heart tube continues to grow, it becomes too long to fit into the pericardial cavity but the arterial and venous ends of the heart tube are fixed. Figure 13.2B shows the result; the tube folds in the region of the bulboventricular sulcus into a U-shaped loop. Figure 13.2C shows how a second fold at the junction between ventricle and atrium makes the heart S-shaped.

As shown in Figure 13.2D, several single chambers can be distinguished by the time folding is complete. Arrows in the Figure 13.2D indicate the blood flow from the **sinus venosus** into the **atrium** which bulges around either side of the bulbus cordis. Blood enters the **ventricle,** then the **bulbus cordis** and the paired dorsal aortae. The bulbus cordis will form the roots of the aorta and pulmonary trunk.

13.2.3 Development of the heart chambers

Between the fourth and eighth week, the heart is divided into its four distinct chambers. The changes occur simultaneously in all chambers, but it is easier to appreciate what happens by looking at the components of the heart tube in turn.

The atria

The sinus venosus is incorporated into the atria as they form and part of it forms the coronary sinus.

Division of the atrium into left and right chambers begins during the fifth week. Figure 13.3 indicates the sequence of changes taking place; Figure 13.3A shows the starting configuration. In Figure 13.3B, **endocardial cushions** grow from the anterior and posterior walls and unite to form the left and right atrioventricular openings. At the same time, a sickle-shaped ridge, the **septum primum**, grows forwards in the sagittal plane from the posterior wall of the atrium towards the atrioventricular opening, separating the right and left atria. As shown in Figure 13.3C, the two atria communicate through the gap under the sickle shape, but this is soon closed as the endocardial cushions grow. A number of small openings in the dorsal part of the septum primum, seen in Figure 13.3.C, unite to form the **ostium secundum** to maintain communication between the atria, shown in Figure 13.3D. Much later during the seventh week, as shown in Figure 13.3E, the **septum secundum** grows down from the roof of the right atrium to the right of the septum primum. The septum secundum is never completed so there is a deficiency in its free border that overlaps the ostium secundum. As illustrated in Figure 13.3F, the combined opening through the two septae is the **foramen ovale**, connecting the two atria.

The communication between the atria is important because oxygenated blood returning from the placenta enters the inferior vena cava. Oxygen-rich blood enters the right instead of the left side of the heart. The foramen ovale allows oxygenated blood from the right (pulmonary) to enter the left (systemic) side of the heart for distribution to the tissues and organs without having to pass through the pulmonary circulation. The foramen persists until just after birth when it closes by fusion of the

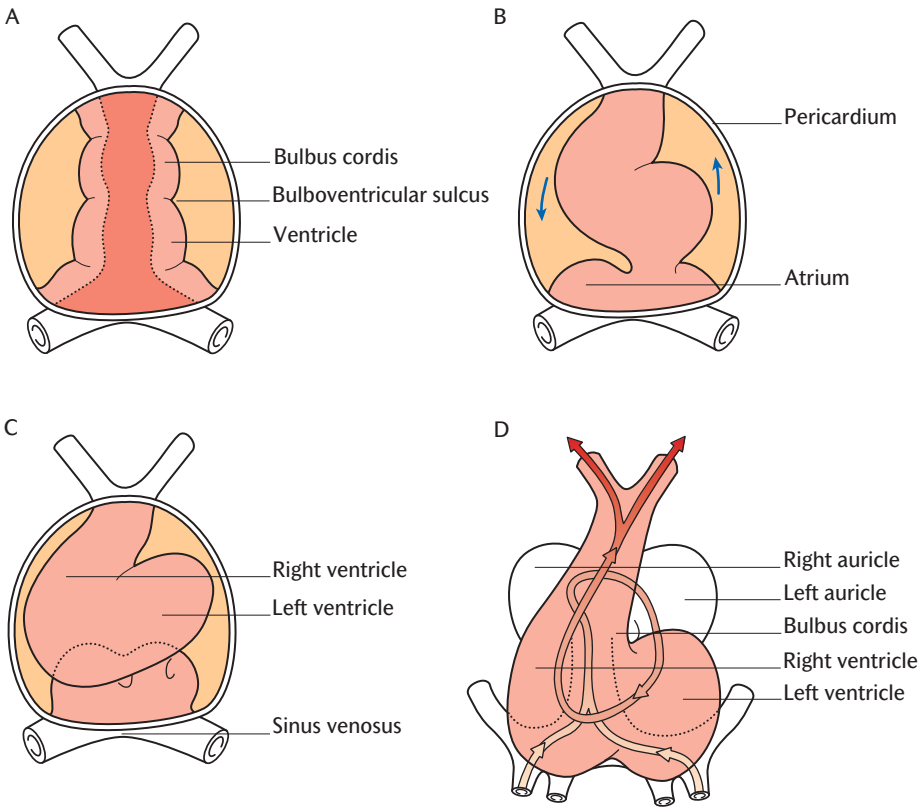

Fig. 13.2 Formation of the heart chambers and folding of the heart tube.

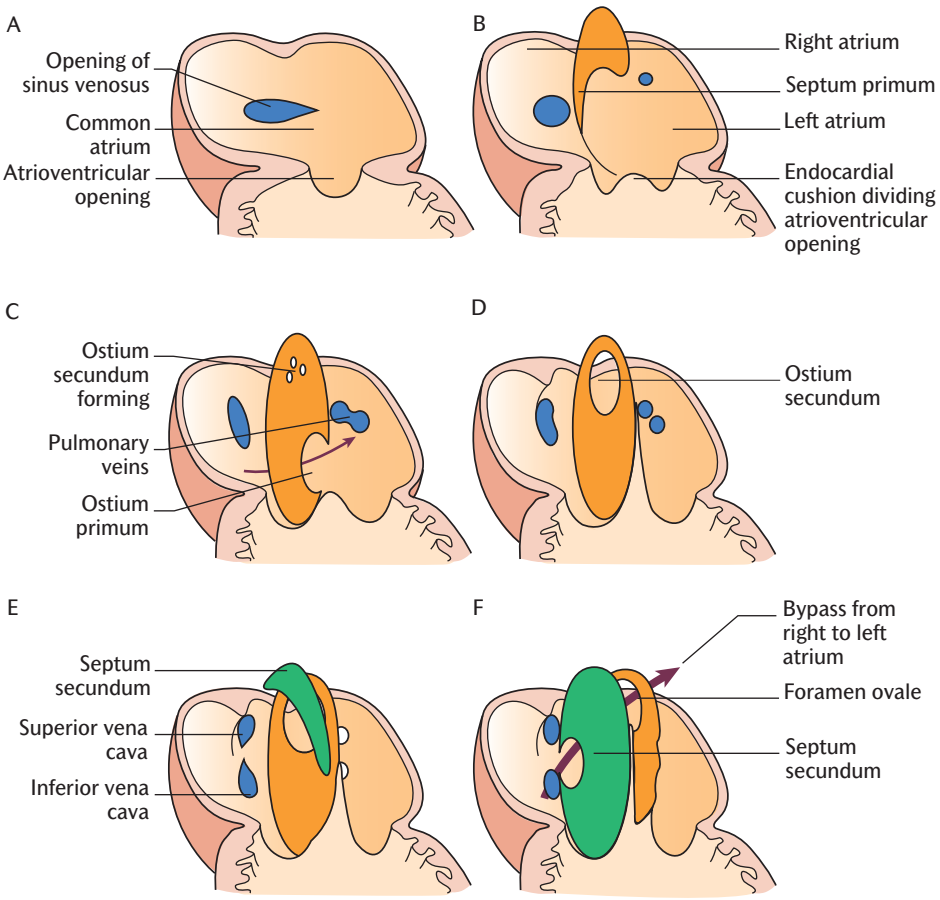

Fig. 13.3 Formation of the atrial septum and foramen ovale.

septum primum and secundum; the **fossa ovalis** marks its position on the atrial septum (see Section 13.2.4).

The ventricles

Division of the common ventricle into right and left chambers is a totally different mechanism from that which divides the atrium. During the fifth week, the right and left sides of the single ventricle expand to form the right and left ventricles. A central band of tissue does not expand and remains as the crescent-shaped interventricular septum which unites with the endocardial cushions and bulbar ridges (see Section 13.2.4).

The endocardial cushions surrounding each atrioventricular orifice thicken to form the atrioventricular valves. The muscle of the ventricular wall below the newly formed valve gradually becomes perforated by larger apertures until the valves only remain attached to the ventricular wall by the **chorda tendinae** and the **papillary muscles**.

The aorta and pulmonary trunk

Around the end of the sixth week, the **bulbus cordis** is divided to form systemic and pulmonary outlets into the aorta and pulmonary arteries. The hydrodynamic flow of blood from the ventricles through the bulbus cordis determines the separation.

As the two ventricles separate, they come to lie side by side with the right slightly anterior to the left. Blood ejected from the right ventricle enters the anterior part of the bulbus and that from the left ventricle enters its posterior part. As shown in Figure 13.4, the two streams spiral round each other. The spiral paths of the two streams of blood from the ventricles mould the two **bulbar ridges** on the walls into spirals. The ridges eventually unite to form a continuous septum. The lower end of this septum

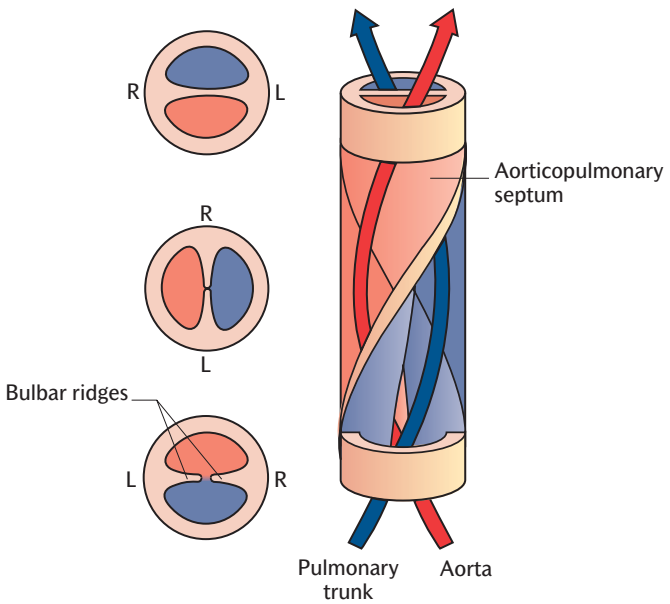

Fig. 13.4 Formation of the aorta and pulmonary trunk.

unites with the ventricular septum. Completely separate channels now exist for blood passing from the right ventricle to the pulmonary trunk and for blood passing from the left ventricle to the aorta. The semilunar valves develop from thickenings at the roots of the aorta and pulmonary trunk.

Development of the conducting system

The developing heart begins to beat before the conducting system and autonomic nervous input develop; the vascular system is still forming at this stage so is incomplete. Contractions spread slowly from the sinus venosus along the simple heart tube. The developing heart lies within the U bend of the intraembryonic coelom (Section 8.3.5) and its beating drives the fluid within the coelom around the embryo, thus providing a temporary nutrient pathway. The folding and rearrangement of the heart renders conduction along a simple tube inadequate so the intrinsic conducting system develops at about 30 days.

13.2.4 The fetal circulation and changes in the circulatory system at birth

Figure 13.5A shows the fetal circulation before birth. Oxygen and nutrients are supplied to the fetus via the placenta which is in the systemic circulation. The position of the placenta within the systemic circulation has two effects. Firstly, it is a large blood reservoir that dramatically lowers the peripheral resistance to systemic blood flow. Secondly, oxygenated blood returns through the systemic circulation to the right atrium as opposed to the left atrium in the post-natal circulation. The right atrium also receives deoxygenated blood from the body tissues as well as oxygenated blood. The pulmonary circulation is non-functional in terms of gaseous exchange, but needs to be established during development, ready for the lungs to function at birth. Because the lungs are collapsed until birth, peripheral resistance in the pulmonary circulation

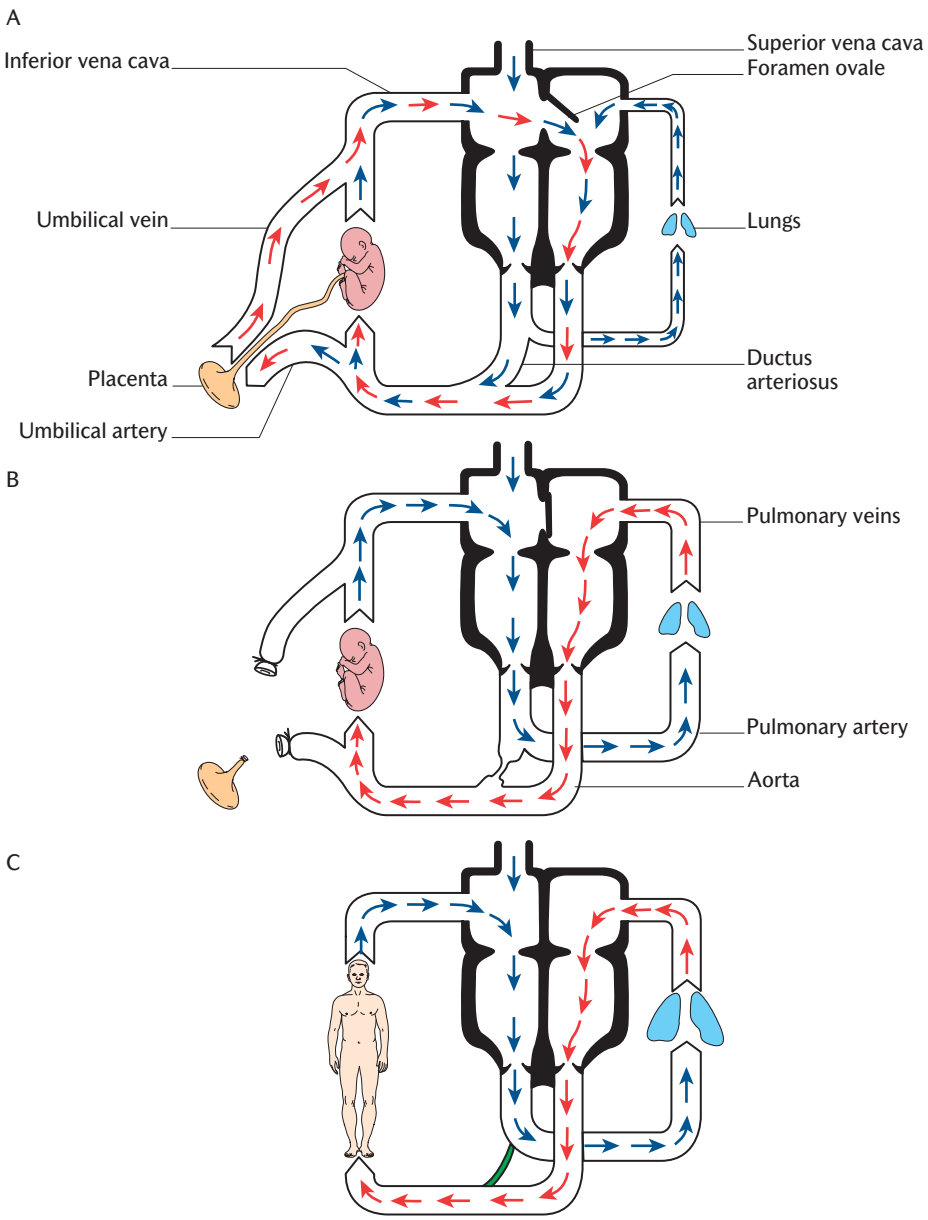

Fig. 13.5 The fetal circulation and changes at birth. A) The fetal circulation; B) Events at birth; C) The post-natal circulation.

Box 13.1 Congenital heart defects

Congenital heart defects occur in about 8 in 1000 live births in the UK. Many congenital heart defects only become apparent shortly after birth, but many are now detected earlier during routine prenatal ultrasound scanning. Heart defects, often commonly referred to as a 'hole in the heart', may manifest shortly after birth or only show later. The usual presenting signs are heart failure and/or cyanosis because of overloading of the heart or inadequate oxygenation of the tissues, respectively. It is now possible to carry out corrective surgery for congenital heart defects at a very early age. Many of these patients will live a long and medically uneventful life as a result. Ventricular septal defects are the most common malformations, accounting for about 20% of cases, followed by atrial septal defects, and patent ductus arteriosus; there are a number of more unusual defects.

Ventricular septal defects

Defects are usually seen in the upper part of the septum close to the atrioventricular valves. After birth, the left ventricle pumps at a higher pressure than the right so blood passes through any septal defect into the right ventricle, thus increasing blood flow to the lungs. The resulting **pulmonary hypertension** will lead to progressive thickening of the walls of the pulmonary arteries which in turn increases the pulmonary blood pressure still further. Sooner or later, a point will be reached when the pressure at which the right ventricle has to pump exceeds that of the left ventricle and blood starts to flow from right to left. Most of the deoxygenated blood will then enter the systemic circulation without passing through the lungs; the tissues will be poorly oxygenated and the patient will develop a bluish tinge in the lips and skin (**cyanosis**).

Any defect in the development of the endocardial cushions that form the left and right atrioventricular orifices, help to form the ventricular septum, and close the developing atrial septum may affect the atrioventricular valves and both septa. Complete failure of fusion will result in multiple defects illustrated in Figure 13.6B.

Atrial septal defects

Post-natal physical sealing of the foramen ovale is incomplete in some 25% of individuals. In most cases, there is no significant passage of blood between the atria and the septal defect is asymptomatic. If you do have access to cadaveric hearts, closely examine the edges of the foramen ovale which may look intact on cursory examination; you may well find small slits or perforations around the margins. As shown in Figure 13.6C, serious atrial defects produce leaks between the atria; as the usual cardiac cycle is disrupted, oxygenation of blood, hence the tissues, is less than normal.

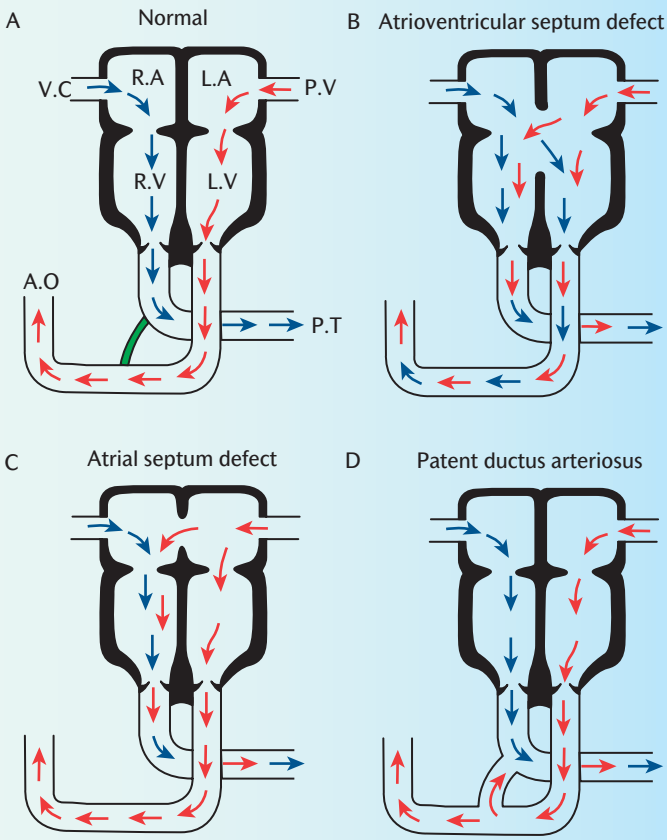

Fig. 13.6 Diagrammatical representations of some congenital heart defects. A) Normal; B) Atrioventricular septum defects; C) Atrial septum defects; D) Patent ductus arteriosus.

Patent ductus arteriosus

The ductus arteriosus normally closes shortly after birth. Physical closure follows during the next 3 months and the ductus degenerates into a fibrous remnant, the **ligamentum arteriosum**. Persistent patency of the ductus commonly occurs, either by itself or in conjunction with other heart abnormalities. The consequences of a patent ductus are shown in Figure 13.6D. Blood is shunted from the high resistance systemic circulation to the low resistance pulmonary circulation with consequent pulmonary hypertension. This may lead to the same changes in the lung vessels as those described above for ventricular septal defects with eventual reversal of the shunt and cyanosis if untreated.

Box 13.2 Development of the arterial system

The first functional vessels, the right and left primitive aortae, arise at about 24 days as superior continuations of the primitive heart tube. These vessels form a few days *after* the heart has started to beat; the early 'circulation' is through the intraembryonic coelom rather than blood vessels.

Follow Figure 13.7A to understand the initial disposition of the aortae and their branches and their development. The two aortae fuse just above the bulbus cordis, but remain as two ventral aortae more cranially. They run forwards from the heart tube, ventral to the developing foregut, and then curve dorsally either side of the gut to run caudally as the paired **dorsal aortae**.

The next stage of arterial development is closely bound up with the formation of the pharyngeal arches which is dealt with in detail in Chapter 21. The first vessels linking the ventral and dorsal aortae are embedded in the ectomesenchyme of the first pair of structures known as the **pharyngeal arches**. To understand the development of the large arteries, you should appreciate that five more pairs of pharyngeal arches develop inferior to the first arch.

Figure 13.7A shows how each pair of developing pharyngeal arches is supplied on each side by an aortic arch arising from the primitive ventral aortae. It curves through the arch to join the corresponding dorsal aorta. This set of vessels allows blood to percolate through the gills of lower vertebrates, but is modified in gill-less mammals to serve other functions. The arches develop from above downwards.

The fate of the aortic arches from each pharyngeal arch is shown in Figure 13.7B. The vessels of the **first** and **second aortic arches** persist only as minor parts of other vessels. The superior arches are disappearing while the lower ones are forming.

The **third aortic arches** make a major contribution to the adult arterial system as shown in Figure 13.7B. They form the **common carotid arteries** and the first part of the **internal carotid arteries**, a remnant of the primitive dorsal aorta. The internal carotid arteries

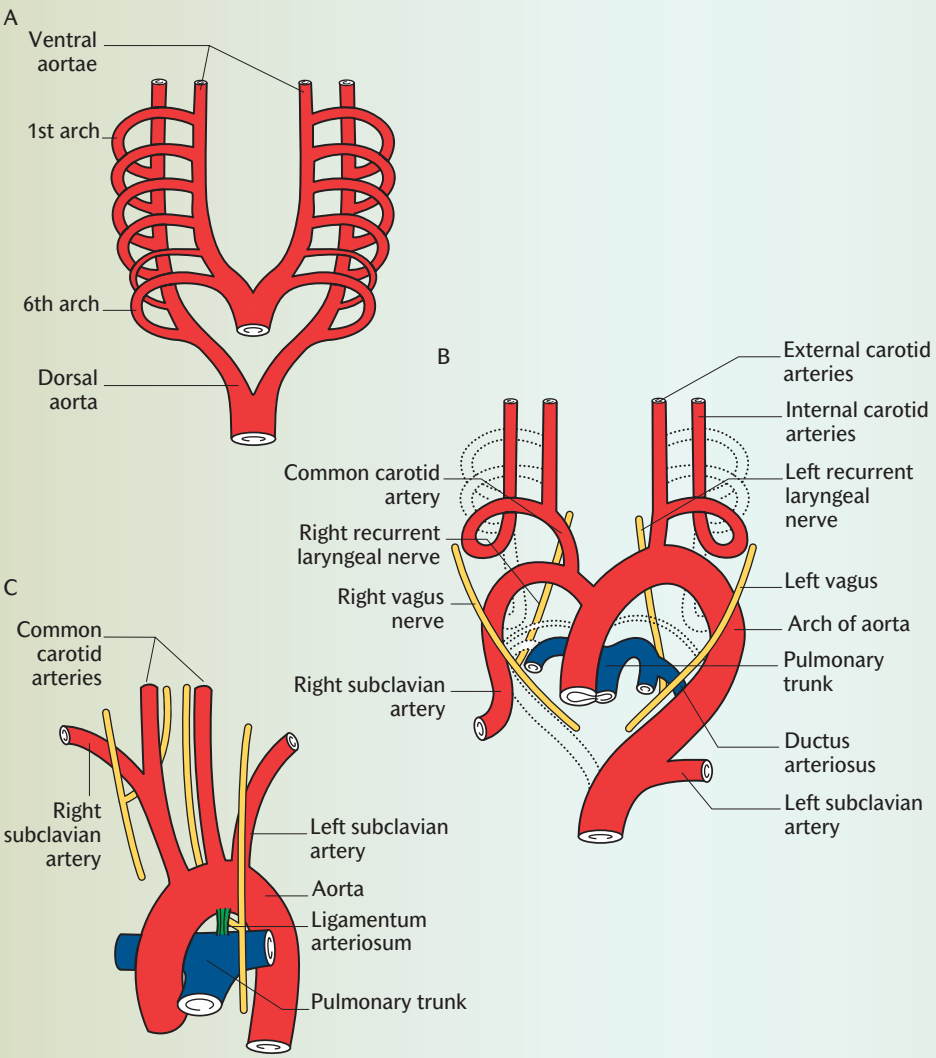

Fig. 13.7 Development of the great arteries. A) The aortic arches in the pharyngeal arches; B) The patterning of the mature arterial system by regression of some embryonic vessels; C) The mature pattern of arteries and the relationship of the recurrent laryngeal nerves.

supply the brain. The origin of the external carotid is uncertain, but it may be a prolongation of the primitive ventral aorta.

Figure 13.7B shows the major asymmetry of the derivatives of the **fourth aortic arch**. On the right, it forms the stem of the **subclavian artery**. The right side of the ventral aorta becomes the **brachiocephalic artery**, leading to the right subclavian and right common carotid arteries. There is no equivalent vessel on the left; the left fourth aortic arch becomes the **arch of the aorta** which maintains its connection to the left dorsal aorta, thus forming the thoracic and abdominal aorta.

The fifth aortic arch vessels leave no trace.

The **sixth** (or **pulmonary**) **aortic arch** vessels form the **pulmonary arteries** indicated in blue in Figure 13.7B. On the left side, the short section of the ventral aorta linking the fourth and sixth aortic arches persists as the **ductus arteriosus.** The septum in the bulbus cordis to divide the aorta from the pulmonary trunk directs blood from the left ventricle into the third and fourth arches and that from the right into the sixth (pulmonary) arch.

All other components of the dorsal and ventral aortae and the pharyngeal aortic arches regress.

The formation of other skeletal and muscular derivatives of the pharyngeal arches causes considerable elongation of the future neck region as a result of which the heart moves from its original cervical position to the chest. Figure 13.7C indicates how the carotid and brachiocephalic arteries elongate to accommodate to these changes and the recurrent laryngeal branches of the vagus nerves are dragged down with the blood vessels. On the left side, this nerve passes around the ductus arteriosus and under the arch of the aorta whereas on the right, it passes around the subclavian artery. The asymmetric course of the two recurrent laryngeal nerves is thus explained by the development of the arteries of the pharyngeal arches.

The development of the venous system is extremely complex and of no importance to dental practice.

is high compared with that in the systemic circulation, the reverse of the post-natal situation.

Oxygenated blood arriving at the right side of the heart needs to be delivered to the tissues through the systemic circulation as rapidly as possible. Circulation through the non-functional lungs is unnecessary so the pulmonary circulation and lungs are bypassed through two channels.

Much of the blood returning to the right atrium is diverted through the **foramen ovale** straight into the left atrium and then into the systemic circulation. This is due to hydrodynamics in the right atrium. Mixed oxygenated and deoxygenated blood entering through the inferior vena cava is almost entirely diverted through the foramen; deoxygenated blood from the superior vena cava tends to enter the right ventricle and pulmonary circulation. Most of the blood leaving the right ventricle through the pulmonary trunk is diverted into the aorta via the **ductus arteriosus** linking the two vessels. The blood could flow through the ductus into the low resistance systemic circulation or try to force its way through the pulmonary arteries to the high resistance pulmonary circulation; most of the blood follows the line of least resistance into the systemic circulation, but some does enter the pulmonary circulation. The post-natal remnant of the ductus arteriosus is the **ligamentum arteriosus** (see Chapter 11 and Figure 12.4).

The pulmonary circulatory system of the lungs is complete by six months but receives only enough blood to keep the vessels patent. The developing lungs receive their oxygen and nutrients via the **bronchial arteries** branching from the aorta (see Section 11.3.2).

Figure 13.5B shows the consequences of cessation of the placental circulation at birth. The lungs are emptied of amniotic fluid, either by the pressure exerted on the chest during birth or by the midwife in attendance, and the lungs fill with air. The expanded lungs decrease peripheral resistance in the pulmonary circulation and blood flow through the pulmonary arteries increases. Isolation of the placenta through ligating and cutting the umbilical cord increases peripheral resistance in the systemic circulation. As a result of these pressure changes, blood flow through the lungs increases and the pressure in the left atrium is raised. The pressure equalization between the two atria forces the septum primum and secundum together, thus closing the **foramen ovale**; fibrous overgrowth shortly after birth produces a physical seal.

Changes in blood pressure in the systemic and pulmonary circulations also cause a momentary drop in the blood flow through the **ductus arteriosus**, the walls of which contract, narrowing the lumen. The closure is augmented by spasmodic contraction as a reaction to temporary anoxia before the baby takes its first breath. The ductus is functionally closed within the next few days. The walls of the umbilical vessels connecting the newborn child to the placenta contract similarly. Developmental heart defects are outlined and illustrated in Box 13.1.

13.2.5 Development of the arterial system

The development of the arterial system is quite complex. It does help to explain the marked asymmetry of blood vessels in thorax which have already been described in Chapter 12 so it is included in Box 13.2 for those who wish to understand their development.

13.3 Development of the respiratory system

The trachea, bronchi, and lungs develop from a pouch, the **respiratory diverticulum**, during the fourth week of development. This appears in the ventral wall of the foregut caudal to the last pharyngeal arch at about four weeks of development.

The sequence of formation of the lower respiratory tract is outlined in Figure 11.1. The diverticulum enlarges to form the laryngotracheal tube that, not surprisingly, will form the larynx and trachea. It continues its growth inferiorly and branches into the right and left bronchi, with the

Box 13.3 Congenital abnormalities of the respiratory system

Major abnormalities, such as the complete absence of lungs or agenesis of one lung, are rare. Most congenital abnormalities of the lungs and bronchial tree, such as the presence of supernumerary lobules, are functionally trivial and make little difference to the efficiency of ventilation.

Abnormalities may arise during the development of the trachea from the foregut. There are several variations, including the formation of a blind oesophagus, the formation of an oesophagus opening into the trachea so that food is transferred to the lung, or the formation of a trachea opening into the oesophagus so that air enters the stomach.

right and left lung buds as seen in Figure 11.1B. With continued growth, each lung bud branches into two lobar bronchi on the left and three on the right. These main bronchi undergo repeated branching until by the time of birth, approximately 17 generations of subdivisions exist. Further divisions take place during post-natal growth of the lungs. In the fetus, the bronchi are closed tubes: when the newborn baby takes its first breaths, the thin-walled respiratory bronchioles are inflated to form the **alveoli**.

The epithelial lining of the respiratory tree is thus endodermal in origin because of the development of the respiratory diverticulum from the endodermal lining of the foregut. The associated smooth muscle and cartilage are derived from the mesoderm surrounding the developing bronchial tree. As the lungs enlarge, they push into the part of the intraembryonic coelom anterior to the foregut. As shown in Figure 11.1D, the coelomic mesoderm adjacent to the lungs becomes the visceral pleura and that adjacent to the body wall becomes the parietal pleura. The space between the two layers of pleura becomes the pleural cavity. Some abnormalities of development of the respiratory system are mentioned in Box 13.3.

Section 3
The central nervous system

Section contents

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14

Introduction to the central nervous system

Chapter contents

14.1 Introduction

Dental students and practitioners need a working knowledge of the central nervous system (CNS) for several reasons.

- A general knowledge of the structure and function of the nervous system is required to understand the major roles it plays in controlling body functions.
- The cranial nerves innervating the head and neck, including the oral cavity, underpin all functions in these areas; knowledge of these nerves, including their connections to the CNS is vital to understanding the anatomy and physiology of this region.
- Clinically, dental students and practitioners will frequently encounter patients suffering from one or other of the many diseases affecting the central and peripheral nervous system. Satisfactory dental management of such patients requires some understanding of their illness which in turn requires knowledge of the general structure of the nervous system.

The anatomy of the nervous system was described long before we understood much of its function. Like all other parts of the body, everything is named; some of the names seem to defy the logic of anatomical nomenclature used to describe structures elsewhere in the body introduced in Chapter 1. Some of the structures visible to the naked eye were named by their fanciful resemblance to everyday objects such as olives; their names, therefore, bear no resemblance to their function. However, the nerve tracts that connect different areas to form functional pathways are described using a consistent system of naming (see Section 3.4.3). Only the most important structures that can be observed in dissected brains or form important landmarks in functional pathways are included in these chapters on the nervous system.

14.2 The study of brain structure and function

It is important to appreciate that much of the detailed structure of the brain can only be observed microscopically. Special microscopical methods are required to show its structure and even then, a practised eye is required to interpret them. Nevertheless, it does help to know the outline of how the connections and functions of the nervous system have been investigated to understand how we have arrived at our present level of knowledge.

14.2.1 Historical methods of investigation

Initially, careful clinical observations of signs and symptoms prior to death were correlated with post-mortem changes in the brain. Diseased parts of the brain revealed at post-mortem implicated that these areas carried out the functions that were observed to be deficient when the subject was still living. Unfortunately, neurological diseases respect neither anatomical nor functional boundaries so many studies, although helpful, were inconclusive. The first designation of function to areas of the cerebral cortex, those involved with language processing, were derived from such methods in the 19th century. In essence, they have stood the test of time but have been significantly modified as new information has become available. Major advances were made from extensive studies of the large numbers of wounded soldiers in the major conflicts of the first half of the twentieth century which were then confirmed and extended by experimental studies.

The next step was to try and repeat clinical findings on experimental animals by artificially injuring key areas and observing the effects. Some advances were made from such experiments. However, they are severely limited in many respects, not the least that it is difficult to investigate higher cognitive functions in animals. In the 1960s and 70s, methods were developed for tracing pathways in the brain. Special dyes that are transported along neuronal processes were introduced into specific areas of the brain in experimental animals. By examining microscopical sections of the brain, it was possible to work out many connections and pathways in the brain by tracing where the transported dyes ended up. Once again, animal studies have their uses.

14.2.2 Modern methods of investigation

In recent years, methods available for studying functional localization in the brain have become very much more powerful and refined. It is now possible to use non-invasive or minimally invasive methods to study the

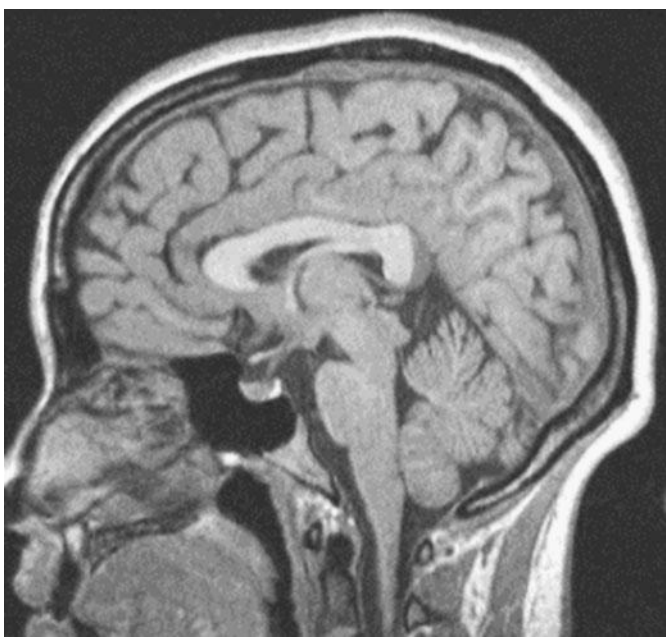


Fig. 14.1 An MRI scan of the human brain (Courtesy of Prof. Patty Cowell, Department of Human Communication Science, University of Sheffield).

living, human nervous system directly rather than have to extrapolate from post-mortem tissue or animal studies. Computer-assisted tomography (**CT scans**) enabled clinicians and neuroscience researchers to investigate the effects of damage and disease in much more detail than previous post-mortem methods and they could be performed on living patients. Magnetic resonance imaging (**MRI scans**) provide a much better resolution of detail; an example is shown in Figure 14.1. MRI has sufficient resolution to distinguish between areas perfused with oxygenated blood and deoxygenated blood because of their different magnetic properties; specific areas of the brain used for a given function will show up on the computer-enhanced images of the brain as oxygenated blood is specifically directed into these areas when they are active. This technique is known as functional magnetic resonance imaging (**fMRI**) and is able to give a dynamic picture of brain function. Transcutaneous magnetic stimulation (**TCMS**) enables investigators to stimulate areas of the brain through the scalp and skull. They can observe the patient's actions and reactions or ask the patient to report the effects.

The use of these methods has changed our views of the structure and functions of the brain and spinal cord and continues to do so. It is humbling to realize that descriptions of many aspects of the structure and function of the CNS will be out of date in the months between the writing of this book and its publication. Investigation of the structure and function of the nervous system is one of the most active areas of research in the whole of anatomy and clinical practice.

14.3 The structure of the nervous system

The structure and function of the component cells of the nervous system and its overall structure and function have been described in Chapter 3. This material should be revised before studying the more detailed information in Chapters 14 to 18. A summary of some of the salient features is outlined in the following paragraphs.

The nervous system contains two main groups of cells, **neurons** and **(neuro)glia**. **Neurons** are specialized cells for the reception of stimuli from external sources or other neurons, which may lead either to excitation or inhibition of the cell. If a neuron is stimulated, it will conduct an impulse (an **action potential**) along its extensive cellular processes, sometimes over considerable distance. Refer back to Figure 3.1 which shows the structure of a typical neuron. Each neuron consists of a **cell body** containing the nucleus and synthetic organelles. A number of processes arise from the cell body. One process, the **axon,** conducts impulses away from the cell body and can be very long. Impulses reach the cell body through one or more short branched processes called **dendrites**.

Neurons connect with each other at **synapses** as shown in Figure 3.3. They are usually formed between axons of presynaptic neurons and the dendrites and/or cell bodies of post-sysnaptic neurons. Synapses are specialized areas between adjacent neurons where the cell membranes of the two neurons are separated from each other by a narrow **synaptic cleft**. The cleft is sufficiently wide to prevent direct electrical conduction from one neuron to another. The synaptic separation is overcome by **neurotransmitters**. These chemical messengers are released from presynaptic axons when action potentials reach their terminal structures. The neurotransmitter then crosses the synaptic cleft to attach to and activate receptors on the post-synaptic neuron. Incoming stimuli from presynaptic neurons can be modified at synapses; this determines whether the post-synaptic neuron is stimulated or inhibited. The axons of some neurons are enclosed by insulating **myelin** sheaths to preserve electrical propagation along axons without change; these sheaths are formed by neuroglia. In the CNS, areas containing a large number of cell bodies, dendrites, and synapses have a greyish appearance and are designated as **grey matter**. The contrasting **white matter** consists mainly of axons, the white appearance being due to the fatty myelin sheaths enclosing many of these processes.

The second major group of cells within the CNS is composed of the non-excitable, supporting (**neuro)glial cells**. These are:

- **Oligodendrocytes** that provide the myelin sheaths for nerve axons within the CNS;
- **Astrocytes** that have a supporting role and are involved in the transport of chemicals and nutrients within the brain;
- **Microglia** that are actively phagocytic and function as part of the macrophage-based, non-specific defence system;
- **Ependymal cells** that form the thin epithelium lining of the ventricles of the brain and the central canal of the brainstem and spinal cord.

Schwann cells are the principal non-excitable glial cells in the peripheral nervous system (PNS) and provide sheaths for the peripheral nerve processes. Smaller unmyelinated processes run singly or in groups in longitudinal grooves in a succession of Schwann cells. The larger processes are surrounded by a myelin sheath formed by Schwann cells.

Turn back to Chapter 3 to revise the important distinctions between:

- The **CNS** comprising the brain and spinal cord and **PNS**, comprising 31 pairs of spinal and 12 pairs of cranial nerves;
- **Motor** and **sensory** components of these systems and their functions;
- **Somatic** and **autonomic** parts of these systems and their functions.

Here is a brief recapitulation of the major regions, subdivisions, and structures within the CNS. The **brain** and **spinal cord** are continuous with each other just below the foramen magnum. The brain itself consists of the **brainstem**, **cerebellum**, and **cerebrum**. The brainstem is the upward continuation of the spinal cord and is clearly divisible, on the basis of both external appearance and internal structure, into **medulla oblongata, pons**, and **midbrain** in ascending order from the spinal cord as shown in Figure 3.8. The cerebellum is attached to the dorsal aspect of the brainstem by the three paired **cerebellar peduncles**. The cerebrum consists of the **diencephalon,** the central core of the cerebrum above the midbrain, and the paired **cerebral hemispheres**. The diencephalon contains such important components as the **thalamus** and **hypothalamus**. The cerebral hemispheres are composed of an external layer of grey matter, the **cerebral cortex,** and an internal mass of white matter. The **basal nuclei** are masses of grey matter located within this internal white matter.

14.4 The function of the nervous system

The key role of the nervous system is to integrate functions throughout the body in response to external stimuli, conscious thought, and unconscious stimuli from within the body itself. It follows, therefore, that the separate structures comprising the nervous system need to be highly integrated into functional systems and pathways of interlinked neurons that may run through several of the various anatomical divisions or individual structures of the CNS.

As you study the nervous system, try to think in terms of *functional connections and pathways* between different components of the nervous system to see the bigger picture of the organization of the nervous system. An appreciation of the general plan makes study and understanding so much easier than learning individual detail.

To understand the CNS and how its structure and function relate to clinical practice, you should ask yourself three essential questions for any new structure or function you encounter:

- **Where is it?**
- **What does it do?**
- **What is going to happen if it cannot carry out its function because of damage or disease?**

15

The structure of the central nervous system

Chapter contents

15.1 The braincase and cranial fossae

It is important to have a picture of the relationship of the brain and spinal cord to the bones of the skull and vertebral column that house and protect them and the protective layers of connective tissues known as the **meninges** that cover the CNS; these lie between the bones and brain and spinal cord.

The brain is housed within the skull which will be described in much more detail in Section 4. As you can appreciate by feeling your own skull, the top, front, sides, and back are smoothly curved. The surface of the brain is similarly curved and conforms to the shape of the bones. Note that, in reality, it is really the other way round—brain shape determines the shape of the bones of the skull vault forming the braincase (see Section 33.3). If the top of the braincase and the brain are removed to reveal the floor of the cranial cavity formed by the bones of the cranial base, it is anything but smooth. Viewed from the lateral aspect and going from anterior to posterior, it is like three descending steps. This structure is shown diagrammatically in Figure 15.1 and shows how different parts of the brain conform to these steps. The first step lies above the nasal and orbital cavities and is known as the **anterior cranial fossa**; it houses the frontal lobes of the cerebral hemispheres. The second step is the **middle cranial fossa** and contains the temporal lobes of each cerebral hemisphere laterally and the midbrain and pons medially. The final step is the **posterior cranial fossa** where the rest of the brainstem and cerebellum lie. The floor of the posterior fossa is pierced by the **foramen magnum** through which the medulla oblongata and spinal cord become continuous.

The **spinal cord** occupies the vertebral canal running in the vertebral column. As you can see in Figure 3.5, in adults, the cord occupies the vertebral canal from the upper border of the first cervical vertebra, the atlas, down to the level of the disc between the first and second lumbar vertebrae. The lower lumbar, sacral, and coccygeal spinal nerves travel down from their relevant spinal cord segments through the lower vertebral canal to exit from their respective intervertebral foramina. For example, the fifth lumbar segment of the spinal cord is at the level of the twelfth thoracic vertebra, fifth lumbar nerve; the roots of the fifth lumbar nerves, therefore, have to run down past all the lumbar vertebrae to their exit from the canal through the intervertebral foramen below the fifth lumbar vertebra. The lower spinal nerves form a bundle of nerves known as the **cauda equina** (Latin = horse's tail). The narrow lower end of the cord is attached to the first coccygeal vertebra by a thin filament, the filum terminale. In the early fetus, the vertebral canal and spinal cord are virtually equal in length; the vertebral column elongates more rapidly than the cord during later fetal life and to a lesser extent, in childhood so the cord does not fill the full extent of the vertebral canal.

15.1.2 The meninges

The meninges are three layers of connective tissue that lie between the bone of the braincase and the external surface of the brain and continue through the foramen magnum to surround the spinal cord within the vertebral canal. Figure 15.2 shows the disposition of the three layers; follow the diagram as you read the description. The outermost layer, the **dura mater**, lies immediately adjacent to bone. The innermost layer, the **pia mater**, is intimately applied to the surface of the CNS. An intermediate layer or **arachnoid mater** lies between the dura and pia. (Although the full name of each layer consists of two words, 'mater' is frequently omitted in practice; the layers thus become the dura, arachnoid, and pia. We will follow this convention in the following descriptions.)

The dura (mater)

Technically, the dura consists of an outer endosteal layer and an inner meningeal layer. As Figure 15.2 demonstrates, these two layers are in fact united except where they separate to enclose the venous

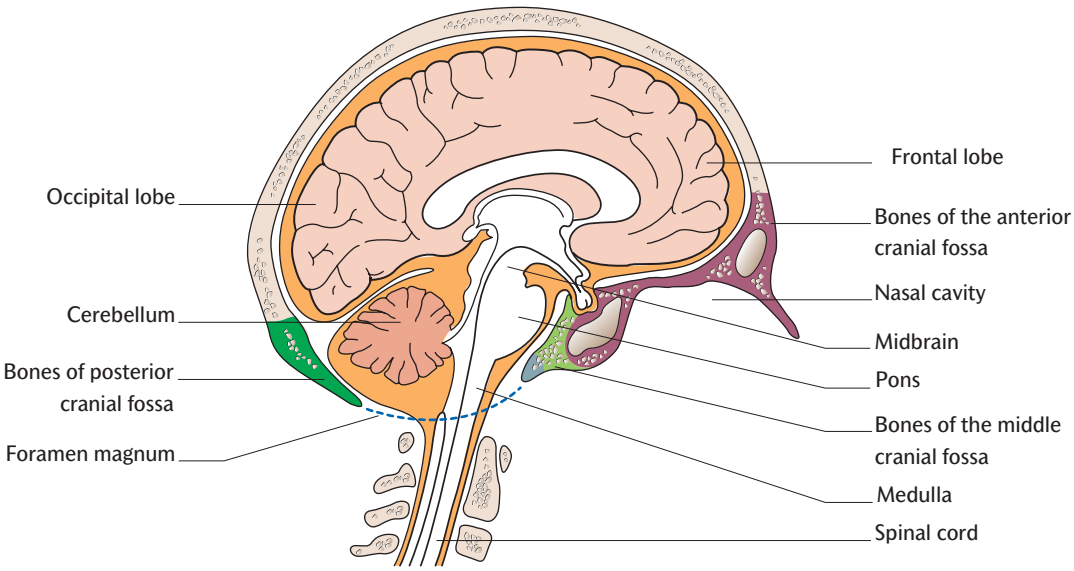


Fig. 15.1 The relationship of the brain to the cranial fossae of the skull.

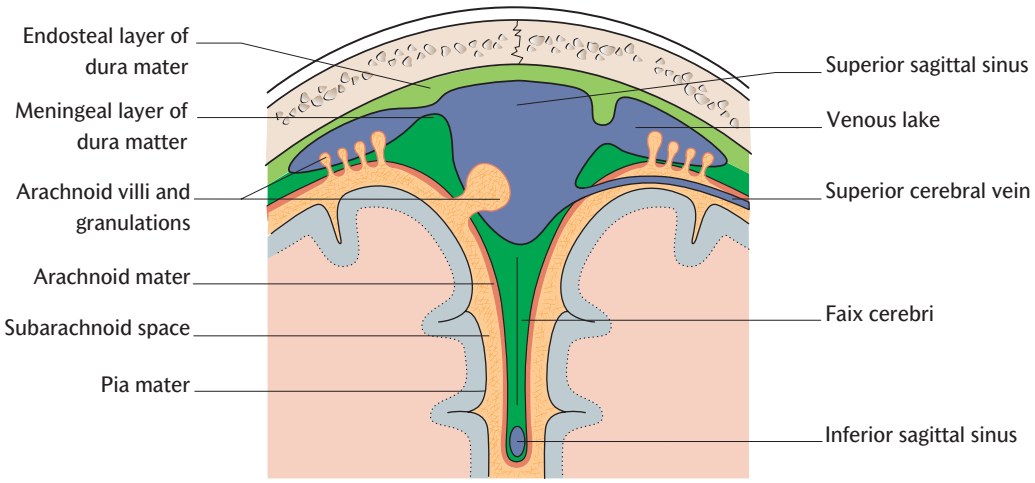


Fig. 15.2 The meningeal layers and formation of venous sinuses.

sinuses, specialized venous channels draining blood from the brain and meningeal blood vessels (see Section 15.1.6).

The endosteal layer is the periosteum of the internal surfaces of the braincase and vertebral canal to which it tightly adheres. This layer continues into the foramina, carrying blood vessels and nerves into and out of the cranial cavity and it blends with the periosteum on the external surface of the braincase. The meningeal layer is tough and fibrous and gives the dura its characteristics. It forms tubular sheaths around the cranial and spinal nerves as they leave the cranial cavity and spinal canal which fuse with the epineurium of the nerves. As illustrated in Figure 15.2, the meningeal layer is reflected at certain points to form double-layered folds that project into the cranial cavity between different parts of the brain and help stabilize and protect the brain; they are described in Section 15.1.3.

The pia (mater)

The pia is a delicate layer of richly vascular connective tissue. The pia is represented in black in Figure 15.2. You can see how it closely covers the surface of the brain and spinal cord at all points; it enters even the deepest sulci and the walls of the ventricles of the brain to form the **choroid plexuses** that produce cerebrospinal fluid.

The arachnoid (mater)

The delicate arachnoid lies against the internal surface of the meningeal layer of the dura mater and the two are separated by the narrow **subdural space** that contains a thin film of tissue fluid. A wider gap separates the arachnoid and the pia. This is the **subarachnoid space** which is filled with **cerebrospinal fluid**. As illustrated in Figure 15.2, fine strands of arachnoid tissue extend across the subarachnoid space to the pia and look like a spider's web; it is this likeness that gives the arachnoid its name (from the Greek = spider). The size of the subarachnoid space varies. It is relatively narrow over convexities of the brain and wider over fissures and other depressions. The subarachnoid space is widest on the underside of the brain in areas called the basal cisterns. If you have the chance to examine a hemisectioned head with the brain still in place, the arachnoid bridging the subarachnoid space is most clearly seen at the cerebellomedullary cistern occupying the angle between the cerebellum and the posterior surface of the medulla.

The arteries and veins of the brain travel within the subarachnoid space before entering or after leaving the brain tissue. As the cranial nerves cross the subarachnoid space, they pick up sheaths of pia and arachnoid. These sheaths extend as far as the exit of the nerves from the skull where they fuse with the epineurium.

The arachnoid herniates through the meningeal layer of the dura mater to protrude into the venous sinuses and venous lakes in certain areas. Small herniations are called **arachnoid villi.** The **arachnoid granulations** are considerably larger and may be seen with the naked eye if the superior sagittal sinus is opened. The villi and granulations are the structures through which cerebrospinal fluid is absorbed into the bloodstream (see p. 131)

15.1.3 Dural reflections

The two largest dural reflections can be seen in Figure 15.3 in which the right cerebral hemisphere and overlying bone have been removed. The **falx cerebri** is a double-layered fold of the dura attached along the midline inside the cranial vault where it is continuous with the meningeal layer of the dura. Study Figure 15.3 to understand its general shape, its attachments to the skull, and its relationship to venous sinuses. It is attached anteriorly to the crista galli, a short bony process above the nasal cavity, and posteriorly it attaches to the superior surface of the tentorium cerebelli. The two leaves forming the falx separate and blend on each side with the tentorium cerebelli to enclose the **straight sinus**. The falx is, therefore, relatively narrow anteriorly and broadens out posteriorly, resembling the shape of a sickle (hence its name from the Latin falx = sickle). At the convex superior attachment, the two leaves of the falx cerebri are separated to form the **superior sagittal venous sinus**. The free concave lower border of the falx contains the **inferior sagittal venous sinus**. The two leaves of the falx are firmly united between the superior and inferior sagittal sinuses to form a strong, inelastic membrane that extends into the longitudinal fissure between the two cerebral hemispheres. The falx, therefore, is ideally positioned to prevent excess movement of the brain if the head is subject to violent turning movements.

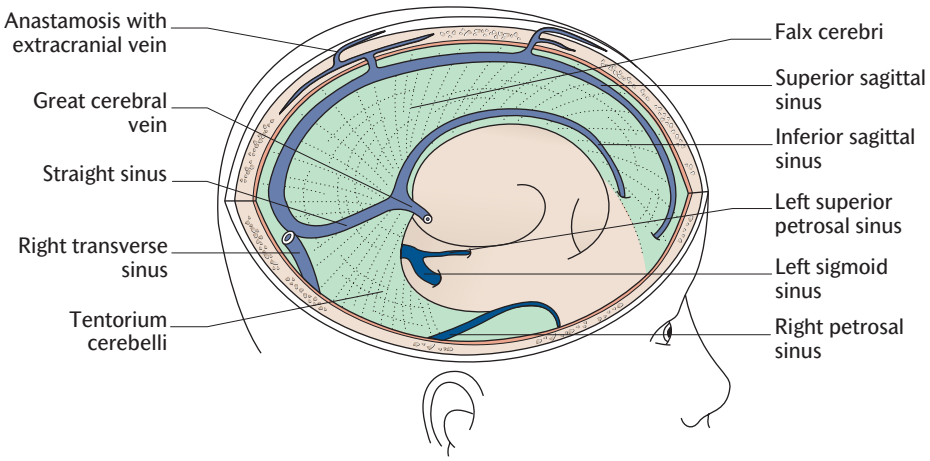


Fig. 15.3 Dural reflections and formation of venous sinuses.

As you can see in Figure 15.3, the **tentorium cerebelli** (Latin = roof (or tent) over the cerebellum) lies in the horizontal plane at right angles to the falx cerebri. The position and attachments of the tentorium is much more easily appreciated when viewed from directly above as in Figure 15.4 which should be studied as the following description is read. Its two leaves separate on each side to blend with the meningeal dura on the inner aspects of the occipital and parietal bones, enclosing the **transverse venous sinus** between them. The course of the transverse sinuses is marked by prominent grooves in these bones. Anteriorly, the tentorium is attached to the posterior superior border of the petrous temporal bones; the **superior petrosal sinuses** are found at these attachments. The anterior attachments extend to just behind the pituitary fossa. Figure 15.4 shows how this arrangement creates short, concave, free inner margins, enclosing an opening occupied by the midbrain. The tentorium projects into the cranial cavity above the cerebellum and below the occipital lobes of the cerebral hemispheres. It is well placed to prevent movement of the brain when the head is moved violently forwards and backwards as in nodding the head.

Near the apex of the petrous bone, the lower layer of the tentorium continues forwards to form a small outpouching between the endosteal and meningeal layers of the dura of the middle cranial fossa. This is the **trigeminal** (Meckel's) **cave** that contains the roots and the sensory ganglion of the trigeminal nerve.

The falx cerebelli is a small curved fold of dura mater which projects into the posterior notch of the cerebellum below the tentorium.

Figure 15.4 illustrates a circular fold of dura, the **diaphragma sellae**, which forms the roof of the pituitary fossa in the sphenoid bone. The diaphragm of dura completely covers the pituitary gland, apart from a small central opening for the pituitary stalk.

15.1.4 Meninges of the spinal cord

The arrangement of the meninges enclosing the spinal cord is illustrated in Figure 15.5. The spinal **dura** forms the external layer and lies close to the periosteum lining the vertebral canal. The **extradural** (or **epidural**) **space** between them contains some adipose tissue and a venous plexus. The dura ensheathes the roots of the spinal nerves as they pass through the intervertebral foramina, then fuses with the covering epineurium.

The **spinal arachnoid mater** lies against the inner surface of the dura. The **pia mater** closely invests the spinal cord and surfaces of the roots of the spinal nerves. There is a substantial **subarachnoid space**

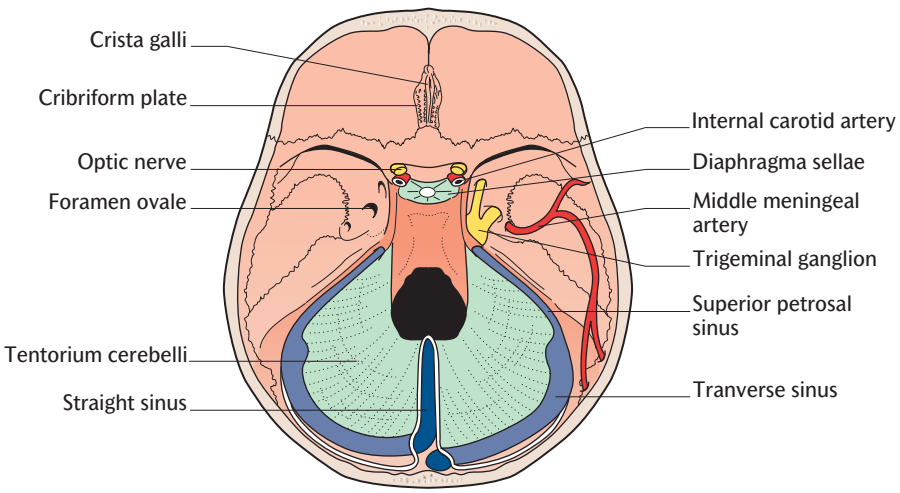


Fig. 15.4 The tentorium cerebelli and associated venous sinuses from above.

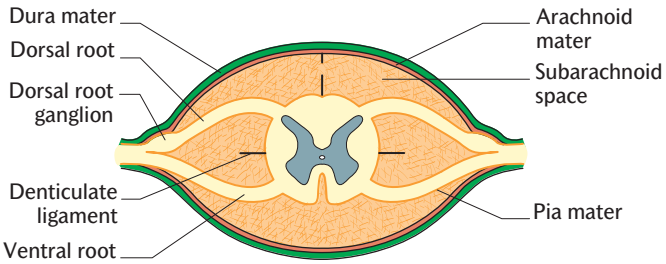


Fig. 15.5 A cross section of the spinal cord and covering meninges.

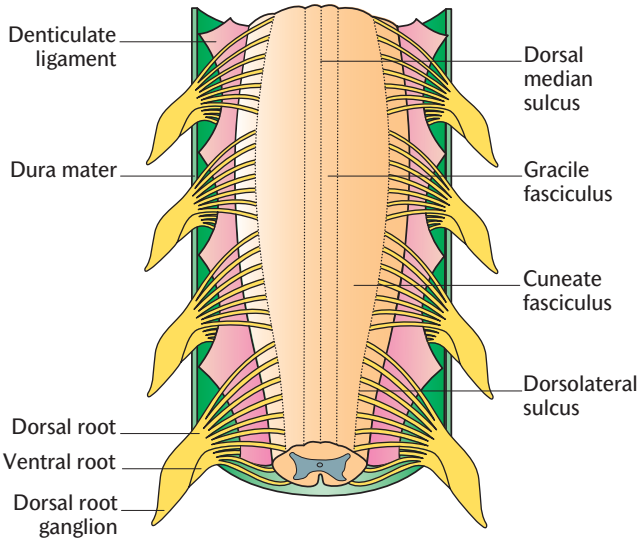


Fig. 15.6 The denticulate ligaments of the spinal cord. The dura mater dorsal to the cord has been removed.

between the pia and the arachnoid containing cerebrospinal fluid, with strands of arachnoid bridging the gap. The spinal cord is suspended within the subarachnoid space by the **denticulate ligaments** shown in Figure 15.6. These are linear structures which run from the upper cervical to the lumbar region on each side of the cord. They extend from the pia to the dura between the exits of successive spinal nerves. Tooth-like projections extend into the interval between the dorsal and ventral roots of the spinal nerves.

The consequences of meningeal infections are described in Box 15.1.

Box 15.1 Infections of the meninges

Inflammation of the pia and arachnoid mater is called **meningitis** and usually involves spinal as well as cerebral meninges. It is most frequently caused by bloodborne infections from microorganisms; infective organisms may also spread from a brain abscess or be introduced through a penetrating wound. The outstanding features of meningitis are headache, fever, rash, and the signs of spinal meningeal irritation (neck rigidity). Some of the organisms that cause meningitis are extremely virulent; meningitis can have an extremely rapid onset and may quickly become fatal unless diagnosed and treated urgently.

15.1.5 Nerve and blood supply of the dura mater

The dura of the anterior cranial fossa are supplied by branches of the ophthalmic divisions of the **trigeminal nerves**; **meningeal branches** of the maxillary and mandibular divisions of the trigeminal nerves supply the middle cranial fossa. The dura of the posterior cranial fossa is innervated by ascending branches from **the upper cervical spinal nerves** entering the cranial cavity through the foramen magnum. The meningeal nerves contain sensory and post-ganglionic sympathetic neurons. The latter may have a vasomotor function and have been implicated in migraine attacks.

The arteries to the dura mater mostly supply the endosteal layer. The principal supply to the dura above the tentorium cerebelli is through the **middle meningeal arteries** which are branches of the maxillary arteries (see Section 24.5.2). They enter the cranial cavity through the foramen spinosum on each side and run laterally across the floor of the middle cranial fossa between the endosteal and meningeal layers of the dura and divide into anterior and posterior branches; they groove the bones that they cross. The subtentorial dura is supplied by meningeal branches of the vertebral arteries (see p. 135). The effects of blood loss into the meninges are outlined in Box 15.2.

15.1.6 The venous sinuses

The venous sinuses have been referred to several times as they are formed between the endosteal and meningeal layers of the dura adjacent to bone and its reflections. The venous sinuses are wide venous channels draining blood from the brain, the bones of the braincase, and the meninges. The sinus walls are tough sheets of dura with no muscular tissue or valves lined by endothelium like any other blood vessel. Furthermore, they are held patent at all times because of their construction; the sinuses will, therefore, not collapse when blood pressure falls, e.g. in shock, meaning that perfusion of the brain should continue.

The arrangement of the venous sinuses can best be pictured by studying Figure 15.7 in conjunction with Figure 15.3 and 15.4.

The **superior sagittal sinus** receives blood from superior cerebral veins draining blood from the superior, lateral, and medial surfaces of the cerebral hemispheres. Venous blood drains backwards to the back of the skull. Figure 15.3 shows what happens at the point where the straight sinus and the superior sagittal sinus meet at a point marked by a slight bulge of bone, the internal occipital protuberance. The superior sagittal sinus usually turns to the right, but occasionally to the left, to become the transverse sinus of that side.

As illustrated in Figure 15.3, the **inferior sagittal sinus** running in the inferior free margin of the falx cerebri receives blood from the falx and ends by opening into the straight sinus formed where the falx attaches to the tentorium cerebella. The straight sinus drains the inferior sagittal sinus and also receives blood from the great cerebral vein which drains the deep parts of the cerebral hemispheres. Figure 15.4 shows that the sinus ends at the internal occipital protuberance by turning to become, in most cases, the left transverse sinus.

The **transverse sinuses** pass forward until they reach the junction of the petrous and mastoid parts of the temporal bone where they curve downwards to become the sigmoid sinuses. The sigmoid sinuses have

Box 15.2 Extradural and subarachnoid haemorrhage

Intracranial haemorrhage may occur either within or outside the brain. Intracranial haemorrhage occurring outside the brain may occur in the extradural, subdural, or subarachnoid space. The effects of haemorrhages inside the brain are described on in Box 15.10.

The **extradural space** is a *potential* space, located between the fused endosteal and meningeal layers of the dura mater. Haemorrhage into this space—**extradural haemorrhage**—is relatively uncommon and is usually the result of tearing of meningeal vessels or venous sinuses consequent upon a fracture of or heavy blows to the braincase. The meningeal vessels are particularly vulnerable to blows to the temple because the bone is relatively thin.

The subdural space is another potential space between the meningeal layer of the dura and the arachnoid mater. Bleeding into this space (**subdural haemorrhage**) is more common, usually due to tearing of one or more of the superior cerebral veins at the point where they enter the superior sagittal sinus. Although the cerebral veins run for most of their extracerebral course in the subarachnoid space, they cross the subdural space to reach the venous sinuses. In blows to the head, the brain may be suddenly moved enough to tear the cerebral veins near where they are fixed where they pass through the dura mater. Blood then enters the subdural space.

Extradural and subdural haemorrhages result in **raised intracranial pressure (ICP).** The principal clinical features are a consequence of brain compression caused by the raised ICP. The rate of onset is extremely variable. Essentially, blood forces its way into a very limited space to produce localized, but extremely intense, pressure which raises the ICP quite rapidly. This, in turn, results in **'coning'**, the compression of the medulla as it is displaced into the foramen magnum; coning compromises the functions of the cardiovascular and respiratory centres of the medulla with life-threatening consequences (see Box 3.4).

The **subarachnoid space** is a real space occupied by cerebrospinal fluid. Blood has room to spread when vessels haemorrhage into this space (**a subarachnoid haemorrhage**). Subarachnoid haemorrhages may follow the rupture of a congenital aneurysm of one or other of the arteries comprising the arterial circle (see Section 15.5.1) or the rupture of cerebral veins by a blow to the head with consequent bleeding into the subarachnoid space. Although ICP will rise eventually, the principal clinical features of subarachnoid haemorrhage are those of meningeal irritation described above because extravasated blood is a powerful irritant of neuronal tissues.

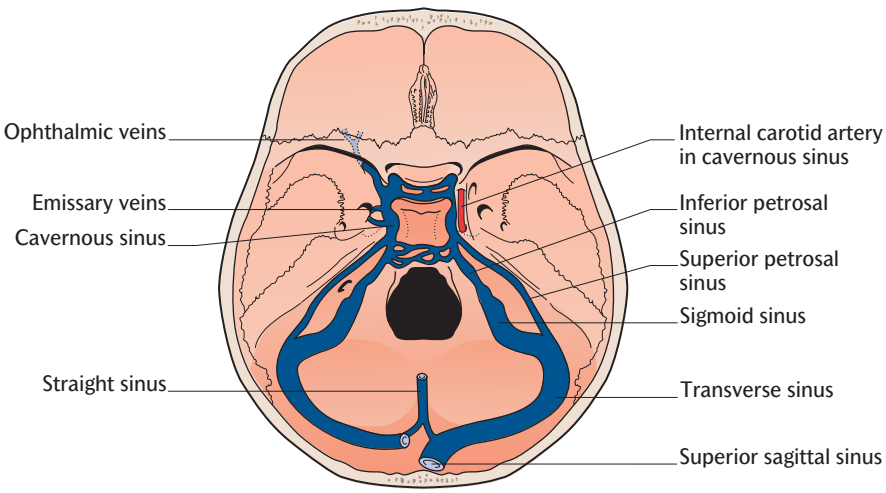


Fig. 15.7 The venous sinuses seen from above after removal of the tentorium cerebelli.

a shallow S shape, seen most clearly in Figure 15.7, as they continue downwards and forwards to the jugular foramina. They deeply groove the endocranial surface of the mastoid part of the temporal bone. The sigmoid sinuses expand to form the jugular bulbs in the posterior part of the jugular foramen; these are continuous with the internal jugular veins below.

15.1.7 The cavernous sinuses

Two important sinuses have not been encountered yet. These are the right and left **cavernous sinuses** on each side of the body of the sphenoid shown in Figure 15.7. If they are viewed in cross section, numerous fibrous partitions cross their cavities, partially dividing them into a series of small caverns—hence the name for these structures. Like most of the other sinuses, they lie between the endosteal and meningeal layers of the dura. The endosteal layer covers the lateral aspects of the body of the sphenoid as might be expected from the position of the cavernous sinuses. The meningeal dura extends laterally from the diaphragma sellae covering the pituitary fossa and then descends to the floor of the middle cranial fossa, forming the roof and lateral walls of the sinuses. Several important structures pass through or along the walls of the sinuses:

- The internal carotid arteries (see Section 15.6.1);
- The third, fourth, and sixth cranial nerves supplying the extraocular muscles that move the eyes (see Chapter 18);

Box 15.3 The venous sinuses and infection

The venous sinuses drain the cranial cavity whereas the structures in the head and neck lying outside the cranial cavity drain into conventional veins. The skin and immediately underlying tissues drain into superficial veins whereas deeper structures drain into the deep veins. The superficial and deep drainage of the head and neck will be described more fully in Section 23.2.2.

It is important to realize that the superficial, deep, and venous sinus components of the venous drainage are all interconnected. These connections can be a route for the spread of infection which can be particularly serious if it enters the cranial cavity where the meninges or venous sinuses may be infected. The cavernous sinus in particular has numerous connections with extracranial veins. The orbit drains through ophthalmic veins into the cavernous sinuses posteriorly, but also into the venous drainage of the face anteriorly; the two routes are connected. The ophthalmic veins also communicate with the pterygoid plexus of veins before entering the sinus; the pterygoid plexus receives venous blood from deep structures in and around both jaws, including most of the dental structures (see Section 24.5.3). Infection may thus spread from the face or from structures drained by the pterygoid plexuses into the cavernous sinuses. Blood flow through the cavernous sinuses is relatively slow because they are partitioned into small interconnected cavities, making it more prone to clot, producing an infected thrombosis. Thrombosis in the cavernous sinuses may produce a retrograde thrombosis into one of the middle cerebral veins that drain deep parts of the brain; retrograde thrombosis is usually fatal. It will also affect the cranial nerves passing through the cavernous sinus, leading to defective eye movements (see Box 18.3).

- The ophthalmic and maxillary divisions of the trigeminal nerve (see Chapter 18).

The cavernous sinuses receive blood from the lateral and inferior surfaces of the cerebral hemispheres and the ophthalmic veins from the orbit and drain via the superior and inferior petrosal sinuses that exit from the posterior part of the cavernous sinuses on each side (Figure 15.7). Each superior sinus runs along the superior border of the petrous temporal bone to enter the junction of the transverse and sigmoid sinuses. Each inferior sinus runs more or less directly downwards into the jugular foramen.

The potential spread of infection through venous sinuses and its consequences are described in Box 15.3.

15.2 The spinal cord

The spinal cord is oval in cross section, being flattened dorsoventrally as outlined in Figure 15.8. There is a general reduction in diameter from the cervical to the coccygeal region, but there are two enlargements where the thick spinal nerves supplying the limbs emerge. The **cervical enlargement** in the lower cervical and upper thoracic region is where the nerves forming the brachial plexus innervating the upper limb arise. The **lumbar enlargement** is the area where lumbar and sacral nerves form the lumbar and sacral plexuses to supply the lower limbs.

The external surface of the cord is shown from the dorsal aspect in Figure 15.6. A shallow **dorsal median sulcus** marks the midline and there are shallow **dorsolateral sulci** laterally. Figure 15.8 indicates the corresponding indentations on the ventral aspect; the **ventral median fissure** is obvious, but the **ventrolateral sulci** are shallow. Thirty-one bilaterally paired **spinal nerves**—eight **cervical**, 12 **thoracic**, five **lumbar**, five **sacral**, and one **coccygeal**—are attached to the cord. Each nerve is formed by the fusion of a dorsal and ventral root. Each root is formed from the confluence of a series of rootlets entering or leaving the corresponding segment of the cord. Dorsal roots exit along the dorsolateral sulcus (see Figure 15.6) and ventral roots along the ventrolateral sulcus. The structure of spinal nerves has already been described in Chapter 3; their relationship to the spinal cord and vertebrae has also been covered above and illustrated in Figure 3.5.

15.2.1 Internal structure

As shown in Figure 15.6 and 15.8, there is a clear distinction between the grey and white matter in a transverse section of the spinal cord which can be seen with the naked eye on anatomical preparations of the spinal cord. The grey matter is H-shaped in transverse section and forms a continuous mass throughout the length of the spinal cord; grey matter contains neuronal cell bodies, dendrites, and synapses. The crossbar of the H is the **central commissure** formed by neuronal processes connecting the right and left halves of the spinal cord. Look carefully at Figure 15.8 and you will see a narrow **central canal** centrally in the commissure; the canal is continuous with the ventricular system of the brain.

Box 15.4 Lumbar puncture

The cauda equina is surrounded by subarachnoid space. When a sample of cerebrospinal fluid is required for examination, it is usually taken from this region by means of a **lumbar puncture.** A needle is inserted between the arches of the third and fourth lumbar vertebrae and advanced until its tip enters the subarachnoid space. Inserting the needle at this level avoids any possibility of damaging the spinal cord and the nerves forming the cauda equina will float away from the needle. The same site is used for **epidural injections** that may be used to alleviate pain during childbirth.

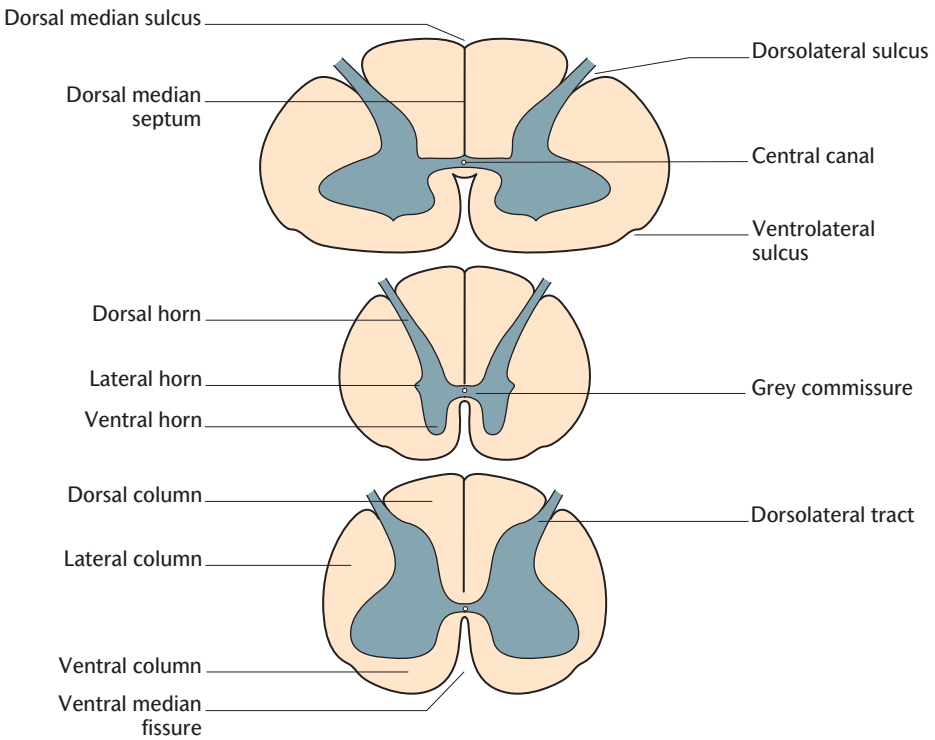


Fig. 15.8 Transverse sections of the spinal cord in the cervical (above), thoracic (middle), and lumbar (below) regions.

The grey matter

The **dorsal** and **ventral horns** of the grey matter project dorsally and ventrally on each side. The dorsal and ventral horns form continuous columns throughout the length of the spinal cord. Figure 15.8 also indicates the position of the small **intermediolateral** (or **lateral**) **horns** between the junction of the dorsal and ventral horns on each side; they only extend from the upper thoracic to the upper lumbar region. As already described in Chapter 3, the dorsal horns are the site of termination of sensory neuronal processes and the ventral horns are the location of the cell bodies of motor nerves and the origin of their axons. The lateral horns are the site of the cell bodies of preganglionic sympathetic axons distributed via the ventral roots of the thoracic and upper lumbar spinal nerves.

The grey matter within the dorsal, ventral, and intermediolateral horns can be subdivided microscopically into layers (the **laminae of Rexed**) on the basis of the cell types and density that constitute each layer. These laminae are shown in Figure 15.9. Laminae I to III from the tips of the dorsal horns inwards are where many incoming primary sensory neurons terminate; neurons terminating in these laminae carry mainly temperature and nociceptive information. Laminae IV to VI, forming the deeper part of the dorsal horns, contain the cell bodies of secondary thalamic projection neurons. The primary and thalamic projection neurons have to be connected to form efficient ascending sensory pathways. This is achieved by interneurons connecting different layers within the dorsal horns. In addition, many thalamic projection neurons have comparatively long dendrites that extend from their cell bodies in the deeper laminae to synapse with the axons of primary sensory neurons entering the upper layers (see Section 16.2).

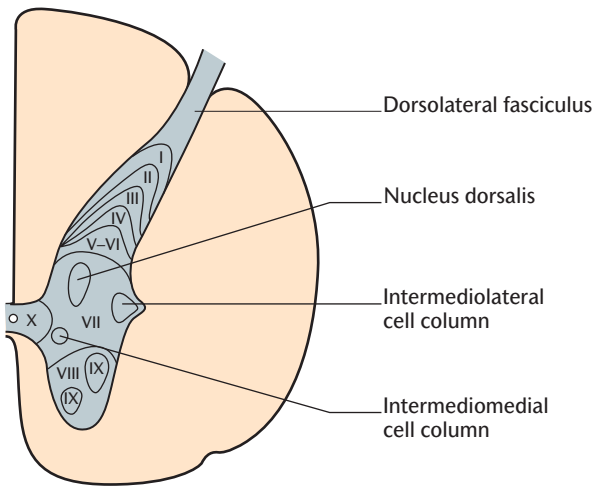


Fig. 15.9 A transverse section of the spinal cord in the thoracic region showing the laminae of the grey matter.

Lamina VII is, by far, the biggest layer. It contains numerous interneurons, but also two important columns of neurons. The intermediolateral cell column contains the cell bodies of autonomic preganglionic neurons. The nucleus dorsalis or Clarke's column is the site where primary proprioceptive neurons synapse with neurons whose axons form the spinocerebellar tracts. Lamina VIII forms the major part of the ventral horns and encloses many groups of large lower motor neuron cell bodies; these groups constitute lamina IX. Lamina VIII is the area in which

descending motor pathways synapse with the dendrites of lower motor neurons found in lamina IX. Lamina X is the central commissure.

The white matter

The white matter consists principally of nerve axons and neuroglia. Figure 15.8 shows its arrangement into three columns on each side incompletely separated by the dorsal and ventral horns. The **dorsal columns** lie between the dorsal grey horn and the posterior median septum, the **lateral columns** are between the dorsal and ventral horns, and the **ventral columns** are between the ventral horns. Note from Figure 15.8 that there is no clear demarcation between lateral and ventral columns anteriorly.

Neuronal processes forming the white matter are aggregated into microscopically distinct tracts with separate functions. Such tracts may be ascending sensory tracts connecting the spinal cord to the brain or descending motor pathways connecting the brain to the cord; other tracts connect different segments of the spinal cord. The distribution and function of these tracts is described in Chapter 16.

15.3 The brainstem

The brainstem is continuous with the spinal cord and contains the upward continuations of many of the structures present in the cord or their equivalents. All the major tracts in the white columns of the spinal cord run through at least part of the brainstem to reach their destinations.

The principal differences between the brainstem and the spinal cord are:

- The grey matter in the brainstem is broken up into discrete nuclei related to the origins of each pair of cranial nerves whereas it forms a single continuous mass in the spinal cord;
- The central canal, which is narrow throughout the cord, is greatly expanded in the middle region of the brainstem to form the fourth ventricle;
- The cranial nerves emerge from the brainstem in a less regular pattern than that in which the spinal nerves leave the cord because the proportion of motor, sensory, and autonomic components differ radically in different cranial nerves;
- The cerebellum is attached to the posterior aspect of the brainstem by three pairs of peduncles.

The brainstem is divided into three parts. As shown in Figure 15.10, they are, in ascending order, the **medulla oblongata, pons**, and **midbrain**. The brainstem lies on the basilar part of the occipital bone and the body of the sphenoid anteriorly; the cerebellum is posterior. Revisit Figure 15.1 to recall that the medulla and pons are situated in the posterior cranial fossa and the midbrain lies in the middle cranial fossa. As outlined in Figure 15.11, the **fourth ventricle** is located posterior to the upper part of the medulla and the lower pons. The fourth ventricle narrows above to enter the **cerebral aqueduct** as it passes through the midbrain to the third ventricle in the cerebrum.

To place the fourth ventricle and cerebral aqueduct in context, the ventricles are a series of chambers within the brain in which cerebrospinal fluid (CSF) is formed and circulates. The first three ventricles are in the cerebrum and connect to the fourth ventricle in the brainstem by the cerebral aqueduct. The fourth ventricle is continuous below with the central canal of the spinal cord. CSF leaves the ventricular system to surround the brain through foramina in the lining of the fourth ventricle. Eventually, CSF is resorbed back into the intracranial venous system. The ventricular system is described more fully on p. 131.

If you turn Figure 15.10 upside down, the pons and medulla together look like a flower bulb, with the pons forming the bulb and the medulla being the shoot. This resemblance accounts for the old name for the brainstem, the **bulb**. The tracts passing from the cerebral cortex to the motor nuclei of the cranial nerves are now called the corticonuclear tracts, but they are also referred to as the corticobulbar tracts. Damage to the corticonuclear tracts produces a condition known as corticobulbar palsy, a term still in clinical use.

15.3.1 External features

The medulla

The junction of the spinal cord and medulla is at the level of the upper rootlet of the first cervical spinal nerve, corresponding to the upper border of the atlas. This is an arbitrary dividing line since the transition in internal structure is not abrupt; in fact, the internal structure of the lower part of the medulla closely resembles that of the cervical region of the spinal cord. The upper limit of the medulla is at its junction with the pons and is clearly marked on the ventral surface of the brainstem. The medulla is about 3 cm long and becomes progressively wider superiorly; it forms a narrow cone with its base on the pons and its 'apex' running into the spinal cord.

Figure 15.10 shows the anterior surface of the brainstem. The **ventral median fissure** and **ventrolateral sulci** continue upwards from the spinal cord on to the medulla. The **pyramids** are elongated swellings on each side between the ventral median fissure and the ventrolateral sulcus. The pyramids are produced by corticospinal axons passing through the medulla close to its ventral surface from the cerebral cortex to the ventral horns of the spinal cord. In the upper part of the medulla, a second elongated swelling, the **olive**, lies laterally to the ventrolateral sulcus on each side. Each bulge is caused by the underlying **inferior olivary nucleus** which is involved in circuits linking motor pathways to the cerebellum (see Section 16.3.3).

The cranial nerves all have names and are also numbered using Roman numerals in the order in which they arise from the brain. The lowest numbered nerves arise most anteriorly from the cerebral hemispheres and the highest numbered from the medulla. As we are exploring the brainstem by ascending from the spinal cord, the highest numbered cranial nerves will be encountered first. Figs 15.10 shows the **hypoglossal nerves** (cranial nerves (CN) XII) leaving the medulla between the pyramid and olive on each side as a group of rootlets. The **glossopharyngeal** (CN IX) and **vagus nerves** (CN X) emerge as

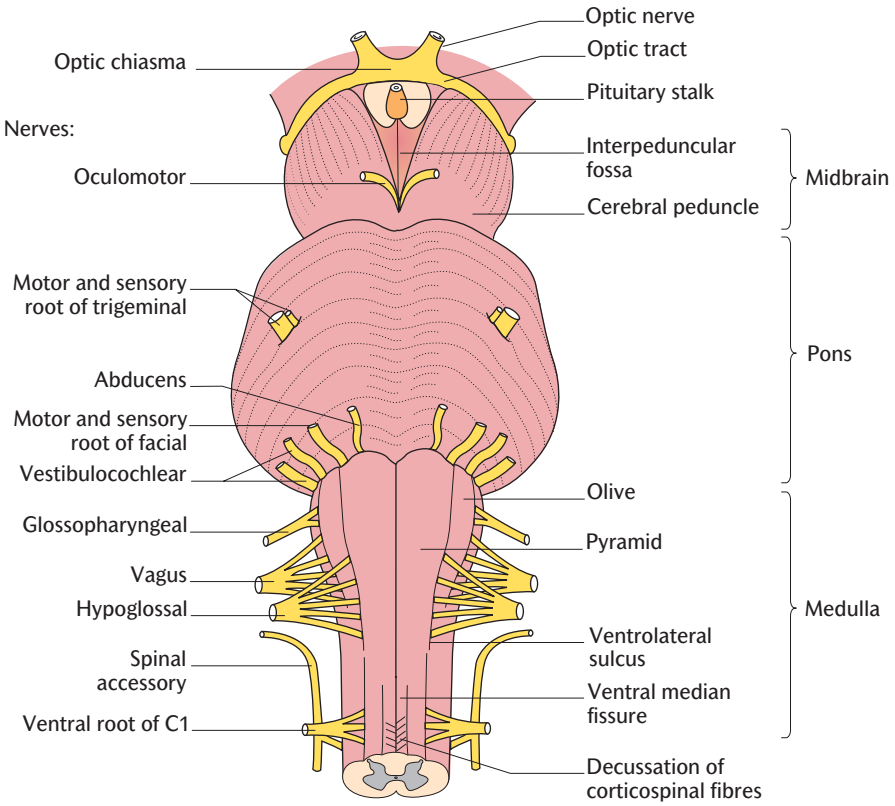


Fig. 15.10 An anterior view of the brainstem.

rootlets lateral to the olive; the ninth rootlets are superior to those of CN X. These rootlets join together to form their specific nerves as they pass through the jugular foramina to exit the skull.

The dorsal surface of the medulla is usually hidden by the overlying cerebellum. Figure 15.11 shows the appearance with the cerebellum removed. The **dorsal median sulcus** can be seen running upwards from the cord on to the dorsal surface of the medulla. The medial **gracile** and lateral **cuneate fasciculi** form bulges on either side of the sulcus. The fasciculi terminate as low swellings, the **gracile nucleus** and **cuneate nucleus**. These fasciculi carry ascending sensory nerve processes from the spinal cord to the two nuclei where they synapse (see Section 16.2.2).

The dorsal surface of the medulla opens out where the inferior part of the roof of the fourth ventricle is situated. This roof is an extremely thin layer of ependymal cells covered by a delicate layer of pia mater; it is almost invariably torn during the removal of the cerebellum to reveal the floor of the ventricle as in Figure 15.11.

The pons

The pons is about 2 cm long and its borders with the medulla below and midbrain above are clearly marked by horizontal grooves. When the brainstem is viewed from its anterior aspect in Figure 15.10, the pons has the appearance of a bridge connecting the right and left cerebellar hemispheres (hence its name from the Latin: pons = bridge). Laterally, the pons is continuous with the cerebellar hemispheres through the middle cerebellar peduncles. The cerebellum also receives cell processes from the cerebrum and from the spinal cord through the superior and inferior cerebellar peduncles, respectively. As you can see in Figure 15.11, each inferior cerebellar peduncle lies medial to the middle peduncle and the superior peduncles lie above it. The posterior surface of the pons forms the superior part of the floor of the fourth ventricle.

Figure 15.10 shows the exit points of the cranial nerves associated with the pons. The **abducens nerves** (CN VI) exit at the junction of pons and medulla and run vertically up the pons. The **facial** (CN VII) and **vestibulocochlear** nerves (CN VIII) leave the brainstem more laterally between the pons, medulla, and cerebellum at the **cerebellopontine angle**. The facial nerve is attached to the brainstem by a large motor and a small sensory root, the **nervus intermedius**, which lies between the motor root and the vestibulocochlear nerve. The **trigeminal nerves** (CN V) leave the ventral surface of the pons. Each trigeminal nerve also has motor and sensory roots; the sensory root is the largest and lies inferior to the motor root.

The midbrain

The midbrain connects the pons to the cerebrum. It is less than 2 cm in length and is approximately cylindrical in cross section. The midbrain is difficult to see on an intact brain as it is hidden by the cerebral hemispheres above and anteriorly and the cerebellum posteriorly. Its position as an upward continuation of the pons can be appreciated on a hemisection of the brain (see Figure 3.7B). The narrow central canal, the **cerebral aqueduct**, linking the third and fourth ventricles is visible in a hemisection.

As seen in Figure 15.10, two prominent vertical bulges form most of the anterior surface. These are the **cerebral peduncles** and contain the major ascending and descending tracts passing between the spinal

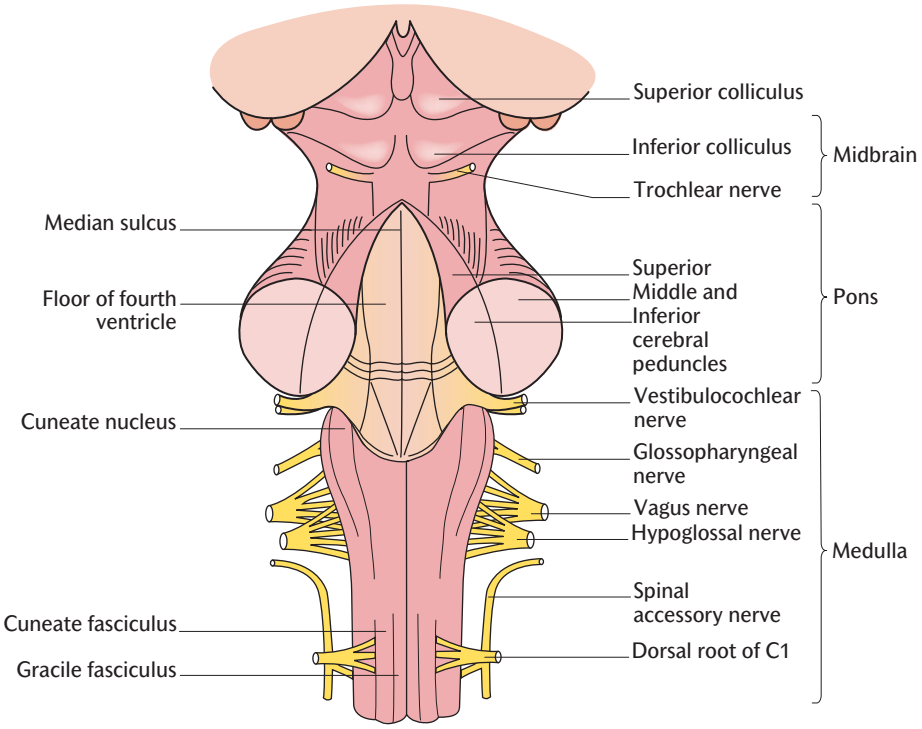


Fig. 15.11 A posterior view of the brainstem with the cerebellum removed.

cord and lower brainstem to the cerebrum and vice versa. The cerebral peduncles are partially separated by a deep midline depression, the **interpeduncular fossa**, as they diverge to enter the right and left hemispheres. On the dorsal surface illustrated in Figure 15.11, two pairs of swellings, the **superior** and **inferior colliculi**, can be seen. These contain nuclei concerned with visual and auditory reflexes, respectively. The **superior cerebellar peduncles** run downwards from the midbrain to the cerebellum.

Figure 15.10 shows the **oculomotor nerves** (CN III) leaving the ventral surface of the midbrain from the depths of the interpeduncular fossa. The **trochlear nerves** (CN IV) also originate in the midbrain, but they are the only cranial nerves to emerge from the *dorsal* surface of the brainstem. As shown in Figure 15.11, they arise immediately below the inferior colliculi and continue round the lateral aspect of the midbrain.

The fourth ventricle

The fourth ventricle is the expanded central canal of the brainstem in the region of the upper medulla and pons. The cavity has expanded posteriorly so that the nervous tissue lies anterior to the ventricle. The roof of the ventricle is formed by a thin layer of ependyma. The ventricle is broadest in its middle region and narrows sharply superiorly into the aqueduct and inferiorly into the central canal of the lower medulla. As you can see from Figure 15.11, the floor of the fourth ventricle is, therefore, diamond-shaped when viewed from above. The inferior ventricular roof is pierced by three apertures—a large **median aperture** (the foramen of Magendie) and two smaller **lateral apertures** (the foramina of Luschka). CSF passes from the ventricular system into the subarachnoid space through these apertures.

15.3.2 Internal features of the brainstem

The components of the brainstem can be divided into four groups:

- The **ascending and descending pathways**, many of which are continuations of the tracts in the spinal cord and together make up the bulk of the white matter in the brainstem (see Chapter 16);
- The **nuclei** of CN III–XII (see Chapter 18).
- Other named nuclei such as the **red nucleus, substantia nigra**, and **inferior olivary nucleus** (see Chapter 16);
- The **reticular formation** occupying those parts of the brainstem not occupied by the named nuclei or tracts (see Chapter 16).

The majority of these structures can only be seen on microscopical sections of the brainstem. Only the red and inferior olivary nuclei and the substantia nigra can be seen with the naked eye.

The medulla

A cross section of the medulla passing through the olives is shown in Figure 15.12. The **inferior olivary nuclei** may be visible with the naked eye, but a magnifying glass shows them more clearly. Each nucleus occupies the area beneath the olives seen on the external surface of the brainstem. In section, the nuclei look like a crumpled bag with their openings facing posteromedially. The inferior olivary nuclei receive descending inputs from the cerebral cortex, the red nucleus, and other areas of the midbrain. Axons from each nucleus cross the midline to reach the cerebellar hemisphere of the opposite side through the inferior cerebellar peduncle. These pathways are involved in the mechanisms used to learn motor skills (see Section 16.3.3).

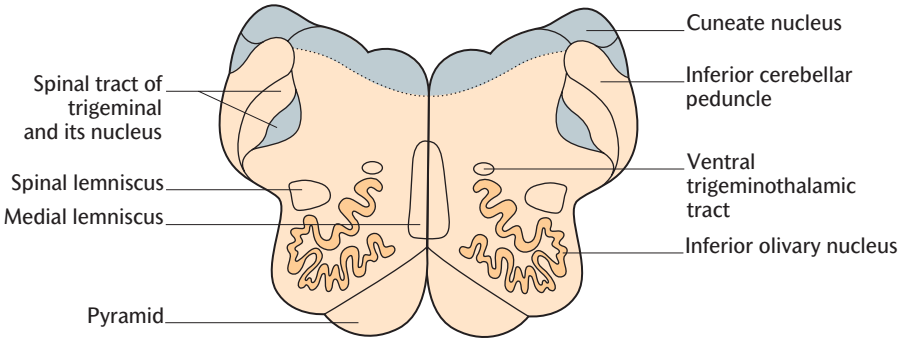


Fig. 15.12 A transverse section through the medulla.

Figure 15.12 illustrates the position of the pyramids immediately adjacent to the midline and medial to the inferior olivary nucleus on each side. They extend virtually the whole length of the medulla and are separated from each other by the ventral median fissure. The pyramids contain corticospinal axons descending from the motor cortex of the cerebral hemisphere of the same side. At the lower end of the pyramid, most of these axons cross sides in the **pyramidal decussation** before continuing downwards into the spinal cord. The ventral median fissure becomes less marked at the decussation.

The pons

The three paired cerebellar peduncles connect the dorsolateral aspect of the pons to the cerebellum. The **inferior peduncles** leave the inferior part of the pons and carry dorsal spinocerebellar (proprioception), olivocerebellar (motor learning), and vestibulocerebellar tracts (balance) to the cerebellum. The **superior peduncles** leave the superior part of the pons and adjacent midbrain and carry neurons from the cerebellum to the red nucleus, thalamus, and cerebral cortex. The large **middle peduncles** are lateral to the inferior and superior peduncles and connect the pontine nuclei to the cerebellum.

In a cross section of the pons, it is possible to see conspicuous bundles of nerve processes as shown in Figure 15.13. Some run longitudinally, but the most obvious bundles run transversely. The longitudinal bundles are descending corticospinal and corticonuclear pathways. They give off numerous collateral corticopontine axons that synapse with neurons in the **pontine nuclei**; these are scattered between the axon bundles and are too small to see with the naked eye. The axons from the neurons in the pontine nuclei form the prominent transverse **pontocerebellar bundles** illustrated in Figure 15.13. These bundles cross the midline and enter the cerebellum through the contralateral middle cerebellar peduncle. Corticopontine axons begin in many areas of the cerebral cortex and play a part in the control of precision of voluntary movements through their connections with the cerebellum (see Section 16.3.3).

The midbrain

A cross section of the midbrain is illustrated in Figure 15.14; note how it is divided into the relatively narrow **tectum** posterior to the cerebral aqueduct and the large **cerebral peduncles** anterior to the aqueduct. A thin band of darkly pigmented granular grey matter, termed the **substantia nigra**, can usually be seen clearly in each peduncle. The **substantia nigra** is functionally part of the basal nuclei (see p. 131) and has inputs and outputs to and from other basal nuclei. This system is involved in the regulation of motor activity (see Section 16.3.4). The substantia nigra uses the neurotransmitter **dopamine**; the dark colouration is produced by granules that accumulate in certain of its cells as a by-product of dopamine synthesis.

The substantia nigra describes a shallow crescent and the red nuclei sit in the concavity on each side. The **red nuclei** can be seen as a pink area contrasting with the white matter in a fresh section of the brainstem; the colour quickly fades and the nuclei become indistinguishable, except from the demarcation of their position by the substantia nigra. The red nuclei receive inputs from the cerebral motor cortex and the cerebellum. A large number of output passes from each red nucleus to form the rubrospinal motor tracts and others connect the red and inferior olivary nuclei.

The paired inferior and superior colliculi are clear bumps on the posterior aspect of a cross section of the midbrain. The **inferior colliculi**

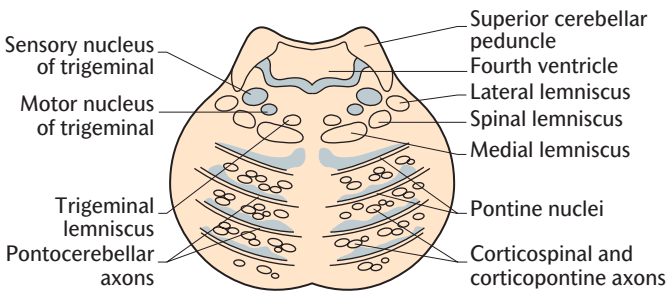


Fig. 15.13 A transverse section through the middle of the pons.

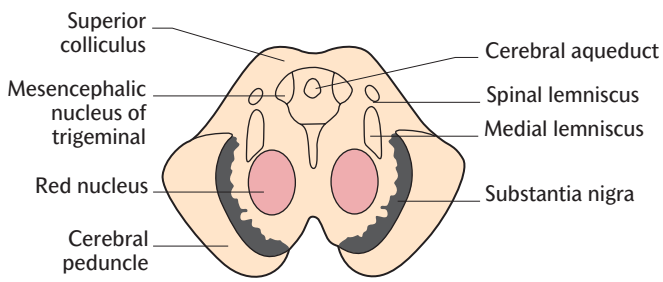


Fig. 15.14 A transverse section through the upper part of the midbrain.

are relay stations in the auditory pathway from the cochlear nuclei in the medulla to the primary auditory area of the cortex in the temporal lobe (See Section 18.8.1). These colliculi are involved with reflex coordination of the eye, head, and body movement towards the source of sounds, in particular, loud, unexpected ones. If you are sitting in a café with your friends and a waiter drops a tray, you will all look up and turn towards the source of noise without even realizing it; those with their backs to the incident will have turned their heads or bodies as required.

The section illustrated in Figure 15.14 passes through the **superior colliculi**. These nuclei are involved in reflex response to visual stimuli and receive inputs from collateral axons branching from those forming the visual pathway connecting the eyes to the visual cortex (see Section 18.4.1). They also receive inputs from the visual association cortex surrounding the visual areas of the occipital lobes of the cerebral hemispheres. Their outputs pass bilaterally to connect with the motor nuclei of the oculomotor, trochlear, abducens cranial nerves that control eye movement. They also connect to motor nuclei of the facial nerves to form the efferent part of the reflex that closes the eyes tight when faced with really bright light. Axons also go to the opposite side of the cervical spinal cord to control head movement so that objects can still be followed as they move out of the visual field.

The reticular formation

In primitive vertebrates, a large part of the brain is made up of a diffuse network of neurons, termed the reticular formation (from the Latin: reticulum = net or network). During the course of evolution, the forebrain has developed enormously to give rise to the cerebral hemispheres and diencephalon. Brainstem structures associated with the enlarged forebrain, such as the ascending and descending tracts and many of the large nuclei, occupy an increasing proportion of its volume until the reticular formation occupies only those areas of the brainstem not taken up by the tracts and nuclei. The reticular formation is a vital component of the mammalian brain and has connections with all levels of the central nervous system. In the human brain, a considerable portion of the dorsal part of the brainstem is occupied by the reticular formation. Its cells have unusually long dendrites which extend widely through the brainstem, making numerous connections with other reticular cells and with neurons in non-reticular structures. The reticular system is involved in:

- Control of voluntary muscle action (Chapter 16);
- Determination of the level of consciousness and arousal of cerebral cortex;
- Perception of pain (Chapter 16);
- Control of visceral function (Chapter 17).

Reticulospinal tracts play an important part in controlling motor function. They receive direct and indirect inputs from other parts of the reticular formation, the thalamus, cerebellum, and cerebral motor cortex and their outputs project to the ventral horns of the spinal cord (see also Chapter 16). Ascending nociceptive pathways have inputs into reticular nuclei. These function in the modulation of nociceptive information through their connections with the thalamus and periaqueductal grey matter around the cerebral aqueduct in the midbrain illustrated in Figure 15.14 (see Chapter 16).

Collections of neurons within the reticular formation are specifically associated with visceral functions; these neurons do not aggregate to form definitive nuclei so they are called 'centres'. The **respiratory centre** regulates rhythm and depth of inspiration and expiration. The **cardiovascular centre** regulates heart rate, cardiac output, and other variables. Both centres are in the medullary part of the reticular system.

15.4 The cerebellum

As you can see from Figure 15.15, the cerebellum lies posterior to the brainstem. It is connected to the different parts of the brainstem by the inferior, middle, and superior cerebellar peduncles as described on p. 124. Its upper surface is separated from the underside of the occipital lobes of the cerebrum by the tentorium cerebelli. As illustrated in Figure 15.15, the cerebellum is highly folded into the folia, a series of narrow transverse folds separated by narrow fissures; the folding greatly increases its surface area. It consists of two hemispheres joined in the midline by an area called the vermis.

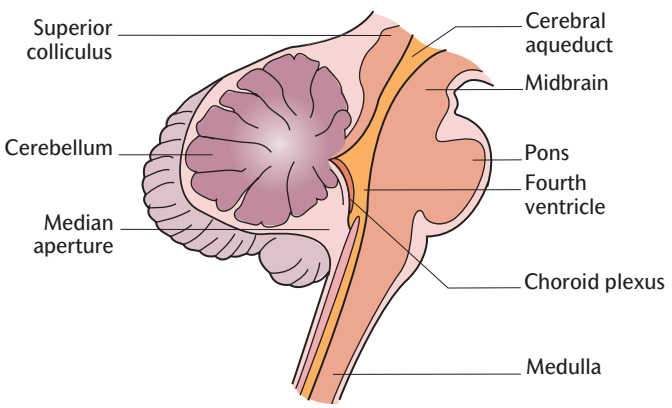


Fig. 15.15 A coronal section through the cerebellum and brainstem.

The cerebellum has a key role to play in motor activities by:

- Maintaining equilibrium (balance);
- Influencing posture;
- Influencing muscle tone;
- Coordinating movement;
- Its role in learning complex motor movements.

The cerebellum receives inputs from ascending spinocerebellar tracts carrying proprioceptive information from muscles and joints, from the inferior olivary nuclei that are involved in motor learning, from the pontine nuclei conveying information from the cortex and vestibular system. Its main outputs are to the red nuclei in the motor

pathways, the motor cortex, and the vestibulospinal tracts (see Chapter 16). Essentially, the cerebellum receives balance and proprioceptive information that it computes to establish the position of the body and head, the state of contraction of muscles, and the position of joints. When motor activities are initiated, the motor cortex sends signals to the cerebellum about the muscles it needs to recruit and the cerebellum modifies the actions of these muscles by feeding information back to the motor cortex or directly on to lower motor neurons. Box 15.5 introduces the consequences of cerebellar dysfunction.

Box 15.5 Cerebellar dysfunction

The cerebellum may be damaged by head injury, alcoholic intoxication, degenerative diseases affecting the cerebellum, and its inputs or generalized degenerative diseases such as multiple sclerosis. A patient with cerebellar damage characteristically has slurred speech (**dysarthria**) and a wide-based staggering gait (**cerebellar ataxia**). Further detail of cerebellar dysfunction is given in Chapter 16.

15.5 The cerebrum

The anterior part of the neural tube has three enlargements during its early stages of development. The most anterior **prosencephalon** gives rise to the cerebrum, the **mesencephalon** produces the midbrain, and the hindbrain (medulla and pons) develops from **rhombencephalon** posteriorly. As the prosencephalon continues its development, it becomes subdivided into the anterior **telencephalon** that rapidly enlarges bilaterally to form the cerebral hemispheres and a posterior **diencephalon**; this does not expand so much, but its lateral walls become thickened to form the thalamus and hypothalamus. The posterior **pituitary gland** (neurohypophysis), optic pathways, and the retinae are also derived from the diencephalon. The development of the CNS will be described in more detail in Chapter 19.

15.5.1 The diencephalon

The diencephalon comprises, on each side, the **thalamus**, the largest component of the diencephalon, the **subthalamus**, and inferiorly the **hypothalamus**. These structures are found close to the midline at the base of the cerebrum just above the midbrain. The thalamus is an important relay station in the sensory pathways en route from the spinal cord and lower brainstem to the cerebral cortex. It also forms important relays between different components and areas of the cerebral hemispheres. The subthalamus is located ventral to the thalamus and is functionally part of the basal nuclei. The hypothalamus is the principal brain area for the control of visceral function. These features are illustrated in Figure 15.16.

The thickening of the lateral walls of the diencaphalon during their development compresses its central cavity into a median, slit-like cavity, the **third ventricle** seen in Figure 15.16. The third ventricle communicates on each side with the two lateral ventricles, one within each cerebral hemisphere, through the **interventricular foramina.**

As shown in Figure 15.16, the anterior wall of the third ventricle is formed by the **lamina terminalis**. This represents the extreme front end of the neural tube before the development of the cerebral hemispheres. It links the developing right and left cerebral hemispheres and during their development, bundles of nerve processes from each hemisphere form tracts connecting the two sides; these are **commissures**. The **corpus callosum** is the major commissural structure and sits above the roof of the third ventricle. It connects the frontal, parietal, and occipital lobes of both hemispheres. The **anterior commissure** is a small bundle of commissural axons in the lamina terminalis, linking the left and right temporal lobes. The floor of the third ventricle contains the **optic chiasma** where the two optic nerves join (see Section 18.4).

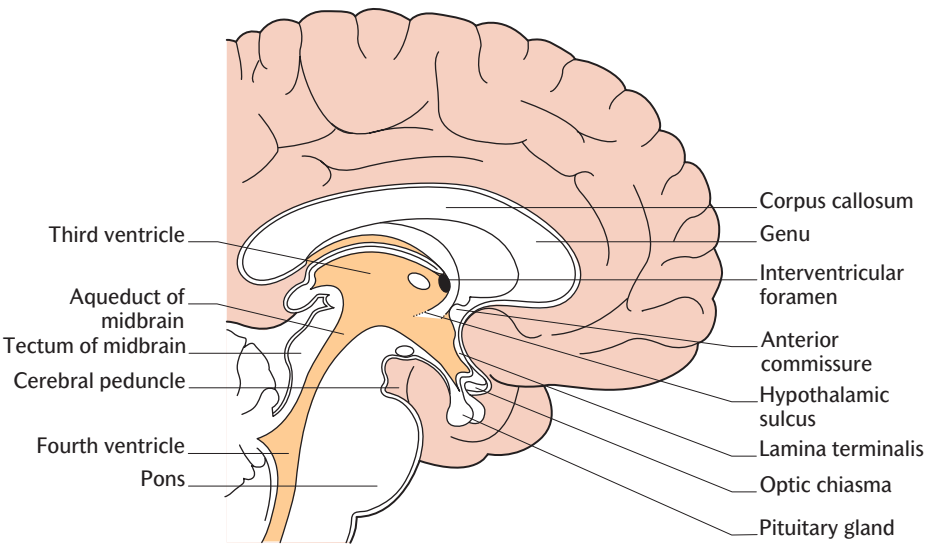


Fig. 15.16 A median section through the diencephalon.

The thalamus

Technically, there are two thalami, one on each side of the midline, but they are usually referred to as one structure. Each side of the thalamus is a large ovoid nucleus of grey matter, forming the lateral wall of the third ventricle.

Each half of the thalamus is divided into three main parts by an internal sheet of white matter which is almost impossible to see with the naked eye. The white matter consists of axons connecting different thalamic regions. Several individual thalamic nuclei can be distinguished microscopically within the three main parts of the grey matter on the basis of their position, cellular structure, and connections to other parts of the CNS. Some are relevant to the understanding of sensory and motor pathways and will be considered in Chapter 16. The thalamus sends outputs to all parts of the cerebral cortex. Reciprocal inputs run from the cortex to the thalamus. In addition, the thalamic nuclei receive inputs from subcortical regions of the brain.

The hypothalamus

The hypothalamus is about the size of a fingernail on a hemisection of the brain and surrounds the lower part of the third ventricle. It plays a critical role in the control of visceral function despite its relatively small size. As indicated in Figure 15.16, the boundary between the thalamus and hypothalamus is marked on the lateral wall of the third ventricle by the shallow **hypothalamic sulcus**, but this can be very difficult to see.

In Figure 15.16, the **pituitary (hypophysial) stalk** is seen connecting the inferior surface of the hypothalamus to the pea-sized endocrine **pituitary gland** (or **hypophysis cerebri**) sitting in the pituitary fossa of the sphenoid bone. The gland consists of two distinct parts—the **posterior pituitary** (or **neurohypophysis**) posteriorly is developmentally a downgrowth of the diencephalon whereas the anterior hypophysis (or **adenohypophysis**) is derived from an outgrowth of the oral ectoderm from the roof of the mouth. If the brain is removed from the skull, the pituitary gland is retained in the pituitary fossa by the diaphragma sellae but the stalk may adhere to the hypothalamus. In many dissection schedules, the head is bisected in the sagittal plane. The pituitary stalk might be seen connecting the gland and the hypothalamus if the head is cut right in the midline but is often on one side. The smaller neurohypophysis can usually be distinguished from the larger adenohypophysis; the anterior pituitary is reddish whereas the posterior pituitary appears paler because it is made of neuronal processes.

The hypothalamus receives inputs carrying information from the viscera through poorly defined pathways within the CNS; many of them relay in the brainstem and thalamus. The hypothalamus also receives information from the olfactory cortex, the thalamus, and the hippocampus which is part of the limbic system (see p. 134). The hypothalamus also monitors the properties of circulating blood, including temperature and levels of nutrients and hormones. This input is particularly important in the control of body temperature and of the release of hormones by the pituitary. The hypothalamus has direct nerve pathways connecting to autonomic cranial nerve nuclei in the brainstem and the intermediolateral horns of the spinal cord.

Pituitary hormones control many functions in the body and also control most of the other endocrine glands in the body. The hypothalamus synthesizes two hormones. **Vasopressin** (antidiuretic hormone) acts on the kidneys where it plays a part on water regulation and acid–base balance. **Oxytocin** stimulates uterine smooth muscle contraction during childbirth and also ejects milk from the breast during suckling. These hormones are actually synthesized by neurons; their cell bodies are in the hypothalamus and their axons pass in the hypothalamo**-**hypophysial tract to form the neurohypophysis. Vasopressin and oxytocin are stored intraneurally and eventually released into the bloodstream in response to stimulation of the nerves that synthesize them.

Other hypothalamic neurons produce **releasing factors** which are passed into capillaries on the underside of the hypothalamus. Blood from these capillaries passes through veins forming the pituitary portal system around the pituitary stalk; these open into a second set of capillaries in the adenohypophysis. Releasing factors stimulate or inhibit the secretion of hormones by the endocrine cells of the adenohypophysis. Releasing factor production is influenced partly by nervous factors, but more importantly, by the blood levels of the hormones produced by the target organs. For example, if the blood level of thyroid hormones is low, the production of thyrotrophin releasing factor by the hypothalamus is increased which, in turn, stimulates increased secretion of thyrotrophin (thyroid-stimulating hormone) by the adenohypophysis and so increases the production of hormone by the thyroid gland. As the level of thyroid hormone increases, the production of thyrotrophin releasing factor in the hypothalamus decreases. Control of thyroid hormone secretion is an example of a negative feedback mechanism.

15.5.2 The cerebral hemispheres

The cerebral hemispheres are very enlarged in all mammals, but especially in humans where they make up by far the greater proportion of the brain. Each hemisphere contains a cavity, the **lateral ventricle**, which communicates with the third ventricle in the diencephalon through the **interventricular foramina** (Figure 15.16). The two hemispheres are partially separated from each other by a deep median cleft called the **longitudinal cerebral fissure.** The falx cerebri extends into this cleft. The **corpus callosum** at the bottom of the longitudinal cerebral fissure connects the left and right hemispheres.

The outer layer of the hemisphere or **cerebral cortex** is a thin rim of grey matter and surrounds a central mass of white matter. The white matter is made up of axons of:

- **Projection neurons**, forming ascending and descending tracts connecting the cortex with structures lower in the CNS;
- **Association neurons** connecting one part of the cortex on the same side with another;
- **Commissural neurons** connecting the two hemispheres.

The **basal nuclei** (or **ganglia**) are a number of large subcortical masses of grey matter buried within the central white matter.

The external surfaces

Each cerebral hemisphere has superolateral, medial, and inferior surfaces. These surfaces of each hemisphere are extensively folded to form wide irregular ridges, the **gyri** (singular = **gyrus**), separated by deep furrows, the **sulci** (singular = **sulcus**). This folding greatly increases the total volume of the cerebral cortex. The anatomical location of some

gyri and sulci is constant from one brain to another and most are named. Only the names of sulci and gyri that have anatomical or functional significance for the study and practice of dentistry will be given.

The superolateral surface

Figure 15.17A shows the superolateral surface of the cerebrum. You need to identify two sulci on this surface because these are the key to orientation of other structures to be described. Find them in Figure 15.17A as you read about them. The **lateral fissure** is clearly visible as a deep cleft that separates the **frontal** and **parietal lobes** above from the **temporal lobe** below. If the lips of the lateral fissure are opened, a buried area of cortex can be seen; this is the **insula**. The position of the insula within the lateral fissure is clearly visible in coronal sections of the hemispheres (see Figure 15.22B). The **central sulcus** is not so clearly identifiable. It runs upwards and backwards from the posterior part of the lateral fissure to the superior border of the superolateral surface, a little posterior to its midpoint, and continues a cm or two on to the medial surface. It is the *only* major sulcus on the superolateral surface that crosses on to the medial surface as shown in Figure 15.17B. This is an aid to its identification if you have access to either a brain for dissection or one of the excellent models of the human brain currently available.

The central sulcus separates the **frontal** from the **parietal lobe** on the superolateral surface. It also separates two important gyri as shown in Figure 15.17A. The **precentral gyrus** lies immediately anterior to the sulcus in the frontal lobe and is the site of the **primary motor cortex**. The **post-central gyrus** immediately behind the sulcus in the parietal lobe is the location of the **primary somatosensory cortex**.

If the lateral sulcus is opened gently, the superior surface of the temporal lobe can be seen. This surface has two or three transversely running sulci that separate the **transverse temporal gyri**; the **primary auditory cortex** is located in the transverse temporal gyri.

The medial surfaces

The medial surfaces of the hemispheres are difficult to see on an intact brain, but can be seen on a hemisection produced by cutting through the corpus callosum and the walls of the third ventricle connecting them across the midline. Figure 15.17B shows the result of

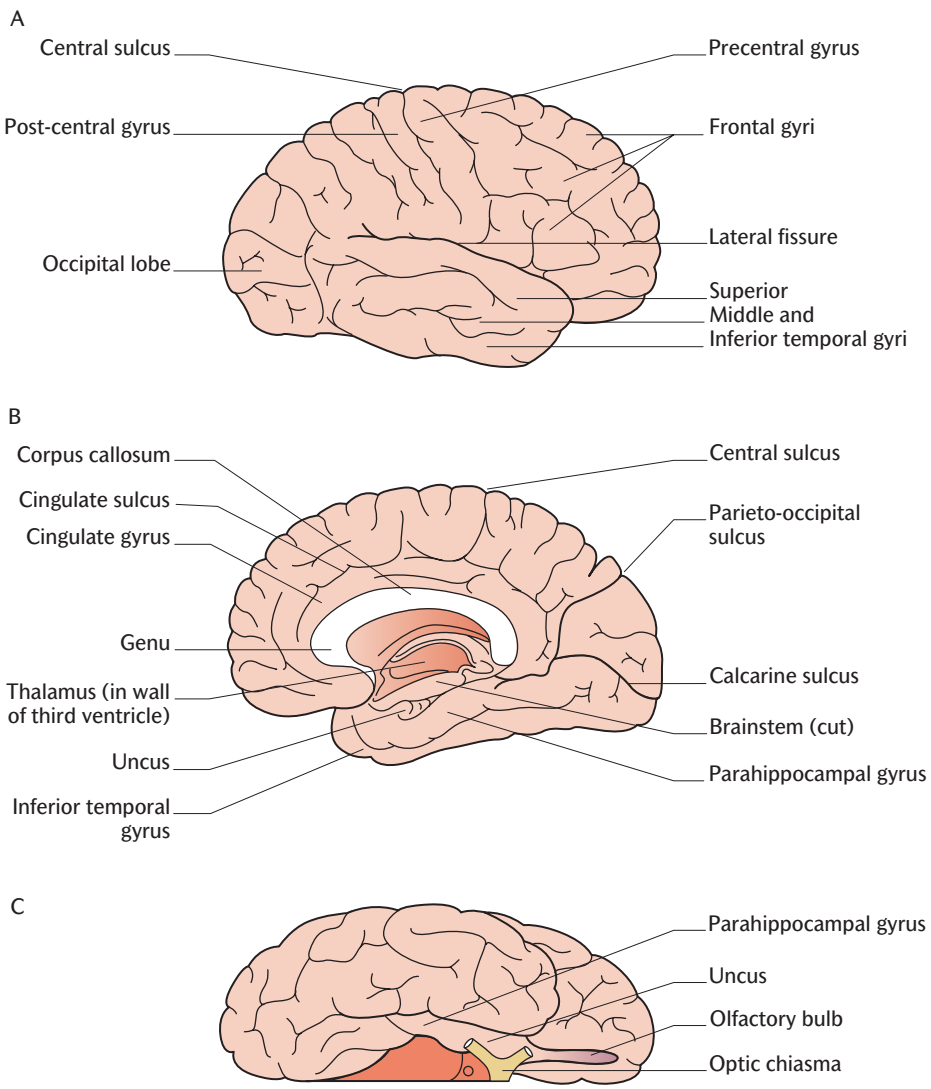


Fig. 15.17 The right cerebral hemisphere with brainstem removed. A) Lateral surface; B) Medial surface of cerebrum; C) Inferior surface.

Box 15.6 Evolution of the cerebral cortex

From an evolutionary and functional viewpoint, the cortex of the mammalian brain can be divided into two basic parts—the ancient **archicortex** and **paleocortex** form one part and the more recently evolved **neocortex** the other. The neocortex accounts for about 90% of the total bulk of the cerebral cortex in humans. It includes the primary areas for somaesthetic sensation, motor function, and the special senses (other than smell) and association cortex which links these primary areas. In the human brain, much of the neocortex is made up of such **association areas** that integrate information from different primary areas and process higher cognitive functions. The paleocortex is the part of the cerebral hemisphere concerned with olfaction and the archicortex forms the **limbic system**. The paleocortex and archicortex have fewer neurons and their organization is simpler than the neocortex.

such an operation. The upwardly convex curve of the **corpus callosum** can now be clearly seen. The **cingulate gyrus** begins below the anterior part of the corpus callosum and curves upwards and backwards above it. This gyrus is an important component of the limbic system (see p. 133).

Two important sulci mark the posterior part of the medial surface and are shown in Figure 15.17B. On the diagram, locate the **calcarine sulcus** which curves upwards and forwards from the occipital pole. The **parieto-occipital sulcus** begins at the upper border of the hemisphere and runs downwards and forwards to meet the calcarine sulcus. As its name suggests, the parieto-occipital sulcus marks the boundary between the **occipital** and **parietal lobes** of each hemisphere. Note that there is no clear-cut boundary between these two lobes on the superolateral surface of the hemisphere. The **primary visual cortex** surrounds the posterior part of the calcarine sulcus as it reaches the occipital pole.

The inferior surface

The inferior surface of the cerebral hemisphere is shown in Figure 15.17C. Note that this figure is orientated exactly the same way as the other two pictures making up Figure 15.17—the anterior part is to the right and posterior aspect to the left. Anteriorly, the inferior surface is made up of the orbital surface of the frontal lobe. The **olfactory bulb** and **olfactory tract** lie against the orbital surface of the frontal lobe. The inferior surfaces of the temporal and occipital lobes are visible posteriorly.

15.5.3 The cerebral cortex

The cerebral cortex consists of a thin rim of grey matter on the surface of the cerebral hemispheres. As you can see in Figure 15.19, the cortex follows every sulcus and gyrus. Specific areas of cerebral cortex have specific functions. See also Box 15.6.

Functional localization within the cerebral cortex

In Figure 15.18A and B, the primary functional areas of the hemispheres are projected on to the superolateral and medial surfaces of the cortex together with the important sulci described in Section 15.5.2 that are required to locate them.

The **primary somaesthetic sensory area** occupies the post-central gyrus (Figure 15.18A). Ascending sensory pathways cross at some point as they ascend through the spinal cord and brain so the *left* side of the body is represented in the *right* sensory cortex and vice versa for the left cortex. The body is represented **somatotopically** on each cortex such that the area of cortex receiving information from each area of the body is proportional to the importance of that area in reception of sensation. Sensory somatopy is illustrated in Figure 15.19; the grotesquely distorted human figure indicates the importance of sensation in each part of the body with larger areas being the most sensitive and the solid bars indicate the amount of cortex devoted to that area. Neurons conveying sensation from very sensitive areas such as the fingers, lips, and tongue occupy large areas of the cortex entirely out of proportion to their actual size as a percentage of the body. As Figure 15.19 indicates, the representation of the body is inverted on the cortex; the area for the head is most inferior just above the lateral sulcus, followed by the upper limbs above, then the trunk, and finally, the reception area for the lower limb and lower part of the trunk being most superior. The leg area extends over on to the medial surface of the hemisphere. Somatotopic representation reflects the fact that the neurons carrying sensation from the various parts of the body maintain an orderly arrangement throughout the ascending pathways in the spinal cord and brainstem. Somatotopic representation is also maintained in synaptic sites in the ascending pathways, including the thalamus and some of the cranial nerve nuclei. The secondary **somatosensory association cortex** where sensory data are integrated and interpreted is posterior to the primary sensory area on the superolateral surface of the hemisphere. The effects of damage to the sensory cortex is outlined in Box 15.7.

The cortical area receiving **taste sensation** is located in the head region of the post-central gyrus just above the lateral sulcus and extends into the insula.

The **primary visual cortex** surrounds the calcarine sulcus (Figure 15.18B). The **visual association cortex** surrounds the primary visual area. Damage here may lead to the inability to recognize objects in the opposite field of vision.

Box 15.7 The effects of trauma or disease on the sensory cortex

Disease or trauma affecting the primary sensory area greatly reduces awareness and localization of sensory stimuli on the contralateral side of the body. If the primary area is intact, but the association area is damaged, it is difficult to interpret the sensory input; for example, the patient may be unable to recognize by feel an everyday object placed in the hand with their eyes closed.

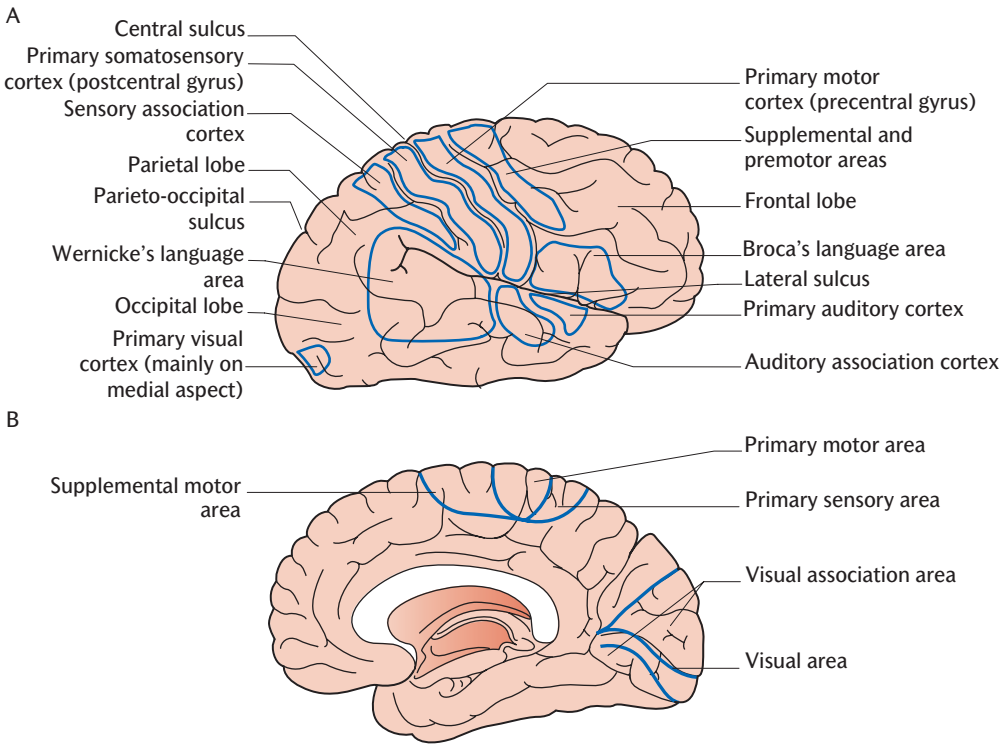


Fig. 15.18 The principal primary functional areas of the right cerebral cortex. A) Lateral view; B) Medial view.

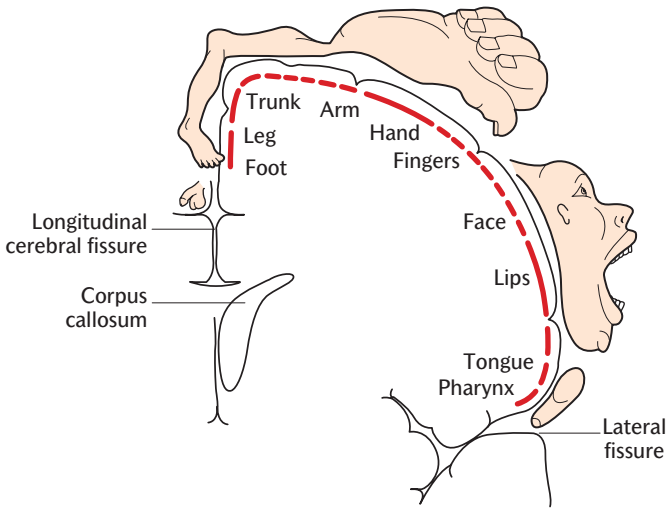


Fig. 15.19 A diagram of the left somatosensory cortex showing the somatotopic representation of the body (the sensory homunculus).

The **primary auditory area** lies in the transverse temporal gyri on the superior surface of the temporal lobe (Figure 15.18A). The auditory cortex has a tonotopic arrangement in which different sound frequencies project to different areas within it. Pathways from the organs of Corti in the inner ears project bilaterally to both auditory cortices. Unilateral lesions involving the auditory area, therefore, lead to little hearing impairment. The **auditory association area** is situated in the floor of the lateral sulcus beneath and behind the primary auditory area.

The **primary motor area** occupies the precentral gyrus (Figure 15.18A). The contralateral side of the body is represented somatotopically in an inverted manner, similar to the somatopical representation in the somatosensory cortex. Destruction of the motor area is followed by the loss of fine control of voluntary muscles on the opposite side of the body. The **premotor** and **supplemental motor areas** are anterior to the motor area and initiate and sequence complex learned motor activities (see Section 16.3.3).

Broca's area is the **executive language area.** As you can see in Figure 15.18A, it is situated in the frontal lobe above the anterior end of the lateral sulcus, anterior to the head area of the primary motor cortex. A lesion at this site results in difficulties in formulation of language, but comprehension remains good. **Wernicke's area,** the **receptive language area**, is more diffuse and less well marked; it is the area in the parietal and temporal lobes around the posterior termination of the lateral sulcus close to the auditory association cortex. Lesions in these sites lead to poor comprehension of language. The language areas are located in the left hemisphere in the majority of people. Language processing in the brain involves other cortical areas outside Broca's and Wernicke's areas and is much more complex than the outline given above. Our understanding of language processing is one area that has advanced enormously in recent years using non-invasive investigations of brain function.

The large cortical areas in the frontal, parietal, occipital, and temporal lobes outside those parts of the cortex with clearly defined functions are less precisely understood. Those in the frontal lobe are concerned with control of behaviour and relating it to judgement and previous experience. The areas in the other three lobes are involved in such complex processes as memory, the experience of emotions, and intellectual processes such as thinking and learning.

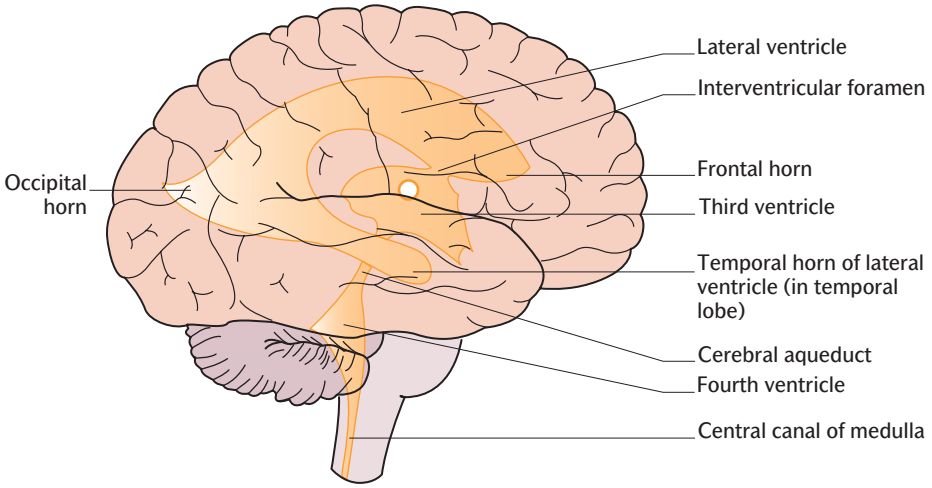


Fig. 15.20 The outline of the ventricular system superimposed on a lateral view of the brain.

The lateral ventricles

Each hemisphere surrounds a central cavity, the **lateral ventricle**. The two lateral ventricles communicate with the third ventricle in the diencephalon through the interventricular foramina. The outline of the lateral ventricles and their communication with the other ventricles is shown in Figure 15.20. Each lateral ventricle does not lie centrally within its hemisphere, but is much closer to the medial surface. As you can see in Figure 15.20, the body of the ventricle has an anterior frontal horn projecting into the frontal lobe, a posterior occipital horn projecting towards the occipital lobe, and an inferior temporal horn extending forwards and downwards into the temporal lobe. Each lateral ventricle contains **choroid plexus** that produces **CSF**.

CSF produced in the lateral ventricles passes into the midline **third ventricle** where more CSF is added. CSF then passes down the cerebral aqueduct into the **fourth ventricle** posterior to the brainstem where yet more CSF is manufactured. CSF exits through the foramina in the fourth ventricle into the **subarachnoid space** surrounding the brain and spinal cord. CSF is eventually resorbed through **arachnoid granulations** into the venous sinuses that drain the brain. The total volume of CSF is about 150 ml.

Cerebrospinal fluid

CSF is a clear, colourless liquid. It contains inorganic salts similar to those in blood plasma and small amounts of protein and glucose. It also contains a few lymphocytes. It is maintained at a pressure of 50–150 mm of water. CSF is secreted quite slowly and is entirely replaced about six times in 24 hours.

The principal functions of CSF are to:

- Provide mechanical support and cushion the CNS (the real average weight of a human brain is about 1500–1700 g, but its apparent weight is reduced by a factor of 10 by floating in CSF);
- Nutrition for the CNS;
- Defend the CNS and meninges against infection.
- Some aspects of CSF in disease are outlined in Box 15.8.

The basal nuclei

Anatomically, the **basal nuclei** (or basal ganglia) are areas of grey matter deep within each hemisphere. They include the **caudate, lentiform**, and **amygdaloid nuclei.** The caudate and lentiform nuclei have extensive interconnections. The bands of axons crossing between them can be seen in sections of the brain cut through these structures. The striped appearance of these axons and the two nuclei they connect give these structures their collective name of the **corpus striatum.** The relationship of the caudate and lentiform nuclei is illustrated in Figure 15.21. Parts of the basal nuclei can be seen as prominent grey areas in horizontal and coronal sections of the brain as shown in Figure 15.22 A and B. Functionally, the basal nuclei also include the subthalamic nuclei and substantia nigra.

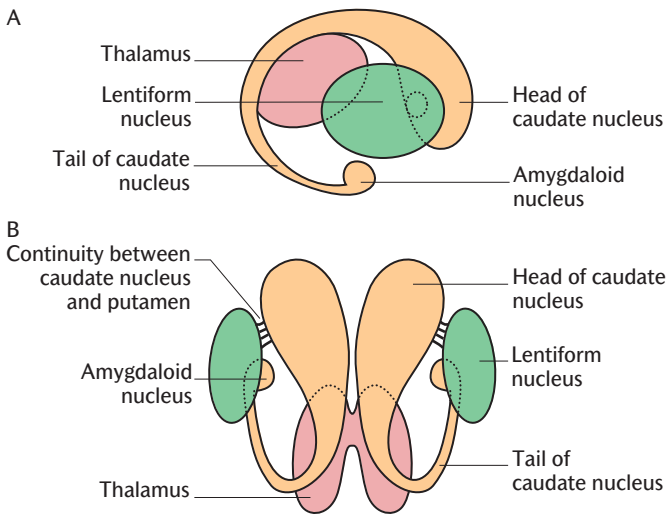


Fig. 15.21 A diagrammatic outline of the basal nuclei. A) Lateral view; B) Superior view.

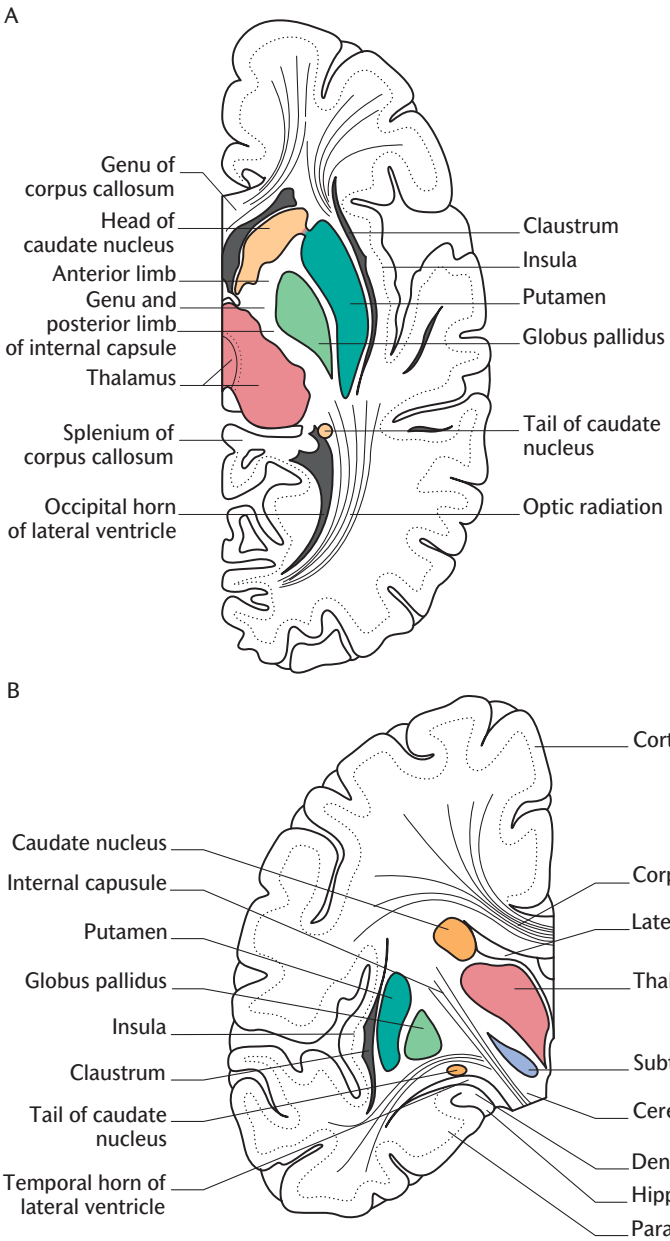
As you can see from Figure 15.21, the **caudate nucleus** has a large bulbous head, a narrow body, and a long tapering tail which gives it its name. On a horizontal section (Figure 15.22A), its head is adjacent to the frontal horn of the lateral ventricle. The body arches above the plane of section, but can be seen adjacent to the lateral ventral on a coronal section (Figure 15.22B). The tail is present as a small grey area near the occipital horn of the lateral ventricle on horizontal sections. Figure 15.21

Box 15.8 CSF in disease

The character of the CSF may change greatly in diseases of the CNS and meninges and alterations in the character of the CSF are of value in diagnosing neurological disease. The number of white blood cells rises during such infections and the predominant cell type depends on the nature of the infecting organism. Red blood cells are present in CSF following subarachnoid haemorrhage. Protein content rises in many conditions, including tumours of the brain and spinal cord. A sample of fluid for examination can be obtained by means of a lumbar puncture (see Box 15.4).

The pressure of the CSF is increased when there are space-occupying lesions such as tumours within the cranial cavity or vertebral canal and after most serious head injuries. This condition is known as **raised intracranial pressure**. Raised intracranial pressure must be treated as a priority, otherwise 'coning' ensues which is life-threatening (see Box 3.4).

indicates that the tail is continuous anteriorly with the amygdaloid nucleus, part of the limbic system (see p. 133). The entire **lentiform nucleus** resembles a biconvex lens on horizontal sections which gives it its name.



In both horizontal and coronal section, a lateral dark part, termed the **putamen**, may be distinguished from a medial paler part, termed the **globus pallidus.**

Look at the horizontal section illustrated in Figure 15.22A and identify a shallow V-shaped strip of white matter, the **internal capsule** (see p. 133), separating the lentiform nucleus from the thalamus posteriorly and the lentiform nucleus from the head of the caudate nucleus anteriorly. On coronal sections such as Figure 15.22B, the internal capsule runs obliquely upwards and laterally between the lentiform nucleus and the thalamus and body of the caudate nucleus.

The **claustrum** is a very thin sheet of grey matter situated in the white matter between the lentiform nucleus and the insula. Its function is unknown.

The **subthalamic nuclei** lie below the thalamus as their name implies. Figure 15.22B shows their position in the internal capsule just before it enters the midbrain.

Functionally, the **substantia nigra** is part of the basal ganglion system. Its position in the midbrain has already been described and illustrated in Figure 15.14.

Functions of the basal nuclei

The basal nuclei are linked to each other by at least five different series of complex neuronal loops which begin and end in the sensory and motor cortical areas, the limbic system, and other cortical association areas. Essentially, the system functions during motor activities to plan complex

Fig. 15.22 A) A horizontal section through the right cerebral hemisphere; B) A coronal section through the cerebral hemisphere.

Box 15.9 Disease of the basal nuclei

Lesions of the basal ganglia produce various effects according to their specific location, but one of the distinguishing features is **dyskinesia**—abnormal movement. This may manifest as overactivity (**hyperkinesia**) or underactivity (**hypokinesia**). Hyperkinesia may show as peripheral **tremor** in Parkinson's disease when the substantia nigra does not function; slow writhing movement (**athetosis**) or dance-like movements (**chorea**) occur when the caudate or lentiform nuclei is involved. Hypokinesia is typified by reduced movements; movements may be slower (**bradykinesia**) or weaker (**poverty of movement**) than normal and there may be muscular rigidity. A good example of poverty of movement is the shuffling gait seen in Parkinson's disease when patients take small steps without the usual arm swinging that occurs in normal walking. The effects of lesions of the basal nuclei are described more fully in Section 16.3.4.

movements; they *amplify* the intended movement and *suppress* unwanted movement. The connections between the basal nuclei with other areas of the cerebral cortex, including the limbic system, are involved in motivation, emotional expression, and social responses (see also Box 15.9).

The cerebral white matter

As you can appreciate from Figure 15.22, much of the interior of the cerebral hemisphere is composed of white matter. As already mentioned, the neuronal processes constituting this white matter may be projection, association, or commissural axons.

Commissural axons crossing from one hemisphere to the other in the body of the **corpus callosum** radiate outwards into the hemispheres on each side, intersecting the association and projection fibres. These connections are concerned with bilateral learning processes such as being able to employ the left hand to carry out a task learned by use of the right hand. As shown in Figure 15.22A, axons in the posterior part of the corpus callosum radiate into the occipital lobes and those in the anterior part radiate into the frontal lobes.

The internal capsule

The majority of projection axons ascending to or descending from the cerebral cortex pass through the **internal capsule** already outlined and illustrated in Figure 15.22. In a horizontal section as shown in Figure 15.22A, it appears as a shallow V shape. The apex of the V is anterior to the thalamus and posterior to the head of the caudate nucleus. The two arms of the V form the **anterior** and **posterior limbs** which are continuous with each other at the angle—the **genu.** The lentiform nucleus sits in the trough of the V. The anterior limb separates the head and body of the caudate nucleus medially from the lentiform nucleus. The posterior limb separates the thalamus medially from the lentiform nucleus laterally.

Figure 15.22B shows what happens to the neuronal processes forming the internal capsule above and below. The ascending and descending projection axons passing to or from the various areas of the cerebral cortex fan outwards above the internal capsule in an arrangement called the **corona radiata**. Inferiorly, the processes from the internal capsule on each side are concentrated together to form the corresponding cerebral peduncle of the midbrain.

The anterior limb of the capsule contains frontopontine axons descending from the cortex of the frontal lobe to synapse in the pontine nuclei and thalamocortical projections that connect the hypothalamus and limbic system with the frontal association cortex. The genu contains somatopically arranged corticonuclear axons travelling to the motor nuclei of the cranial nerves. The posterior limb contains corticospinal axons en route to lower motor neurons in the ventral horn of the spinal cord; these are also arranged somatopically in an anteroposterior sequence—those innervating the upper limb lie anteriorly adjacent to the genu, next are those supplying the trunk muscles, and finally, those related to the lower limb. The internal capsule also contains other descending tracts connecting to the corpus striatum, substantia nigra, red nucleus, and inferior olivary nucleus. The posterior limb conveys tertiary projection neurons from the thalamus to the primary sensory area in the post-central gyrus. Figure 15.22A shows the backward, more or less horizontal, extension of the posterior limb of the internal capsule. This is the **optic radiation** that forms part of the visual pathway carrying neurons from the lateral geniculate nucleus of the thalamus to the visual cortex in each occipital lobe. The functions of the tracts passing through the internal capsule are described in the next chapter.

The limbic and olfactory systems

The **limbic system** is a group of structures related to emotional and instinctive behaviours that have high survival value for both the individual and the species; it is also involved in the processes of memory.

The major part of the cerebrum in lower vertebrates is formed from the evolutionary oldest archicortex and ancient paleocortex (see Box 15.6); both these structures are associated with olfaction, reflecting the importance of the sense of smell in lower vertebrates in the early stages of vertebrate evolution. In higher mammals, the neocortex (new cortex) is greatly expanded so the paleocortex and archicortex are much reduced in *relative* size. The paleocortex is still concerned with olfaction, but the archicortex has become included in the limbic system.

The olfactory pathways

The **olfactory pathways** are illustrated in Figure 15.23; they consist of the olfactory cells of the nasal mucosa, the olfactory bulbs and tracts, and the olfactory areas of the cerebral cortex. The olfactory cells are neurons specialized to respond to minute quantities of chemicals in the inspired air. Their axons pass through foramina in the cribriform plate of the ethmoid bone forming part of the roof of the nose and synapse in the olfactory bulb on the dendrites of mitral cells which form the bulb. The axons of the mitral cells travel in the olfactory nerve and tract to terminate in the primary olfactory area in the temporal lobe, a region called the **uncus**. The olfactory areas have numerous projections to other parts of the brain, especially the hypothalamus and the limbic system. These connections explain the powerful effects that odours can have in stimulating or inhibiting visceral functions such as appetite and in arousing emotions.

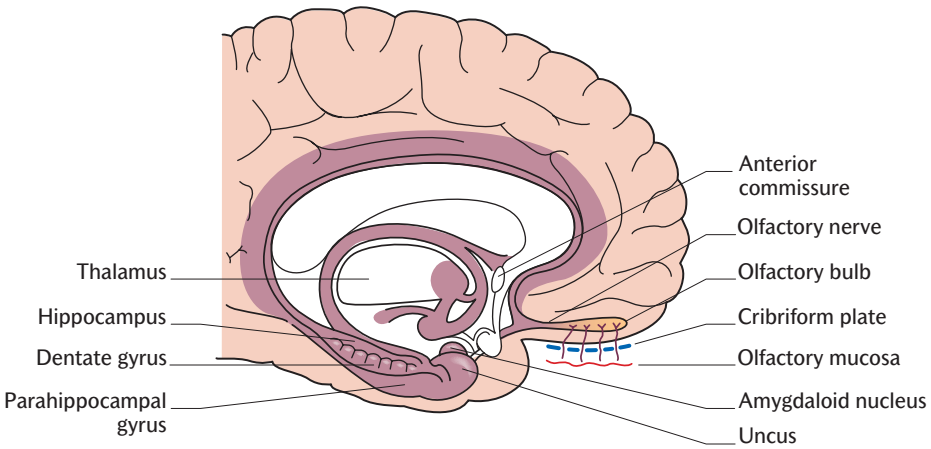


Fig. 15.23 A diagrammatic representation of the limbic and olfactory systems in the left cerebral hemisphere.

The limbic system

The **limbic system** comprises a number of structures located around the junction of the diencephalon and cerebral hemispheres. These structures are shown diagrammatically in Figure 15.23 and include the **cingulate gyri** arching up over the corpus callosum and their continuations into the temporal lobes, the **parahippocampal gyri**. The hippocampus and dentate gyrus adjacent to the parahippocampal gyrus form the **hippocampal formation**; the dentate nucleus is a narrow strip of grey matter situated between the hippocampus and the parahippocampal gyrus These structures can also be seen on the coronal section in Figure 15.22B in the floor of the temporal horn of the lateral ventricle; 'hippocampus' is a Greek word meaning 'sea horse' and it was the supposed resemblance of these structures to a sea horse that gave them their name. The amygdaloid nuclei, the mammillary bodies of the hypothalamus, and the anterior nuclei of the thalamus also contribute to the limbic system.

The hippocampal formation receives numerous inputs from the olfactory areas and the cingulate gyrus. It also has extensive inputs from the association areas in the cerebral cortex. These inputs link complex behaviour to the basic instincts and memory governed by the limbic system. The principal output pathway from the limbic system ends in the hypothalamus. The limbic system influences motor activity as well; smiling when we meet a friend is controlled from the limbic system whereas smiling when asked to is directed from the motor cortex.

15.6 Blood supply of the brain

The brain receives blood from two sources, the **internal carotid arteries** and the **vertebral arteries**. The internal carotid and vertebral inputs may be connected with each other on the under surface of the brain by a circle of small arteries termed the **arterial circle** (of Willis). The venous return from the brain is generally into the nearest venous sinus of the dura mater.

The arteries and veins of the brain run within the subarachnoid space inside the cranial cavity. The arteries tend to lie deep inside the sulci of the cerebral hemispheres whereas veins are more superficial.

15.6.1 The arteries

If you have the opportunity to examine a cadaveric human brain, you will notice that the arteries look different from those you will have encountered elsewhere in the body; they look more transparent and quite thin-walled. They are, in fact, thin-walled because the outermost fibrous coat (the tunica adventitia) is poorly developed or absent. These thin-walled arteries are more prone to rupture than arteries elsewhere (see Box 15.10). The cerebral arteries also differ from arteries supplying most other organs in that they branch repeatedly *before* their terminal branches enter the brain. The cerebral arteries are true *end arteries*, each terminal branch supplying a circumscribed area of the brain with no alternative supply (see Section 4.1.3). If a cerebral arterial branch should rupture or become blocked, a small area of the CNS will lose its blood supply and will die because there is no alternative anastomotic or collateral circulation. The functions carried out by the dead area of brain tissue will no longer operate (see Box 15.10).

The internal carotid arteries

As described in Section 12.5.2, the **common carotid arteries** arise from the arch of the aorta, the right being a branch of the brachiocephalic trunk and the left one being a direct branch (see Figure 12.10). Each common carotid artery divides into external and internal carotid arteries high in the neck. Each **internal carotid artery** passes without branching through a bony canal in the base of the skull, the **carotid canal**, into the cranial cavity where they pass through the corresponding cavernous sinus to emerge through its roof.

The branches of the internal carotid arteries are illustrated in Figure 15.24 which should be followed as the following description is read. Each

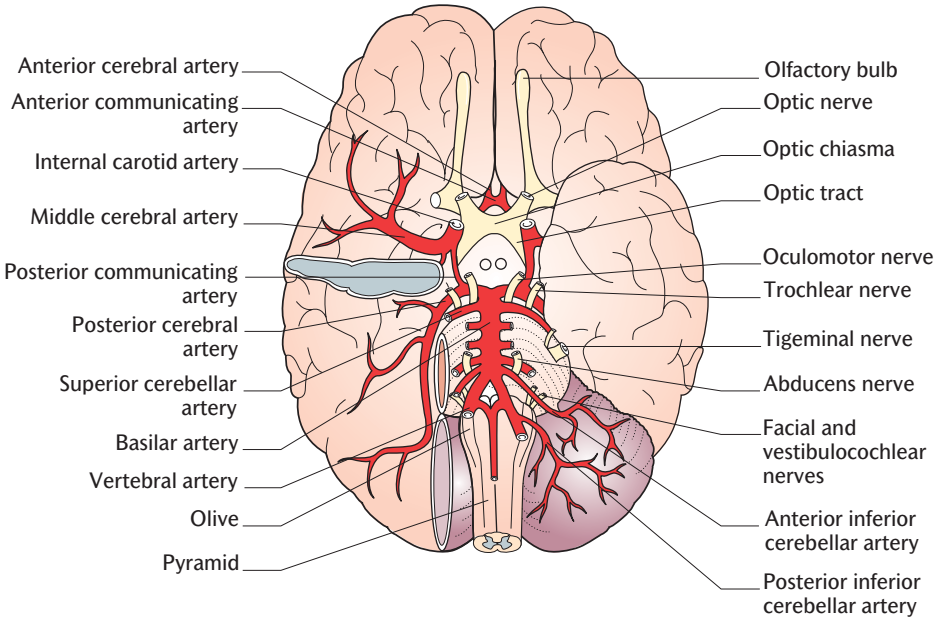


Fig. 15.24 The arterial supply of the brain seen from below. The anterior part of the right temporal lobe and right cerebellum have been removed for clarity.

internal carotid artery gives off an **ophthalmic artery** (not illustrated) just above the point where it leaves the cavernous sinus; these arteries run anterolaterally to enter the orbits through the optic canals. A **posterior communicating artery** may arise from the posterior aspect of the internal carotid artery; if present, it runs backwards to join the posterior cerebral artery. The internal carotid artery on each side terminates by dividing into the **anterior** and **middle cerebral arteries**. This division occurs below the **anterior perforated substance,** an area on the inferior surface of the cerebral hemisphere lateral to the optic chiasma. The anterior perforated substance is perforated by numerous small perforating arteries that arise from the terminal branches of the internal carotid arteries and supply deep structures of the brain such as the striate cortex.

As can be seen in Figure 15.24, each **anterior cerebral artery** passes forwards and medially above the optic nerve before disappearing deeply into the longitudinal fissure separating the two cerebral hemispheres. Just before entering the fissure, the left and right arteries may be joined by the short **anterior communicating artery.** The anterior cerebral arteries then turn to run upwards and backwards above the corpus callosum, across the medial surfaces of the corresponding hemispheres as far as the parieto-occipital sulcus. Figure 15.25 indicates that each anterior cerebral artery supplies the medial surface of each hemisphere and overlaps about 2 cm on to the lateral surface.

Each **middle cerebral artery** is larger than the anterior cerebral artery. Each artery passes laterally into the lateral fissure to supply the lateral surface of each cerebral hemisphere up as far as the area supplied by the anterior cerebral artery and down as far as the upper surface of the temporal lobe, an extensive area as you can see in Figure 15.25. Although there is no obvious boundary line on the lateral surface, the middle cerebral artery is distributed only as far as a line corresponding to the parieto-occipital sulcus on the medial surface.

The vertebrobasilar system

The **vertebral arteries** are branches of the subclavian arteries. Each one ascends through the neck in the foramina in the transverse processes of the upper six cervical vertebrae, winds around the lateral mass of the atlas forming the articulation with the underside of the skull, and enters the cranial cavity through the foramen magnum.

The intracranial course and distribution of the vertebral arteries is illustrated in Figure 15.24. The vertebral arteries run up alongside the medulla and join together at the lower border of the pons to form the single midline **basilar artery**. It ascends to the upper border of the pons where it terminates at a T-junction by dividing into the right and left **posterior cerebral arteries**. These arteries turn back between the inferior surface of the temporal lobes and the cerebellum and as shown in Figure 15.25, they supply most of the temporal lobes and all of the occipital lobes. The **posterior perforated substance** lies above the bifurcation of the posterior cerebral arteries in the interpeduncular fossa of the midbrain (see Figure 15.10). Small perforating arteries enter here to supply deeper structures, in this case, the thalamus and posterior areas of the basal ganglia.

As you can see in Figure 15.24, the vertebral and basilar arteries have several important branches as they travel from the foramen magnum to their termination.

The vertebral arteries give rise to:

- **Spinal branches** which pass downwards into the vertebral canal to supply the spinal cord;

- A **posterior inferior cerebellar artery** to the cerebellum and lateral aspects of the medulla;
- Short **medullary** branches to the anterior part of medulla oblongata immediately either side of the midline.

The basilar artery also has important branches:

- The **anterior inferior cerebellar** arteries arise from either side of the artery just after its formation;
- **Pontine branches** arise as it passes over the pons together with a **labyrinthine** branch to the internal ears;
- The left and right **superior cerebellar arteries** branch off just before the basilar artery terminates as the posterior cerebral arteries.

Note that the anterior, inferior, and superior cerebellar arteries not only supply the cerebellum as their names imply, but also the lateral aspects of the pons.

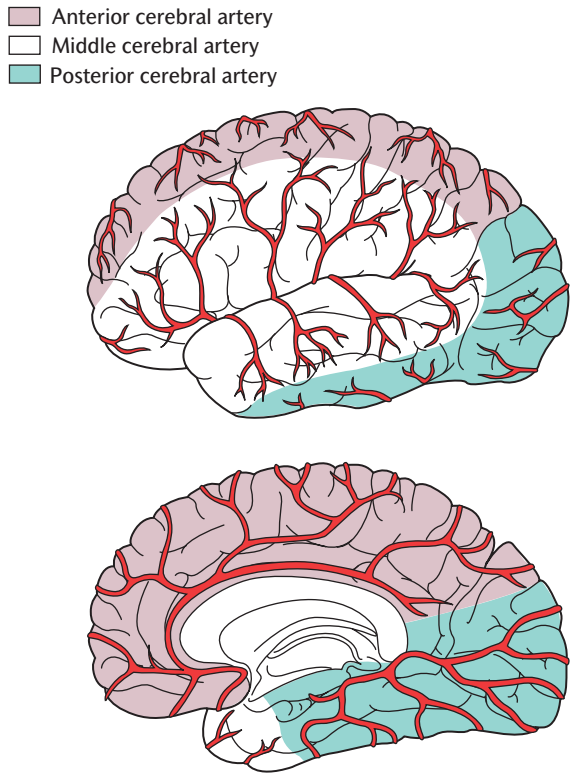


Fig. 15.25 The distribution of the cerebral arteries to the cerebral hemispheres. A) Lateral view; B) Medial view.

The arterial circle (of Willis)

The complete arterial circle as shown in Figure 15.26 is formed by the **anterior communicating artery** between the anterior cerebral arteries and the **posterior communicating arteries** between the internal carotid arteries and posterior cerebral arteries. The communicating vessels are small and the circle is only complete in about 60% of people. Even if all the communicating branches are present, they are usually too small to maintain adequate circulation to the brain if one or other of the major arteries entering the brain is suddenly blocked. They are, however, capable of expanding if blockage occurs more slowly; adequate cerebral blood flow may be maintained even if one or more of the feeders into the arterial circle should become completely occluded. It is interesting that cerebral angiograms show that there is little or no flow through the communicating arteries in normal subjects; a tracer dye injected into the vertebrobasilar system through the subclavian artery will only enter the brainstem, cerebellum, and areas of the cerebrum supplied by the posterior cerebral arteries. Likewise, an injection into one internal carotid artery will be limited to the anterior and middle cerebral arteries on the side of the injection only.

15.6.2 The veins

The veins returning blood from the brain are extremely thin-walled because they lack a muscular layer; they also have no valves. They open into the venous sinuses of the dura mater. The cerebral veins comprise two groups, external and internal, draining the superficial and deep parts of the cerebral hemispheres, respectively. The veins draining the cerebellum and brainstem open into adjacent venous sinuses adjacent to these structures.

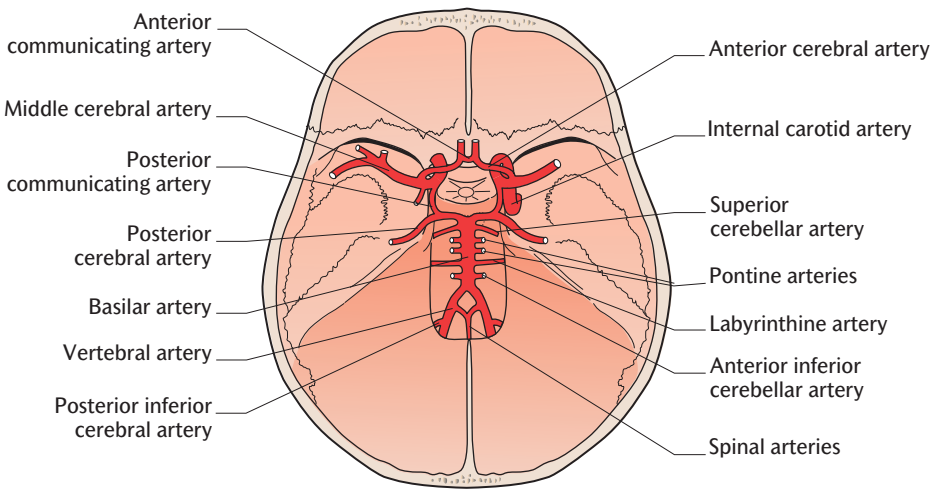


Fig. 15.26 An enlarged view of the arterial supply of the brain seen from above showing the arterial circle of Willis and branches of the vertebral and basilar arteries.

Box 15.10 Cerebrovascular accident (CVA) or 'stroke'

The thin-walled arteries of the brain are prone to rupture (**aneurysm**) as a result of degenerative changes in their walls. In addition, the arterial branches that actually penetrate into the brain tissue are relatively small and are, therefore, particularly liable to thrombosis. These events are referred to as a **cerebrovascular accident (CVA)** or 'stroke' in lay terminology because the cause of blood loss to a particular area of the brain is not usually known immediately. Irrespective of the actual cause of blood loss to the brain, it is important to emphasize that lesions of this sort respect neither anatomical nor functional boundaries. In other words, they can spread extensively and adversely affect quite wide areas.

This is more the case with aneurysm than thrombosis because blood escapes from the cardiovascular system (**extravasation**). When blood makes direct contact with neural tissue, it is extremely toxic. The high molecular weight materials that are required for blood functions such as albumin and haemoglobin actually dehydrate neural tissues. The initial signs and symptoms of 'stroke' are often far worse than the long-term results because extravasated blood is damaging tissues. Such damage is reversible; these areas may well recover when natural mechanisms of blood removal and clot resolution have taken place. On the other hand, any brain area that does not receive a blood supply starts to degenerate irreversibly very rapidly. Thus the areas distal to the site of a blood clot or aneurysm in an artery will be deprived of blood and nutrients and will be unlikely to recover.

When assessing the likely consequence of a CVA, it is, therefore, important to determine which functional areas of the brain may have been affected. For example, a CVA in the middle cerebral artery may possibly lead to degeneration of most of the motor, sensory and auditory cortices, and Broca's and Wernicke's areas, depending on how far from its origin the lesion is sited. Loss of these cortical areas would lead in turn to spastic paralysis of arms, trunk and head, sensory loss over the same areas, hearing problems, and problems interpreting or encoding language.

The arterial blood supply to the internal capsule is by perforating **striate arteries** that branch from the middle and posterior cerebral arteries; these arteries are small and prone to thrombosis. If the subject survives, which is more likely with a thrombosis than with a haemorrhage, there will be some destruction of descending and ascending pathways in the internal capsule. The actual effects will depend on the extent of the lesion and its location in the internal capsule, but motor and sensory deficits are likely. As will be described in more detail in Chapter 16, the effects will be contralateral if ascending or corticospinal pathways are damaged, but the effects are bilateral in most instances if corticonuclear pathways are affected by a lesion close to or in the genu.

In the brainstem, CVA in one or other of the cerebellar arteries is likely to produce some degree of cerebellar ataxia. However, these arteries also supply the lateral aspects of the brainstem and the nuclei in which cranial nerves originate or terminate will also be affected. The effect on specific cranial nerves depends on the level of the lesion. For example, a posterior inferior cerebellar CVA would be most likely to affect the lower ninth, tenth, and twelfth cranial nerves arising from the lower medulla.

16 Major sensory and motor systems

Chapter contents

16.1 Introduction

The previous chapter provided an overview of the anatomy of the CNS, concentrating on structures that can be seen during dissection of the human brain and spinal cord or the study of anatomical models of these structures. Some indication of the function of different components of the CNS has been given in Chapter 15, but this chapter shows how the various anatomical components of the CNS are functionally linked together through sensory and motor pathways. These pathways enable the nervous system to convey information over considerable distances, to integrate the information, and formulate functional responses that coordinate activities of different parts of the body. It will be necessary to introduce some other structures in addition to those described in Chapter 15 during the description of major pathways; most are not visible to the naked eye and even when seen in microscopical sections, they require considerable practice to distinguish them. However, they are important landmarks or relay stations in the central nervous pathways and you need to know of them for a full understanding of pathways.

As emphasized in Chapter 14, our views of the structure and function of many aspects of the nervous system are constantly subject to revision in the light of new clinical and experimental observations and methods of investigation. This applies to nerve pathways just as much as any other aspect of the nervous system. This chapter presents a summary of current views on somatic sensory and motor functions and their application to the practice of dentistry. The special sensory pathways of olfaction, vision, and hearing are described in Chapter 18 in the context of the cranial nerves that form the first part of these pathways.

16.2 General sensory pathways

The information conveyed from the periphery by the sensory components of spinal and cranial nerves is destined to reach the cerebral cortex or the cerebellum. You will be conscious of sensory information that reaches the cerebral cortex, but mostly unaware of information that does not travel to the cortex. However, this does not mean that sensory information that does not attain cortical levels is of no value. For example, sensory neurons or their collateral processes form the afferent limbs of many reflex arcs. We simply could not function efficiently without proprioception informing us about the state of muscles and joints—another source of information that does not reach consciousness.

Sensory pathways may be classified in several different ways. They can be classified by the type of sensory stimulus they react to such as touch or temperature. Alternatively, they can be described in terms of the response to that sensory information. We can **react** to a stimulus, e.g. by retracting away from a noxious stimulus or taking a coat off if a room is too hot. Peripheral receptors responding to noxious, temperature, light touch, and pressure stimuli provoke a reaction. We can use sensory information to make fine judgements and **discriminate** to determine if we are holding a dental instrument tight enough, for example. Receptors for fine touch and vibration convey information used for discriminative purposes. **Proprioceptive** information from muscles, joints, and ligaments are also required for discrimination. Proprioception is also vital if we are to make meaningful and accurate motor responses to sensory stimuli; we need to know where our bodies are in space, where individual components are, what state our muscles are in, and what position joints are in. The sensory pathways that convey information from receptors sensitive to stimuli that produce a reaction follow one route through the CNS, the **spinothalamic pathway,** whereas those carrying discriminative information follow another route, the **dorsal column–medial lemniscus pathway**. Some proprioceptive information reaches the cerebral cortex by the latter pathway to aid discrimination, but the majority is conveyed only as far as the cerebellum by separate **spinocerebellar pathways**.

The basic plan of reactionary and discriminative pathways is the same. Typically, there are three principal neurons in each pathway between the peripheral receptor and the cerebral cortex.

- **Primary** sensory neurons are components of spinal or cranial nerves that convey information from the peripheral receptor to the CNS.
- **Thalamic projection (**or secondary) sensory neurons transfer sensory information within the CNS from the spinal cord or brainstem to the thalamus.
- **Thalamocortical** (or tertiary) neurons transmit information from the thalamus to sensory cortex.

The arrangement of both pathways is shown in Figure 16.1 and 16.5. Follow the general arrangement of the three-neuron chain in these figures as you read the following description.

In spinal nerves, sensory processes enter the spinal cord through the dorsal roots of the spinal nerves. The cell bodies of primary sensory neurons form swellings on their dorsal roots, the **dorsal root ganglia;** peripheral and central processes arise from these cell bodies. The peripheral processes travel in spinal nerves to the peripheral receptors and their central processes pass into the spinal cord through the dorsal roots to connect with thalamic projection neurons. The arrangement is essentially similar in the cranial nerves. Those cranial nerves that contain sensory neurons have ganglia near to their connections with the brain, containing the sensory cell bodies; their peripheral processes connect to receptors in the head and neck and their central processes enter the brainstem.

In the spinothalamic pathway, the cell bodies of thalamic projection neurons are situated within the dorsal grey matter of the spinal cord or in sensory cranial nerve nuclei in the brainstem. Note that cranial nerves are not all identical in composition; some are purely motor nerves whereas others are mixed. They only have the equivalent of dorsal and ventral horn grey matter where it is required; the grey matter thus occurs as discrete areas, the **cranial nerve nuclei**. The cranial nerve nuclei are explained more fully in Chapter 18.

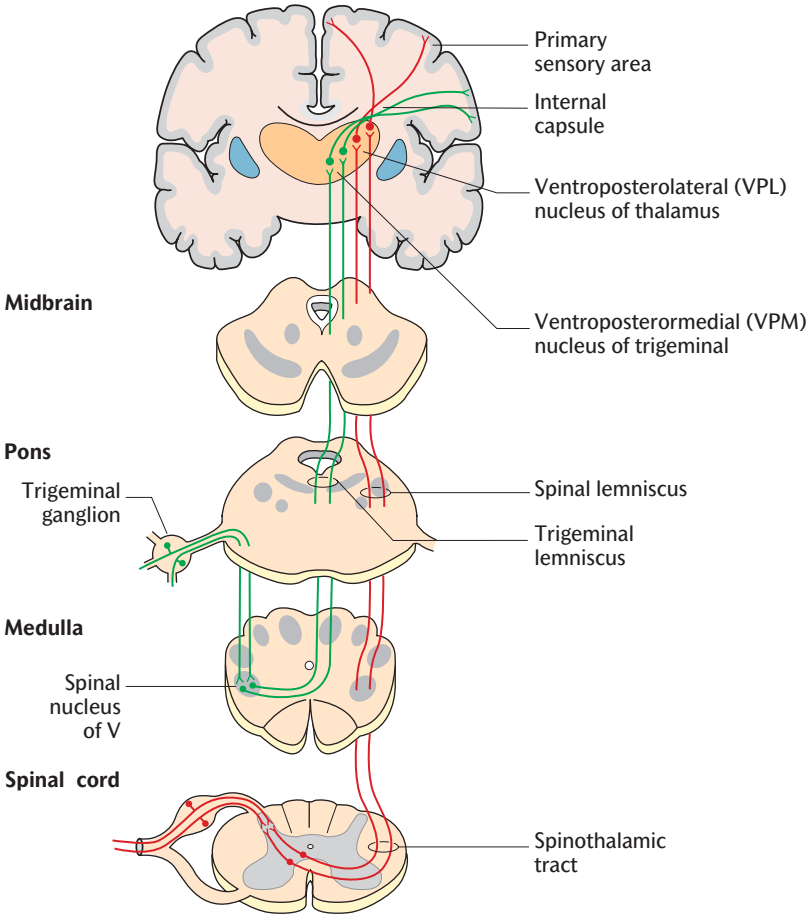


Fig. 16.1 The spinothalamic pathway for temperature, simple touch, and pressure.

Thalamic projection neurons for the dorsal column pathway are located in specific dorsal column nuclei in the lower brainstem. Irrespective of the pathway, axons of thalamic projection neurons cross the midline, usually at the level of their synapse and ascend to the thalamus where they synapse with tertiary neurons. The axons of the tertiary neurons run through the posterior limb of the internal capsule and the corona radiata to reach the somatosensory area of the cerebral cortex in the post-central gyrus. As described in Section 15.4.2 and illustrated in Figure 15.16, sensory projections to the primary sensory cortex are somatotopically represented such that each part of the body is represented in a specific area of the cortex.

The three-neuron pathway outlined above is a simplification. Interneurons occur at the synaptic sites between the principal neurons of the sensory pathways in the spinal cord or brainstem and in the thalamus. These multiple synapse sites enable convergence, divergence, amplification, or inhibition of sensory information as outlined in Chapter 3.

The detailed structure of the two pathways and differences between will now be outlined.

16.2.1 The spinothalamic pathway (the anterolateral pathway)

This pathway conveys noxious, temperature, simple touch, and pressure stimuli to the CNS. It is called the spinothalamic pathway because the secondary neurons pass from the spinal cord to the thalamus. The

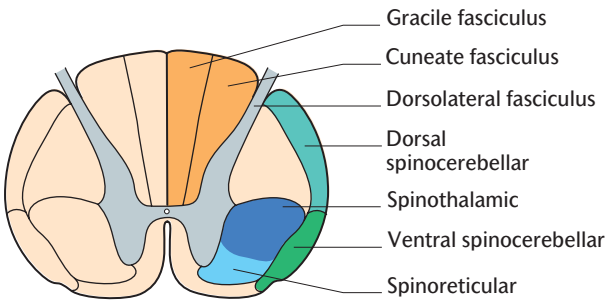


Fig. 16.2 A cross section of the spinal cord to show the positions of the major sensory pathways.

alternative name indicates the position of this tract in a cross section of the spinal cord; as shown in Figure 16.2, it lies in the anterolateral white matter adjacent to the ventral horn. The spinothalamic pathway is illustrated in Figure 16.1; the pathway should be followed on the diagram as the description is read.

The skin and other tissues contain many types of sensory endings to which the peripheral process of primary sensory neurons connect. Different receptor types have a high degree of selectivity for different sensory modalities.

Nociception is the sensation that arises from stimuli that cause tissue damage. Noxious stimuli generate the unpleasant sensation or

emotion that we call **pain** when they reach consciousness. Unencapsulated free nerve endings in the skin epidermis, other epithelia, and deeper tissues such as ligaments and joint capsules respond to noxious stimuli. Temperature receptors are also free nerve endings. There are both unencapsulated and encapsulated receptors for simple touch. Unencapsulated endings contact specialized cells in the basal layer of epithelia and surround hair follicles. Encapsulated endings of different types are located in the dermis, subcutaneous tissues, and in the capsule of many synovial joints. The processes from the receptors for simple touch and pressure are medium calibre and myelinated. As shown in Figure16.1, the cell bodies of all the types of sensory process already mentioned are located in the dorsal root ganglia of spinal nerves or the corresponding sensory ganglia of those cranial nerves that contain somatic sensory neurons, irrespective of the type of receptor the processes connect to. The sensory ganglia of cranial nerves are usually named after the nerve in question.

Continue tracing these pathways in Figure 16.1, noting that their central processes from cell bodies in the dorsal root ganglia form the dorsal roots of the spinal nerves. As they enter the spinal cord, they divide into ascending and descending branches which travel for one or two segments before entering the dorsal grey horn through the dorsolateral tract (see Figure 15.8).

The primary sensory neurons of the spinothalamic system synapse in different locations, depending on whether they are conveying noxious stimuli or touch and temperature stimuli. Nociceptive primary sensory neurons synapse in the superficial laminae of the dorsal horn directly with thalamic projection neurons that only respond to high threshold noxious stimuli. Neurons that convey thermal and touch information synapse with cells in lamina V of the dorsal horn (see Figure 15.9). These lamina V cells have conventional short dendrites within lamina V as you may expect, but they also have long dendrites that travel right back into lamina I. These cells may, therefore, be stimulated by either high threshold noxious stimuli synapsing in lamina I or low threshold tactile or thermal stimuli synapsing in lamina V referred to as wide dynamic range neurons. The dual stimulation of the thalamic projection cells in lamina V is important in pain localization (see Section 16.2.2.).

There are numerous interneurons between the terminals of primary neurons and the thalamic projection neurons. The primary sensory neurons also make synaptic connections for spinal reflex responses. The thalamic projection neurons receive numerous other inputs, notably from collateral branches of neurons responding to discriminative touch, neurons descending from the brainstem reticular formation, and parietal association cortex. All these connections probably play a role in the modulation of sensation passing through the spinal cord, especially pain.

Figure 16.1 illustrates that the axons of thalamic projection neurons cross the midline at the same level as their cell bodies and pass through the commissural areas to ascend in the contralateral **spinothalamic tract** located in the anterolateral part of the white matter. Some sources divide the spinothalamic tract into an anterior component carrying touch and pressure and a lateral part conveying noxious and thermal stimuli. The upward continuation of the spinothalamic tract in the brainstem is known as the **spinal lemniscus** which can be traced in Figure 16.1. Thalamic projection neurons conveying thermal and tactile information synapse in the **ventroposterolateral nuclei** of the thalamus with tertiary neurons; the processes of the tertiary neurons pass to the primary sensory cortex through the posterior limb of the internal capsule. In contrast, thalamic projection neurons conveying noxious stimuli synapse with tertiary neurons in the **posterior nuclei** of the thalamus. These tertiary neurons do not project to the somatosensory primary cortex, but to several areas of the cortex and other cerebral structures comprising the **pain matrix** (see Section 16.2.2.)

The trigeminal (fifth cranial) nerves convey most of the primary sensory neurons carrying nociceptive, thermal, simple touch, and pressure stimuli from the head; their cell bodies are located in the trigeminal ganglia. The glossopharyngeal (ninth) and vagus (tenth) nerves also have sensory functions in the head and neck and their sensory cell bodies are in ganglia on the corresponding nerves. The central processes of the primary sensory neurons of all these three cranial nerves synapse with thalamic projection neurons in the spinal part of the **trigeminal sensory nuclear complex**. The trigeminal sensory nuclear complex is described more fully in Section 18.6.

The processes of trigeminal thalamic projection neurons decussate and ascend in the **trigeminal lemniscus** of the opposite side as shown in Figure 16.1. They synapse in the **ventroposteromedial nucleus** of the thalamus and are thus kept separate from neurons ascending in the spinothalamic tract. Tertiary neurons project from this nucleus to the head region of the primary sensory cortex through the posterior limb of the internal capsule.

16.2.2 Pain

In the description of the spinothalamic tract, you will have noticed that nociceptive stimuli take different routes and make different connections from thermal and tactile information. There are many interesting facets to the transmission of nociceptive stimuli and the perception of pain which require further consideration. After all, one of the main functions of dental practitioners is to put their patients at ease through a combination of good interpersonal skills and pain management when required. Pain is an entire study in itself and many aspects of pain are outside the scope of this book.

The pathways from nociceptors are shown in Figure 16.3. This figure shows nociceptors connecting to the cell bodies of primary sensory neurons by small diameter unmyelinated C processes and finely myelinated Aδ processes. Aδ processes conduct action potentials faster than unmyelinated C processes. It is now generally accepted that the Aδ processes conduct the initial noxious sensation that is interpreted as sharp pain (often referred to as **first pain**) while the unmyelinated processes convey the wave of duller, but intensely disagreeable, pain which follows (**second pain**).

The incoming Aδ and C axons of primary sensory neurons make separate connections in the spinal cord as shown in Figure 16.3B. Aδ axons connect with lamina I and lamina V thalamic projection neurons as described in the main description of the spinothalamic tract whereas C axons only synapse with lamina I neurons. Furthermore, the thalamic projection neurons responding to incoming nociceptive information carried by C axons form their own **spinoreticular tract** in the anterolateral aspect of the spinal cord (see Figure 16.2). This

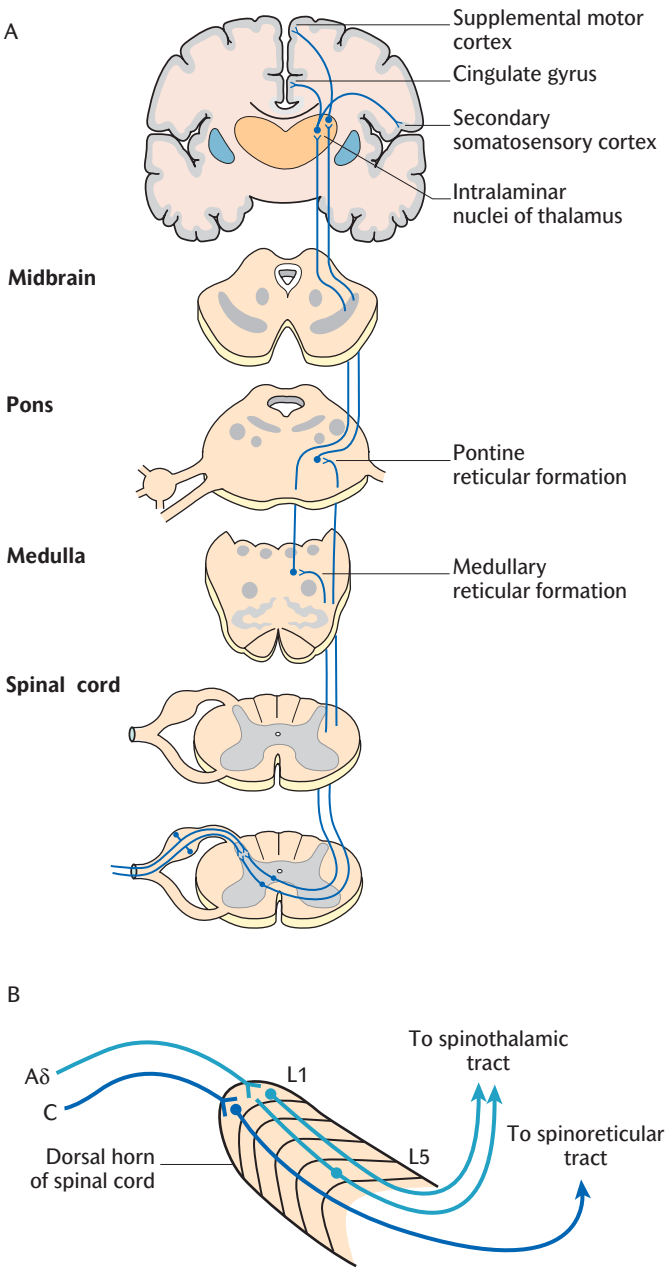


Fig. 16.3 The spinoreticular pain pathways.

tract appears earlier in the course of evolution than the spinothalamic tract and is also referred to as the paleospinothalamic tract to distinguish it from the spinothalamic tract or neospinothalmic tract. As its name implies, the spinothalamic tract or spinoreticular tract does not terminate directly in the thalamus but, as illustrated in Figure 16.3, has intermediate synapses in the medullary and pontine reticular formation. The thalamic projection components of this tract may, therefore, be subdivided into a first secondary neuron from the spinal cord to the reticular formation and a second secondary neuron from the reticular formation to the thalamus. When they reach the thalamus, their axons synapse in the intralaminar nuclei.

Nociceptors served by both Aδ and C processes have wide receptive fields so localization of the stimulus is poor. The posterior and intralaminar nuclei of the thalamus where these stimuli are ultimately directed do not project to the primary somatosensory cortex so they have no somatopical representation which compounds the problem of localization. However, the dual connections of Aδ processes to high threshold nociceptive and wide dynamic range tactile thalamic projection cells enable pain to be localized accurately in most cases.

Central projection of noxious stimuli

If the intralaminar and posterior nuclei of the thalamus do not project to the primary somatosensory cortex, where do they send their tertiary neurons to? Both sets of nuclei connect to widespread areas of the cerebral cortex, including the insula and basal ganglia which is not terribly instructive about the terminal destination of nociceptive pathways. It should also be borne in mind that only about 10% of thalamic neurons relay sensory information of all types to the cerebrum. The other 90% are concerned with controlling how much information and what specific information is allowed through the thalamus. A substantial amount of modulation of noxious stimuli probably takes places here and it is believed that general anaesthetics exert their actions at the thalamic level.

Returning to the destination of nociceptive pathways, recent research involving brain scanning of volunteers has identified the **pain matrix** referred to earlier. The pain matrix is the term used to refer to the different cortical areas that are activated by painful stimuli. As Figure 16.3 shows, these include the **secondary somatosensory cortex** situated just below the primary somatosensory cortex on the underside of the lateral fissure, the **insula**, the **anterior cingulate gyrus**, the **cerebellum**, and **supplementary motor areas**. Taken together with lower connections made from the reticular formation, the role of some of these projections starts to make sense when we think about our reactions to pain. The reticular formation connects to cardiovascular and respiratory centres that can produce changes in blood pressure and respiratory activity in response to pain. The anterior cingulate cortex is part of the limbic system (Section 15.4.2) which is involved in the emotional response to pain. Other connections are made from the reticular formation to the limbic system which is believed to be involved in learning; through experience, we learn which events produce a noxious stimulus and pain and thus how to avoid them. The connections to areas concerned with motor activity planning may be concerned with the responses required to withdraw from the stimulus. The secondary somatosensory cortex and the insula are particularly interesting; the emotional response to pain is reduced if these areas are inoperative but discrimination is preserved. It would appear that several brain structures are required to detect and localize nociceptive stimuli and to perceive these stimuli as pain with all the uncomfortable and emotional ramifications that accompany it. Some aspects of pain peculiar to toothache are outlined in Box 16.1.

Pain modulation

Pain can be modulated or abolished by a variety of pharmacological agents such as analgesics and local and general anaesthetics. Sedation is valuable in the treatment of many over-anxious patients, the root of the anxiety for many being the prospect of potentially painful dental treatment. Pain can be also physiologically modulated by numerous neural mechanisms within the CNS. Hypnotherapy and acupuncture are also

Box 16.1 Toothache

Patients with toothache often have some difficulty identifying the tooth that is giving them pain. They can usually indicate whether it is an upper or lower tooth, whether it is on the right or left side of the mouth, and whether it is a front or back tooth. However, they are usually unable to identify the exact tooth more precisely unless there are other cues such as a large cavity that they can feel with their tongue. Teeth are only innervated by Aδ and C neurons and the dual mechanism operating through Aδ processes most likely operates in the trigeminal nuclei and beyond to give some localization. However, as shown diagrammatically in Figure 16.4, there is often branching of peripheral nerve processes to adjacent teeth and considerable convergence of primary sensory neurons on to thalamic projection neurons in the trigeminal sensory nuclear complex that confuses localization.

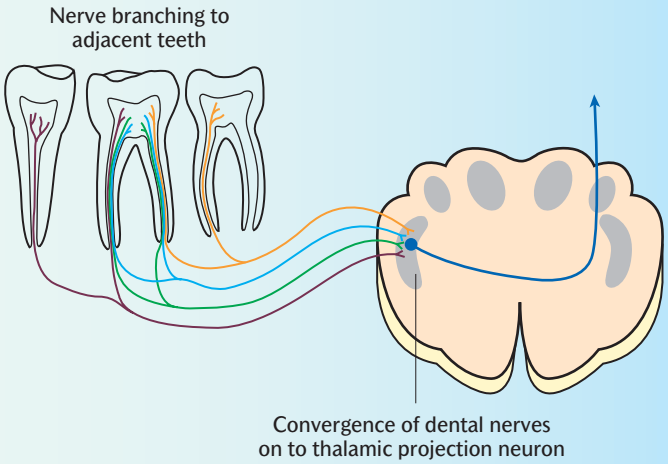


Fig. 16.4 Peripheral branching and central convergence of dental sensory nerves.

used by some dental practitioners with some success for some patients and may stimulate some of these neural mechanisms. One mechanism we are all familiar with is 'rubbing it better' when we receive a painful knock. Aβ sensory nerves carrying discriminative stimuli (see Section 16.2.3) are stimulated by rubbing the painful area. As they enter the dorsal columns, they give off collateral branches which act at high threshold synapses in lamina I of the spinal cord to decrease the likelihood of neurotransmission from primary to thalamic projection neuron and, therefore, the upward conveyance of the stimulus. Pathways descending from the cortex and reticular formation follow the spinothalamic tracts in the reverse direction. These pathways modulate pain and are implicated in the mechanisms underlying acupuncture. Unfortunately, although these mechanisms are present and have been shown to work in experimental situations, no one has yet discovered how to harness them for effective pain relief.

Pain is a complex, but clinically important, subject and many aspects of it are outside the scope of an anatomy book. You should consult textbooks of neurophysiology, neurophysiology, or some of the texts on clinical pain management for more information.

16.2.3 The dorsal column–medial lemniscus pathway

This is the pathway for discriminative touch, proprioception, and vibration and is illustrated in Figure 16.5. Follow this figure as you read the description below and compare it with Figure 16.1 to appreciate the major differences between the dorsal column and the spinothalamic systems. The two pathways differ in:

- The route taken by the central processes of primary sensory neurons once they enter the CNS and as a consequence of this different route;
- The pathway that thalamic projection neurons follow to reach the thalamus.

Proprioceptive receptors include muscle spindles, Golgi tendon organs, and other endings in the capsules and ligaments of the joints. Encapsulated nerve endings are the principal receptors involved in discriminative touch and vibrational sense. Irrespective of the receptor type, the peripheral processes of primary sensory neurons for this pathway are myelinated fast-conducting large-calibre Aβ processes. As Figure 16.5 shows, their cell bodies are in dorsal root ganglia or cranial nerve ganglia so are no different from the general pattern outlined for sensory pathways.

Surprisingly, the central processes of spinal nerve neurons in this pathway do not enter the grey matter of the spinal cord. Instead, they enter the dorsal white columns and divide into long ascending and short ascending and descending branches. As shown in Figure 16.2 and 16.5, the long ascending branches travel upwards without synapsing in the ipsilateral dorsal white column right through the length of the spinal cord to enter the medulla oblongata. New central processes of primary sensory neurons are added to the lateral side of each dorsal column as it passes through each successive segment of the cord. As you may anticipate, there is a massive influx of central processes into the spinal segments where nerves from the arm enter. These are so numerous that they can be recognized as a separate tract in the dorsal column from the mid-thoracic level upwards. As shown in Figure 16.2, the dorsal column is divided into a medial **gracile fasciculus** containing the processes of neurons from the lower half of the body and a lateral **cuneate fasciculus** containing the processes of neurons supplying the upper body and limbs. The short descending and ascending branches synapse with interneurons to mediate spinal reflexes and with projection cells whose ascending axons contribute to the spinocerebellar tracts (see Section 16.2.4).

The long ascending processes forming the two dorsal columns finally synapse in the dorsal column nuclei in the posterior part of the lower medulla as illustrated in Figure 16.5. There are two nuclei on each side, the **gracile nucleus** and **cuneate nucleus,** corresponding to the two columns. Here, the primary sensory neurons synapse with thalamic projection neurons. As you follow the tract further in 16.5, you will see that the axons of the secondary neurons decussate at the level of their nuclei to form the **medial lemniscus** which ascends on each side to the **ventroposterolateral** (VPL)

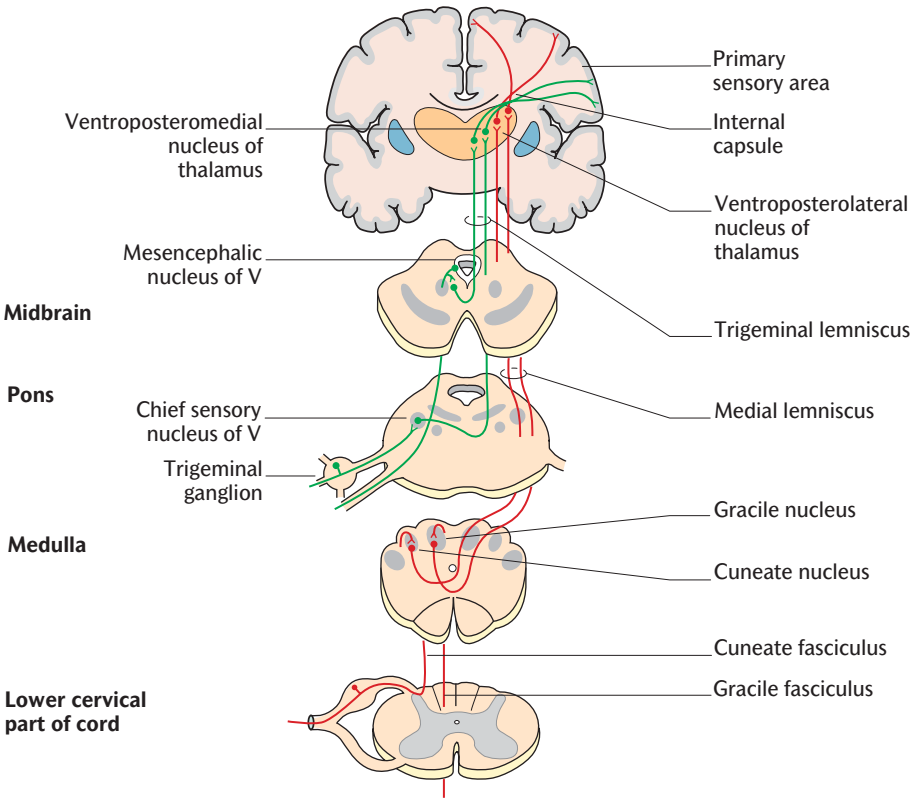


Fig. 16.5 The dorsal column–medial lemniscus pathway for discriminative touch, vibration sensation, and proprioception.

nucleus of the thalamus. The tertiary neurons project to the somatosensory cortex through the posterior limb of the internal capsule.

The long ascending processes in the gracile and cuneate fasciculi are concerned with discriminative touch and vibration sense from the whole body. There is comparatively little convergence so stimuli are transmitted very faithfully throughout the whole pathway, thus preserving fine discrimination. By analysing stimuli travelling through the dorsal column pathway, the primary sensory cortex is able to determine aspects of discriminative touch such as texture; **stereognosis,** the ability to determine the shape of an object by touch, can be achieved by combining tactile and proprioceptive information, an invaluable skill for dentists.

Figure 16.5 illustrates the primary sensory neurons forming the first part of the pathways for discriminative touch and proprioception in the head and neck; they are carried mainly in the trigeminal nerves, the major sensory nerves to the head. The cell bodies of primary sensory neurons for discriminative touch are contained within the trigeminal ganglion which corresponds to the dorsal root ganglion in a spinal nerve. The central processes enter the brainstem to synapse in the **chief sensory nucleus of the trigeminal** on each side (see Figure 18.8). Sensory processes from the glossopharyngeal and vagus nerves that carry the discriminative supply from other structures in the head and neck also terminate in the same trigeminal nucleus. Follow Figure 16.5 and trace the axons of the thalamic projection neurons as they cross to the opposite side of the brainstem to form the **trigeminal lemniscus** medial to the medial lemniscus carrying information from the body. As in the spinothalamic tract, information from the body and head is segregated in the brainstem and this separation is maintained in the thalamus. The trigeminal lemniscus terminates in the **ventroposteromedial** (VPM) **nucleus** of the thalamus. Tertiary neurons project from the thalamus through the internal capsule to the head region of the somatosensory cortex.

Box 16.2 describes the effects of damage to and disease of the sensory pathways.

Box 16.2 The effects of disease or damage on the sensory pathways

As we have seen, the two major sensory pathways follow different routes in the spinal cord and brainstem. In certain diseases it is possible for one pathway to be damaged while the other pathway is unaffected. **Syringomyelia** is a disease in which the central canal of the spinal cord, usually the cervical area, becomes enlarged. This leads to compression of the axons of thalamic projection neurons of the spinothalamic system as they decussate just in front of the spinal canal. Noxious and temperature sensations from the upper limbs are lost bilaterally; this has potentially severe consequences because the sufferer lacks the protective effects of these sensory modalities and may, e.g. burn their hand without even realizing it.

Although touch and pressure sensations are also lost, their effects are less noticeable because discriminative touch, carried in the dorsal columns, is still intact and there is some functional overlap between the different types of touch neurons.

The most frequent cause of lesions that affect the dorsal columns without affecting the spinothalamic tracts are demyelinating diseases, particularly **multiple sclerosis**. Once again, the cervical region of the spinal cord seems most vulnerable; the fasciculus cuneatus is usually affected, producing serious defects in proprioception and discriminative touch from the upper limbs. This seriously impairs manual dexterity and stereognosis. The medial lemniscus pathways may also be interrupted in the brainstem as a result of demyelinating disease, diminished blood supply, or trauma; the effects will be seen at all levels *below* the lesion.

If the spinal cord is damaged by disease or trauma, the passage of information for all types of sensation will be interrupted at the level of the injury; sensation is thus lost below this point. Partial section affecting only one half of the cord is rare, but its effects are interesting and reinforce the differences between the two major sensory pathways. On the side of the injury, the *uncrossed* axons of the primary sensory neurons of the medial lemniscus system ascending in the dorsal column will be interrupted and so will the *crossed* thalamic projection neurons originating from the contralateral side of the spinal cord ascending in the spinothalamic tract. Thus, discriminative touch and positional sense will be lost on the side of the hemisection whereas noxious and temperature sensation will be absent on the opposite side. Hemisection will also interrupt the descending pathways described in the next section with consequent effects on the muscles on the affected side of the cord below the level of the lesion. This combination of sensory and motor defects is known as the **Brown–Sequard syndrome**.

The somatotopic pattern of body representation is maintained as the thalamocortical neurons pass through the internal capsule and corona radiata so that neurons originating from the same part of the body, but ascending through the two major sensory pathways travel together from thalamus to cortex. Lesions of the ascending pathways at this level will affect all modalities of sensation from a particular part of the body, but on the contralateral side as both pathways have crossed by this stage. A CVA (stroke) in one of the perforating arteries is a relatively common cause of sensory disruption (see Box 15.10). The internal capsule will be deprived of its blood supply with consequent interruption of the processes ascending and descending through it. The precise area affected will depend upon the size and position of the lesion.

16.2.4 Spinocerebellar pathways

The spinocerebellar pathways carry proprioceptive information from peripheral muscles to the cerebellum. As illustrated in Figure 16.2, two tracts, the dorsal and ventral pathways, are present on each side of the spinal cord. The complete tract is illustrated in Figure 16.6. Notice that there are only two principal neurons in each pathway.

As outlined in the description of the dorsal column pathway, primary sensory neurons originate from muscle spindles and other proprioceptive receptors in joints and associated structures; their cell bodies are located in dorsal root ganglia. The central processes of these primary sensory neurons branch into long ascending axons and short ascending and descending branches. The long ascending axons continue upwards, forming the dorsal column pathways described in the previous section. The short ascending and descending branches synapse in lamina VII of the spinal grey matter (see Figure 15.8) with neurons that form the dorsal spinocerebellar tract as shown in Figure 16.6. The secondary neurons form the dorsal spinocerebellar pathway close to the dorsolateral surface of the spinal cord. As illustrated in Figure16.6, the **dorsal spinocerebellar tract** travels ipsilaterally to enter the cerebellum through the inferior cerebellar peduncles.

The **ventral spinocerebellar tract** is directly anterior to the dorsal spinocerebellar tract as shown in Figure 16.2. Many primary sensory neurons synapse with lower motor neurons to form reflex arcs, controlling muscle response to sensory stimuli at the spinal level (see Section 16.3.1). Collateral axons from the central processes of these primary sensory neurons synapse in lamina VII. The secondary neurons decussate to form the contralateral ventral spinocerebellar tracts as shown in Figure 16.6.

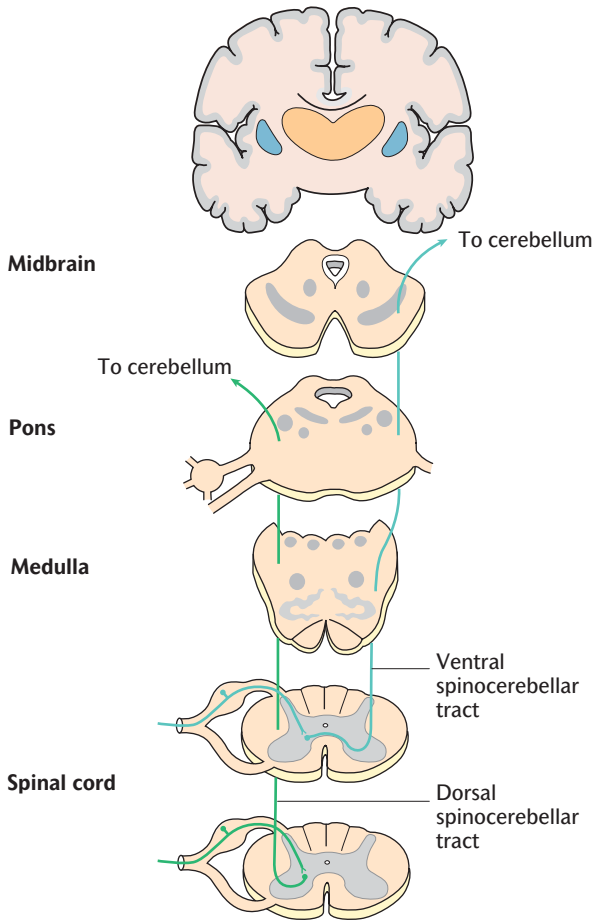


Fig. 16.6 The spinocerebellar pathways.

Box 16.3 Friedreich's ataxia

Friedreich's ataxia is an inherited recessive neurodegenerative disease that usually begins in childhood. The spinocerebellar tracts are affected in the early stages of the disease. There is a wide-based staggering gait (**ataxia**) and a marked **intention tremor** when the patient attempts a manoeuvre, especially with their hands or fingers. As the disease progresses, other structures in the CNS are affected; progressive cerebellar degeneration occurs and the trigeminal and vagus nerves demyelinate.

As can be seen, these neurons enter the cerebellum through the superior cerebellar peduncles and supply the cerebellum with information on spinal reflexes.

The effects of damage or disease on the spinocerebellar tracts are described in Box 16.3.

Proprioception from the head

The proprioceptive pathways in the cranial region are not completely understood. In the trigeminal nerve, the primary sensory neurons for proprioception are unique primary sensory neurons because their cell bodies are located *within* the CNS and not in the sensory ganglion of the nerve. They are found in the **mesencephalic nucleus** of the trigeminal nerve where neurons conveying proprioceptive information from muscles of mastication, the temporomandibular joint, and periodontal tissues supplied by the fifth cranial nerves synapse. Furthermore, electrophysiological evidence suggests that proprioceptive information from muscle spindles in the extraocular muscles supplied by the third, fourth, and sixth cranial nerves, and possibly also from the facial, laryngeal, and lingual muscles supplied by the seventh, tenth, and twelfth cranial nerves, respectively, all terminate in the trigeminal mesencephalic nucleus as well (see Section 18.6.1). Central branches of the neurons in the mesencephalic nucleus connect with cells in the motor nucleus of the trigeminal nerve for reflex actions to the thalamus via the trigeminal lemniscus and the cerebellum.

16.3 Motor pathways

The study and practice of dentistry involve learning lots of new and exciting subjects. These include the exploration of the structure and function of the human body covered in this book with subsequent application of this knowledge to surgery and disease processes. You will also learn new practical skills at the same time as you study anatomy or when you have successfully completed that and other associated courses, depending on the structure of your particular dental course. As you master these manual skills, you will be able to administer local anaesthetics, cut and fill cavities during restorative treatment, and carry out surgical procedures such as tooth extraction or gingival surgery, to mention but a few. Acquisition of the manual skills necessary for successful dental practice is dependent upon motor functions within your CNS and PNS.

Most motor skills have to be learned, but many will eventually become second nature. You will be unable to recall the trials of tribulations involved in acquiring some essential motor skills, but your parents will be able to regale you with stories of your infantile mishaps and errors as you tried to walk and talk, things you now take for granted. As we have already seen, the CNS is hierarchical with the complexity of function increasing as we progress from the spinal cord to the brainstem to the diencephalon to the cortex. This hierarchy applies just as well to motor activity as any other neural functions.

Motor function in the spinal cord involves control of basic muscle function and some simple reflexes. One aspect of motor function that is often overlooked is the control and maintenance of posture. A secure posture provides the firm platform on which other voluntary movements are anchored. Posture is controlled by **medial motor pathways**; some of these pathways descend from cortical locations whereas others originate from brainstem structures. Voluntary movement is regulated by the primary motor cortex and its associated cortical areas through **lateral motor pathways.** Other cortical structures also regulate motor reflexes and postural activities as well as playing a major role in learning and memorizing new motor skills; the **basal ganglia** are key structures in these functions as is the brainstem structure, the **cerebellum.**

16.3.1 Spinal motor activity

As described in Chapter 2, muscles that produce a particular movement at a specific joint are the **agonists** whereas those that produce the opposite movement are **antagonists**. Most people are familiar with the biceps muscle on the front of the upper arm that flexes the elbow. Its antagonist muscle is the triceps on the back of the upper arm which extends the elbow. Biceps is the agonist and triceps is the antagonist when you flex your elbow, but the roles are reversed when you extend it; triceps becomes the agonist while biceps becomes the antagonist.

Most muscles in the body are not limp when not being used, but minimally contracting to maintain **muscle tone**. If you wish to use a muscle as an agonist, its antagonist must be prevented (inhibited) from contracting. **Reciprocal inhibition** is one of the reflexes that occur at the spinal level; incoming activity from proprioceptors in the agonist inhibits activity in the antagonist. As soon as the antagonist becomes active, the agonist is now inhibited as their roles reverse. If you could make this switch happen automatically and repeatedly, you could generate a pattern of activity. This is exactly what happens as you walk. You stand on one leg using mainly extensor muscles while moving the other leg forward using flexor muscles. As the legs change from swinging to standing, the opposite sets of muscles become active. The alternating phases of activity are produced by **central pattern generators** formed from networks of spinal interneurons. Basically, one group of muscles is stimulated while another is inhibited, then the pattern generator switches off the first group and switches the second group on. Activity of spinal central pattern generators for locomotion is initiated from the medullary reticular system and carried by the reticulospinal tracts (see p. 148). This demonstrates how one part of the CNS cannot act

in isolation. The basic rhythms set up by medullary and spinal pattern generators can be modified by higher cortical areas. If you see your bus approaching the stop, you will quickly change from a leisurely walk and break into a run to attempt to catch it.

16.3.2 Posture

Postural mechanisms stabilize the body so that it can act as a steady platform for other activities. The weight bearing components of the skeleton, the lower limbs, pelvis, and vertebral column are arranged so that an upright posture can be maintained using minimal muscular force and, therefore, less energy. The joints of the lower limb 'lock' when the optimum position is attained and the muscles can then relax. Every schoolboy knows this without necessarily understanding the underlying mechanisms. Our schoolboy notices another boy standing at ease and sneaks up behind him. He gives an unexpected push behind the knee of his victim, disrupting the optimum 'locked' position; the victim begins to fall, but equilibrium is quickly restored as reflex mechanisms stimulate the requisite muscles to contract to restore the optimum position.

This familiar schoolboy trick is only one of the various forces that can upset posture and destabilize the body. Some of these will be unexpected such as the example above or when you trip over a raised paving stone. Other destabilizing movements will be expected as you pivot exquisitely on your left leg to kick the ball with your right leg into the net for that crucial winning goal. In the unexpected case, postural reflexes will right the body, if possible, often before you are even aware of what has disrupted your posture. In the second scenario, you have already learned the required skills and your brain anticipates what is necessary to maintain posture because you have mastered the complex movements.

Posture is vitally important to dentists because without good stable posture, it is very difficult to carry out complex skilled movements accurately and continuously. Back pain is an occupational hazard of dentists as they are required to sit over their patients for long periods of time while carrying out treatment. Although the detailed anatomy of the vertebral column is outside the scope of this book, it is designed for efficient posture. Placing the vertebral column or any other joint, for that matter, in awkward positions for any length of time does the joints, the associated muscles, and their proprioceptive mechanisms no good at all. This may not be apparent to a young and fit dental student, but the effects are often cumulative rather than of sudden onset; back pain does not necessarily manifest itself until well into a dental career. The sooner habits of good posture are developed, the better are the prospects for a long, successful practice life, free of back trouble.

The major sensory inputs enabling us to execute postural reflexes are from:

- The vestibular apparatus in the inner ear;
- Proprioception from muscles and joints;
- Visual cues from the eye via the tectum of the midbrain.

The pathways that convey these inputs to lower motor neurons travel through the anterior white matter close to the midline as shown in Figure 16.7. These pathways are known collectively as the **medial motor pathways** and their course is illustrated in Figure 16.8.

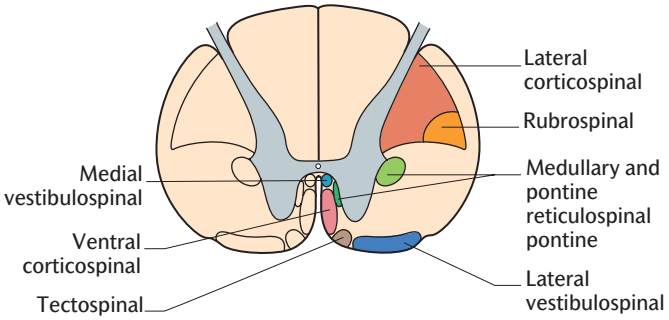


Fig. 16.7 A cross section of the spinal cord to show the positions of the major motor pathways.

The tectospinal pathways

The **tectospinal pathways** originate from the superior colliculi of the midbrain as shown in Figure 16.8. Inputs are from the axon collaterals of neurons, forming the visual pathways. Each tract crosses over and descends in the anterior white matter to the cervical segments of the spinal cord. For postural control, these pathways adjust head position so that the eyes maintain their usual position of looking forward and being level with each other. They are involved in the coordination of head movements to track visual stimuli moving across the visual fields.

The vestibulospinal pathways

The vestibular apparatus in the inner ear consists of the utricle and saccule that monitor static head position and the semicircular canals that monitor movement of the head in three dimensions. Sensory nerves from these inner ear components form the vestibular part of the eighth cranial nerve, the vestibulocochlear nerve (see Section 18.8.2). Most of the vestibular nerve processes synapse in the **vestibular nuclei** of the brainstem but some pass directly to the cerebellum. Axons from cells in the vestibular nuclei make connections with the cerebellum, spinal cord, brainstem, and cerebral cortex.

Neurons from the vestibular nuclei connect with the abducens, trochlear, and oculomotor nuclei in the brainstem; movements of the eyes are coordinated through these connections to maintain visual fixation (see Section 18.5). The cerebellar projections maintain equilibrium through the influence that the cerebellum exerts on muscle tone (see p. 150). Neurons from the vestibular nuclei descend in the **vestibulospinal tracts** in the ventral white column of the spinal cord illustrated in Figure 16.7 and 16.8. Bilateral projections to the cervical segments of the spinal cord excite muscles on the same side while inhibiting contralateral muscles to adjust head position to accommodate to movement.

One force that tends to disrupt posture is gravity; the pull of gravity should make us collapse in a heap. **Antigravity muscles** resist gravitational forces and are stronger than the muscles that act with gravitational pull. The major antigravity muscles are the extensors of the vertebral column that keep your back straight and extensors of the lower limbs, particularly the quadriceps muscles forming the large muscle masses on the front of your thighs; these muscles extend and lock the knees. Follow the vestibulospinal tracts in Figure 16.8 as they

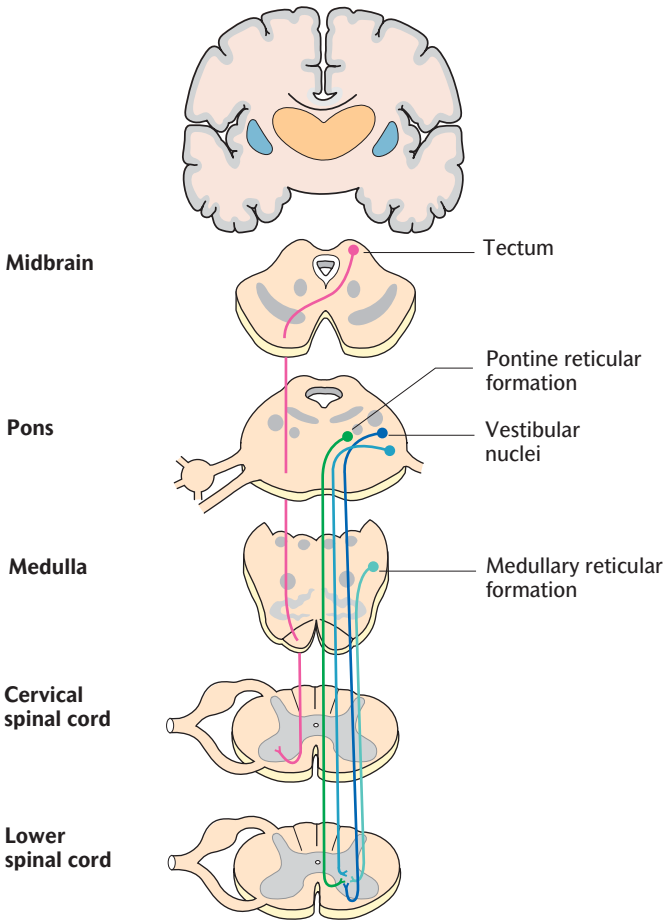


Fig. 16.8 The medial motor pathways. The tectospinal pathway is illustrated on the right side and the vestibulospinal and reticulospinal pathways on the left side for clarity.

project ipsilaterally to the lower segments of the spinal cord and stimulate the antigravity extensor muscles while inhibiting flexor muscles to maintain posture.

The reticulospinal tracts

Reticulospinal tracts are also involved in maintenance of posture. These tracts arise from the reticular nuclei in the brainstem which receive inputs from muscle spindles and joint receptors, particularly those from the intervertebral joints. There are two reticulospinal tracts on each side of the spinal cord as shown in Figure 16.7 and 8. The **medial reticulospinal tract** from the medullary reticular formation excites extensor, but inhibits flexor muscles. The **lateral reticulospinal tract** originates from the pontine part of the reticular system; it receives inputs from the midbrain region, controlling locomotor patterns and projects to local central pattern generators at different levels of the spinal cord. Reticulospinal pathways are also involved in the rhythmic control of inspiration and expiration during ventilation.

Postural adjustment in anticipation of a movement which we know is going to disturb posture has to be learned. These so-called feedforward mechanisms are complex and involve inputs from the motor cortex and cerebellum (see p. 150).

16.3.3 Voluntary movement

Voluntary movements are those made in response to sensory stimuli or higher cognitive processes. The majority of voluntary movements are highly skilled, requiring a significant number of muscle groups and individual muscles to cooperate in moving various joints in a given sequence with the correct amount of force applied at the appropriate rate and moving over the required range to achieve the intended outcome.

The motor cortex

As already outlined in Chapters 3 and 15, the **primary motor cortex (M1)** is situated in the precentral gyrus in the posterior part of the frontal lobe. The opposite side of the body is represented somatopically. There are two other components of the motor cortex; the **premotor (PM) cortex** and **supplemental motor area (SMA)**. These are shown in Figure 15.18A. The PM is concerned with initiation of movement and receives a large input from the parietal association cortex that receives and associates visual and somatosensory information; the PM is thus wired to initiate movement in response to specific visual or sensory cues, often called goal-directed movement. The SMA is involved with sequencing movement before specific muscle groups and individual muscles are recruited by M1. PM and especially SMA have somatotopic representations of both halves of the body, which enables coordinated movement of both sides. If a person is asked to perform a movement while their brain is scanned, M1 is active when they make a simple movement; all three motor areas become activated in the sequence PM–SM–M1 if they make a more complex movement; if they are asked to imagine and mentally rehearse making the movement without actually performing it, only PM is active.

The lateral (corticospinal and corticonuclear) pathways

These pathways play a pivotal role in the voluntary control of movement; as shown in Figure 16.7, they ultimately travel through the lateral white matter of the spinal cord in contrast to the medial pathways—hence their name. They begin in the **motor areas**, but processes from adjacent zones of the cerebral cortex, particularly the parietal lobe, also contribute to them.

Recall that the primary motor cortex is somatopically arranged as shown in Figure 15.16; the area controlling muscles of the limb is most superior, followed by the trunk and upper limb areas with the head area at the lower end of the precentral gyrus just above the lateral fissure. Axons from the limb and trunk areas join to form the **corticospinal tracts**. Axons from the head region of the primary motor area form the **corticonuclear (**or **corticobulbar) tracts**. Both tracts are illustrated in Figure 16.9. Note that they share a common pathway initially through the corona radiata and the posterior limb of the internal capsule to enter the midbrain through the cerebral peduncles; they maintain their somatotopic arrangement as they pass from cortex to brainstem. These two pathways constitute the **upper motor neurons**, a term still in current clinical use.

The corticospinal tracts

The **corticospinal tracts** form several discrete bundles as they pass through the pons. As shown in Figure 16.9, they reunite in the medulla to form the **pyramidal tracts**, demarcated by the two

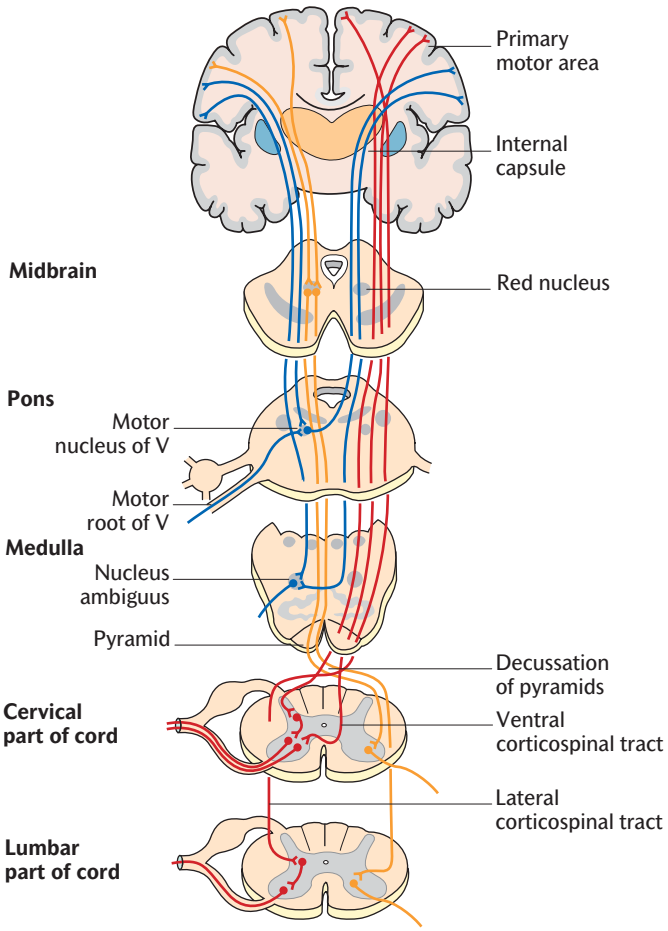


Fig. 16.9 The corticospinal, corticobulbar and rubrospinal motor pathways. For clarity corticospinal pathways (red) are only shown originating from the left hemisphere and the rubrospinal pathways (orange) from the right side. Corticonuclear pathways (blue) are shown originating from both sides.

prominent ridges either side of the anterior midline. At the lower end of the medulla, about 80% of the axons decussate in the pyramidal decussation and continue downwards into the spinal cord as the **lateral corticospinal tracts.** These tracts terminate in the ventral horn of the spinal cord where they synapse with alpha and gamma motor neurons, mostly through interneurons, although some synapse directly with lower motor neurons, especially those innervating muscles in the distal aspect of the limbs involved in fine skilled movements.

The remaining 20% of axons in the pyramidal tracts continue downwards in the ipsilateral ventral white matter as the **ventral corticospinal tracts** and only cross to the opposite side at the level they synapse with lower motor neurons. Ultimately therefore, all corticospinal tracts supply lower motor neurons on the contralateral side of the body. Corticospinal axons have many collateral branches as they descend from the cortex past the diencephalon and through the brainstem; these connect to the thalamus, basal ganglia, red nucleus, pontine nuclei, and the reticular formation of the brainstem.

The corticonuclear pathways

The **corticonuclear tracts** descend through the brainstem on the medial side of the corticospinal tract. They rapidly decrease in size as axon bundles leave the tracts to supply the motor nuclei of different cranial nerves at various levels of the brainstem. Like corticospinal neurons, most corticonuclear axons connect through interneurons with the lower motor neurons forming the motor component of the relevant cranial nerves, but some synapse directly with motor neurons. Some corticonuclear axons make ipsilateral connections to the motor nuclei of the cranial nerves, but also cross to the opposite side at the level of the nuclei in question. This means that each cranial nerve motor nucleus receives inputs from *both* motor cortices (see Figure 18.6). There are two exceptions to this bilateral pattern of supply which will be described more fully in Chapter 18. Briefly, they are the lower part of the motor nuclei of the facial nerves and the hypoglossal nuclei. The hypoglossal nuclei are at the level of the pyramidal decussation and the connections are predominantly, if not exclusively, from the contralateral corticonuclear tract. The bilateral innervation of the cranial nerve motor nuclei produces different effects after damage to the corticonuclear tracts from those seen after damage to corticospinal pathways to the ventral horn of the spinal cord (see Box 18.4 and Figure 18.6).

The neurons forming the corticonuclear and corticospinal tracts begin in the cortex and end in a motor nucleus of a cranial nerve in the brainstem or in the ventral horn of the spinal cord; these are the **upper motor neurons**. As you can see if you follow either of these tracts to their destinations in the brainstem or spinal cord in Figure 16.9, a second neuron beginning in a brainstem motor nucleus or the ventral horn of the cord is required to convey the information to the muscle being innervated. This is the **lower motor neuron** and forms the motor component in the peripheral cranial or spinal nerve to the muscle. The term 'upper motor neuron' now includes other descending pathways involved in the voluntary control of muscle action, not just the corticospinal or corticobulbar tracts.

The rubrospinal pathways

Some neurons from the motor areas travel in the corticospinal and corticonuclear tracts only as far as the **red nuclei** in the midbrain where they synapse. Other axons which continue downwards also send collateral axons to the red nuclei. The neuronal processes that arise in the red nuclei form the **rubrospinal tracts**. These travel with the corticonuclear and corticospinal tracts in humans. The two tracts appear to have subtly different functions in control of movement. The rubrospinal tracts carry out learned voluntary movements that have become automated whereas the corticospinal pathways are used as we are learning such actions. When a skilled sequence of movements has been mastered, activity switches from the corticospinal tracts to the rubrospinal tracts. Activity can be switched the other way when a learned movement requires modification. The switch is in the **inferior olivary nuclei** in the medulla (see p. 151).

The effects of damage to upper and lower motor neurons have already been outlined in Section 3.4.3 and illustrated in Figure 3.13. The subject is revisited in Box 16.4.

Box 16.4 Upper and lower motor neuron injuries

Disorders of movement take many forms. The simplest example is that produced by a lesion affecting **lower motor neurons**. These neurons are the final common pathway through which information from all levels of the CNS is transmitted to the muscles. A lower motor neuron lesion effectively denervates the associated muscles, producing **flaccid paralysis** characterized by muscle flaccidity and absent tendon reflexes. If lower motor neurons are damaged beyond repair and do not regenerate, after about 6 months the muscles begin to atrophy, producing **muscle wasting**. Muscular wasting may produce cosmetic defects as well as function deficits if the affected muscle is superficial. In the longer term, after about a year, denervated muscles degenerate and are replaced by fibrous tissue. The fibrous tissue shrinks by about 20% of its length as it forms. This has the equivalent effect of the muscle contracting by the same length and produces a fixed **contracture** of the muscle. Contracture is not only functionally debilitating, but can cause serious cosmetic problems, especially in areas such as the face where contracture is obvious.

Upper motor neuron lesions produce absence of voluntary movement with increased muscle tone (**spastic paralysis**) and enhanced tendon reflexes (**hyperreflexia**) or weak voluntary movements of the muscles (**paresis**); wasting of the muscles is not marked. Dentists are most likely to encounter patients with upper motor neuron lesions due to the effects of a CVA (a 'stroke') disrupting the blood supply to the lateral motor pathways; CVAs occurring in the posterior limb of the internal capsule often affect these pathways. After the initial effects wear off, the victim is left with some degree of spastic paralysis and hyperreflexia on the *opposite* side of the body to which the lesion occurred due to the crossing of corticospinal pathways. The flexor muscles are stronger than the extensors in the upper limb while the reverse is true in the lower limb. If spastic paralysis affects the lower limb, a characteristic gait is produced in which the leg is swung forwards with the knee and ankle joint extended; in other words, the leg is kept straight. If the arm is affected, it is carried with the elbow flexed and the forearm held across the chest and the hand tightly bunched into a fist.

The reason that spastic paralysis is observed in upper motor neuron lesions is because proprioceptive information from muscle spindles in the affected muscles can still reach the spinal cord through the intact peripheral spinal nerve and form reflex connections with the lower motor neurons. However, this information cannot be modified by inputs from higher centres in the brain; the unmodified proprioceptive information produces the characteristic spastic paralysis and hyperreflexia.

It was, at one time, thought that **upper motor neuron lesions** were caused specifically by interruption of the lateral motor pathways. However, experiments in animals have indicated that damage to these pathways only produces reduced tone in the contralateral muscles with little or no effect upon the tendon reflexes. The principal deficit appears to be clumsiness in the use of the contralateral limbs. Lesions limited to the corticospinal pathways are rare in humans; clinical observations suggest that they produce deficits similar to those in experimental animals when they do occur. This has led to the view that the principal function of the corticospinal and corticobulbar pathways is in the control of highly skilled movements. It is now known that upper motor neuron lesions also involve corticoreticular pathways which, although functionally part of the medial motor pathways, travel from the motor cortex to the brainstem reticular nuclei alongside the lateral motor pathways. Corticoreticular pathways are, therefore, also involved in lesions such as internal capsule CVA. One function of reticulospinal pathways is to act to inhibit spinal reflexes. When this action is lost, the result is hyperreflexia and spasticity.

The outcome from **upper motor neuron lesions** affecting the **corticonuclear pathways** is somewhat different and is described in Box 18.4.

16.3.4 The role of subcortical structures in motor control

The voluntary control of muscle action is still not fully understood. Electrical stimulation of M1 produces much simpler movements than normal voluntary movements under conscious control, indicating that many more areas of the cortex are involved in motor activity. **Closed motor loops** between the motor areas, basal ganglia, cerebellum, and thalamus play an important role in initiation and coordination of complex movement sequences although their precise functions are still being clarified.

One closed loop originates in the SMA which projects to the basal ganglia and these in turn project back via the thalamus to the SMA. Another loop originates in the motor areas and travels with the corticospinal and corticonuclear pathways. These pathways give off collateral axons that synapse in the pontine nuclei; the pontine nuclei project to the cerebellum that in turn sends neuronal processes back to M1 and PM via the thalamus.

The cerebellum

The **cerebellum** plays an important part in the maintenance of equilibrium and posture and the control of muscle tone and coordination of muscle activity. Through its connections with the motor cortical areas, the cerebellum ensures that voluntary movements are carried out smoothly and precisely in the correct sequence, using the necessary rate, range, and force of muscle contractions to execute a given movement. It also plays a vital part in learning motor skills and probably has some role in cognitive activities.

The cerebellum receives inputs from:

- The vestibular, visual, and auditory system via the midbrain colliculi;
- The proprioceptive endings in muscles, tendons, and joints that supply information about the state of muscle contraction and joint position

Box 16.5 Cerebellar dysfunction

There are many causes of cerebellar dysfunction, ranging from tumours or other space-occupying lesions, traumatic head injuries, degenerative diseases such as multiple sclerosis or Friedrich's ataxia (Box 16.3) that affects spinocerebellar tracts and the cerebellum specifically. Alcoholic intoxication interferes with cerebellar function and we have all probably observed or may be experienced these short-term effects at some time. An inebriated person has difficulty standing, walks with a wide-based staggering gait, has slurred speech, and often 'misses' when trying to perform actions such as putting a key into a lock. These signs and symptoms sum up the major effects of cerebellar disease or damage.

Most of the causes of cerebellar damage listed above affect the whole cerebellum, but localized tumours give insight into the effects of disease and damage on the subdivisions of the cerebellum. Vestibulocerebellar damage is responsible for the staggering gait (**cerebellar ataxia**) and a rapid side-to-side eye movement (**nystagmus**). A patient with spinocerebellar damage has extreme difficulty with walking and standing, thus keeps falling. They also cannot coordinate the complex motor activities required for speech which becomes slurred as a result (**dysarthria**). Another manifestation of spinocerebellar dysfunction is **intention tremor,** large amplitude shaking when a movement is attempted, resulting in 'misses' during motor activity. Damage to the cerebrocerebellum causes delay and overshoot of movement that is most noticeable with complex movement involving several joints.

Bear in mind that in the majority of cases involving cerebellar dysfunction, all components of the cerebellum are affected so the patient is likely to show many of the signs and symptoms described above. **Charcot's triad** of nystagmus, dysarthria, and intention tremor is strongly indicative of disease affecting cerebellar function.

through spinocerebellar pathways and corresponding pathways from cranial nerves; these pathways also carry other sensory information, including discriminative touch;

- The cerebral motor cortex via the pontine nuclei;
- A relatively small, but nevertheless important, input is from the inferior olivary nuclei which receives inputs from the red nuclei, motor cortex, and spinal cord.

By computing the information from all these sources, the cerebellum feeds back to motor areas of the brain and into both the lateral and medial motor pathways to ensure smooth coordinated muscular activity.

Three functional parts of the cerebellum can be distinguished by their connections, activities, and their order of appearance in the evolutionary scale.

The evolutionary oldest part is the **vestibulocerebellum** (or archeocerebellum) which occupies a small area known as the flocculonodular lobe. As its name suggests, it receives direct inputs from the vestibular apparatus in the inner ear and sends outputs to the vestibular nuclei in the brainstem from which the vestibulospinal tracts arise; these are part of the medial pathways as described on p. 147. It also connects with reticulospinal components of the medial pathways.

The next oldest part is the **spinocerebellum** (or paleocerebellum) which occupies the midline area of the cerebellar cortex. As might be anticipated from its name, this area receives inputs from dorsal spinocerebellar tracts carrying proprioception and touch sensations and ventral spinocerebellar tracts conveying information from spinal motor circuits. It also receives inputs from several other sources, including the vestibular apparatus, visual and auditory pathways, and a massive input from the pontine nuclei carrying information from the motor and sensory cortices. This is also the area that receives inputs from the inferior olivary nuclei. The output from the spinocerebellum operates via two routes. One goes to the vestibular nuclei and hence to the medial motor pathways. The other output projects back to the motor cortical areas via the thalamus and to the red nuclei; both routes affect motor output through the lateral motor pathways.

The lateral parts of each cerebellar hemisphere form the **cerebrocerebellum** (or neocerebellum). Once again, the name of this area indicates where it receives its major inputs from; they are from the cerebral cortex via the pontine nuclei. Note that inputs via this route are not restricted to motor information; there is a considerable input from cerebral areas concerned with cognitive activity. The output is to the motor areas of the cerebral cortex via the thalamus and to lateral motor pathways via the red nuclei. The output also goes to cortical areas involved in cognitive functions.

Essentially, the cerebellum computes information from many sensory sources and compares it with the intentions of the motor systems. The actions of the motor system at all levels from spinal activity to motor cortex can be modified to correct errors of specific movements. Over a longer period, these mechanisms operate during learning of new motor tasks.

Box 16.5 describes the effects of disease and trauma on cerebellar function.

The basal nuclei (basal ganglia)

The precise functions of the basal nuclei and their connections are still being discovered, but clinical findings show that they play essential roles in the control of motor activity. As indicated above, the basal nuclei are one of a group of structures connected to the cortical motor areas through closed motor loops. In this case, inputs from the motor cortical areas enter the basal nuclei and then pass back via the thalamus to the motor areas.

The basal nuclei comprise the caudate and lentiform nuclei in the deep parts of each cerebral hemisphere (see Figure 15.20 and 15.21), the subthalamic nuclei in the diencephalon, and the substantia nigra in the midbrain. There are several complex interconnections between the cerebral cortex and different components of the basal nuclei, creating several separate loops. Two of these loops are directly involved with

Box 16.6 Basal nuclei dysfunctions

The functions of the basal nuclei are not fully understood, but they appear to amplify the intended movement while suppressing extraneous movement. Involuntary movements (**dyskinesia**), abnormal motor activity, and alterations in muscle tone characterize lesions of the basal nuclei and related nuclei, usually due to degenerative diseases. Dyskinesia includes intermittent, purposeless twitching of individual muscles (**choreiform movements**), slow sinuous writhing most obvious in the limbs (**athetoid movements**), or rapid short amplitude movements of the limb extremities at rest (**tremors**). These involuntary movements are believed to arise as a result of the inhibitory pathways in the basal ganglia failing to operate efficiently to suppress unwanted movement. Abnormalities of movement usually show as slowness (**bradykinesia**) or weak and incomplete actions known as **poverty of movement** or **hypokinesia** because the intended movement is not amplified.

Parkinson's disease is due to progressive degeneration in the substantia nigra. It produces tremor in the extremities, most noticeably the hands, accompanied by poverty of movement and muscular rigidity. The patient has a shuffling gait, stooped posture, lack of facial expression, and often fails to complete verbal statements because of the hypokinesia. **Huntington's disease** is progressive degeneration of the basal ganglia, usually the caudate nucleus and putamen, characterized by choreiform movement and progressive dementia.

motor function; one is involved in the overall control of motor activity and the other is specifically concerned with eye movements. The other loops are involved in emotion, memory, and cognitive functions. The numerous interconnections of the individual basal nuclei with each other and with other areas of the brain indicate that much integrative activity occurs in these structures.

The closed motor loop concerned specifically with movement has two parallel circuits that involve neurons originating from the **substantia nigra** that use **dopamine** as their neurotransmitter. These act on two populations of neurons that look identical, but carry two different dopamine receptors; one receptor excites the cells whereas the other inhibits them. It appears that the excitatory loop amplifies movement intentions signalled from the motor cortex whereas the other circuit inhibits extraneous unintended movement. The effects of damage or disease on the basal nuclei are described in Box 16.6.

17

The autonomic nervous system

Chapter contents

17.1 Introduction

A large part of the nervous system is dedicated to the control of the internal viscera and their functions. Much of the activity of these organs is controlled reflexly at the brainstem level, e.g. the **cardiovascular** and **respiratory centres** (the vital centres) in the reticular formation of the medulla controlling cardiac and respiratory activity. There are also centres in the cerebrum, notably the **hypothalamus** in the diencephalon. Somatic and visceral functions are closely integrated at these higher levels; think of the effect that emotional factors or somatic stimulation can have on heart rate, blood pressure, and gastrointestinal activity when we are nervous or are in pain. The nerves involved in these activities are described as **visceral sensory** or **visceral motor** nerves because they control visceral function; this distinguishes them from somatic sensory nerves from peripheral receptors and somatic motor nerves controlling voluntary function.

17.2 Visceral motor neurons

Visceral motor neurons innervate smooth muscle and secretory cells of the gastrointestinal and respiratory systems, the smooth and cardiac muscle of the cardiovascular system, the sweat glands and arrector pili muscles of the skin, and the muscles of the ciliary body and iris of the eyeball. In many cases, there is a dual supply from the **sympathetic** and **parasympathetic** divisions of the autonomic nervous system.

In both divisions of the autonomic nervous system, there is a sequence of two neurons between the CNS and the effector organ which synapse in peripheral autonomic ganglia. The neurons from the CNS to the synapse in the ganglion are the **preganglionic neurons** and those from the ganglia to the effector organs are the **postganglionic neurons**. The **enteric plexus** is a third set of neurons interposed between the post-ganglionic neurons and the effector cells in the gastrointestinal tract.

Figure 17.1 compares the general arrangement of the sympathetic and parasympathetic nervous system. The cell bodies of **sympathetic** visceral preganglionic motor neurons are located in the intermediolateral horns of the thoracic and upper lumbar segments of the spinal cord while those of the **parasympathetic** visceral preganglionic (secretomotor) neurons are in the nuclei of four of the cranial nerves and the sacral segments of the spinal cord.

The functional effects of the two divisions of the autonomic system on a target tissue or organ are **antagonistic;** under normal circumstances, there is a balance between them, which maintains an appropriate level of visceral activity to maintain bodily **homeostasis**. In states of alarm or anger, there is a massive stimulation of the sympathetic outflow which completely overrides the parasympathetic effects and results in the body being placed in a state of activity suitable for violent physical activity, the so-called **'fight or flight reaction'**. This effect is backed up and prolonged by increased secretion of adrenalin from the adrenal medulla. Excess levels of adrenalin may cause fainting as outlined in Box 17.2.

As indicated in Figure 17.1, there are also pharmacological differences between the two components of the autonomic nervous system. **Acetylcholine** is the neurotransmitter in the synapses between pre- and post-ganglionic neurons in both divisions. The same neurotransmitter is used at the synapses between post-ganglionic parasympathetic neurons and the target organs. The neurotransmitter between post-ganglionic sympathetic neurons and their targets is **noradrenalin;** those to sweat glands are, however, cholinergic. Many pharmacological agents can interfere with autonomic neurotransmission as described in Box 17.1.

17.2.1 The sympathetic nervous system (thoracolumbar outflow)

The general distribution of the sympathetic nervous system is shown in Figure 17.2 which should be followed as the description is read.

Sympathetic preganglionic neurons are located in the intermediolateral horns of the spinal grey matter. The intermediolateral horns only extend from the first thoracic to third lumbar segments of the spinal cord. The preganglionic axons exit the CNS through the ventral roots of the *first thoracic to third lumbar spinal nerves* with somatic motor neurons, hence the term **thoracolumbar outflow**. As shown in Figure 12.11, sympathetic neurons leave the spinal nerves via **white**

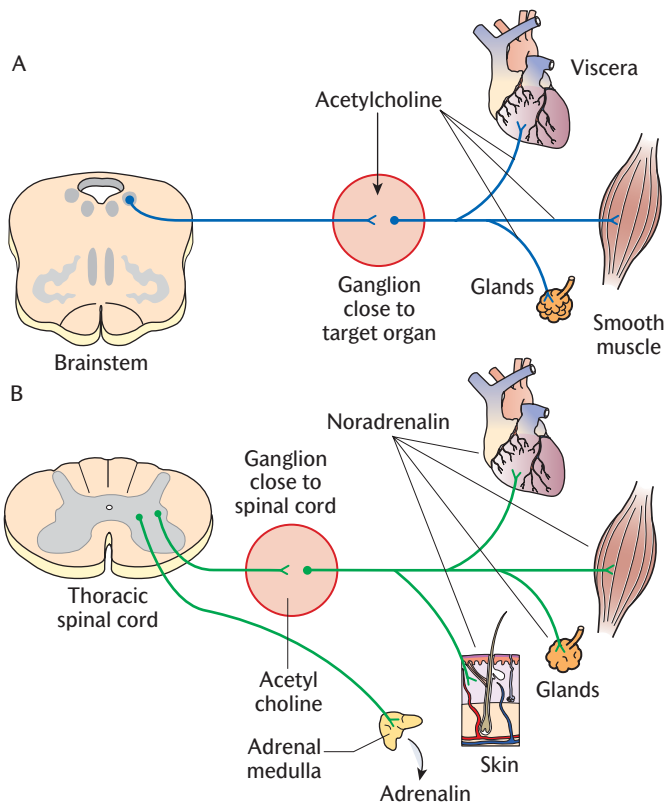


Fig. 17.1 The general arrangement of: A) The sympathetic; B) The parasympathetic nervous systems.

Box 17.1 Drug side effects and the autonomic nervous system

Knowledge of the structure and function of the autonomic nervous system is that it is the basis for understanding the side effects of many important drugs used in clinical practice that act on the sympathetic or parasympathetic systems or mimic their actions. Somewhere in the order of 500 generic and patent drugs in the British Pharmacopoeia have **sympatheticomimetic** actions. They either bind to adrenergic receptors, truly mimicking sympathetic stimulation or block cholinergic receptors, thus leaving sympathetic action unopposed. The main cause for concern from a dental practitioner's point of view is that these actions inhibit or reduce salivary flow.

Reduction of salivary flow below 50% of normal secretion results in **xerostomia** (dry mouth). Natural oral hygiene is reduced without efficient salivary flow because there is not sufficient saliva to wash away food debris. Saliva has a crucial buffering action which neutralizes acid produced by bacteria metabolizing food debris; other antibacterial actions of saliva are also reduced. Dental caries becomes rampant and periodontal disease also occurs. There are often oral infections caused by opportunistic pathogens. These organisms are normally kept in check by salivary actions but can become active if this is lost; oral thrush caused by *Candida* species of fungi is a good example of an opportunistic infection. Many antipsychotic drugs, ß-blockers to control blood pressure, and respiratory decongestants are just some of the classes of pharmaceuticals with a sympatheticomimetic action.

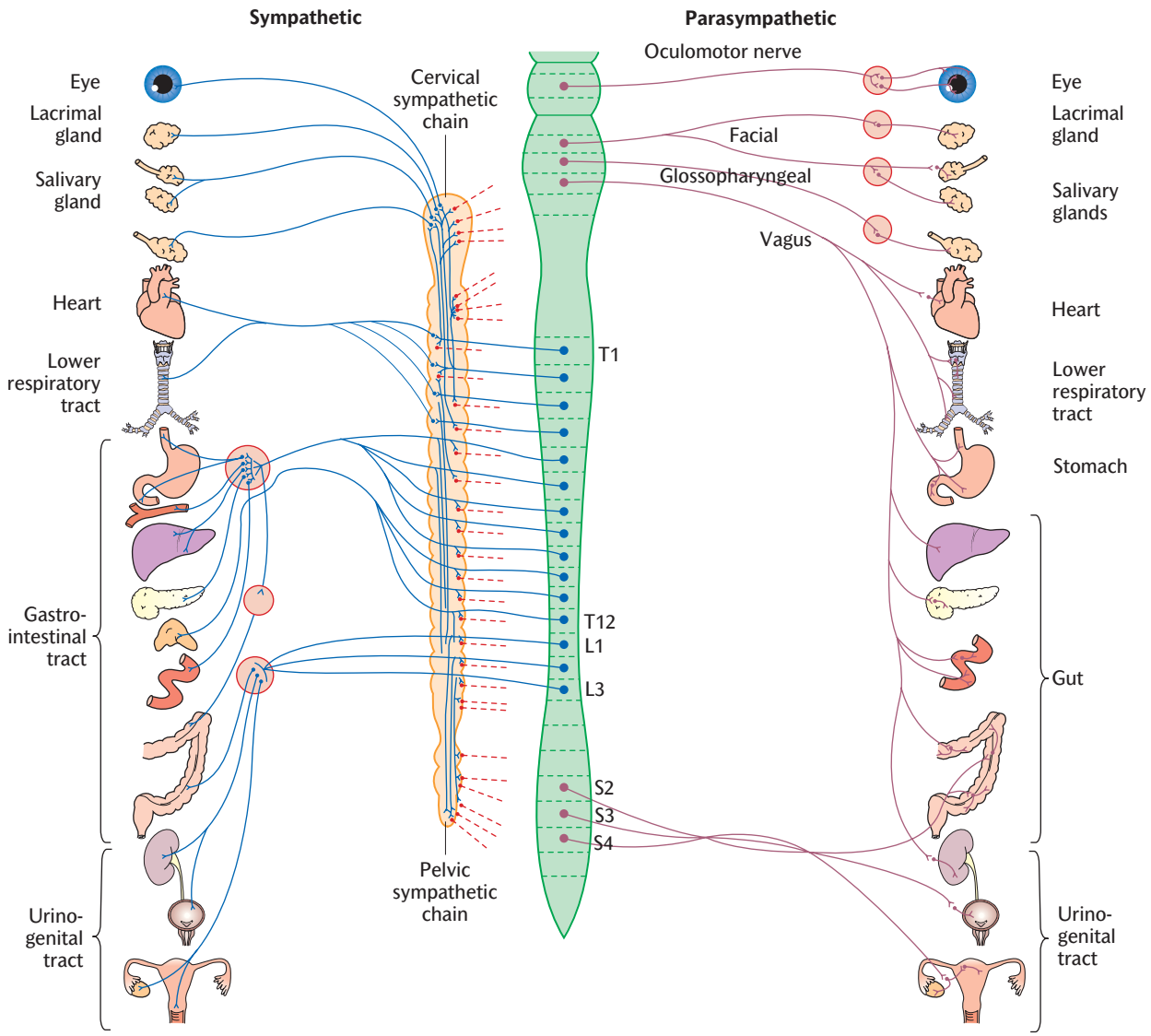


Fig. 17.2 The distribution of the autonomic nervous system. For clarity, the sympathetic nervous system is shown only on the left and the parasympathetic only on the right.

Box 17.2 Is your patient going to faint?

As mentioned in the main text, the sympathetic nervous system may be stimulated entirely when people are terrified or angry—the 'flight or fight reaction'. Even if you have the best interpersonal skills in the world, most patients attending your dental surgery will be somewhat apprehensive and will already have some adrenalin in their circulation due to the flight or fight reaction. Dental local anaesthetics usually contain noradrenalin as well as the active anaesthetic agents. The noradrenalin causes vasoconstriction in the area of administration and, therefore, slows the bloodborne removal of anaesthetic from the site, prolonging working time for the dentist. Sometimes, administration of local anaesthetic can add enough noradrenalin to cause the patient to faint.

You are not in a position to monitor heart rate and many other warning signs that accompany the flight or fight reaction and precede a faint, but you can see the warning signs that occur in the patient's face. Their skin will become very pallid as blood is directed away from skin to muscles and their pupils will dilate. If you see these signs, you should stop the procedure immediately, take preventative action, and reassure the patient. Simply being aware of their nervousness and manipulating the dental chair so that their feet are above the level of their head is often sufficient to prevent a faint.

The patient's medical history should *always* be checked before starting any dental procedure requiring local anaesthetic, especially for the presence of cardiovascular or respiratory disease. Anaesthetics without noradrenalin should be used for compromised patients to prevent any potential adverse effects on the cardiovascular or respiratory systems from the noradrenalin.

rami communicantes shortly after the dorsal and ventral roots fuse to form the spinal nerve. They form a chain of nerves (**the sympathetic trunks**) lateral to the vertebral column, interconnecting the sympathetic ganglia which contain the cell bodies of the post-ganglionic axons.

Sympathetic innervation is required throughout the body. As shown in Figure 17.2, the preganglionic axons from the upper thoracic nerves extend upwards into the neck to form the **cervical sympathetic trunks** and from the lumbar nerves extend downwards into the pelvis as the **pelvic sympathetic trunks**. In the thoracic and lumbar regions, there is usually a ganglion associated with each spinal nerve, but in the cervical and sacral region, the ganglia are often fused. There are usually three ganglia in the cervical region and four or five in the sacral region in addition to 12 in the thoracic region and four in the lumbar region.

Post-ganglionic sympathetic axons may leave the ganglia by several roots. They may connect to the corresponding spinal nerve by a **grey ramus communicans** as shown in Figure 12.11. In the cervical region where fusion of ganglia has occurred, the ganglia may connect by multiple grey rami to several spinal nerves. In addition, post-ganglionic axons may be distributed through vascular and visceral branches. **Vascular branches** leave many of the ganglia of the sympathetic trunk and pass to nearby arteries; they form plexuses around the vessels which can constrict them as they are distributed along the vessels to the peripheral target tissues. Vascular branches are the most important route for the distribution of post-ganglionic sympathetic axons supplying the head and neck. The **visceral branches** pass to large autonomic plexuses in the thoracic and abdominal cavities to innervate the viscera of the trunk.

The post-ganglionic axons are small diameter and mostly unmyelinated. They may ascend or descend to a higher or lower level of the sympathetic trunk before leaving through one of the routes mentioned above.

The adrenal medulla is supplied directly by preganglionic sympathetic neurons and, therefore, the secretion of adrenalin is coordinated with the activity of the sympathetic nervous system.

17.2.2 The parasympathetic system (craniosacral outflow)

As Figure 17.2 illustrates, parasympathetic preganglionic neurons originate from specialized cranial nerve nuclei and from the intermediolateral horns of the sacral segments of the spinal cord, hence the alternative name, the **craniosacral outflow**. At first sight, these origins suggest the parasympathetic system has a rather limited distribution. Furthermore, it seems that there is a huge gap in the distribution of parasympathetic nerves with no innervations between the head and the pelvis where most of the important viscera of the body are located. These organs clearly require a parasympathetic nerve supply if control of visceral function and homeostasis is to be maintained. This gap is filled by the distribution of parasympathetic preganglionic nerves to the thoracic and most of the abdominal organs through the **vagus nerves,** the tenth cranial nerves. The Latin word 'vagus' means 'wanderer'—appropriate for a nerve that originates in the medulla, branches in the head, and then wanders through the trunk.

As you can see in Figures 17.1 and 17.2, the preganglionic parasympathetic neurons are considerably longer than the post-ganglionic neurons and travel with the cranial or sacral nerves with which they originate to reach the ganglia. There is no equivalent to the sympathetic trunks in the parasympathetic division of the ANS; instead, the ganglia are individual structures close to or in the organs being supplied. The post-ganglionic axons are usually short.

Parasympathetic stimulation produces secretion from visceral glands and motility in the gastrointestinal tract among other activities; because of these actions, the neurons are often referred to as **secretomotor** neurons.

The cranial parasympathetic nerves

The parasympathetic preganglionic neurons in the cranial region begin in motor nuclei of the brainstem equivalent to the lateral horn of the spinal cord. They leave in cranial nerves III (**oculomotor**), VII (**facial**), IX (**glossopharyngeal**), and X (**vagus**). A fuller account of

the parasympathetic components of the cranial nerves is given in Chapter 18.

Briefly summarized, the parasympathetic components in the:

- **Oculomotor nerves** supply the ciliary body that controls lens thickness for focusing and the sphincter pupillae muscle of the eyeball to decrease pupillary diameter and thus regulate the amount of light falling on the retina;
- **Facial nerves** stimulate the secretion from salivary glands in and around the oral cavity and the tear-producing lacrimal glands in the orbit;
- **Glossopharyngeal nerves** stimulate the secretion from the parotid salivary glands;
- **Vagus nerves** innervate the respiratory and cardiovascular system and the gastrointestinal tract as far as the left flexure of the large intestine.

The preganglionic axons in the:

- Oculomotor nerve relay in the **ciliary ganglion**;
- Facial nerve in the **pterygopalatine** and **submandibular ganglia**;
- Glossopharyngeal nerve in the **otic ganglion**.

The anatomy and connections of these ganglia will be described in Section 4. The corresponding synaptic sites between pre- and post-ganglionic neurons in the vagus nerve are found in the autonomic plexuses and ganglia in or close to the thoracic or abdominal organ being supplied.

The sacral parasympathetic nerves

The gastrointestinal tract below the left colonic flexure and the remaining pelvic viscera receive their parasympathetic innervation through preganglionic neurons which leave the spinal cord in the second to fourth sacral spinal nerves. These connect with the post-ganglionic neurons in ganglia close to the viscera.

17.3 Visceral sensory neurons

Visceral sensory neurons may be divided into two main groups, **general visceral sensory neurons** from the viscera and blood vessels and **special visceral sensory neurons** concerned with taste. The pathways for taste travel through cranial nerves and are, therefore, described in Chapter 18.

General visceral sensory neurons convey information which does not usually reach consciousness. They are the peripheral processes of cells located in the dorsal root ganglia of spinal nerves (**T1 to L3**) which contribute visceral motor neurons to the sympathetic nervous system and in the sensory ganglia of the **glossopharyngeal** (ninth) and **vagus** (tenth cranial) nerves. These processes vary in diameter and may be myelinated or unmyelinated.

The peripheral processes pass from the dorsal root ganglia into the spinal nerves, then via the white rami communicantes to the sympathetic ganglia. They do not synapse in the ganglia, but pass straight through to be distributed through visceral and vascular branches to unencapsulated sensory endings in their targets. The central processes enter the spinal cord through the dorsal roots and synapse in the grey matter of the thoracic and upper lumbar segments and ascend in the dorsal columns (see Section 16.2.2). Collateral branches at the spinal level connect either directly or through interneurons with the preganglionic sympathetic neurons in the lateral horn, thus establishing pathways for reflex visceral action. Some general visceral sensory neurons convey nociceptive information to the pain matrix (see p. 142); the resulting visceral pain is outlined in Box 17.3.

The **vagus nerves** carry a large number of general visceral sensory neurons. The cell bodies are located in the inferior vagal ganglia. Their peripheral processes are widely distributed through the branches of the vagus nerves, the area of supply corresponding broadly with that of the parasympathetic secretomotor components of the vagus. Their central processes end in the inferior part of the nucleus of the tractus solitarius (see Section 18.10). The **glossopharyngeal nerves** convey general visceral sensory neurons from the **carotid sinuses** and **carotid bodies** at the bifurcation of the common carotid arteries. These terminate in the cardiovascular centres in the medulla.

Box 17.3 Visceral pain and diseases affecting the autonomic nervous system

Noxious sensations from the viscera may reach consciousness under certain pathological conditions. Possibly the best known example is ‘stomach ache’. Unlike pain caused by peripheral stimulation, e.g. a kick on the shin, the pain is diffuse and poorly localized. It may be felt in the region of the organ affected or may be referred to the area of skin which receives somatic sensory innervation from the same segments of the spinal cord. The central pathways followed by these nociceptive visceral sensory neurons are not well understood.

Diseases of the autonomic nervous system are not common; it is more usual for parts of the system to be affected by lesions in adjacent organs. The cervical sympathetic trunk may be interrupted by compression of the upper part of the thoracic sympathetic trunk or the cervical part of the trunk itself. One cause of such compression in the thorax is enlargement of mediastinal lymph nodes as a result of the spread of bronchial carcinoma from the lung. Similarly, cancerous lesions in the neck (laryngeal cancer, for example) can cause similar compression of the cervical sympathetic trunk. Interruption of the trunk produces ipsilateral:

- Constriction of the pupil (**miosis**) due to paralysis of the dilator pupillae and unopposed action of the sphincter pupillae muscle;
- Drooping of the upper eyelid (**ptosis**) because of paralysis of the part of levator palpebrae superioris composed of smooth muscle (see Section 23.1.5);
- Flushed dry facial skin due to vasodilatation and absence of sweating (**anhydrosis**);
- And possibly slight retrusion of the eyeball (**enophthalamos**).

This group of clinical signs is known as **Horner's syndrome** and may be the first clinical manifestation of the primary disease.

18 The cranial nerves

Chapter contents

18.1 Introduction

The cranial nerves are *the* most important neural structures relevant to dental students and practitioners. The cranial nerves are the nerve supply to all the structures in the head and neck and underpin of the anatomy and function of these regions—the head and neck will not work without them. In a wider context, correct functioning of the cranial nerves is a very good indicator of the health or otherwise of the CNS; it may be necessary to test the function of some, or even all, of the cranial nerves at times to assess neural function. In addition, many of the cranial nerves may be involved in various diseases of the head and neck.

As outlined in Chapter 3, 12 pairs of cranial nerves arising from the brain form one major component of the peripheral nervous system, the 31 pairs of spinal nerves forming the other. Each pair of cranial nerves has a name and number. Conventionally, they are numbered using the Roman numerals I to XII. The nerves are numbered from one to 12, according to their origin from the brain; nerves with the lowest numbers arise from the most anterior aspect of the brain (the forebrain) whereas those with highest numbers arise from the lowest part (the medulla).

Several aspects of any nerve anywhere in the body are required to describe its anatomy and function in complete detail:

- Its origins and terminations in the CNS;
- Its neuronal components—are they motor, sensory, or autonomic?
- Its course to and from its target tissues;
- Its distribution to specific areas and structures through specific branches;
- Its overall functions and specific functions of its component parts.

In addition, if the clinical significance is going to be appreciated, we will also need to consider:

- The effects of damage or disease on the nerve;
- Its important relationships to other structures;
- How to test whether the nerve is functioning correctly.

Given that there are 12 pairs of nerves, does a competent dentist need to know everything in the two lists about every cranial nerve? The answer, you will be relieved to hear, is 'no'. To paraphrase George Orwell—'all cranial nerves are important, but some are more important than others'. You should be able to list, for *all* 12 pairs of cranial nerves:

- Their component types of neurons;
- Their function;
- How to test their function;
- And outline their distribution.

More detail than this bald outline is required in some of the different headings for some nerves (**III, IX, X, and XII**), but precise detail is required for a few (**V and VII**). Why have certain nerves been emphasized? The nerves that require detailed knowledge are those nerves that dentists will be working on everyday, either by anaesthetizing them or trying to avoid damage to them during surgical procedures.

18.2 General anatomy of the cranial nerves

Cranial nerves are similar to spinal nerves in many respects, but differ significantly in others. As we have seen in Chapters 3 and 15, the general plan of all spinal nerves is identical; they are mixed nerves containing motor and sensory processes. As described in Chapter 17, some spinal nerves originating from the thoracic, lumbar, and sacral regions of the spinal cord also contain autonomic neurons. Cranial nerves are not all identical in composition; some are purely motor nerves, some are mixed somatic sensory and motor nerves, and others contain autonomic components. Some purely sensory cranial nerves are specialized to serve specific sensations such as olfaction, vision and hearing, and balance (the **special senses**). In addition, some cranial nerves contain neuronal types that are not found in spinal nerves such as those conveying the sensation of taste.

Despite these differences, some similarities can still be seen. The cell bodies of somatic sensory components in cranial nerves are found in ganglia lying outside the CNS, analogous to the dorsal root ganglia of spinal nerves.

It should also be pointed out that of the 12 pairs of cranial nerves, only the third to twelfth nerves are true peripheral nerves, the distinction being that their processes are ensheathed by Schwann cells as opposed to glial cells. The first two pairs of cranial nerves develop as outgrowths from the brain and carry their covering glial cells and meninges with them. They are, in fact, components of the CNS rather than the PNS, but are still considered as cranial nerves.

18.2.1 Neuronal types in cranial nerves

Table 18.1 lists the types of neurons found in each of the cranial nerves. Do not just learn the table. Read the following explanation to understand the distinctions between the different neuronal types, then use the table for quick reference and a reminder of the salient points.

The first point to note is that cranial nerves I (olfactory), II (optic), and VIII (vestibulocochlear) do not fall within the general plan in Table 18.1 because they are nerves conveying the special senses of smell, vision and hearing, and balance respectively.

The muscles of the head and neck are derived embryologically from mesoderm just like muscle tissue anywhere else in the body (see Chapter 8). Most of these muscles are voluntary muscle, therefore, we should expect them to be innervated by **somatic motor nerves**. This is clearly the case for muscles supplied by cranial nerves III (oculomotor), IV (trochlear), VI (abducens), and XII (hypoglossal). The third, fourth, and sixth cranial nerves supply the extraocular muscles that move the

Table 18.1 Neuronal types in the third to twelfth cranial nerves

	Somatic motor	Branchiomotor	Parasympathetic (Secretomotor)	General Visceral sensory	Taste (Special visceral sensory)	Somatic sensory
III Oculomotor	X		X			
IV Trochlear	X					
V Trigeminal		X				X
VI Abducens	X					
VII Facial		X	X		X	
VIII Vestibulocochlear	Special senses of hearing and balance					
IX Glossopharyngeal		X	X	X	X	X
X Vagus		X	X	X	X	X
XI Spinal accessory	X					
XII Hypoglossal	X					

eyes within the orbital cavities. These muscles are derived from preotic somites in the anterior part of the developing head. The hypoglossal nerves supply the muscles of the tongue derived from occipital somites that develop near the base of the skull formed by the occipital bone (see Section 21.8). Cranial nerve XI (spinal accessory) supplies two muscles in the neck whose embryonic origins are uncertain, but these nerves certainly comprise somatic motor neurons.

During development of the face and neck, a series of paired sausage-like structures grow out from around the hindbrain as ectomesenchymal cells migrate as the neural tube closes (see Section 8.3.4). These structures are the **pharyngeal** (or branchial) **arches** (see Section 20.1). Initially, there are six pairs of arches, but the lower three pairs rapidly coalesce to form complexes known as the fourth arches. Certain muscle groups in the head and neck derive from mesoderm within each arch, each group being derived from a specific arch. Each pair of arches is innervated by a specific pair of cranial nerves and any structures derived from those arches are innervated by those cranial nerves. Muscles from arch 1 is supplied by cranial nerve V (trigeminal), arch 2 by cranial nerve VII (facial), arch 3 by cranial nerve IX (glossopharyngeal), and arch 4 by cranial nerve X (vagus). These cranial nerves also contain somatic motor nerves, sometimes referred to as branchiomotor nerves (see Box 18.1).

As already outlined in the introduction to this chapter and Chapter 16, four pairs of cranial nerves (III, VII, IX, and X) contain autonomic **parasympathetic** motor neurons, often called **secretomotor neurons**.

Some cranial nerves deal specifically with special senses as we have seen. However, the other cranial convey **somatic sensory** innervation from the skin of the head and mucosa lining the respiratory and gastrointestinal tracts as they pass through the head and neck. Cranial nerves V, IX, and X have major somatic sensory components.

As we have seen in Chapter 17, visceral sensation is somewhat different from somatic sensation; much of the sensation from the viscera is unconscious and functions to control the function of internal organ by monitoring their stretch or distension, for example. Two pairs of cranial nerves (IX and X) contain **general visceral sensory** nerves, controlling cardiovascular, respiratory, and gastrointestinal function.

The final neuronal type found in cranial nerves is designated as **special visceral sensory**; this is just a special term for neurons that convey the sensation of **taste**. Cranial nerves VII, IX, and X contain taste neurons.

Box 18.1 Mistaken origins of pharyngeal arch muscles

When anatomists and embryologists were first working out the developmental relationships between the muscles of the pharyngeal arches and the cranial nerves, they believed that muscles that developed in the pharyngeal arches were derived from a different source of mesoderm than that which forms voluntary muscles elsewhere in the body. They thought that the muscles derived from mesoderm that also produced some of the internal organs so they distinguished the nerves innervating these muscles as **branchiomotor** (or special visceral motor) **nerves**. We now know that these pioneer anatomists were wrong, but the name is still used in many textbooks. You may think it is an arbitrary distinction, but you will see why the distinction is important when we examine the cranial nerve nuclei in the next section. Under this classification, a given cranial nerve can contain either somatic motor or branchiomotor components, but not both.

18.2.2 Cranial nerve nuclei

You should now appreciate from the description above and Table 18.1 that the cranial nerves differ quite markedly in structure and function. It follows from this that the terminations of each nerve in the brain will

differ, depending on their functions. As described in Chapter 15, the spinal cord has the same overall structure along its entire length. The spinal cord has grey matter dorsal horns throughout its length that receive incoming somatic sensory processes that all spinal nerves contain. Likewise, the ventral horns extend the length of the spinal cord spinal nerves because all spinal nerves contain somatic motor components that originate from cell bodies in the ventral horns. Some areas of the spinal cord also have intermediolateral horns where autonomic axons arise.

In contrast to spinal nerves, cranial nerves only need areas in the CNS for the synaptic termination of the specific types of neurons they carry so that appropriate connections can be made. To put it another way, if a cranial nerve lacks a particular component, then there is no need for a connecting area. For example, a cranial nerve that has purely motor functions has no need for the equivalent of dorsal horns for reception of sensation.

The dorsal and ventral horns of the spinal cord develop from structures called the alar and basal laminae as shown in Figure 18.1A. The internal structure of the brainstem from which the third to twelfth cranial nerves arise is actually analogous to the spinal cord, but the structures are displaced. As shown in Figure 18.1B, the grey matter is split vertically and pushed laterally by the presence of the fourth ventricle. Sensory, motor, and autonomic areas of grey matter still maintain their relationships relative to each other, but sensory areas in the brainstem lie laterally to the motor areas; there is a corresponding intermediate area that serves general visceral functions.

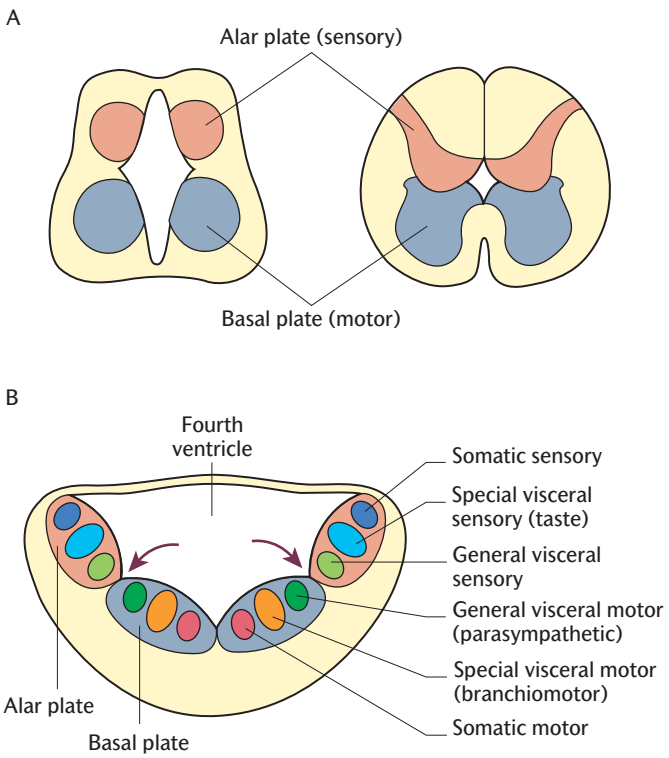


Fig. 18.1 A) Development and structure of the spinal cord. B) Development and structure of brainstem.

The grey matter in the brainstem is, therefore, discontinuous because there are only areas specific to each different function of each cranial nerve. These discrete areas of grey matter are the **cranial nerve nuclei**. If you imagine a series of columns extending into the brainstem from the grey horns of the spinal cord, it emphasizes the similarity between the arrangement of the grey matter of the spinal cord and brainstem. Each column will then be found to contain nuclei with functions similar to those of the grey horn with which they align. One column can be imagined as an upward extension of the dorsal horn, containing nuclei associated with somatic sensory neurons like the dorsal horn itself. A column in line with the ventral horn contains nuclei where the cell bodies of somatic motor neurons are located as in the ventral horn. Six columns of cranial nerve nuclei are shown in Figures 18.1 and 18.2:

- Column 1 (most laterally) contains **somatic sensory nuclei**;
- Column 2 contains **taste** (special visceral sensory) nuclei;
- Column 3 contains **general visceral sensory** nuclei;
- Column 4 contains **parasympathetic** (general visceral motor) nuclei;
- Column 5 contains (branchiomotor) **motor nuclei** for nerves that innervate muscles derived from the pharyngeal arches;
- Column 6 (nearest the midline) contains **somatic motor nuclei** to muscles derived from somites.

Columns 1 and 2 are sensory, 3 and 4 autonomic, and 5 and 6 sensory. However, as Figure 18.2 shows, these nuclei occupy only parts of their respective columns with long stretches where no nuclei are present.

The position of cranial nerve nuclei

The position and number of nuclei for each cranial nerve (excluding those serving the special senses) can be worked out using a few basic rules. As we all know, rules are made to be broken as we will see as each nerve is described. There are exceptions to these general rules and some cranial nerves make use of nuclei of other nerves for similar functions. The rules for working out the cranial nerve nuclei for nerves III to VII and IX to XII are as follows:

1. There is **one nucleus for each function** of the cranial nerve on each side;
2. Sensory nuclei lie **lateral** to motor nuclei with autonomic nuclei in between (see Figure 18.1B);
3. The nuclei are at **the level** at which the cranial nerves attach to the brainstem.

Using Table 18.1 which outlines the function of each component of the cranial nerves as a basis and using information about the level of origin of each pair of cranial nerves described in Chapter 15 and illustrated in Figure 15.10, we can build up a scheme for the cranial nerve nuclei using the three rules.

We will use the hypoglossal nerve as an example. From Table 18.1, we can see that this is a pure somatic motor nerve so rule 1 tells us there should be one motor nucleus on each side of the brainstem. Rule 2 tells us that each nucleus should lie close to the midline. The hypoglossal

nerves are attached to the lower medulla just above the spinal cord so the application of rule 3 tells us the nuclei should be low down in the medulla. Look at Figure 18.2 that shows the actual cranial nerve nuclei in a schematic diagram and confirm that these predictions are correct; the hypoglossal nuclei are indeed adjacent to the midline low in the medulla. If we take another example of the trigeminal nerve (CN V), there should be two nuclei in each half of the brainstem, a somatic sensory and a motor nucleus, according to rule 1 and the information in Table 18.1. The sensory nucleus should lie laterally to the motor nucleus (rule 2) and they should be at the level of the pons (rule 3). Now look again at Figure 18.2. There are two nuclei and the sensory nucleus is lateral to the motor one. Clearly, the motor nucleus is in the pons; note that it is slightly lateral to the hypoglossal nucleus, reflecting the different embryological origins of the two nerves. Look at the sensory column. What *is* going on with the sensory nucleus shown in Figure 18.2? It extends way beyond the pons where we would expect it to be located down into the spinal cord and up through to the midbrain. The reason for the vast expansion of the trigeminal sensory nucleus is that it receives sensation from areas of the head that are very sensitive and highly discriminative such as the tongue, lips, and eyelids. If discrimination is to be preserved, then incoming nerves cannot converge and must retain their individuality. They simply will not pack into the available space in the predicted location in the pons and so expand above and below that level.

If Figure 18.2 is examined against the information in Table 18.1, several other anomalies will be apparent. Some are anatomical whereas others are functional. Anatomically, the parasympathetic nucleus of the **oculomotor nerve** (the Edinger–Westphal nucleus) is displaced more medially than it should be. Similarly, the motor nucleus of the vagus is more laterally than it should be; this nucleus is called the **nucleus ambiguus** because its position is ambiguous. The ninth (glossopharyngeal) and tenth (vagus) nerves should have somatic sensory nuclei in the medulla, but they are clearly missing from Figure 18.2. These nerves use another nucleus, in this case, the sensory nucleus of the trigeminal nerve. The rules also predict separate nuclei for the termination of taste neurons travelling in the facial, glossopharyngeal, and vagus nerves. However, these all converge into a single area, the upper part of the nucleus of the **tractus solitarius**. General visceral sensory neurons, principally in the vagus, but also in the glossopharyngeal nerve, converge to terminate in the lower part of the nucleus of the tractus solitarius. As can be seen in Figure 18.2, this part inclines medially to lie in the general visceral sensory column.

In the following sections of this chapter, an overview of the individual cranial nerves is presented. The main emphasis is on the types of neurons in the cranial nerves, the central connections between cranial nerve nuclei and other parts of the brain, their functions, and clinical tests. Only an outline of the course and distribution of each of the cranial nerves will be given here. Where further detail is required, it will be described in Section 4 on the regional anatomy of the head and neck.

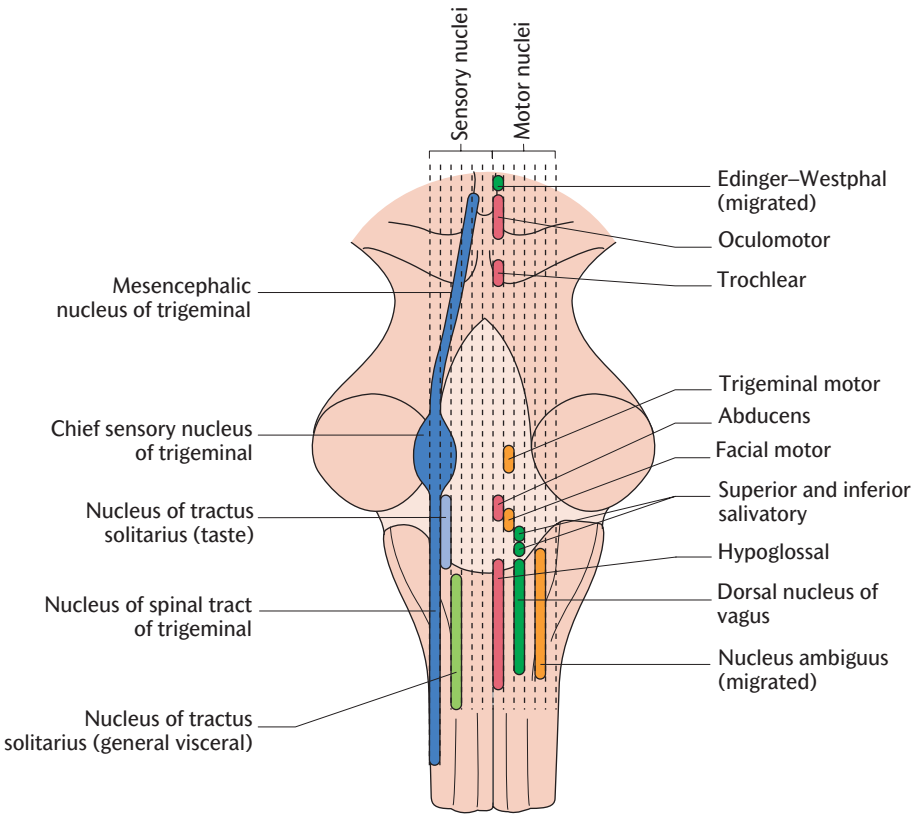


Fig. 18.2 The arrangement of the cranial nerve nuclei in a dorsal view of the brainstem. Motor nuclei are shown on the right and sensory nuclei on the left for clarity.

18.3 The olfactory nerves (CN I)

The olfactory nerves convey the sense of smell from the nose to the olfactory areas in the temporal lobes (see Section 15.4.2). The olfactory nerves are usually clearly visible, running along the underside of each frontal lobe as seen in Figure 15.17C. As described in Chapter 15, each nerve ends in the olfactory bulb, a bulge created by the presence of cell bodies positioned in the anterior cranial fossa above the nasal cavities. Olfactory mucosa only covers a small area in the roof of each nasal cavity and comprises specialized neurons. Their axons pass through perforations, forming the cribriform plate of the ethmoid bone to synapse in the olfactory bulb. The axons of the olfactory bulb cells form the olfactory nerves that run back to enter the temporal lobes and terminate the olfactory cortex. The connections of the olfactory cortex with other parts of the brain have been described in Chapter 15.

18.3.1 Clinical testing of the olfactory nerves

If required, the function of the olfactory nerves are tested by holding a strong smelling substance, such as smelling salts, under each nostril in turn while occluding the other nostril.

18.4 The optic nerves (CN II)

The optic nerves are the first components in a series of neurons that form the **visual pathways**, conveying vision from the retina of the eyeballs to the visual cortex in the occipital lobes.

Cells called rods and cones are the photoreceptors in the retina in each eyeball. The optic nerves are the axons of cells that connect indirectly to the rods and cones. These axons emerge as the optic nerve from the posterior aspect of each eyeball and have a long course inside the bony orbits before passing through the optic canals into the middle cranial fossa. As Figure 15.24 shows, the optic nerves can be seen very clearly on the underside of the brain medial to the internal carotid arteries. This is not surprising as the optic nerves are the largest of the cranial nerves, each nerve containing about a million axons.Figure 18.3 indicates that they have quite short intracranial course before the two nerves merge with each other to form the **optic chiasma**.

18.4.1 The visual pathways

The rest of the visual pathway is illustrated in Figure 18.3 which should be followed as you read this description. As the two optic nerves converge at the optic chiasma, the axons from the nasal half of each retina decussate whereas those from the temporal half of the retina do not cross. The axons passing through the optic chiasma diverge again to form the **optic tracts**, the continuations of the optic pathway. Each optic tract contains temporal axons from the eyeball of its own side and nasal axons from the opposite eyeball. Most of the axons in the optic tract terminate in the **lateral geniculate nucleus** of the thalamus. Post-synaptic axons form the **optic radiation** on each side which passes through the posterior limb of the internal capsule to the **visual cortex** in the upper and lower lips of the calcarine sulcus of the occipital lobe.

As Figure 18.3 shows, some of the axons in the optic tract have collateral axons that branch off before the lateral geniculate nucleus to end in the **pretectal area** and **superior colliculus** of the midbrain. These parts of the midbrain also receive collateral axons from the optic radiation and neurons that pass back from the visual cortex. These pathways to the midbrain are involved in reflex responses to light, including the **pupillary light reflex** (constriction of the pupil in response to bright light) and the **accommodation reflex** (ocular convergence, pupillary constriction, and change in the curvature of the lens when viewing close objects) as well as reflex movements of the eyes and neck to track objects moving across the visual fields.

It is important to understand that the retinal images of the visual fields are *inverted* and reversed right to left by the optics of the cornea and lens of the eye. In clinical practice, **visual fields** (or **fields of vision**) are referred to rather than the parts of the retina. As Figure 18.3 demonstrates, information from the temporal field of vision actually ends up on the nasal part of the retina. Due to the decussation of nasal axons and the continuation of temporal axons, each optic tract and radiation contains information from both eyes, which is important for stereoscopic vision. The representation of each eye in the optic tract and radiation also explains why light reflexes are **consensual** and will occur in both eyes at the same time—a bright stimulus delivered to one eye will cause pupillary constriction in both eyes, for example.

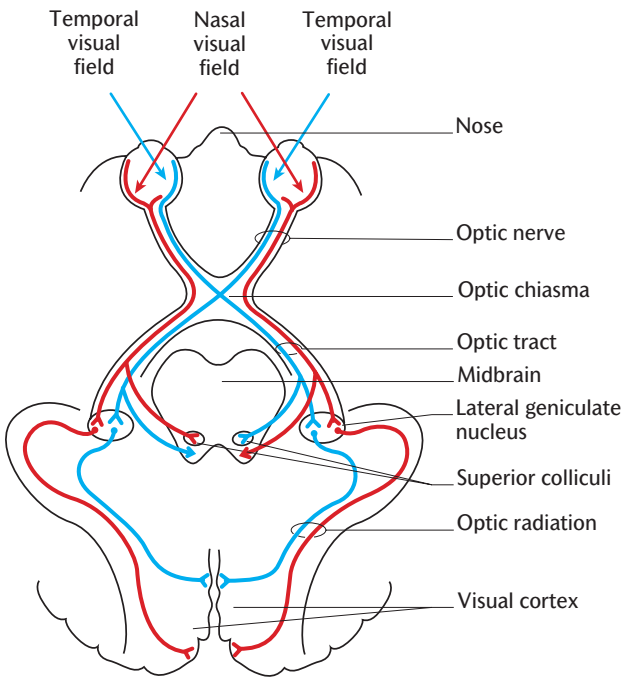


Fig. 18.3 The visual pathways.

Box 18.2 The effects of damage and disease on the visual pathways

It should be apparent from the above account that lesions at different parts of the optic pathway will produce different visual defects. A lesion in one eye or the optic nerve may lead to blindness in that eye. A common cause of damage to the optic nerve is compression due to raised intraocular pressure due to **glaucoma**. Damage to the optic chiasma, usually due to the pressure from an enlarged pituitary gland caused by tumourous growth, produces a condition known as **bitemporal hemianopia** (= half blindness in both eyes in the temporal fields of vision). The pressure on the chiasma will tend to interrupt conduction in the decussating axons from the temporal visual field while the nasal visual fields are unaffected. Consequently, the patient will lose sight in the left and right temporal fields of vision.

Depending on the exact level of the lesion, damage to the optic tract, radiation, or visual cortex of one side will produce loss of different parts of the visual fields of both eyes in quite complex manners, which are beyond the scope of dental practice.

Clinical testing of the optic nerves and visual pathways

Tests of visual acuity and visual loss are the specialist realm of optometrists. However, most dental students and practitioners will be familiar with many of the routine tests employed as they will have had their own eyes tested at some stage in their career. If you have not had your eyes tested yet, better sooner than later.

18.5 The oculomotor (CN III), trochlear (CN IV), and abducens nerves (CN VI)

The third (oculomotor), fourth (trochlear), and sixth (abducens) cranial nerves are treated together because as shown in Table 18.1, they are all somatic motor nerves supplying extraocular muscles controlling eye movement. In addition, the table indicates that the oculomotor nerve also contains parasympathetic neurons that control the pupillary diameter and lens curvature within the eye; these movements are reflex responses to light conditions and focusing requirements. The meaning of oculomotor should be fairly obvious—motor to the ocular apparatus (eye). However, the names given to the fourth and sixth cranial nerves are much less obvious; all will become clear as these nerves are described.

18.5.1 The extraocular muscles

Before the functions of the nerves are described, a brief explanation of the extraocular muscles is required, although they will be described more fully in Section 30.4.1. There are six extraocular muscles in each orbit and their dispositions are shown in Figure 18.4. The four rectus muscles (= straight) turn the eyeball (globe) in the same direction as their names imply:

- The superior rectus turns the eye upwards;
- The inferior rectus turns the eye downwards;
- The lateral rectus turns the eye laterally;
- The medial rectus turns the eye medially.

Note that the superior and inferior recti also exert a medial pull on the globe because the longitudinal axis of the orbits is at an angle to the midline whereas the eyes look straight ahead as shown in Figure 30.7.

The superior and inferior oblique muscles have more complicated and less obvious actions determined by their position of attachment to the globe. Both oblique muscles turn the eye laterally, but the superior oblique muscle turns the eye downwards, and the inferior rectus turns it upwards; these two muscles have the opposite effect to their name on vertical eye movement.

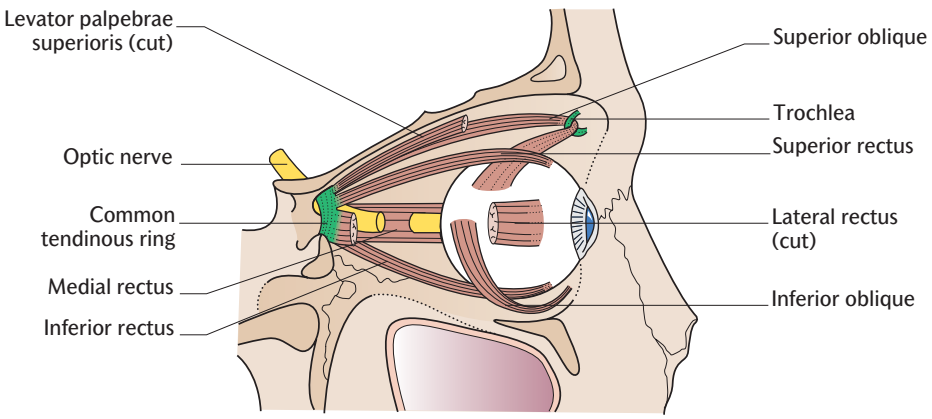


Fig. 18.4 The extraocular muscles.

18.5.2 The oculomotor nerves

The oculomotor nerves supply the superior, inferior and medial rectus muscles, and the inferior oblique muscle. They also carry the major motor nerve supply to the **levator palpebrae superioris muscle** elevating the upper eyelid.

The somatic motor neurons to these extraocular muscles originate in the **oculomotor nucleus** which is situated in the midbrain at the level of the superior colliculus as shown in Figure 18.2. The motor units in the extraocular muscles are very small, allowing very precise control of eye movements; each axon supplies about six muscle fibres. As can be seen in Figure 18.5, the oculomotor nerves are usually visible, exiting the under surface of the midbrain. They can usually be seen between the posterior cerebral and superior cerebellar arteries as shown in Figure 15.24 on an intact brain; a short stump may be visible, entering the cavernous sinus when the brain has been removed from the skull.

The oculomotor nucleus receives inputs from the cerebral cortex for the voluntary control of eye movements. It is easy to forget that in any direction of gaze where there is a horizontal component, each eye is moving in an opposite direction. As you are reading this text from left to right, your right eye is moving laterally, but your left eye is moving medially. If you were watching a game of tennis, you would only be able to follow the ball so far by eye movement alone; you would eventually have to turn your head in the direction of travel and may even have to turn your body. Of course, when you have been lucky enough to obtain those tickets to the final, you will not be thinking of eye, head, and body movement; they will be happening subconsciously so you do not miss the action. Visual inputs via the superior colliculus and pretectal area and from the vestibular nuclei (see Section 18.8.2) give information about movement in the visual fields and head position, respectively. Outputs allow eye, head, and body movement to be coordinated through connections to nuclei controlling the other extraocular muscles, including those of the other side, and the relevant segments of the ventral horns of the spinal cord.

The oculomotor nerves also carry parasympathetic preganglionic axons to the sphincter pupillae and ciliary muscle of the eyeball. These nerves originate from the **Edinger–Westphal nucleus** close to the oculomotor motor nucleus shown in Figure 18.2. They synapse in the **ciliary ganglion** in the back of the orbit. Post-ganglionic axons travel with the short ciliary nerves of the ophthalmic trigeminal nerve to enter the eyeball. The Edinger–Westphal nucleus receives inputs from the pretectal area and superior colliculus which are the areas in which collateral axons from the optic radiations terminate. The optic nerves and oculomotor nerves form reflex arcs for the pupillary light and accommodation reflexes through the tectal areas of the midbrain. The sphincter pupillae muscles constrict the pupils in response to bright light to prevent damage to the retina. The ciliary muscles thicken the lens and increase its curvature. This has the effect of focusing near objects on to the retina and is part of the **accommodation reflex** that occurs when changing your view from a distant object such as the screen in a lecture theatre to a near object such as your notepad on the bench. The accommodation reflex also involves pupillary constriction and convergence of the eyes.

18.5.3 The trochlear nerves

Each trochlear nerve innervates only the superior oblique muscle on each side. The trochlear nerves are consequently very thin, about the size of a strand of cotton, and, therefore, difficult to observe. The trochlear nerves

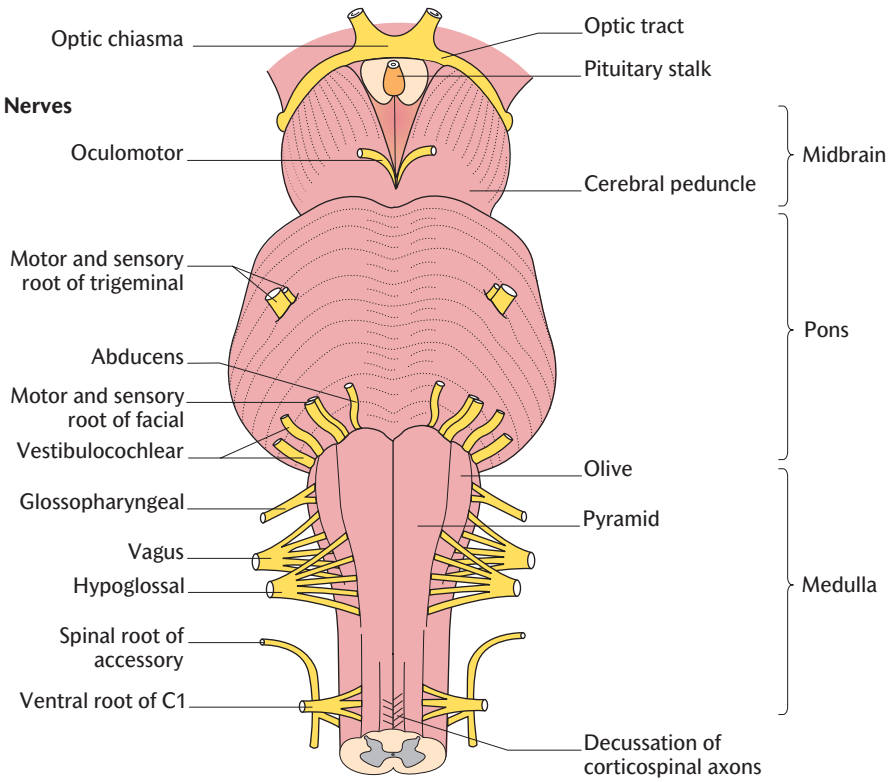


Fig. 18.5 The origin of the cranial nerves from the brainstem.

are unique in that all the axons beginning in the **trochlear nuclei** cross the midline within the midbrain to supply the superior oblique of the *contralateral* eye. Furthermore, as shown in Figure 15.12, they are the only cranial nerves that emerge from the dorsal surface of the brainstem where they emerge below the contralateral inferior colliculus.

As shown in Figure 18.4, the superior oblique muscles attach to the common tendinous ring in the posterior part of the orbit and run forwards between the medial wall and roof of the orbit. They then pass through a fibrous pulley and turn back on to the globe. This apparently bizarre course converts a backward pull into the correct direction. The Latin word for pulley is 'trochlea', hence the name of this nerve. Because the trochlear nerves are so flimsy and exit from the posterior midbrain, they often break as the brain is removed from the skull. Often, no trace is seen on the brain; they usually adhere to the free edge of the dura forming the opening in the tentorium cerebelli.

Their connections within the CNS are similar to those of the oculomotor nerves.

18.5.4 The abducens nerves

Each abducens nerve also supplies only one muscle, the lateral rectus muscle. The abducens nerve is so-called because the lateral rectus muscle it supplies moves the eye laterally; the correct anatomical term for any movement away from the midline is *abduction*, thus the abducens nerve abducts the eye.Figure 18.2 shows the **abducens nuclei** low down in the pons. As you can see in Figure 18.5, each nerve emerges at the junction of the pons and medulla; it has a long intracranial course on the base of the skull and alongside the sphenoid bone in the area of the pituitary gland before entering the orbit through the superior orbital fissure. This long course is clinically significant (see Box 18.3).

Box 18.3 describes the effects of damage and disease on the function of the third, fourth, and sixth cranial nerves and the clinical tests used to assess their function. The effects of damage to corticonuclear pathways, including those supplying the nerves to the extraocular muscles, are covered in Box 18.4.

Box 18.3 The effects of damage and disease on the third, fourth, and sixth cranial nerves

Damage to any of the nerves supplying the extraocular muscles will impair eye movement. These nerves are often traumatized in fractures of the middle third of the face that include the orbital walls; displaced bone fragments may trap the nerves. Maxillofacial surgeons treating midfacial injuries should always be aware that the extraocular muscles and their innervations may also be compromised by such fractures. Irrespective of whether the oculomotor, trochlear, or abducens nerve is injured, the first symptom apparent to the patient will be double vision (**diplopia**) as they move their eyes.

Damage to the **oculomotor nerves** has potentially the most far reaching effects as they supply more muscles than the other two nerves. If one oculomotor nerve is affected by trauma or disease, all the extraocular muscles on that side will be paralysed except the superior oblique and lateral rectus muscles. Both the unaffected muscles exert a lateral pull on the globe, which will be unopposed because the other muscles are inactive. Eventually, the eye on the injured side will acquire a **lateral strabismus** (squint) and will be fixed laterally and usually downwards because of the pull of superior oblique. In addition, the pupil will be dilated because the action of the dilator sympathetic innervation of the pupil will be unopposed because of the absence of parasympathetic innervations to the sphincter papillae muscle through the oculomotor nerve.

Patients with **abducens nerve** damage will experience diplopia, especially when trying to move their eye laterally, and may eventually acquire a **medial strabismus**. The medial pull of the medial rectus aided by the inferior and superior recti overcomes the lateral pull of the superior and inferior oblique muscles, thus pulling the eye medially. As mentioned above, the abducens nerves have the longest intracranial course of all the cranial nerves and are in contact with bone forming the cranial base and pituitary fossa for most of that course. When intracranial pressure is raised following head injury or brain trauma, the abducens nerves are often compressed between the expanding brain and the base of the skull. Double vision accompanied by headache and confusion (Box 15.3) are often the first signs of raised intracranial pressure.

Following **trochlear nerve** injuries, the patient may have difficulty negotiating stairs as they cannot turn their eye downwards and laterally to see the step; they will also experience diplopia. They often hold their head sideways towards the opposite side to the injury to compensate for the absent pull of superior oblique. Because the trochlear nerves exit from the posterior aspect of the midbrain, they can be injured in 'whiplash' injuries. A whiplash injury is caused when the head continues to accelerate forward or backwards when the body stops. The most frequent cause is in road traffic accidents when the patient's car is hit from the front or behind; the patient's body is held by the seat belt, but their head may 'whip' violently forwards or backwards, causing neck injury and possible trochlear nerve damage. Modern cars fitted with head restraints and air bags minimize the likelihood of whiplash injuries.

Clinical tests for the third, fourth, and sixth cranial nerves

Following tests are used clinically to investigate the integrity of the nerves innervating the extraocular muscles. The patient is asked to follow the tip of a pen or the light of a pen torch as the clinician moves it vertically, horizontally, and diagonally across the visual fields and observes which directions the patient exhibits difficulty in following. To test the pupillary light reflex, a pen torch is shone into each eye in turn and the reaction of *both* pupils is observed. The reflex will be absent from the eye on the injured side. Trochlear nerve injuries may be quite difficult to detect using eye movement alone.

Box 18.4 Corticonuclear lesions

A frequent site for the interruption of transmission in corticonuclear pathways is as they travel through the internal capsule. CVA (stroke) in the narrow perforating arteries supplying this area (see Chapter 15) is the usual cause. You should recall from Box 16.4 that damage to upper motor neurons forming descending corticospinal pathways and supplying spinal nerves produces spastic paralysis; this manifests on the contralateral side to the lesion because these pathways cross at the pyramidal decussation to the opposite side of the body. **Corticonuclear pathways** supply the motor nuclei of cranial nerves before the pyramidal decussation is reached so you may anticipate that any effects of damage to these pathways will be on the same side as the lesion. However, many corticonuclear axons remain uncrossed, but others decussate about the level of the nucleus in question. This is illustrated in Figure 18.6; note that red axons from the right cortex and blue axons from the left innervate both nuclei—both nuclei receive inputs from both sides of the motor cortex. If the corticonuclear tract is damaged by disease or trauma on one side only, there is still some innervation from the contralateral side. The 'X' in Figure 18.6 indicates damage by a stroke in the internal capsule interrupting the red axons arising from the right hemisphere. However, the motor nuclei will still receive some input from the blue axons originating from the other cortex. This input is not as profuse as normal, but still means that the lower motor neurons comprising the cranial nerve receive some information. The result is that damage to upper motor neurons forming corticonuclear pathways controlling cranial nerves does *not* cause a classical upper motor neuron lesion. Instead, because there is some residual innervation, the muscles supplied by the nerve act weakly, a condition known as **paresis**. Paresis is usually apparent on both sides, but may exhibit more strongly on one side.

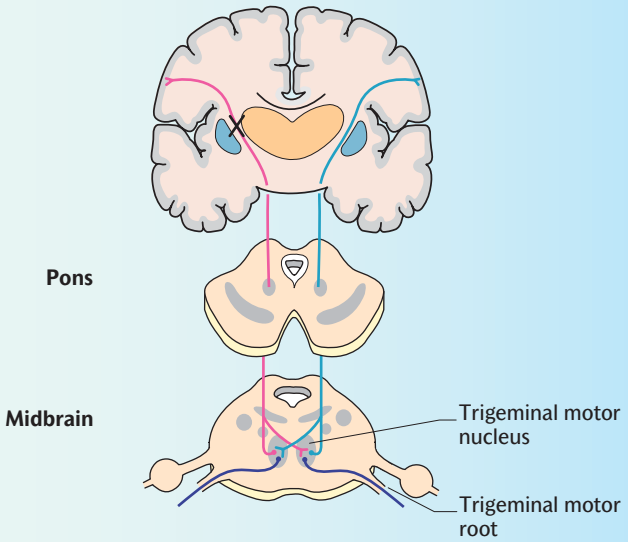


Fig. 18.6 Corticonuclear pathways. The trigeminal pathways are illustrated as an example.

Paresis may affect any of the cranial nerves which have a motor component. In the case of supranuclear palsy affecting the corticonuclear supply to the motor nuclei supplying the extraocular muscles, eye movements are weaker and slower than normal, but the eyes move together; there is therefore no diplopia.

18.6 The trigeminal nerves (CN V)

The trigeminal nerves are the second largest of the cranial nerves after the optic nerves. Functionally, the trigeminal nerves are straightforward but their anatomy is complex. Their name is derived from the Latin term trigemini (= triplets) because the nerves are distributed through three large divisions on each side of the head, called the **ophthalmic, maxillary**, and **mandibular** divisions from above downwards; these are outlined in Figure 18.7.

The trigeminal nerves are the major somatic sensory nerves of the head receiving sensory processes from:

- The skin of the face and the conjunctiva and cornea covering the eyes;
- The eyeball;
- The lining of the nasal cavities and associated paranasal sinuses;
- The mucosa lining the oral cavity, the teeth and their supporting tissues and;
- Most of the dura mater.

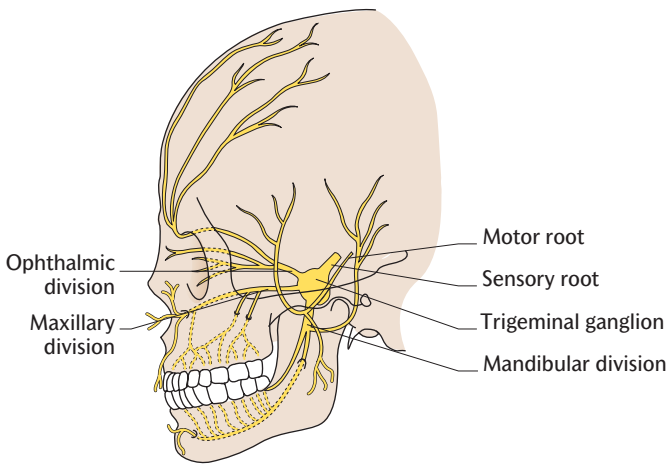


Fig. 18.7 The major divisions of the trigeminal nerve and some of its branches.

The trigeminal nerves also convey proprioceptive information from the muscles of mastication, responsible for movement of the lower jaw and some other muscles and the temporomandibular joint to the CNS. It can be appreciated from the long list above of sensory reception that the trigeminal nerves cover large and extremely important areas for dentists.

The trigeminal nerves also convey motor neurons to muscles derived from the first pharyngeal arch, the major group being the muscles of mastication. Only the mandibular division contains motor axons; the ophthalmic and maxillary divisions are purely sensory.

As shown on Figure 18.5, a large **sensory** and a small **motor root** arise from the lateral aspect of the pons on each side to form each trigeminal nerve.Figure 18.7 shows the large **trigeminal ganglion** on the sensory root which contains most of the sensory cell bodies. The ganglion is located within the dural trigeminal cave in the middle cranial fossa. Each nerve divides into its three divisions distal to the ganglion. The motor root bypasses the ganglion and joins the mandibular division.

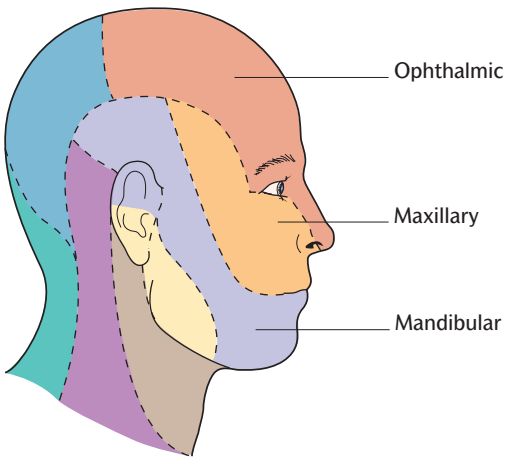


Fig. 18.8 The cutaneous distribution of the trigeminal nerve.

Most of the major branches of the trigeminal nerve are illustrated in Figure 18.7. Follow the diagram as you read the description. The **ophthalmic** and **maxillary divisions** of the trigeminal nerve leave the upper part of the ganglion and pass forwards into the lateral wall of the cavernous sinus. The ophthalmic division leaves the cranial cavity and enters the orbit through the superior orbital fissure. The maxillary division exits through the foramen rotundum into the pterygopalatine fossa. The **mandibular division**, the largest of the three divisions, emerges from the lower part of the ganglion and passes through the foramen ovale into the infratemporal fossa after a very short intracranial course.

A simple way to recall the areas and structures supplied by the three divisions of the trigeminal is to remember the cutaneous distribution of the nerve—the areas of facial skin supplied by each division. These are illustrated in Figure 18.8. As you can see, the ophthalmic division supplies skin from the vertex (highest point) of the skull down to the upper eyelid and a narrow strip down the nose. If you literally fall flat on your face, the ophthalmic division will signal to you that your nose has met the floor. The maxillary division covers the area between the lower eyelid and upper lip and extends back on to the temple. The mandibular division supplies the skin of the lower lip, chin, and over the mandible and continues up just anterior to the ear on to the temple.

The cutaneous distribution pattern is so useful because any structures lying deep to the cutaneous distribution is supplied by branches of the *same division* of the nerve. Look at Figure 18.7 again. You can see that the eyes lie within the ophthalmic domain as does the upper part of the nasal cavity and some of the air sinuses; these structures are innervated by branches of the ophthalmic division. The majority of the nasal cavity, the roof of the mouth, and upper teeth fall within the distribution of the maxillary division and are innervated by it. Figure 18.7 shows the branches of

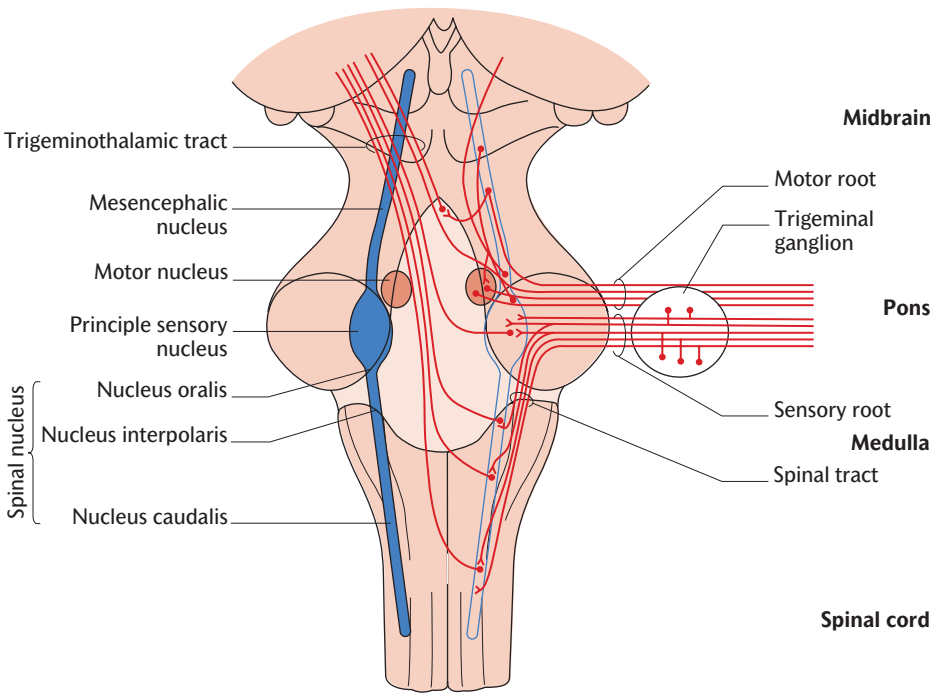


Fig. 18.9 The trigeminal sensory nuclear complex and some of its connections.

the mandibular division distributed to the lower teeth, tongue, and floor of the mouth within the cutaneous area covered by this division.

18.6.1 The trigeminal nuclei

Using the rules for locating cranial nerve nuclei, the trigeminal nerves should have a sensory and motor nucleus on each side located in the pons.Figure 18.2 shows that this is the case, but as outlined above, each trigeminal sensory nucleus extends up into the midbrain and down even beyond the medulla to extend into the upper few segments of the spinal cord. The reason for this spread well beyond the expected region is that the trigeminal nerves have such a large and important area to cover where detailed discrimination of sensory stimuli is absolutely crucial to efficient function. Just think how sensitive your lips and tongue are and how you can sense the tiniest piece of food lodged between your teeth. As we saw in Chapter 3, fine discrimination is only possible if there is little or no convergence of peripheral processes when they synapse with thalamic projection neurons in the CNS. A large number of projection neurons are, therefore, required to ensure accurate localization and discrimination of sensory information; this number of neurons will simply not fit into the pons and, therefore, extend into adjacent areas of the brainstem.

The trigeminal sensory nuclei (often referred to as the **trigeminal sensory nuclear complex** or **TSNC**) are subdivided into different components on the basis of their position and microscopic structure. The subdivisions and their connections are shown in Figure 18.9 and the diagram should be followed as you read the following description.

Large diameter processes conveying discriminative touch from primary sensory neurons synapse in the **principal sensory nucleus** of the trigeminal which is located in the dorsolateral part of the pons. Some of the smaller diameter processes for simple touch and pressure also synapse in this nucleus.

The spinal nucleus

Most of the central processes for simple touch and pressure in the sensory root and the small-calibre processes for pain and temperature turn inferiorly as they enter the brainstem. These processes form the **spinal tract of the trigeminal** lateral to the TSNC; the tract descends through the brainstem as far as the upper three segments of the spinal cord. The portion of the TSNC medial to the trigeminal tract is the **spinal nucleus** which is continuous above with the chief sensory nucleus and below with the dorsal horn of the upper part of the spinal cord. The spinal tract gradually diminishes as it progresses downwards as sensory processes enter successive levels of the nucleus. The processes forming the spinal tract are somatotopically arranged; those from the ophthalmic divisions run in its anterior part, those from the mandibular division in its posterior part, and those from the maxillary division in between the other two.

The spinal nucleus is further subdivided into three main parts as illustrated in Figure 18.9; they are called, from above downwards, the **nucleus oralis**, **nucleus interpolaris**, **and nucleus caudalis**. The processes synapsing on the small and medium-sized thalamic projection cells in the upper two subdivisions are mostly for simple touch and pressure, but may be the area of termination of some taste processes. The function of the nucleus interpolaris is still not clear. It may be the terminal site of descending axons from the sensory cortex that regulate the passage of sensory information in other parts of the TSNC. The nucleus caudalis has a similar cellular composition to the dorsal horn of the spinal cord. Most of the processes ending in this part of the nucleus convey nociceptive or temperature sensations. The representation of the face in the nucleus caudalis is somatotopic. The innervation of the face may be divided into a series of semicircular zones, spreading concentrically from around the mouth as indicated in Figure 18.10. The lips

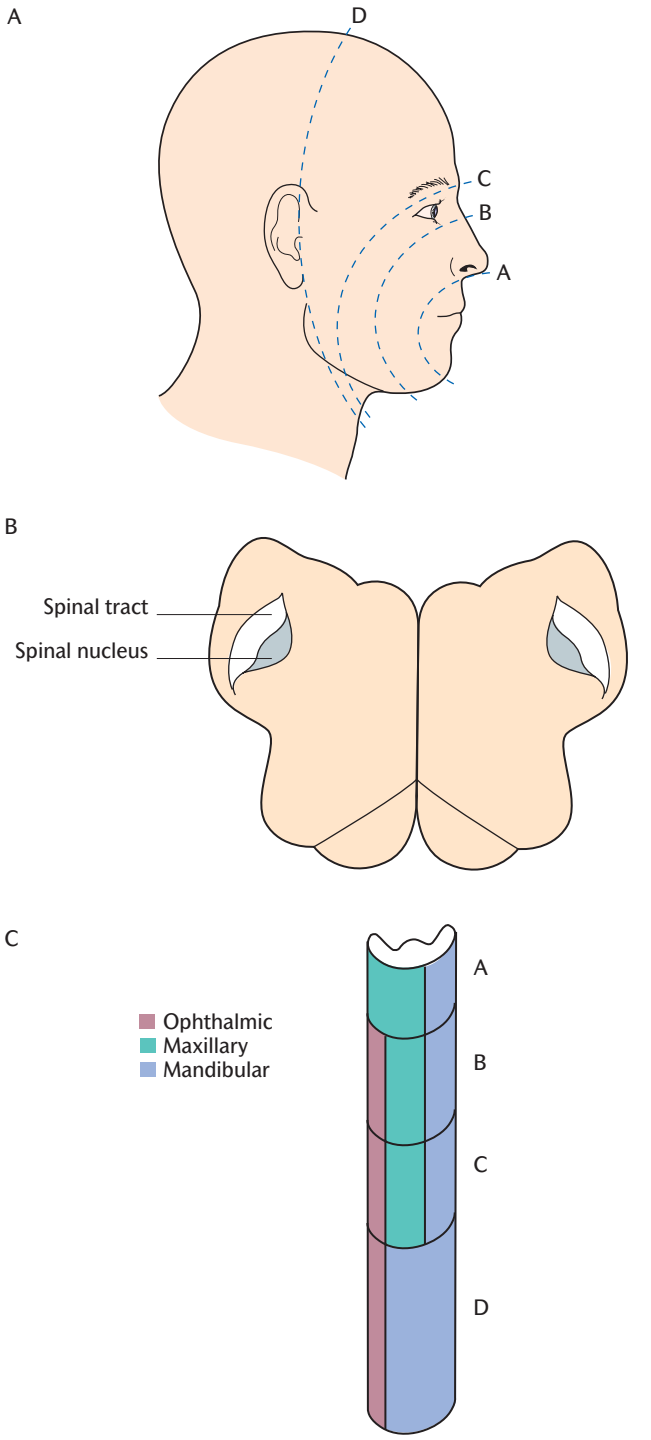


Fig. 18.10 The somatotopic arrangement of the trigeminal spinal nucleus. A) The 'onion skin' pattern of sensory loss. B) The position of the spinal nucleus in the medulla. C) The somatotopic representation in the nucleus. (Redrawn after Shigenaga, Y. *et al. Journal of Comparative Neurology* 243: 388–408 (1986)).

form the innermost area and project to the uppermost segment of the nucleus caudalis. The succeeding zones are represented in successively more inferior segments of the subnucleus. The outermost zone stretching from the lower border of the mandible up across the anterior parts of the auricles to the top of the scalp is represented in the lowermost segment of the subnucleus. This arrangement is often referred to as an 'onion skin' representation of the facial zones as they are arranged rather like the layers of an onion. The somatotopic arrangement of the trigeminal nucleus caudalis is believed to apply to only nociception and temperature. The most caudal part of the nucleus caudalis blends with the upper cervical segments of the dorsal horn and also receives processes conveying nociceptive and temperature information from the upper cervical spinal nerves.

Thalamic projection neurons from the chief and spinal nuclei cross over in the brainstem and travel to the contralateral thalamus in the **trigeminothalamic tract**. They synapse in the **ventroposteromedial (VPM) nucleus** of the thalamus, keeping them separate from neurons conveying sensation from below the head which synapse in the ventroposterolateral (VPL) nucleus. Some neurons projecting from the spinal nucleus connect directly with motor nuclei of various cranial nerves for reflex responses to stimulation of sensory areas supplied by the trigeminal nerves. Reflex salivation in response to the presence of the dentist's fingers and instruments in the patient's mouth is a good example of such connections; sensory stimuli from the oral mucosa are carried in the maxillary and mandibular divisions of the trigeminal and are then conveyed to the salivatory nuclei associated with the facial and glossopharyngeal nerves to cause salivation (see Sections 18.7.1 and 18.9).

The mesencephalic nucleus

Figure 18.9 shows the TSNC extending superiorly from the principal sensory nucleus into the lateral part of the midbrain as the **mesencephalic nucleus of trigeminal**. Trigeminal processes conveying proprioception are unusual in that their cell bodies are located in the mesencephalic nucleus rather than in the trigeminal ganglion as expected. These processes carry information from muscle spindles in the jaw muscles and proprioceptive endings in the temporomandibular joint and the periodontal tissues of the lower teeth in the mandibular division of the trigeminal nerve. Processes travel from endings in the supporting tissues of the upper teeth in the maxillary division. It is thought that proprioceptive information from muscle spindles in the extraocular muscles, and possibly also from spindles in the muscles of the face, tongue, and larynx, also projects to the mesencephalic nucleus. The central processes of the neurons whose cell bodies are located in the mescencephalic nucleus synapse with neurons in the trigeminal motor nucleus for reflex control of jaw movements and also cells in the reticular formation from which axons pass to the thalamus.

The motor nucleus

Trigeminal lower motor neurons begin in each **motor nucleus** which is located in the pons medial to the chief sensory nucleus as shown in Figure 18.9. The axons of these cells run in the motor root of the nerve to the mandibular division through which they are distributed to the muscles of mastication, mylohyoid, anterior belly of digastric, tensor veli palatini, and tensor tympani; all these muscles are derived embryologically from the first pharyngeal (mandibular) arch (see Section 21.5). The motor nucleus receives inputs from both the ipsilateral and contralateral corticonuclear tracts (see Section 16.3.3). As indicated above and in Figure 18.9, the motor nuclei also receive inputs from the trigeminal sensory nuclear complex for reflex responses to stimulation of the areas covered by the sensory distribution of the trigeminal nerve.

The effects of damage and a disease on the trigeminal nerves and the clinical tests used to determine their integrity are described in Box 18.5.

Box 18.5 The effects of damage and disease on the trigeminal nerve

The importance of the trigeminal nerve in the practice of dentistry cannot be emphasized too strongly. Because it is the major sensory nerve to structures in and around the oral cavity, the trigeminal nerve is the target for local anaesthetic procedures designed to lessen or remove pain from dental procedures. The anatomy of local anaesthesia will be explained in more detail in Section 25.5.

Clinical testing of the trigeminal nerves

As with all the cranial nerves, it may be necessary to test the function of the trigeminal nerve, its divisions, or individual branches in different circumstances. Lack of sensation is tested using 'sharp–blunt tests' over the skin of the head and other areas supplied by the nerve. A long pin that has a small ball on the opposite end is applied to the areas of skin under test. The pin is rotated so that either the sharp (pin) or the blunt (ball) end is applied to the area in question. It is important that they are not simply alternated (sharp–blunt, sharp–blunt . . .), otherwise the patient will anticipate the test. The tests should be as random as possible and the patient should be asked after each application of the test probe if they felt anything and if so, what sort of touch. Note that only the *blunt end* of the test probe should be used if testing sensation in the mouth or nose as most patients find the sharp sensation too unpleasant in these areas. If it is necessary to test sensation of the conjunctiva or cornea, this is done using sterile cotton buds or similar instead of the standard probe; the test object must be introduced from the side because if the patient sees it coming, they will reflexly blink.

Lack of motor function is tested by asking the patient to stick out their chin. Normally, the chin will protrude in the midline with no deviation to either side. If the motor component of the trigeminal is damaged on one side, the chin will deviate to the injured side; the muscles that protrude the jaw on the injured side will not function whereas those on the contralateral side will produce their normal action and move the jaw forward, resulting in *deviation to the injured side*.

Diseases affecting the trigeminal nerves

The trigeminal nerves may also be involved in a number of medical conditions. These may lie outside the province of dental practitioners in terms of their treatment but may first be seen in patients attending for dental treatment.

Trigeminal neuralgia is a particularly unpleasant condition in which the patient suffers spasms of violent intractable pain in the area of distribution of one or more of the divisions of the trigeminal nerve. For unknown reasons, this condition seems to affect the maxillary division more than the ophthalmic and mandibular divisions. The spasm is often set off by innocuous stimulation of a particular part (the 'trigger' area) of the skin of the face such as gentle touching or a light breath of air. The cause of trigeminal neuralgia is unknown. The problem may reside in the trigeminal ganglion but, more recently, pressure on the sensory root from a dilated superior cerebellar artery have been suggested. Anticonvulsant drugs that block sodium channels can produce effective pain relief; there are however problems of side effects from long-term administration of such powerful drugs. Microvascular surgery to relieve compression of the nerve roots by aberrant vessels can also be very successful.

Herpes zoster is the virus that causes chickenpox which usually occurs in childhood. The virus often lies latent in sensory nerve ganglia following a previous infection. Reactivation of the virus, usually in adulthood, causes the condition known as **shingles**. Crops of numerous small lesions similar to those seen in chickenpox infections appear on the skin along the course of the sensory nerves whose cell bodies lie in the affected ganglia. It also causes pain in the affected areas. The trigeminal ganglion may also harbour the latent virus and become involved in shingles. When it affects the trigeminal ganglion, the pain and lesions occur on the face and in the mouth, their exact distribution depending upon which divisions of the nerve are involved. If the ophthalmic division is affected, the lesions may occur on the cornea of the eye and without appropriate preventative treatment, may be produce scarring with permanent impairment of vision. More seriously, the virus may move centrally rather than peripherally and enter the cranial cavity, producing viral encephalitis (see Box 15.1).

Any of the three divisions of the trigeminal nerve may be damaged by facial fractures. The ophthalmic division divides into several branches within the orbit and these may be damaged in fractures involving the orbital bones (see Box 30.4). The maxillary division and its branches are almost entirely enclosed within the maxillary bone and may be damaged by fractures of the middle third of the face (see Section 24.5.1). The inferior alveolar branch of the mandibular nerve supplying the lower teeth and their supporting structures runs through the mandible and is, therefore, vulnerable in fractures of the mandible. The lingual branch supplying the mucosa of the tongue with somatic sensation also runs close to the mandible and is also vulnerable, especially during surgical extraction of lower third molar (wisdom) teeth (see Section 24.5).

18.7 The facial nerves (CN VII)

The facial nerves are composed of several different neuronal types.

- Motor neurons are most numerous and innervate the muscles of facial expression and other muscles derived from the second pharyngeal arch (see Section 21.5).
- Parasympathetic preganglionic secretomotor axons supply the lacrimal gland, submandibular and sublingual salivary glands, minor salivary glands in various parts of the oral cavity, and glands in the mucosa of the nasal cavity, paranasal air sinuses, and nasopharynx.
- Special sensory processes convey taste sensations from the taste buds on the anterior two-thirds of the tongue.

The facial nerves arise from the lateral aspect of the brainstem at the junction of pons and medulla together with the vestibulocochlear nerves as shown in Figure 18.5. It is usually possible to distinguish the large **motor root** from a smaller root lying between the motor root of the facial nerve and the vestibulocochlear nerve on each side. This small root is sometimes referred to as the **nervus intermedius** and carries secretomotor and taste neurons. The course of the facial nerve through bone and the branches given off during this part of its course are shown schematically in Figure 18.11 and should be followed as you read the description. The two roots soon unite as they enter the **internal acoustic meatus** in the petrous temporal bone in company with the vestibulocochlear nerve. The vestibulocochlear nerves supply the structures in the inner ear, mediating hearing and balance (see Section 18.8), but the facial nerves continue into the middle ear. Each facial nerve takes a marked change of course here, turning 90° inferiorly to run across the medial wall of the middle ear cavity. This bend is known as the **genu** and is the site where the cell bodies of the sensory taste nerves form a slight bulge called the **geniculate ganglion**. It is also the point at which some parasympathetic neurons diverge from the main nerve trunk to the lacrimal gland, glands in the nose, and upper part of the oral cavity. As the main nerve descends through the middle ear, the remaining parasympathetic nerves and the sensory taste components branch off to form a separate nerve, the **chorda tympani**. The chorda tympani supplies taste buds in the anterior tongue and parasympathetic innervation to the major and minor salivary glands in the floor of the mouth; it actually joins the lingual branch of the mandibular trigeminal nerve close to its emergence from the skull and travels with it to the mouth; the two nerves are indistinguishable once they have joined. The main nerve now contains only motor axons. After it leaves the skull, each nerve travels through the parotid gland where it branches into five divisions to supply different groups of muscles of facial expression (see Section 23.2.4).

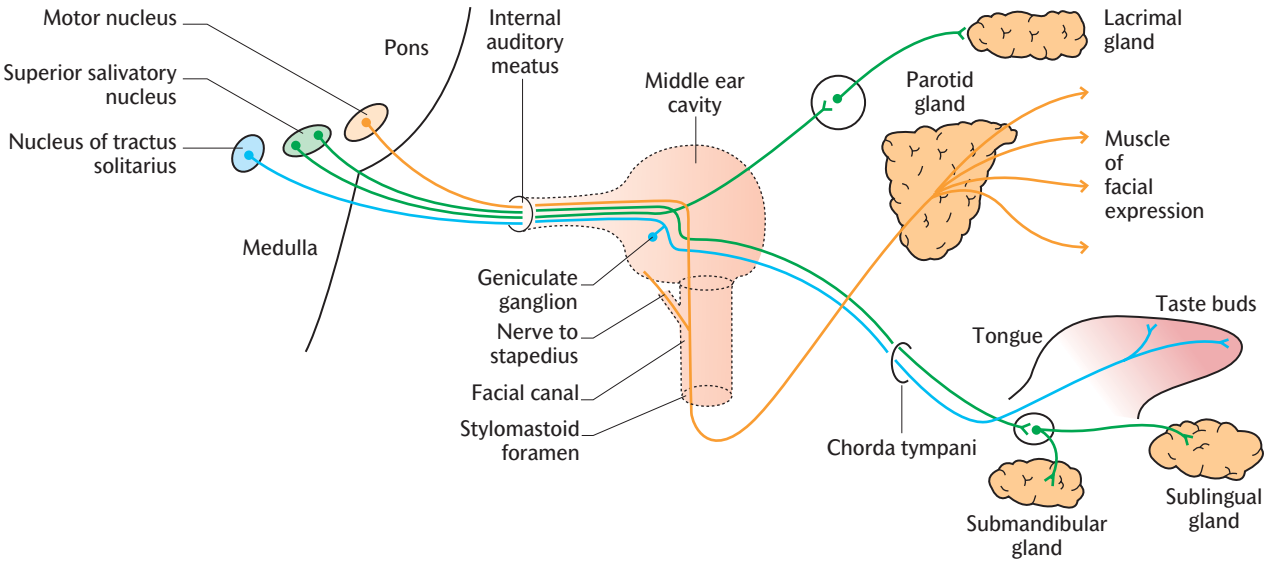


Fig. 18.11 The course of the facial nerve and its branches.

18.7.1 Facial nerve nuclei

The criteria for predicting cranial nerve nuclei indicate that there should be a sensory, parasympathetic, and motor nucleus from lateral to medial on each side of the brainstem for each facial nerve. This is indeed the case as can be seen in Figure 18.2.

The tractus solitarius (gustatory nuclei)

The central processes of taste neurons leave the geniculate ganglion to enter the brainstem through the nervus intermedius. They run inferiorly in the **tractus solitarius** and synapse in the upper part of the **nucleus of the tractus solitarius** which receives taste input; this area is sometimes known as the **gustatory nucleus**. The nucleus has numerous connections with the hypothalamus, the salivatory nuclei of the seventh and ninth cranial nerves, and the dorsal nucleus of the vagus for reflex responses to taste. Thalamic projection neurons from the gustatory nucleus project bilaterally to the VPM nucleus of the thalamus from where tertiary neurons pass to the taste area in the lower part of the post-central gyrus, the area concerned with sensation from the head.

The motor nuclei

The motor neurons begin in the **motor nucleus of the facial nerve** and constitute the whole of the motor root of the nerve. They innervate the muscles of facial expression and other muscles derived from the second pharyngeal arch. As mentioned in Chapter 16, cranial nerve motor nuclei generally receive bilateral inputs from corticonuclear pathways. The facial motor nucleus is an exception; the upper part of the motor nucleus receives inputs from the corticonuclear tracts of both sides whereas the lower part of the nucleus only receives crossed corticonuclear neurons. This has important functional consequences when corticonuclear pathways supplying the facial nerves are damaged (see Box 18.8). The nucleus receives inputs from several other sources, including the tectum of the midbrain and the trigeminal sensory nuclei.

The salivatory nuclei

Preganglionic parasympathetic neurons commence in the **superior salivatory nucleus** situated close to the motor nucleus. Their axons leave in the sensory root of the nerve. Axons destined for the lacrimal gland and the glands of the nasal cavity, nasopharynx, paranasal air sinuses, oral surface of the palate, upper lip, and upper part of the cheek travel in the greater petrosal branch of the facial nerve (Figure 18.11) and synapse in the **pterygopalatine ganglion** (see Section 24.5.3). Post-ganglionic neurons are distributed to their target tissues though branches of the maxillary trigeminal nerve running close to the ganglion. Neurons innervating the submandibular and sublingual glands and the small glands in the floor of the mouth leave the facial nerve in the **chorda tympani**, join the lingual nerve, and relay in the **submandibular ganglion**.

The superior salivatory nucleus receives inputs from the nucleus of the tractus solitarius, the trigeminal sensory nuclei, the olfactory system, and hypothalamus. The part of the nucleus supplying the lacrimal gland (sometimes referred to as the lacrimal nucleus) receives inputs from the trigeminal spinal nucleus for the reflex production of tears in response to corneal and conjunctival stimulation.

18.7.2 Facial nerve damage

The most obvious sign of damage to the facial nerve is **facial paralysis** or **palsy** because the motor components of the facial nerve are the largest components; the delicate superficial branches to individual muscles of facial expression on the face are particularly vulnerable. Observation of the presence and extent of facial paralysis is the basis of clinical testing of the integrity of the facial nerve.

There are several potential causes of facial paralysis and each cause tends to affect a specific point along the course of the facial nerve or the corticonuclear pathways connecting to the motor nuclei. Essentially, there are differences in clinical presentation following injury to lower motor neurons (infranuclear paralysis) described in Box 18.6 and 7 and upper motor neurons (supranuclear paralysis) described in Box 18.8.

Box 18.6 Facial palsy

As shown in Figure 18.11, the facial canal carries the motor branches of the nerve from the middle ear cavity to the stylomastoid foramen where it exits from the skull. If the nerve is compressed in the lower part of the canal, the patient suffers complete flaccid paralysis of all the muscles on the side of the face supplied by the damaged facial nerve. If there is no other aetiology, the resulting facial paralysis is termed **Bell's palsy**. It usually arises spontaneously with no obvious cause but may result from oedema of the tissues lining the canal caused by a viral infection. There is usually a slow recovery of muscle function which is frequently incomplete.

It can be notoriously difficult to observe any paralysis or asymmetry in the face when a patient is expressionless. To test the extent of damage to the facial nerve, the patient is asked to make exaggerated facial gestures to test facial nerve function.*Exaggerated* gesture is important because the patient may still be able to perform some weak facial movements under certain circumstances which could mask the underlying condition. For example, smiling when recognizing a friend is driven by the limbic system whereas a friendly smile to a new patient is driven by conventional motor pathways. Ask the patient to screw their eyes tightly as if faced with a bright light or to purse their lips; you could ask the patient to try to whistle if they are uncertain of what is meant. If the intended movements do not take place, then there is damage to the facial nerve. In complete unilateral facial palsy, they are unable to close the eye, wrinkle the forehead, or puff the cheek; the corner of the mouth may droop on the affected side.

The facial nerve or its branches may be interrupted in the parotid gland or face by trauma, including maxillofacial surgery (see Box 23.9). In such cases, paralysis will only occur in the muscles supplied by the particular branches affected by the trauma, producing flaccid paralysis in the short term. If nerves are damaged beyond repair, atrophy and contracture will eventually follow (see Box 3.2). Muscle atrophy on the face is not noticeable as the muscles of facial expression are very flimsy. Contracture is, however, very obvious, causing serious aesthetic problems as well as functional impairment.

Temporary ipsilateral facial paralysis may occur if local anaesthetic solution is inadvertently introduced into the parotid gland when attempting to give an inferior alveolar block (see Section 25.5).

Box 18.7 Deep damage to the facial nerve

A lower motor neuron lesion may occur at any point along the peripheral course of the facial nerve. If the lesion is close to the CNS, the majority of branches will be affected. A more distal lesion will spare proximal branches which will, therefore, still be functional. It is diagnostically useful to be able to locate the site of the lesion accurately by assessing the extent of damage.

The facial nerve may be affected by middle ear infections (**otitis media**) in its passage through the middle ear. In such cases, there will be facial paralysis as described in Box 18.6, but the branches arising in the middle ear will also be affected. The **chorda tympani** will also be involved, resulting in ipsilateral loss or impairment of taste in the anterior two-thirds of the tongue and secretion by the salivary glands in the floor of the mouth; decrease in salivary output may not be noticed as other salivary glands are still functional. Taste is tested by placing a drop of strong tasting substance (lemon juice or vinegar) laterally on to the suspected injured side of the tongue with a cocktail stick. The patient will be unable to identify the taste immediately, but after a few seconds, the taste will be identified as the substance moves through the saliva film in the mouth to other taste buds that are still innervated.

All functions of the nerve are lost if the facial nerve is affected proximal to the geniculate ganglion. In addition to facial paralysis and loss of taste described above, the nerve supply to the stapedius muscle will be affected. The **stapedius** is a tiny muscle in the middle ear attached to the stapes bone; when loud noises are encountered, it reflexly damps vibration of the ear ossicles to protect the inner ear. If stapedius is paralysed, the patient suffers from **hyperacuity**; sounds seem abnormally loud. There will also be impaired secretion of tears because the secretomotor supply to the **lacrimal gland** is interrupted as well. If the eyes are not efficiently lubricated with tears, the cornea will become sore and may ulcerate—a very unpleasant condition requiring frequent administration of eye drops if the condition persists. One cause of damage to the facial nerve in this part of its course is an **acoustic neurofibroma**, a benign tumour of the vestibulocochlear nerve in the internal acoustic meatus which compresses the facial nerve.

Box 18.8 Supranuclear facial palsy

The most frequent cause of upper motor neuron lesions affecting the cranial nerves is interruption of the corticonuclear pathways as they travel through the internal capsule, e.g. a CVA (stroke) in the narrow perforating arteries supplying this area (see Section 15.5 and Box 15.10). Corticonuclear tract damage results in **paresis** of the affected muscles because of the bilateral innervation of the cranial nerve motor nuclei described in Box 18.4.

The lower part of the facial motor nucleus and the hypoglossal nucleus do not follow this general rule of bilateral innervations through the corticonuclear pathways. The hypoglossal nuclei are low down in the medulla; the corticonuclear axons supplying the hypoglossal nerves cross with corticospinal axons at the pyramidal decussation.

The facial nucleus is an anatomical anomaly. The upper part of each facial motor nucleus receives a bilateral supply from both crossed and uncrossed supranuclear axons as expected. However the supranuclear axons supplying the lower part of the facial motor nucleus, which in turn supplies the muscles of the lower part of the face, receives axons *only* from the opposite side. If these are damaged, there is no alternative input. Following unilateral supranuclear damage, the muscles of facial expression in the forehead and around the eye are still partially innervated so the stroke patient can still wrinkle their forehead and close their eye on both sides to some degree. In contrast, the muscles in the lower part of the face show marked paralysis on the *contralateral* side to the lesion, usually showing as drooping of the corner or the mouth and puffing of the cheek.

Spastic paralysis of the muscles of the lower part of the face, together with weakness in the tongue on the opposite side to the lesion, is consequently a frequent clinical feature in strokes.

18.8 The vestibulocochlear nerves (CN VIII), auditory, and vestibular pathways

The **vestibulocochlear nerves** (also known as the **auditory** or **acoustic** nerves) are the nerves that convey the special senses of hearing and balance from the inner ear to the brainstem; their course is very short. The eighth cranial nerves are the first part of the chain of neurons that form the **auditory pathways** conveying auditory information to the auditory cortex and the **vestibular pathways** sending information about balance to several locations.

18.8.1 The ear

Sound waves are collected by the pinna (or auricle) of the **external ear** and funnelled into the **external auditory meatus** in the petrous temporal bone (see Section 21.3). The **tympanic membrane** (ear drum) separates the external ear from the **middle ear** at the medial end of the external auditory meatus.

The middle ear cavity is illustrated in Figure 18.12A. Sound waves cause the tympanic membrane to vibrate which transfers the vibrations through a chain of small bones (the ear ossicles) to the inner ear. As you can see in Figure 18.12A, the **malleus** (Latin = hammer) is attached to the tympanic membrane and to the **incus** (Latin = anvil). The incus is attached to the tiny **stapes** bone (Latin = stirrup) and the footplate of the stirrup is attached to the **oval window** forming the entrance to the fluid-filled inner ear. The size ratio of the tympanic membrane to the oval window is 15:1 and the leverage through the ossicular chain is around 2; sounds reaching the tympanic membrane are therefore amplified by a factor of 30 when they reach the oval window (15 x 2).

As illustrated in Figure 18.12A, the **inner ear** comprises the snail-like **cochlea** and the **vestibule** containing the three semicircular canals.Figure 18.12B shows the **hair cells** of the **organ of Corti** within the cochlear part of the inner ear which transform sound waves from physical form to electrical impulses. These connect with the peripheral processes of sensory neurons whose cell bodies are located in the **spiral** (or **cochlear**) **ganglion** within the cochlea. The central processes of these cells constitute the **cochlear nerves**; each leaves the inner ear through the **internal acoustic meatus** where it unites with the nerve from the vestibular part of the internal ear to form the vestibulocochlear nerves. They enter the brainstem at the junction of the pons and medulla, the **cerebellopontine angle**, alongside the components of the seventh nerves as shown in Figure 18.5.

18.8.2 The auditory pathways

Each central process of the cochlear parts of the eighth cranial nerves divides as it enters the brainstem. One branch goes to the **dorsal cochlear nucleus** and the other one to the **ventral cochlear nucleus**. These nuclei are not illustrated in Figure 18.2; they do not fall into the usual

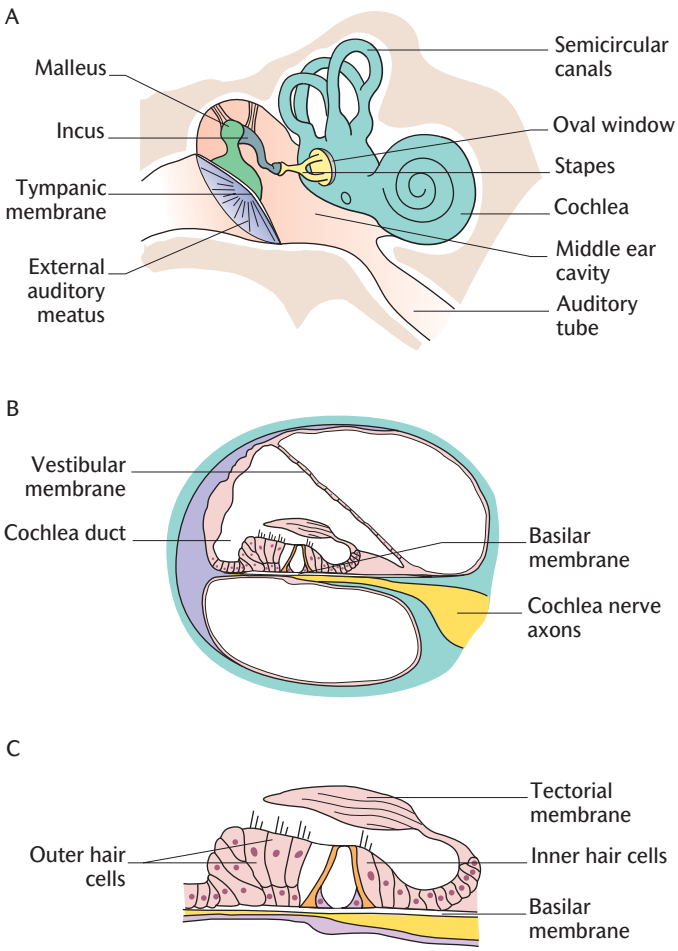


Fig. 18.12 A) The outer, middle, and inner ear. B) The cochlea in cross section. C) The organ of Corti showing hair cells.

scheme for working out the position of cranial nerve nuclei because they serve special sensory functions.

The neurons constituting the auditory pathway ascend from the cochlear nuclei to the **inferior colliculi** of the midbrain. Many neurons make intermediate synaptic connections in the superior olivary nuclei located at the level of the pontomedullary junction. The **superior olivary nuclei** determine the delay between sounds from each ear and thus determine the direction of the origin of the sound. The directly projecting neurons synapse in the contralateral inferior colliculus, but those that synapse in the superior olivary nucleus project to both colliculi. Post-synaptic neurons project from the inferior colliculus to the **medial geniculate nucleus** of the thalamus. Neurons project from the thalamus via the sublentiform part of the internal capsule to the **auditory cortex** in the temporal lobe of the cerebral hemisphere. The neurons constituting the auditory pathways exhibit a complex series of uncrossed and crossed connections, with decussations occurring at most levels of the pathway. The result is that both ears project to both cortices.

The superior olivary nuclei and inferior colliculi connect with the nuclei of other cranial nerves controlling eye movement and with the ventral horn of the cervical spinal cord controlling head movements. These connections enable reflex responses to auditory stimuli such as turning the eyes or head towards the source of sound. Other important connections are with the area of motor nucleus of the seventh nerve controlling the **stapedius muscle** and a similar area in the motor nucleus of the fifth nerve controlling the **tensor tympani muscle**. As already mentioned in the context of the facial nerve, the stapedius muscle is connected to the stapes bone and the tensor tympani to the tympanic membrane; the tensor tympani is connected to the tympanic membrane at the other end of the ossicular chain. Both these muscles contract reflexly when loud sounds are detected; they stiffen the ossicular chain in the middle ear, damping their vibration and protecting the delicate bones and mechanisms within the inner ear.

18.8.3 The vestibular pathways

The structures in the inner ear served by the vestibular components provides information to the CNS, which plays a major part in maintaining equilibrium, together with information from the visual system and from proprioceptive endings scattered throughout the body. The vestibular labyrinth consists of the **utricle** and **saccule** that monitor the static position of the head and the three **semicircular canals** which detect movements of the head.

The vestibular parts of the vestibulocochlear nerves originate from receptor cells in the various parts of the vestibular labyrinth and join with the cochlear portion to follow the same route to the brainstem. Their cell bodies form the **vestibular ganglion** close to the lateral end of the internal acoustic meatus. Their central processes constitute the **vestibular nerve.** Most of the central processes synapse in the **vestibular nuclei** of the brainstem which make connections with the cerebellum, spinal cord, brainstem, and cerebral cortex, but some pass directly to the cerebellum. The vestibular nuclei are the origins of the vestibulospinal tracts which are part of the lateral motor pathways; their course and functions have been described in Section 16.3.

The effects of damage to or disease of the vestibulocochlear nerve or the special sensory receptors in the inner ear are described in Box 18.9.

Box 18.9 Vestibulocochlear and inner ear damage and disease

Damage to the vestibulocochlear nerves through trauma or disease is relatively uncommon because of their short course deep within the robust petrous temporal bones. However, the sensory components in the inner ear serving hearing and balance are often affected.

Hearing loss takes two forms. **Conductive hearing loss** (**CHL**) occurs when conduction of sound through the outer and middle ears is impeded. There are several potential causes, varying from accumulation of water or build up of excess wax in the outer ear to infections of the middle ear (**otitis media**) or arthritic changes in the ear ossicles that impede movements of the bones and thus diminish efficient sound conduction to the inner ear. **Sensorineural hearing loss** (**SNHL**) occurs when transduction of sound is compromised, usually by damage to **hair cells** in the inner ear, but, more rarely, through damage to the eighth cranial nerves. The hair cells may be damaged by some antibiotics but most frequently through age-related hearing loss (**presbyacusis**) or environmental noise damage such as failing to wear recommended ear protection when using machinery or even playing personal music devices too loudly. In SNHL, there is usually selective loss of certain frequencies because hair cells are arranged tonotopically within the cochlea; hair cells responding to high frequencies are located at the base of the coiled cochlea nearest the oval window whereas those responding to low frequencies are near the apex. For example, older people suffering from presbyacusis find it most difficult to hear female and children's voices as these are of higher frequency than male voices. In addition, some specific components of speech become more difficult to distinguish because these are at higher frequencies.

Clinical testing of vestibulocochlear nerve function

CHL and SNHL may be distinguished by using a tuning fork. If hearing is normal, a vibrating tuning fork held close to the ear should be audible. If it is not, the flat base of the tuning fork is placed against the mastoid process of the temporal bone which can be felt just below the ear (see Figure 20.4). If sound can now be heard, this is because it is being conducted through bone to the inner ear. The absence of air conduction but successful bone conduction indicates that the patient has CHL; the inner ear is transducing sound successfully, but something is impeding conduction of sound in the outer or middle ear which would warrant further investigation.

If hearing difficulty is still encountered even through bone conduction, this strongly suggests SNHL. Further investigation of this

condition is carried out by audiologists using special equipment who determine the range of frequencies lost and the sensitivity at each frequency.

Otitis media can often affect the vestibular components of the inner ear. If fluid movement within the vestibule is stimulated by heating effects rather than actual movement of the head by introducing warm water into the outer ear, for example, the patient will feel dizzy and nauseous because the brain thinks the head is moving when in fact it is not. Otitis media, just like any other inflammatory condition, produces swelling, pain, redness, and heat. The heat produced in inflammatory reactions can set up conduction currents and movement of fluid within the vestibule that stimulate the receptors that determine head position and movement, causing dizziness and nausea.

18.9 The glossopharyngeal nerves (CN IX)

The glossopharyngeal nerves have several functions, but are mainly sensory. They:

- Carry **somatosensory sensations** of touch, pain, and temperature from the posterior part of the tongue (Greek 'glossus' = tongue) and pharynx, hence the name of these nerves; they are also sensory to the middle ear cavity;
- Supply **taste buds** in the posterior one-third of the tongue and adjacent areas of the pharyngeal wall with taste sensory neurons;
- Supply the baroreceptors in the carotid sinus and the chemoreceptors in the carotid body through **general visceral sensory** neurons;
- Supply **parasympathetic** secretomotor innervation to the parotid salivary glands;
- Supply a single pair of muscles, the stylopharyngeus muscles.

Given that these nerves have a large sensory role with only minor motor functions, you might anticipate the glossopharyngeal nerves would have large sensory and small motor nuclei. However, you will search in vain for glossopharyngeal nuclei in Figure 18.2; these nerves mainly use the nuclei of other cranial nerves or nuclei which are named by their function.

The glossopharyngeal nerves exit as fine rootlets from the upper part of the medulla as seen in Figure 18.5. They join to form definitive nerves as they exit the skull through the jugular foramina. Each nerve passes down the pharyngeal wall before breaking up into numerous fine nerves that constitute the **pharyngeal plexus** together with branches from the vagus nerves. The lingual branch to the tongue and the carotid branch to the carotid bodies arise before the main nerve becomes the pharyngeal plexus.

The cell bodies of the somatosensory neurons from the tongue, pharynx, and middle ear are situated in the small superior glossopharyngeal ganglion. Their central processes pass via the trigeminal spinal tract to end in the **spinal nucleus** of the **TSNC.**

The cell bodies of the sensory neurons from taste buds in the posterior one-third of the tongue and adjacent areas are located in the inferior glossopharyngeal ganglion and the central processes terminate in the upper part of the nucleus of the **tractus solitarius** with taste neurons from the facial nerve.

The cell bodies of general visceral sensory neurons are also situated in the inferior glossopharyngeal ganglion. Their central processes end in the lower part of the **nucleus** of the **tractus solitarius** where reflex connections are made with the cardiovascular and respiratory centres in the medulla and with the hypothalamus.

The **inferior salivatory nuclei** are the only nuclei that can be clearly ascribed to the glossopharyngeal nerves and are the origin of preganglionic parasympathetic neurons to the parotid glands. Each inferior salivatory nucleus lies immediately below the superior salivatory nucleus. Both salivatory nuclei have connections with the nucleus of the tractus solitarius, trigeminal sensory nuclei, olfactory system, and hypothalamus. These connections are responsible for reflex salivary secretion in response to taste, jaw movement, oral sensation, smell, or emotion, respectively. Preganglionic parasympathetic axons from the inferior salivatory nuclei supply the parotid glands, minor salivary glands in the lower lip and lower cheek, and glands in the mucous lining of the oral and laryngeal parts of the pharynx.

The motor neurons to the stylopharyngeus muscles begin in the upper part of the **nucleus ambiguus** of the vagus nerve described in the next section.

Box 18.10 describes the effects of damage to the glossopharyngeal nerves and the clinical tests for their function.

Box 18.10 The effects of damage to or diseases of the glossopharyngeal nerves

The course of the glossopharyngeal nerves is relatively deep so these nerves are relatively immune from direct trauma. The most frequent causes of damage to the nerves are by compression from space-occupying lesions in the pharynx or posterior tongue or invasion by malignant tumours in these areas. Possibly, the most prevalent cause of disruption of glossopharyngeal nerve function is damage to their central sensory components within the brainstem following brainstem stroke.

The sensory nerves from the pharynx are the afferent components of the **gag reflex** which is stimulated by touching the pharyngeal wall with a blunt object with the mouth open. If the same area is touched by food with the mouth closed, the **swallowing reflex** is stimulated (see Chapter 29). The clinical test for function of the glossopharyngeal nerves is to test the **gag reflex**.

18.10 The vagus nerves (CN X)

We have already encountered the vagus nerves in the context of the autonomic nervous system in Chapters 3, 12, and 17; these frequent references emphasize that the vagus nerves are the most important **parasympathetic** nerves of the body, regulating the function of all the thoracic and most of the abdominal organs. The nerves also contain a large number of **general visceral sensory** neurons from the viscera of the thorax and abdomen, including baroreceptors in the aortic arch and the chemoreceptors in the aortic bodies.

The vagus nerves also have important functions in the head and neck where they carry:

- Motor neurons to muscles of the soft palate, pharynx, and larynx;
- Sensory neurons from the larynx;
- A few taste buds on the epiglottis.

The vagus nerves leave the medulla as a group of rootlets below those of the glossopharyngeal nerves as seen in Figure 18.5. These rootlets join to form each vagus nerve as they pass through the jugular foramen on each side. Each vagus nerve travels down through the neck between the carotid artery and internal jugular vein to enter the thorax. **Pharyngeal** and **superior laryngeal nerves** arise as it passes through the neck. Each **recurrent laryngeal nerve** arises in the thorax as described in Chapter 12 and illustrated in Figure 12.11.

In Figure 18.2, the dorsal nucleus of the vagus is obviously associated with the vagus nerve. The nucleus ambiguous is also a major nucleus of the vagus. These are the nuclei from which parasympathetic and motor neurons originate, respectively. The sensory functions listed above are carried out through the nuclei of other cranial nerves.

The parasympathetic preganglionic neurons begin in the **dorsal nuclei of the vagus**, a large collection of cells extending on each side throughout much of the medulla; they are below and in line with the salivatory nuclei. Preganglionic neurons are distributed through the many thoracic and abdominal branches of the vagus and synapse with post-ganglionic neurons in autonomic ganglia or plexuses in or close to the organs being supplied. The dorsal nuclei receive inputs from the hypothalamus, the nucleus of the tractus solitarius, and the cardiovascular and respiratory centres, enabling the vagal parasympathetic neurons to maintain appropriate levels of visceral activity to meet functional demands and maintain homeostasis.

The motor neurons to the skeletal muscles of the pharynx, larynx, and soft palate begin in each **nucleus ambiguus**. This nucleus migrates during embryonic development and so lies more laterally in the brainstem than might be anticipated for a motor nucleus; its ambiguous position gives its name. The motor neurons are distributed through **pharyngeal nerves** to the muscles of the pharynx and soft palate and via the **superior laryngeal** and **recurrent laryngeal nerves** to the muscles of the larynx. Vagal motor neurons which also begin in the nucleus ambiguus also supply striated muscle in the upper two-thirds of the oesophagus. The nucleus ambiguus receives inputs from corticonuclear tracts, the sensory nuclei of the trigeminal nerve, and the nucleus of the tractus solitarius. The last two connections provide the central reflex pathways involving the muscles of the larynx, pharynx, and soft palate, including swallowing and coughing.

The cell bodies of the neurons carrying visceral sensory information are located in the inferior ganglion of each vagus nerve. Their central processes end in the inferior part of the **nucleus** of the **tractus solitarius** close to those from the glossopharyngeal nerves. As described in Section 18.9, this nucleus has connections of the hypothalamus and the cardiovascular and respiratory centres of the brainstem. These connections are involved in the reflex control of cardiovascular, respiratory, and alimentary activity.

The cell bodies of the few neurons from taste buds are in the inferior vagal ganglion and their central processes end in the upper part of the **nucleus of the tractus solitarius** with other taste neurons from the seventh and ninth nerves.

The vagus nerves contain sensory neurons carrying touch, temperature, and nociception from the pharynx, oesophagus, and larynx. The vagus nerves also supply neurons to a small area of the external ear and part of the eardrum through the auricular branches. The cell bodies of somatic sensory neurons are in the small superior ganglion of the vagus and their central processes end in the **spinal trigeminal nucleus** like those from the glossopharyngeal nerve.

The effects of damage to the vagus nerves and clinical tests used to ascertain their function are described in Box 18.11.

Box 18.11 The effects of damage to or diseases of the vagus nerves

The vagus nerves are relatively deep and are affected mainly by space-occupying lesions compressing the nerves or invasion by malignant tumours. The major cause of damage to the vagus nerves is usually through damage to their nuclei through brainstem stroke.

Loss of innervation to the laryngeal muscles will produce **dysphonia**, problems with the phonatory component of speech production. Phonation is the production of noise within the larynx by vibration of the vocal folds and is a component of all vowel sounds and about half of the consonants in spoken English (see Chapter 29). Loss of motor innervation to the larynx also makes it difficult to close the vocal folds sufficiently to build up pressure required to cough. Paralysis of the muscles of the soft palate will also affect those components of speech requiring movement of the soft palate, producing some degree of **dysarthria**. Deficiency of muscle activity in the pharynx produces difficulty with swallowing (**dysphagia**).

If the sensory supply to the larynx is compromised, the patient is unaware of foreign material in the larynx. The presence of such things as food or drink in the larynx will normally elicit a cough reflex. If this does not happen, the patient is likely to aspirate the food into the lower respiratory tract; this is **silent aspiration**. The food will lodge in the bronchial tree and may become infected which, in turn, can cause pneumonia with possible serious consequences.

Clinical tests of vagal function

There are two straightforward clinical tests for the vagus nerves. For the first test, the patient is asked to say a prolonged 'Aaaaa' which involves raising the soft palate. Normally, the soft palate should elevate symmetrically. If only one side raises this is **palatal insufficiency** and indicates that there is damage to the vagus nerve on the immobile side. The second test is asking the patient to cough as strongly as possible. If the cough is weak or absent, this is indicative that the vagal supply to the muscles of the larynx is deficient; they are unable to close their larynx forcefully enough to build up the required air pressure for a cough.

18.11 The accessory nerves (CN XI)

The accessory nerves are usually described as having cranial and spinal roots. They have different embryological and anatomical origins. The **cranial accessory** roots which arise from the nucleus ambiguus of the vagus are aberrant roots of the vagus nerve and join with it in the jugular foramen. They contribute to the motor supply of the muscles innervated by the vagus nerves.

The spinal accessory nerves are distinct entities; they are not true cranial nerves because they do not arise from the brain. They are motor nerves that begin in the lateral part of the ventral grey horn of the first to fifth cervical segments of the spinal cord and supply the trapezius and sternocleidomastoid muscles in the neck (see Section 23.1.4). The axons contributing to the spinal accessory nerves do not exit from the spinal cord in the ventral roots as might be anticipated, but by distinct accessory roots between the dorsal and ventral roots. Axons from the different segments combine to form the nerve.

Each spinal accessory nerve ascends on the lateral aspect of the spinal cord to enter the cranial cavity through the foramen magnum alongside the spinal cord and its meningeal coverings. Each nerve then loops back to exit from the cranial cavity through the jugular foramen with the glossopharyngeal and vagus nerves.

The clinical tests for accessory nerve function are described in Box 18.12. The possible reasons for the bizarre course of the spinal accessory nerves are explored in Box 18.13.

Box 18.12 Clinical testing of the spinal accessory nerves

It is a principle of clinical testing of motor nerve injuries that the muscles controlled by the nerve under test are made to move *against resistance* wherever possible. This eliminates compensatory trick movements the patient may have developed and the effect of other muscles with different innervations that may produce the same action. If the nerve is functional, the muscle will either stand out if superficial or can be palpated if not too deep.

The spinal accessory nerve may be tested by asking the patient to raise their shoulders against the resistance of the examiner's hands pressing the shoulders down; this tests the action of the trapezius muscles. Alternatively or additionally, the patient can be asked to turn the head to one side against resistance of the examiner's hand; this tests one of the actions of the sternocleidomastoid muscles. If the patient cannot raise the shoulders or cannot turn their head and the trapezius or sternocleidomastoid muscles cannot be seen to contract, then the accessory nerve is damaged on the affected side.

Box 18.13 The origin and course of the spinal accessory nerves

The reasons for the peculiar origin and course of the spinal accessory nerves are still disputed. The embryological origin of trapezius and sternocleidomastoid muscles is still unclear. These muscles may be of pharyngeal arch origin (see Chapter 21) and the spinal accessory may be a detached part of the vagus nerve. The fact that the motor neurons supplying the trapezius and sternocleidomastoid first differentiate close to the cells that will form the nucleus ambiguus may support this view. An alternative view is that the spinal accessory is the fused ventral roots of a number of cervical spinal nerves which the anatomical evidence supports. However, it is difficult to explain why the trapezius and sternocleidomastoid muscles are not simply supplied by motor neurons of the first to fifth cervical spinal segments exiting conventionally through their ventral roots, but have nerve supplies that follow a separate and somewhat bizarre course.

18.12 The hypoglossal nerves (CN XII)

The hypoglossal nerves are purely somatic motor nerves supplying the muscles of the tongue. We would, therefore, anticipate only one motor nucleus on each side low down in the brainstem which is, in fact, the case. As shown schematically in Figure 18.2, each **hypoglossal nucleus** is located in the medulla in the grey matter surrounding the central canal. It is the most inferior of the column, containing somatic motor

Box 18.14 Damage to the hypoglossal nerves and the clinical tests

The actions of the hypoglossal nerves may be affected by supranuclear problems such as brainstem stroke interrupting the corticonuclear pathways. Unilateral interruption of the corticobulbar tract causes weakness and spasticity of the *opposite* side of the tongue. This may seem to contradict what was said about the effects of supranuclear damage in Box 18.4. The hypoglossal nuclei are, in fact, so low down in the brainstem that the corticonuclear axons destined for these nuclei run with corticospinal axons and actually cross over in the pyramidal decussation.

Damage to the nucleus by brainstem stroke or to the nerve in its peripheral course by malignant lesions in the floor of the mouth, for example, produces paralysis and atrophy of the lingual muscles on the side of the lesion.

The hypoglossal nerve is tested by asking the patient to stick out (protrude) the tongue. If one hypoglossal nerve is damaged, the tongue deviates to the *affected* side because of the unopposed action of the normal functioning muscle on the other side.

nuclei. As you can see in Figure 18.5, axons from the nucleus emerge from the brainstem as a series of rootlets between the pyramid and olive on each side. These rootlets join together to form the main nerve trunk which leaves the cranial cavity through the hypoglossal canal and unite just outside the skull.

The hypoglossal nuclei receive most of their inputs from the contralateral corticonuclear tract which cross with the corticospinal pathways in the pyramidal decussation. They also receive inputs from the trigeminal sensory nuclear complex and the nucleus of the tractus solitarius to enable coordination of tongue movements with those of other muscle groups involved in chewing and swallowing.

Box 18.14 describes the effects of damage to the hypoglossal nerves and the clinical tests for their function.

19

Development of the central nervous system

Chapter contents

19.1 Introduction

The early development of the nervous system, the process of **neurulation**, has already been outlined in Chapter 8 and illustrated in Figure 8.4. To briefly recap, an area of dorsal ectoderm is induced by the underlying notochord to form the **neural plate** during the third week of development. The lateral edges of the neural plate rise to form the **neural folds** which eventually fold over and unite in the midline by the end of the fourth week to produce the **neural tube**. A distinct cell population on the crest of the neural folds, the **neural crest,** migrates from the forming neural tube to form various structures, including components of the peripheral nervous system (see p. 183). The closed **neural tube** consists of a large diameter anterior portion that will become the brain and a longer cylindrical posterior section, the future **spinal cord.**

19.2 Differentiation of the spinal cord

Initially, the neural plate is a single cell layer, but concentric layers of cells can be recognized by the time the neural tube has closed. An inner layer of ependymal cells surrounds the central spinal canal. **Neuroblasts**, the precursors of neurons, make up the bulk of the neural tube called the **mantle layer**; this will become the **grey matter** of the spinal cord. Neuroblasts do not extend processes until they have completed their differentiation. When the cells in a particular location are fully differentiated, the neuronal processes emerging from the neuroblasts form an outer **marginal layer** which ultimately becomes the **white matter** of the spinal cord.

Figure 19.1B shows that the neural tube changes shape due to proliferation of cells in the mantle layer. This figure also indicates two midline structures in the roof and floor of the tube, known as the **roof plate** and **floor plate**. They are important in the determination of the types of neurons that develop from the mantle layer. The **floor plate** is induced by the expression of a protein product of a gene called **sonic hedgehog (SHH)** produced by the underlying notochord; the floor plate then expresses the same gene itself. Neuroblasts nearest to the floor plate receive a high dose of SHH protein and respond by differentiating into motor neurons; as seen in Figure 19.1B, these cells group together to form bilateral ventrolateral **basal plates.** These plates are the future ventral horns seen in Figure 19.1C. Neuroblasts further away receive a lesser dose of SHH and become interneurons. As the neural tube closes, signalling molecules known as **bone morphogenic proteins (BMPs)** from the overlying ectoderm induce the formation of the **roof plate**. As illustrated in Figure 19.1B, the roof plate itself then secretes BMPs which induce the formation of dorsal horn thalamic projection neurons within the **alar plates** which will become the dorsal horns. Induction from the notochord and ectoderm determines the dorsoventral distribution of cell types within the developing CNS. The differential distribution of sensory and motor functions seen in the mature CNS is thus determined remarkably early in embryonic life.

As we have seen in Chapter 18, the lower cranial nerves originate from and terminate in their associated sensory and motor nuclei in the medulla. A similar arrangement of grey and white matter might be expected in the medulla because it is an upward continuation of the spinal cord. In the lower part of the medulla oblongata which is directly continuous with the spinal cord, the pattern of grey matter is indeed similar to that in the spinal cord; the basal plates forming motor nuclei tend to lie ventrally and the alar plates making up sensory nuclei are dorsal. Figure 19.2 demonstrates how this arrangement is altered when the central canal expands to form the

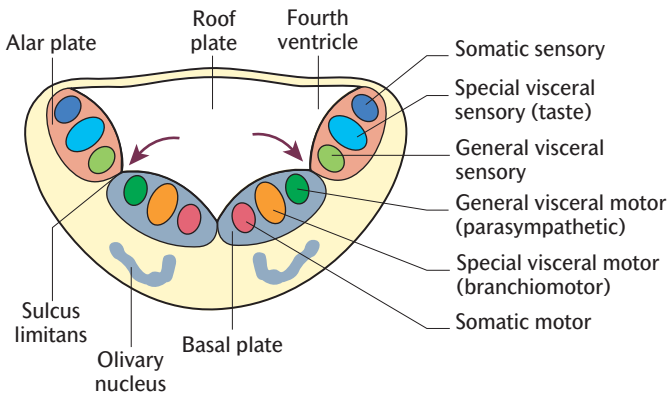


Fig. 19.2 Development of the brainstem and the location of cranial nerve nuclei. The arrows indicate the sulcus limitans.

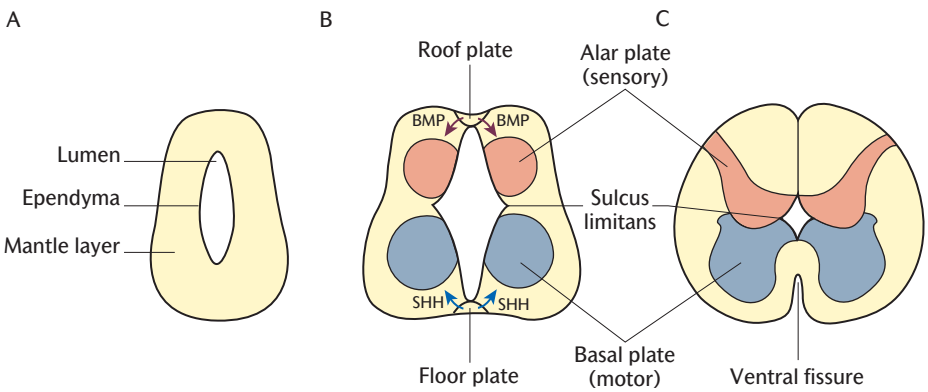


Fig. 19.1 Development of the spinal cord.

fourth ventricle in the upper part of the medulla and the pons. The alar and basal plates are splayed out away from the midline plates which are pushed laterally, but the arrangement of the two plates relative to each other remains the same. Compare the position of the sulcus limitans separating the alar and basal plates in Figure 19.1 and 19.2. The alar plates now lie lateral to the basal plates. The result is that motor nuclei developing from the basal plates lie ventrally near the midline whereas sensory nuclei arising from the alar plates lie more laterally and dorsally; this explains Rule 2 for determining the position of cranial nerve nuclei given in Section 18.2.2).

The **neural crest cells** that form during neurulation contribute to a large number of adult structures, especially in the head and neck (see Chapter 21). They contribute to the PNS throughout the body. In the trunk, neural crest tissue aggregates to form the dorsal root ganglia between somites and also autonomic ganglia. Similarly, neural crest cells form the sensory ganglia of cranial nerves and parasympathetic neurons in the head (see Chapter 21). Peripheral and central sensory and postganglionic autonomic neuronal processes develop from the ganglia.

19.2.1 Vertical specification of the central nervous system

The dorsoventral organization of the nervous system begins very early in development as described in Section 8.3.3. As described in Chapter 8, organization of the embryo along the longitudinal axis begins even earlier as mesodermal tissues are formed during gastrulation.

Recall from Section 8.3.3 that embryonic ectodermal cells are exposed to doses of retinoic acid (RA) as they move through Hensen's node and the primitive streak to become mesenchymal cells. **Homeobox genes** are activated by RA as they pass through the node. The genes closest to the 3' end of the chromosome respond to low doses of RA whereas those nearest to the 5' end are activated by higher doses. The first cells to pass through the primitive streak receive a low dose and end up towards the future head end of the embryo; later migrating cells do not migrate so far. As Figure 19.3 shows, homeobox genes are expressed in a specific sequence along the anteroposterior axis of the part of the neural tube that will become the hindbrain (pons and medulla); the 3' genes are more anterior than the 5' genes because of their exposure to different doses of RA as they migrate. The hindbrain develops a series of segments, the **rhombomeres**, which can be seen clearly under a microscope; as Figure 19.3 shows, the anterior edge of each segment corresponds to the expression boundary of different Hox genes. The hindbrain is the only area of the developing CNS that shows overt segmentation although similar segments can be located between different parts of the developing

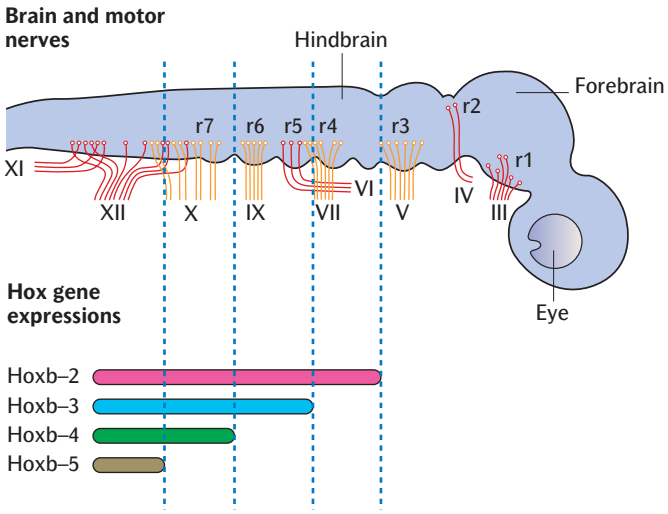


Fig. 19.3 The division of the developing hindbrain into rhombomeres and the expression boundaries of homeobox genes. (Redrawn after Noden, D.M. and Trainor P.A. *Journal of Anatomy* 207: 575–603 (2005)).

brain when gene expression boundaries are examined, but are not visible even under the microscope. As shown in Figure 19.3, neural crest-derived ectomesenchymal cells migrate from particular rhombomeres to populate specific pharyngeal arches. The sensory cranial nerves develop from the neural crest cells and their motor components develop from the basal plate of rhombomeres. The Hox gene coding carried by the ectomesenchymal cells identifies each pharyngeal arch. Cranial nerves will only innervate derivatives of their own arch. The formation of the pharyngeal arches will be covered in more detail in Chapter 21.

The spinal cord is not segmented, but the paraxial mesoderm forming the somites alongside the spinal cord does carry a homeobox gene code that gives the somites at different levels their specific identities. The central sensory processes developing from each dorsal root ganglion and peripheral axons of motor neurons leaving the spinal cord are channelled along specific routes through somites. They can pass through the superior part but cannot pass through the inferior area of each somite. The nerves are thus directed to form bundles in the upper part of the somite which then enter or leave the spinal cord at the same level; the level of attachment of the sensory and motor components of the spinal nerves demarcates the spinal cord segments.

19. 3 Development of the brain

19.3.1 Divisions of the brain

In the early embryo, the head end of the developing neural tube tends to fold ventrally as shown in Figure 19.4A. The **cephalic flexure,** marked by the **ventral sulcus**, probably occurs because the neural tube is growing faster than the tissues forming below it. The area anterior to the ventral sulcus enlarges into the **prosencephalon** or **forebrain**.

Figure 19.4B indicates that a second **pontine flexure** occurs a little later in development posterior to and in the opposite direction to the ventral sulcus. The pontine flexure, also known as the **isthmus**, separates the **midbrain (mesencephalon)** superiorly from the **hindbrain (rhombencephalon)** inferiorly. The isthmus is an organizer region

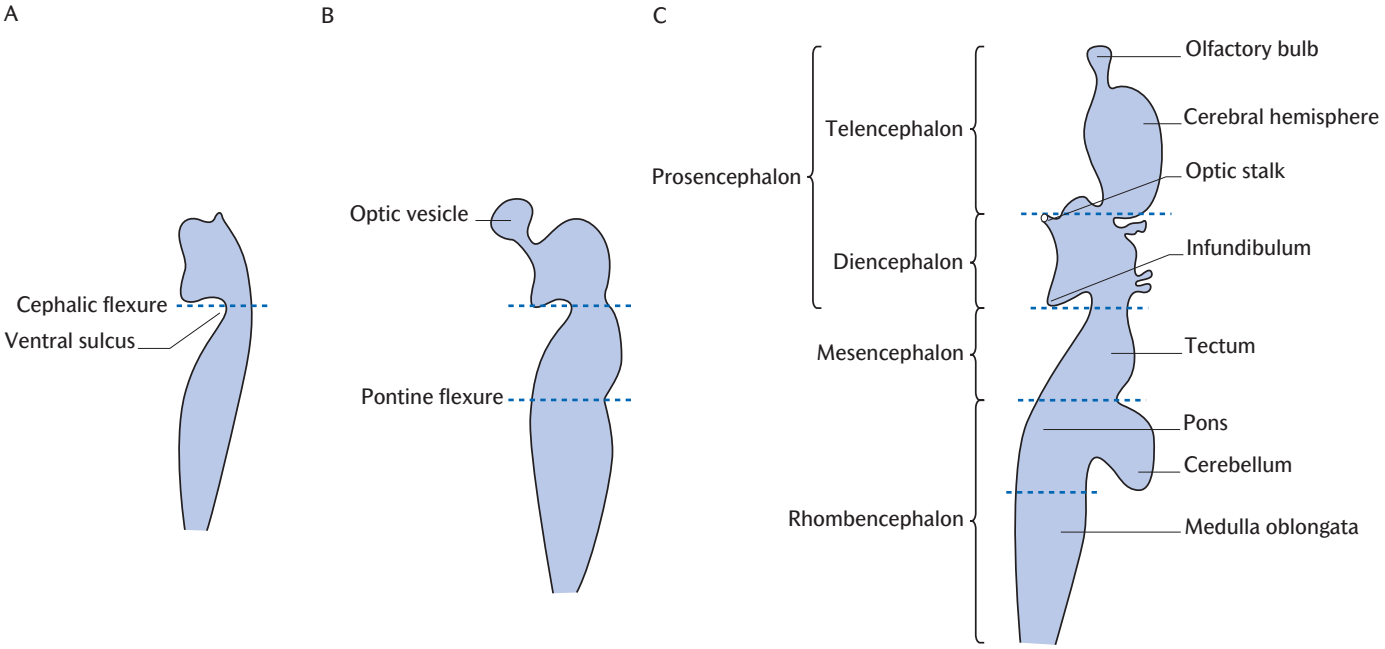


Fig. 19.4 Development of the brain areas from the neural tube.

that secretes signalling molecules that specify neuroblasts superiorly to form midbrain structures and those inferior to the isthmus to form the pons and cerebellum. The boundaries between the isthmus and rhombomeres in the hindbrain prevent the movement of developing neurons between segments so that cells specified to become certain structures remain in the correct location and do not become contaminated with cells from other sources.

In primitive vertebrates, the three gross divisions of the brain are associated with specific sensory inputs—olfaction from the nose to forebrain, vision from the eye to midbrain, and hearing and balance from the ear to hindbrain. During the course of evolution, each division of the brain has developed a posterior extension to increase the number of neurons needed to cope with increasingly large inputs and to develop more sophisticated skills and functions. As shown in Figure 19.4C, the **cerebral hemispheres** developed from the forebrain, the **tectum** from the midbrain, and the **cerebellum** from the hindbrain. The basic vertebrate structure of the brain is maintained during its further embryonic development. The front part of the hindbrain, (the metencephalon**)** forms the **pons** with its dorsal outgrowth, the **cerebellum**. The cerebellum develops in the roof of the rhombencephalon above the anterior part of the fourth ventricle. The roof is wide posteriorly and very narrow anteriorly in this region. The narrow anterior area thickens to form the **cerebellar plate**. By the twelfth week of development, the developing cerebellum begins to resemble the mature structure; paired **cerebellar hemispheres** have started to develop laterally to the small midline vermis. The remainder of the rhombencephalon gives rise to the **medulla oblongata**.

The midbrain tectum has lost its function as the primary visual cortex in higher animals; echoes of its evolutionary history still remain as shown by the function of the **superior colliculi** as centres coordinating reflexes in response to visual stimuli (see Section 15.3.2). The inferior part of the tectum (the **inferior colliculi**) provides relays for the auditory pathway from the cochlear nuclei to the thalami.

The changes in the forebrain are even more dramatic. The **diencephalon** is the original unpaired precursor of the forebrain. As shown in Figure 19.4C, paired outgrowths from the diencephalon then grow forwards to form the **cerebral hemispheres** and the **olfactory bulbs**. These structures together make up the **telencephalon**. Optic vesicles extend from the diencephalon remaining attached by the **optic stalks** which later develop into the **optic nerves**. The distal end of each optic vesicle comes into contact with a dense sheet of surface ectoderm called the lens placode which will form the lens of the eye; the optic vesicle becomes the retina. More inferiorly, the infundibulum, a precursor of the pituitary stalk, grows down towards the roof of the developing oral nasal cavity. These will later meet to form the **pituitary gland**.

The sequential development and changes in relationships and position of the forebrain, midbrain and hindbrain, and their derivatives are illustrated diagrammatically in Figure 19.5.

19.3.2 Development of the cerebral hemispheres

As we saw in Chapter 15, the cerebral hemispheres are the largest components of the human brain. As you can see in Figure 19.5, the developing hemispheres overgrow the diencephalon which becomes buried. There is a limit to the increase in volume of the cerebral cortex which can be achieved by simple expansion. Further increase is most efficiently achieved by folding. As described and illustrated in Chapter 15, the surface of the cerebral hemispheres in the human brain and those of more

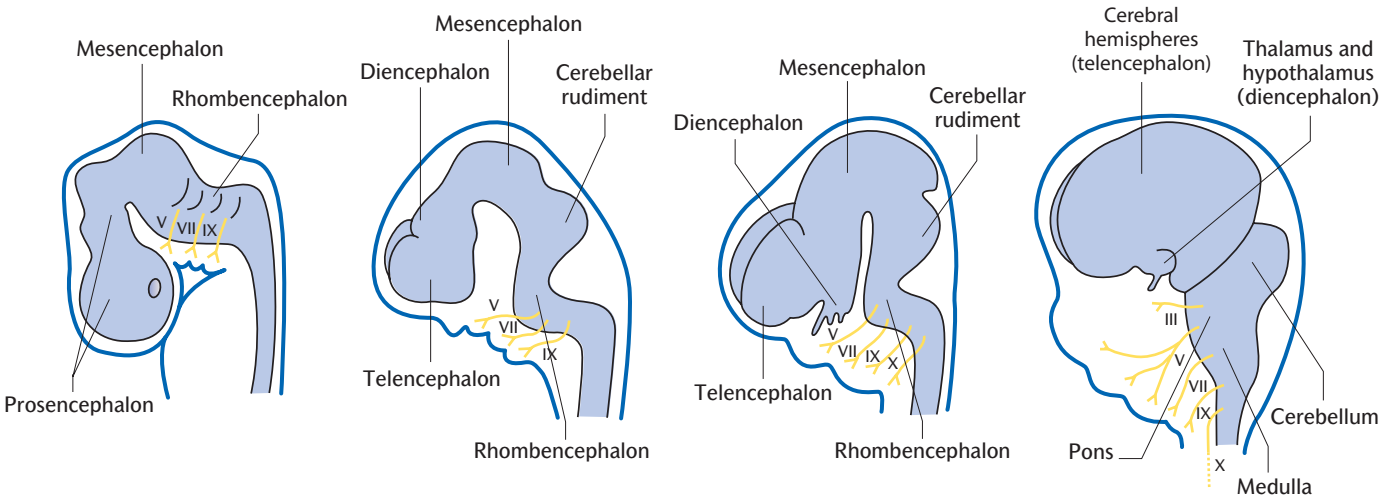


Fig. 19.5 The development of the brain.

advanced types of animals are folded into gyri separated by sulci; the cerebral surface is relatively smooth in primitive mammals. The gyral folds increase the surface area without increasing the overall volume.

The original cavity of the neural tube persists within the brain as a series of spaces filled with cerebrospinal fluid. As the cerebral hemispheres grow outwards, the cavity expands into them to form the **lateral ventricles**. Each of these connects with the cavity of the diencephalon, the **third ventricle**. The cavity of the midbrain is reduced to a narrow canal, the **cerebral aqueduct** running from the third ventricle to the **fourth ventricle** in the pons and medulla oblongata.

19.4 Neuronal connectivity

One of the most intriguing questions in developmental biology is how the billions of neurons that constitute the CNS make all the requisite connections with the correct structures. The same question can be asked of the connections of the central and peripheral processes of PNS neurons with the CNS and their target tissues, respectively; on the face of it, this looks a simpler problem to solve.

This is a huge topic and only a brief outline is given. As we have already seen, in the CNS, the type of neurons and their fate is determined by their developmental position in the superior to inferior and dorsal to ventral axes; different populations of neurons carry identification badges in the form of cell surface molecules. Essentially, the growing processes of developing neurons called **growth cones** carry receptors specific for short-range signalling molecules released by the tissues that they are advancing through. These signalling molecules attract or repel the growing neurons and, therefore, determine the direction in which growth cones advance as we have already seen for the growth of neurons through somites. Long-range guidance cues and chemical attractants released by target neurons or target tissues act in similar ways to ensure correct connections are established between neurons and their targets. Many short-range and long-range signalling molecules actively repel unwanted neurons so that only the correct neurons get through. The way in which neurons and targets are 'wired up' is quite well worked out for some systems but we only have a few tantalizing clues at present for others.

As neurons encounter their target and more processes join them, neuronal tracts are formed within the CNS. The growing neurons also promote the differentiation of glial cells that, in turn, release inhibitory factors that stop axons from the wrong sources joining tracts as they become established. This is an incredibly useful mechanism during development, but, as mentioned in Chapter 3, is unfortunately not turned off when development is complete. This explains why neurons in the CNS do not re-establish connections after trauma or disease as outlined in Box 19.1.

Box 19.1 Why do CNS neurons not regenerate after injury?

Glial inhibitory factors repel neuronal processes from the wrong source away from developing tracts and pathways. Their continued presence in the mature CNS essentially prevents regeneration of neurons and establishment of successful connections following CNS nerve injury from trauma or disease. Damaged neurons will form growth cones on the end of any severed processes which will endeavour to make connections. However, they are inhibited at more or less every turn so wander aimlessly about, trying to make progress; they usually form tangles of blind-ending neurons close to the site of the lesion called a **neuroma**.

One thing that is often overlooked during consideration of neuronal development is the very small distances that developing neuronal processes have to travel between their parent cell body and their intended target in the embryo compared with the distances that separate the two ends in the mature organism. Once connections have been made, often over quite short distances, neuronal processes can grow to accommodate the increasing distance between origin and target by adding material to their cell membranes, thus maintaining the link. This enables neurons to follow the migration and changes of position of their target tissues as they develop and grow and this is often marked by the course of nerves in the adult; the phrenic nerves supplying the diaphragm (Chapter 10) and the recurrent laryngeal nerves have already been cited as examples of these phenomena (Chapter 12).

Section 4
Head and neck

Section contents

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20 Introduction and surface anatomy

Chapter contents

20.1 Introduction

The head and neck contain the structures that are the most significant to the practice of dental surgery. These regions are not as easy to study from dissection as other areas because an 'onion skin' approach has to be adopted. Layers are dissected from the most superficial subcutaneous structures to the deepest internal structures, the brain, and spinal cord; structures that appear at one level may not show up again until the dissection has advanced to much deeper layers. It is important to have a general understanding of the structures forming the head and neck to build up a coherent picture of their relationship to each other.

20.1.2 An outline of the major structures

The skull is the structural basis of the head. The skull comprises the **cranium,** formed from 27 bones joined together by fibrous joints known as **sutures**, and the separate **mandible** that articulates with the cranium at the **temporomandibular joints (TMJ)**. The skull houses and protects the brain in the cranial cavity. It also protects other delicate structures vital for the reception of the special senses; the orbital cavities contain the eyes and dense bones in the cranial base house the internal ears. The entrance to the respiratory tract is the bony and cartilaginous nasal cavity; it can also be accessed together with the gastrointestinal tract through the oral cavity between the cranium and mandible.

The major skeletal component of the neck is the cervical part of the vertebral column formed by seven vertebrae. The lower five cervical vertebrae conform to the general pattern of vertebrae outlined in Section 10.1.1, but the upper two cervical vertebrae are specialized; the **atlas** articulates with the underside of the skull for nodding movements and the second vertebra, the **axis**, articulates with the atlas for shaking movements of the head. The **hyoid bone** in the upper anterior neck and the **laryngeal cartilages** below it form the laryngeal skeleton.

There are several important muscle groups in the head. The **muscles of facial expression** are small superficial muscles beneath the skin of the face; they alter facial expression in response to emotion, but also play a part in chewing, swallowing, and speech. The **muscles of mastication** are bulky powerful muscles that move the mandible relative to the upper jaw during mastication, swallowing, and speech. The **muscles of the tongue** alter the position and shape of the tongue during oral functions. The **extraocular muscles** within the orbit move the eyeballs and **intraocular muscles** within the eyeballs control eye functions such as focusing. Tiny muscles within the middle ear cavity reflexly adjust hearing to accommodate loud sounds and prevent damage to the inner ear.

The **pharyngeal constrictor** muscles form the walls of the pharynx and do what their name indicates; they constrict the pharynx during swallowing to propel food through it into the oesophagus. The pharynx and its constrictor muscles begin in the head, but pass down into the neck. The **laryngeal muscles** are small muscles attached to the laryngeal cartilages which they move to close and open the larynx during swallowing; they also control the length, tension, and thickness of the vocal folds for production of voice. Two groups of muscles lie superficially in the anterior neck; one group, the **suprahyoid muscles**, lies above the hyoid bone and the **infrahyoid muscles** are below it. They raise and lower the hyoid bone and larynx, respectively, during swallowing and also play a significant role in opening the mouth.

Figure 20.1 shows a cross section of the neck; examine it as you read the description below. Observe that the cervical vertebrae are centrally placed and form a considerable amount of the neck. Large bulky muscles posterior to the vertebrae extend the neck and head and smaller flexor muscles are immediately anterior to the bones. Lateral vertebral muscles run on each side from the cervical vertebrae to the first and second ribs. These groups are not important to the practice of dentistry and will not be considered in any further detail. There are, however, two postural muscles in the neck that provide useful landmarks that will be described in Section 20.3.2. In Figure 20.1 you can see the **oesophagus** anterior to the vertebrae and flexor muscles and the **larynx** and **trachea** most superficially. The **thyroid gland** wraps round the front and sides of the

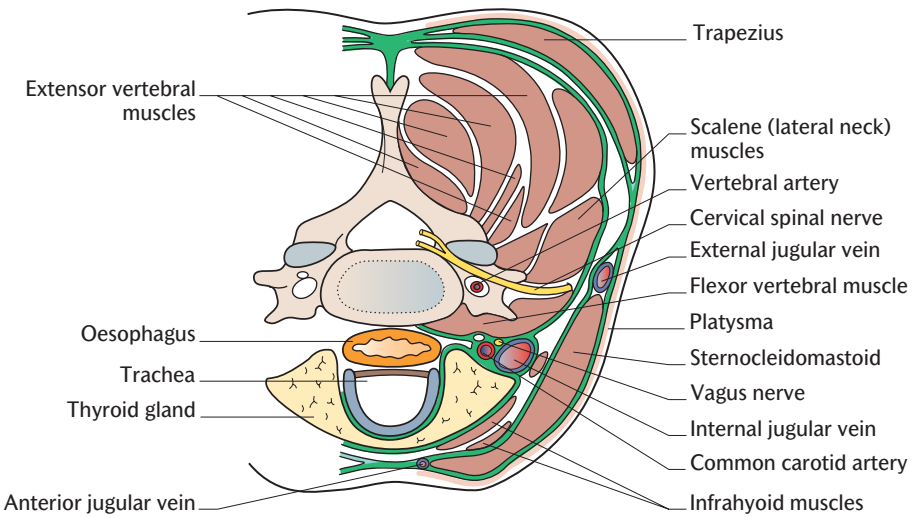


Fig. 20.1 A horizontal section of the neck at the level of the sixth cervical vertebra to show the arrangement of the structures within the neck.

upper part of the trachea. In Figure 20.1, you can also see the major blood vessels supplying the head lateral to the trachea and oesophagus.

As outlined in Chapter 12, the head and neck are supplied by two pairs of arteries, the **common carotid arteries** and the **vertebral arteries**. The left common carotid artery arises directly from the aortic arch but the right one is a branch from the brachiocephalic artery (see Figure 12.10). The common carotid arteries divide high in the neck into the external and internal carotid arteries. Each **internal carotid artery** passes into the cranial cavity where it branches to supply the brain as already described in Section 15.5.1. Each **external carotid artery** has several branches in the upper neck and head; they mainly supply structures in the head although two branches supply structures in the neck. The **vertebral arteries** are branches of the subclavian arteries and also supply the brain as described earlier in Section 15.5.1; they have important branches supplying structures in the neck.

Many of the veins draining the head and neck correspond to the arterial supply of the same area or structure. However, the larger veins do not correspond; in fact, there is only one major vein on each side, the **internal jugular vein**, which drains the brain and the head and neck. Now you have mastered anatomical terminology, you will be asking, 'If there is an *internal* jugular vein, shouldn't there be an *external* one too?' You are right; there is an external vein on each side, but this vessel is superficial and quite variable in size and there is, however, no corresponding artery.

You will realize from Chapter 18 that the structures in the head and neck will not function without the cranial nerves. A brief recap of that chapter will serve as a prelude to the more details of the anatomy of the cranial nerves of the head and neck and orientate you to the location and function of the cranial nerves. Recall the **olfactory** (I) supplies the olfactory mucosa in the nose, the **optic** (II) the retinas, and the **vestibulocochlear** (VIII) the vestibular apparatus and cochleae; these nerves serve the organs of special sense. The **oculomotor** (third), **trochlear** (fourth), and **abducens** (sixth) cranial nerves supply the extraocular muscles of the eye and are, therefore, restricted to the orbital cavities. The **hypoglossal** nerves (XII) are the motor supply to the tongue muscles and are mainly encountered within the mouth. The tongue is a very large organ extending across the floor of the mouth into the pharynx; sensation from its pharyngeal part is carried by the **glossopharyngeal** nerves (IX) as is sensation from the pharynx itself. The **vagus** nerves, as described several times already, have a major parasympathetic component to viscera in the thorax and abdomen. In the head and neck, the vagus nerves supply branches to the muscles of the soft palate, pharynx, and larynx, mainly distributed to the neck rather than the head. The two remaining cranial nerves play major roles in the innervation of the head. The **facial** (VII) nerves are the motor supply to the muscles of facial expression and parasympathetic secretomotor supply to several glands. The **trigeminal** nerves (V) are the major somatic sensory nerves of the head. They convey sensation from the facial skin, the eyeballs, and mucosal linings of the nasal cavities and oral cavity to the CNS. They are also the motor nerve supply to the muscles of mastication and other muscles. The upper **cervical spinal nerves** combine to form the **cervical plexus** which supplies the skin of the neck and infrahyoid muscles.

There are, of course, other structures that make up the head and neck such as salivary, thyroid, and lacrimal glands, not to mention teeth and their supporting structures. These will be met in the appropriate context in subsequent chapters.

There are two excellent ways for you to reinforce some of the concepts introduced above and to familiarize yourself with the overall structure of the head and neck. The first exercise is to gain a general idea of the skull and how it underpins the anatomy of the head using the diagrams provided in this book together with a dried human skull or a plastic model skull. The second exercise is to study the surface anatomy of the head and neck. You can sit in front of a mirror and use yourself as the subject or you can find a partner willing to be the examination subject; some examinations of surface anatomy are much easier to perform on a 'patient' than yourself.

20.2 Introduction to the skull

Look at Figure 20.2 and a skull if you have access to get a general impression of its structure. It does not require any detailed anatomical knowledge to distinguish the smooth curved bones forming the **braincase** from the more irregular bones forming the **facial skeleton**. Figure 20.2 is a view of the skull from the front. Orientate your skull the same way. Below the smooth forehead formed by the **frontal bone**, you should be able to distinguish the two round **orbital cavities** and the triangular **nasal cavity**. Observe the nasal cavity extending up between the orbits. You can also distinguish the upper (maxillary) teeth with their roots embedded in the **maxillary bones** that form the bulk of the facial skeleton between the orbits and upper teeth. The **mandible** forms the lower jaw and houses the lower teeth. The mandible is usually attached to the braincase by springs on dried or model skulls so that the mouth can be opened; these movements occur at the two **TMJs**. Notice the proportions of the adult skull; the orbits are positioned about a third of the way down from the crown and the mandible occupies the lower third of the height of the skull. The maxillae and associated bones occupying the intervening area and are referred to clinically as the **middle third of the face**.

Figure 20.3 shows the skull from above. Observe the junctions between the bones forming the roof of the braincase are formed by wavy lines called **sutures**; look closely to see how the bones on each side interlock with each other through small finger-like processes. In life, the sutures are filled with a small amount of fibrous tissue. In Figure 20.3, the suture running from left to right across the crown of the skull is the **coronal suture** and joins the frontal bones to the two **parietal bones** that form most of the cranial vault. The **sagittal suture** joins the two parietal bones. The back of the braincase is formed by another curved smooth bone, the **squamous part** of the **occipital bone**. (You will encounter the term 'squamous' several times in the context of the skull, but also as a descriptive term for epithelial tissue; 'squamous' is derived from a Latin word, meaning scale or roof tile and is used to indicate flat smooth structures.) The **lambdoid suture** links the occipital and parietal bones. Sutures are relatively easy to distinguish between

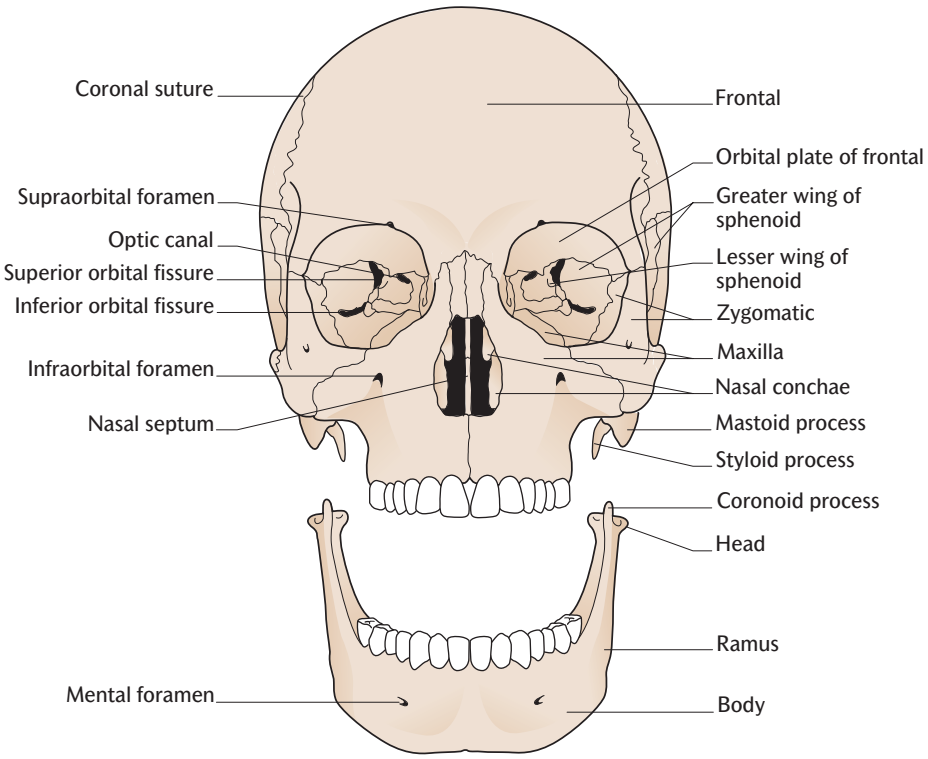


Fig. 20.2 An anterior view of the skull.

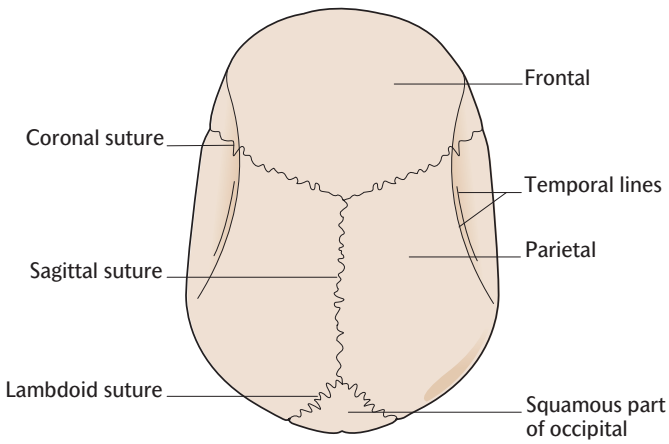


Fig. 20.3 A superior view of the skull.

the smooth bones of the braincase, but you may have to look a little more closely to see them joining the bones of the facial skeleton and other areas of the skull .

Figure 20.4 is a view of the skull from the side. Observe that smooth curved bones form the roof, back, and sides of the cranial vault. Look particularly at the side walls; the suture lines indicate that the parietal bones form the upper part whereas several bones contribute to the lower part of the side wall; these are the **greater wing of the sphenoid** and the **squamous part of the temporal bone.** The temporal and sphenoid bones have a complex shape and have several components, some of which we will meet very shortly. Observe the bar of bone that starts behind the lateral margin of each orbit and extends backwards to the temporal bone on the side of the braincase; this is the **zygomatic arch**. You can see a large hole just behind the posterior root of the arch; this is the **external auditory meatus** and is the bony tube conducting sound from the external ear into the temporal bone that houses the middle and inner ear cavities. The side view of the skull shows that the facial skeleton is attached below the anterior part of the braincase. The cervical vertebrae are attached beneath its posterior part.

Now view the skull from its underside as shown in Figure 20.5. The first thing that should strike you is that the underside looks very irregular compared with the other views of the skull. The second thing that should be obvious is that the underside of the braincase is peppered with lots of foramina that transmit blood vessels and nerves. Locate the relatively smooth squamous occipital bone forming the posterior part of the underside of the cranium. Follow it forward and you will see a very large midline foramen, the **foramen magnum**, that transmits the spinal cord and associated structures. Note the two smooth hemispherical **occipital condyles** either side of the foramen magnum. These are the articular surfaces of the atlanto-occipital joints between the cranium and the atlas, the first cervical vertebra, where nodding movements of the head takes place. The **basal part of the occipital bone** (or basiocciput) is the bone anterior to the foramen magnum and is one of the bones that form the **cranial base.** The thick central bar of bone continues forward as the **body of the sphenoid**. The sphenoid is the *pivotal bone* of the whole skull to which the other bones are attached either directly or indirectly. It has a complex shape

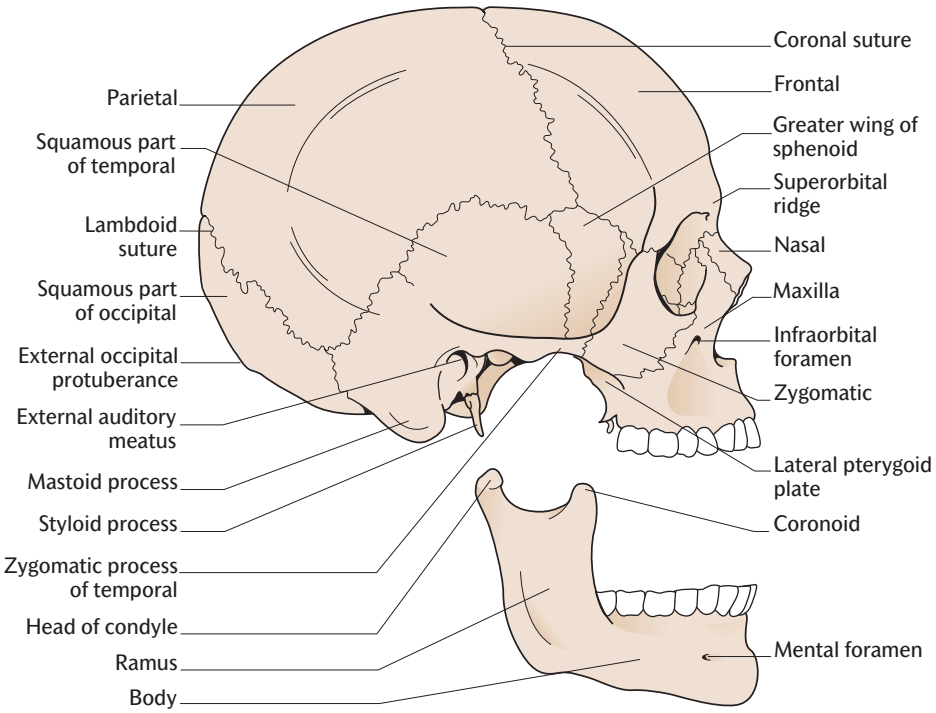


Fig. 20.4 A lateral view of the skull.

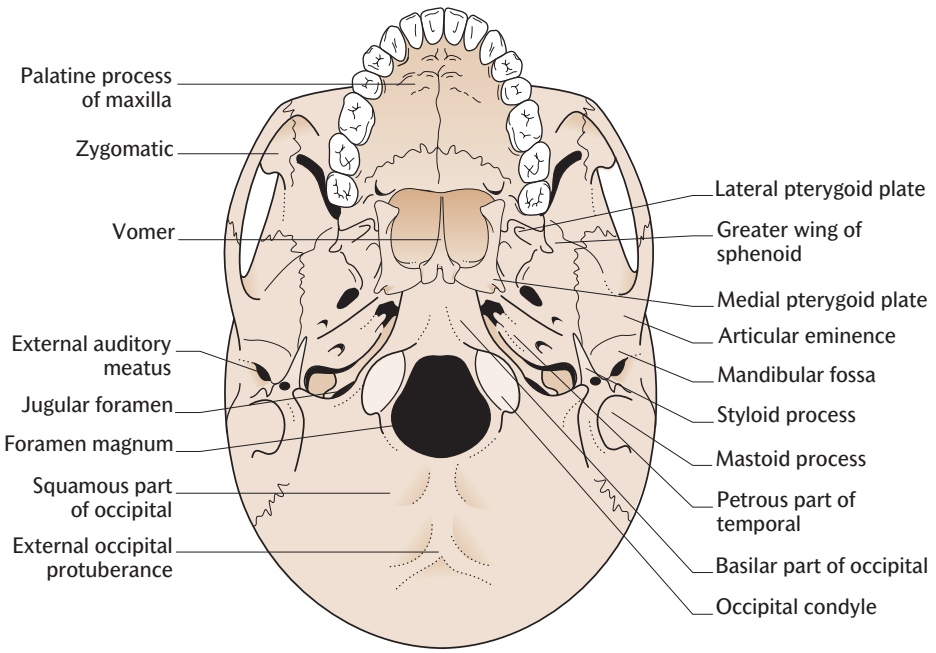


Fig. 20.5 The cranial base viewed from below.

with two wings projecting on either side of the body and processes hanging beneath it.

In Figure 20.5, use the external auditory meatus as a landmark to locate the **styloid process** of the temporal bone, a prominent spike of bone medial to the meatus on the underside of the cranium. The styloid process is often broken off dried skulls but its stump should still be visible. Locate on Figure 20.5 and the skull a thick wedge-shaped bone running medially and anteriorly from the styloid process towards the body of the sphenoid. This is the very robust **petrous temporal bone** that houses the delicate working parts of the middle and inner ear and is also a component of the cranial base. We cannot follow the cranial base any further by examining the underside of the skull because it is masked by the facial skeleton.

The mandible has been detached from the skull in Figure 20.5. This enables you to see the most obvious feature of the underside of the facial skeleton, the U-shaped arch formed by the upper teeth with the

bony **palate** in between. The posterior opening of the nasal cavity is above the posterior free edge of the bony palate. Notice that the lateral walls of the posterior nasal entrance are formed by two vertical plates of bone on each side, the **pterygoid plates of the sphenoid**. Look inside the posterior nasal entrance in Figure 20.5 or a skull and you will see that it is divided into two by a midline nasal septum of thin bone. You can also see the septum if you look inside the nasal cavity from anteriorly, the aspect shown in Figure 20.2. The most anterior part of the septum is formed from cartilage which is lost when dried skulls are prepared; the bony septum starts a little way into the nasal cavity. Another thing to note about the nasal cavity in Figure 20.2 is that the lateral walls comprise curls of thin bone known as the **conchae**; they increase the surface area of the nasal cavity to improve the efficiency of warming, cleansing, and humidifying inspired air.

Dried and plastic skulls usually have a detachable skull cap. When this is removed, the interior of the cranial cavity can be observed and will resemble Figure 20.6. The floor of the cranial cavity is arranged in three steps as illustrated in Figure 15.1; the shallowest anterior step is the **anterior cranial fossa**, followed by the deeper **middle cranial fossa**, and the deepest **posterior cranial fossa** punctured by the foramen magnum. The anterior cranial fossa is mainly formed by the orbital plates of the **frontal bone.** Notice the midline area between the orbital plates contains lots of small foramina; this is the **cribriform plate** of the ethmoid bone and is where olfactory nerves pass through to olfactory mucosa in the roof of the nose. The **ethmoid bone** is the most anterior of the bones of the cranial base and has a quite complex shape with components also contributing to the skeleton of the nasal and orbital cavities. The posterior edge of the anterior cranial fossa is formed by the lesser wings of the sphenoid laterally and the body of the sphenoid medially. The suture lines between the lesser wings and frontal bones are usually obvious as shown in Figure 20.6, but it is extremely difficult to see the join between the ethmoid and sphenoid body in the midline. The petrous temporal bone, greater wing, and body of the sphenoid and basiocciput visible on the underside of the skull described above can be located in the middle and posterior cranial fossae in Figure 20.6.

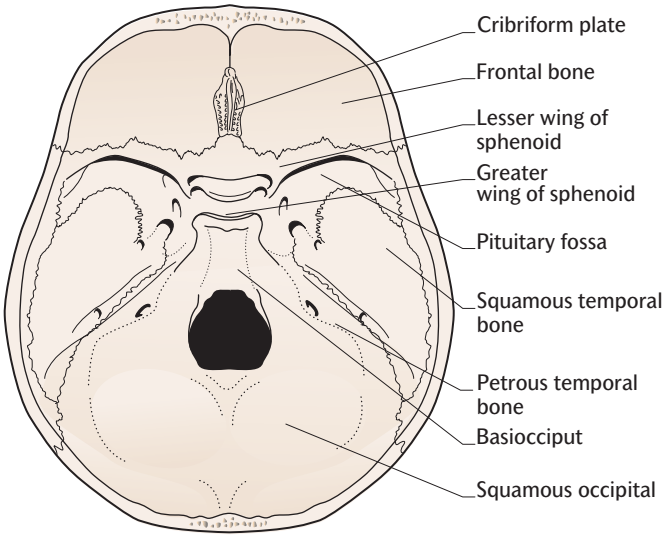


Fig. 20.6 The cranial base viewed from the interior of the cranial cavity.

As different regions of the head and neck are studied in the following chapters, we will revisit the skull to refine our knowledge and add more detail that aids to understand the anatomy of the associate soft tissues and its relevance to clinical dental practice.

20.3 Surface anatomy

20.3.1 The head

The braincase and facial skeleton are the skeletal components of the head. Posteriorly, the neck extends up to the floor of the braincase in the occipital region of the skull. Anteriorly, the neck ends at the inferior border of the mandible. The contours of the skull are the major determinants of facial and head profile because the superficial structures of the head are relatively thin and there is little subcutaneous fat compared with the rest of the body. The thickness of the subcutaneous tissues is relatively constant, although there are some variations with age. It is this constancy that enables medical artists to reconstruct facial appearance from skull profiles for forensic investigation and victim identification. As we have already seen, the contours of the neck are largely determined by muscles surrounding the cervical vertebrae and the laryngeal cartilages.

The skull is palpable through the skin over most of the head. The left side of Figure 20.7 shows the major features of the skull and on the right side, the overlying tissues have been added. Notice that there is not a lot of difference between the two sides besides the addition of some soft tissue structures to the right. Begin your examination of the head by feeling your forehead formed by the **frontal bone**. Pass your hand down towards your orbits and note the **supraorbital ridges** that lie

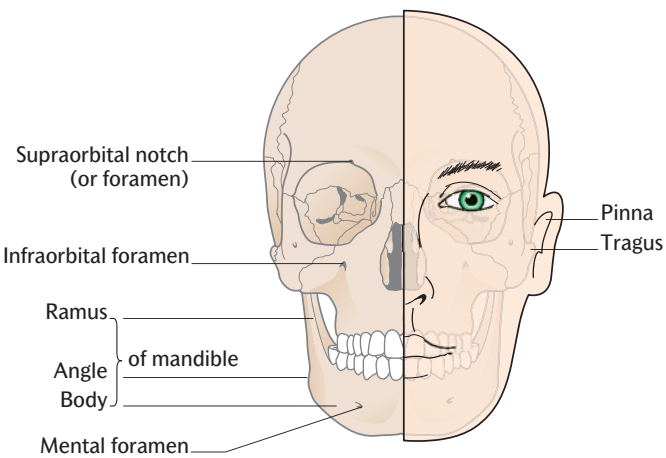


Fig. 20.7 The relationship between the skull (left) and the surface features of the face (right).

under your eyebrows; these ridges are more prominent in post-pubertal males than females. You can feel the whole extent of the orbital margins if you run your finger around the orbit. You should be able to feel an indentation about a finger's breadth from the junction of medial and superior borders of the orbit. This is the **supraorbital notch** which transmits correspondingly named nerve branches of the trigeminal nerve and vessels (see Figure 20.2). In many cases, the notch is replaced by a foramen which is less easy to palpate. If you are having difficulty locating the notch, look straight ahead and the notch should be in line with your pupil.

You can feel the **nasal bones** forming the bridge of the nose between the orbits and the **nasal cartilages** extending forward from the nasal bones to form the external nose. The nasal cartilages are made of elastic cartilage, an excellent adaption should you literally fall flat on your face; your nose will be squashed then will spring back into shape when you pick yourself up. Note on the left side of Figure 20.7 that there is a very large nasal aperture on the skull because the cartilages are lost when dried skulls are prepared. In life, the anterior nasal apertures, the nostrils, are quite small and point downwards. Observe in Figure 20.7 how far the nasal cavities extend vertically; they reach from just above the upper lip as far as the superior margin of the orbits.

The middle third of the face between the orbits and oral cavity is formed by the paired maxillae and zygomatic bones. Each **maxilla** forms the major part of the middle third of the face between the lower orbital margin and upper teeth. The **infraorbital foramen** lays a finger's breadth below the inferior orbital border in line with the supraorbital notch as shown in Figure 20.7. The infraorbital nerve, a branch of the maxillary division of the trigeminal nerve, emerges here to innervate the skin of the lower eyelid, cheek and upper lip. Each **zygomatic bone** forms the point of the cheek and the lateral orbital margin. Several **muscles of facial expression** lie between the facial bones and skin; these muscles are too small and flimsy to be palpated.

The zygomatic bone extends backwards to meet a bar of bone running forward from the temporal bone; these two processes form the **zygomatic arch** that stands off from the skull as shown very clearly in Figure 20.5. The zygomatic arch can be palpated throughout its length from the lateral wall of the orbit to the anterior border of the external ear. The side of the skull above the zygomatic arch is covered by the **temporalis muscle** covered by the temporalis fascia attached to the upper border of the arch. The fascia extends upwards and posteriorly to the **superior temporal line** indicated in Figure 20.8; this may be palpable as a faint ridge arching round on the side of the braincase. When the jaws are clenched, the muscle can be felt contracting beneath the fascia.

The maxillary teeth housed in the maxillae can be felt through the upper lip. Note the 'cupid's bow' outline of the upper lip with the **philtrum**, a broad groove running down the midline. The deep pink area of the lips is the **vermilion border**. It is a transitional zone between hairy skin on the outside and oral mucosa on the inside. The epithelium is relatively thin, allowing the colour of underlying blood vessels to show through.

The **mandible** and the lower dentition can be easily palpated for most of the extent of the mandible. As seen in Figure 20.4, the mandible comprises a horizontal **body** and a vertical **ascending ramus** on each side; the **angle of the mandible** is where the two meet. The inferior border of the mandible is palpable right to the angle and the posterior border of the ramus can also be felt very easily. The **mental foramen** is about a finger's breadth above the inferior border of the mandible between the roots of the first and second premolar teeth. It is usually palpable and is in line with the supraorbital and infraorbital foramina. The mental nerves emerging from this foramen are branches of the mandibular trigeminal nerve that innervate the skin of the lower lip and chin.

The anterior border of the ramus just behind the last mandibular tooth is masked by the bulk of the **masseter** muscle. This muscle is one of the muscles of mastication and attaches between the zygomatic arch and the superficial aspect of the angle of the mandible, thus covering the upper part of the ramus. You can feel this muscle very easily if you bring your teeth together (occlusion); alternatively, clench and relax your jaw; you can feel masseter contracting and relaxing. Feel the whole extent of the muscle to verify its attachments. You may feel a hollow anterior to the anterior border of masseter, but the hollow may be partially obscured by a variable amount of fat. This is the **buccal fat pad** and is especially well developed in infants where it is called the **suckling pad**.

Follow up the posterior border of the ramus of the mandible with your finger and note that it ends in a rounded prominence shown in Figure 20.8, the **condyle** (condylar head) of the mandible. This process articulates with the underside of the temporal bone to form the **TMJ** beneath the posterior part of the zygomatic arch just in front of the ear. Palpate the condyle and open your mouth; the condyle slides forwards and downwards as the mandible is depressed. If you open your mouth wide, you should be able to feel a hollow behind the head where the condyle fits into at rest; this is the **mandibular fossa**. The condyle contacts the articular eminence as it slides forward.

The ear is an obvious feature on the side of your head. The **pinna** or **auricle** is the visible part surrounding the **external auditory meatus**. The **tragus** is a small flap of skin and cartilage that partially covers the opening of the meatus. The external auditory meatus is 2–3 cm long and terminates medially at the tympanic membrane. The pinna and external auditory meatus together constitute the **outer ear**. The **mastoid process** is a prominent lump of bone which can be palpated just behind the ear; this is visible in Figures 20.7 and 20.8 and is part of the temporal bone. It is one of the upper attachments of the sternocleidomastoid muscle of the neck (see Section 20.3.2). The **superior nuchal line** seen in Figure 20.8 runs backwards on each side and marks the junction of the head and neck posteriorly. This line marks the outer attachment of the neck extensor muscles to the underside of the skull.

Each **parotid gland,** the largest of the major salivary glands, occupies the wedge-shaped space between the ramus of the mandible in front and the mastoid process and the attached sternocleidomastoid muscle behind. Each gland extends on the face; its extent is quite variable, but its approximate position is outlined in Figure 20.8. The **parotid duct** is also indicated in the figure**;** it runs forwards from the anterior aspect of the gland across the masseter, then turns inwards to pierce the buccinators muscle forming the cheek to open into the mouth opposite the crown of the second upper molar tooth. If the masseter is tensed, the duct can be palpated as a hard cord about a finger's breadth below the inferior border of the zygomatic arch.

A number of arteries can be seen or felt in the head and neck and the course of a number of others can be represented by lines drawn with reference to surface landmarks. The **facial artery** is a branch of the external carotid artery arising in the upper neck. Figure 20.8 shows its course on the face. Notice that it only becomes superficial as it crosses the inferior border of the mandible to enter the face. With care, its pulsations may be felt where it crosses the bone at the anterior border of the masseter muscle. As Figure 20.8 shows, the artery travels diagonally across the face towards the medial canthus of the eye, passing about 1 cm behind the angle of the mouth where the upper and lower lips meet. There is an accompanying facial vein. The **superficial temporal artery** is one of the terminal branches of the external carotid artery on each side. It emerges from the cover of the parotid gland and branches across the temporal region. As indicated on Figure 20.8, its branches follow tortuous courses within the subcutaneous tissue of the temple; these may be visible, especially in bald men. Small groups of lymph nodes are found at several sites within the head (see Section 23.2.8). These may be palpated when enlarged by disease processes, especially where the nodes overlie bone. The **mastoid nodes** lying superficial to the mastoid processes and the **occipital nodes** on the superior nuchal line in the occipital region of the skull can be readily palpated when enlarged.

20.3.2 The neck

Figure 20.8 illustrates the bulky strap-like **sternocleidomastoid muscle**, running obliquely downwards across each side of the neck from the mastoid process to the sternum and clavicle. The **sternocleidomastoid muscle** can be made to stand out by turning the head towards the opposite side against resistance (push against the direction you are trying to turn to with your hand). It attaches to the sternum through a fibrous tendon and to the medial third of the clavicle by a fleshy attachment. These attachments stand out when the muscle is contracted against resistance. Each **trapezius muscle** is a sheet of muscle superficial to the extensor muscles on the back of the neck. Each muscle runs obliquely upwards from the junction of the middle and lateral thirds of the clavicle and scapula to attach to the skull at the **superior nuchal line** as shown in Figure 20.8. The lateral margin of each trapezius muscle can be seen or palpated if the shoulders are raised against downwards resistance; superiorly, the muscle thins considerably and its edge are less easily identifiable. Each trapezius muscle also extends downwards from the scapula to insert into the lower thoracic vertebrae. The combined triangular outlines of each muscle describes a trapezoid outline, hence the names of these muscles.

For descriptive purposes, the neck is divided by the sternocleidomastoid muscles into areas known as the **triangles of the neck**. The posterior and anterior triangles are marked in Figure 20.8. The posterior border of the sternocleidomastoid muscle and the lateral edge of trapezius demarcate the **posterior triangle**, with its apex just behind the mastoid process and its base formed by the medial one-third of the clavicle. The **anterior triangle** of the neck on each side is the triangular area enclosed by the inferior border of the mandible above, the medial border of the sternocleidomastoid muscle posteriorly, and the midline of the neck anteriorly. The structures in the anterior triangle are important to the study and practice of dental surgery whereas the posterior triangle and its contents are not.

Begin your examination of the anterior triangle by placing your finger on your chin in the midline and tilting your head back. We will trace the structures encountered as you run your finger backwards from the mandible, keeping to the midline down to the suprasternal notch. Follow Figure 20.8 as you do so to discover what structures you are feeling. The floor of the mouth within the mandible is formed by some of the **suprahyoid muscles** and, therefore, feels soft. At the junction of the head and neck, you will feel a prominent transverse bar of bone, the **hyoid bone**. The hyoid bone is unusual because it does not articulate with any

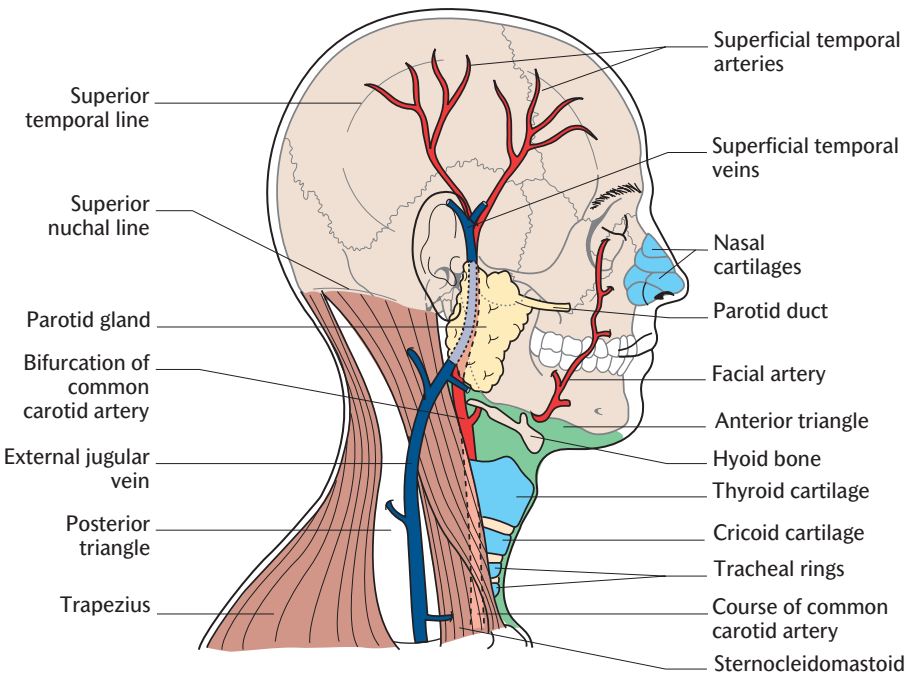


Fig. 20.8 The position and surface markings of some structures of the head and neck. The anterior triangle is shaded green.

other skeletal structures. Instead, it is attached by muscles and ligaments to the mandible and base of skull above and to the laryngeal cartilages, sternum, and scapula below. If you run your finger laterally along each side of the hyoid bone, you will feel that it is a U-shaped bone almost like a small version of the mandible. The backward extensions of the U are the **greater cornua;** Figure 20.8 illustrates that they extend posteriorly almost to the anterior border of the sternocleidomastoid muscle.

Continue tracing down the midline. As Figure 20.8 illustrates, below the hyoid bone, you will encounter the **thyrohyoid ligament**, a band of soft tissue, before you meet the **laryngeal prominence** of the **thyroid cartilage**. The prominence is much more conspicuous in adult men than in women and children, hence its more familiar name of 'Adam's apple'. One of the secondary sexual characteristics acquired at puberty is the enlargement of the thyroid cartilage in males; this enlargement also lengthens the vocal folds which accounts for the voice 'breaking' in pubescent males. Feel the thyroid cartilage and verify that it is formed from two flat laminae meeting in the midline. Run your finger down the anterior border of the thyroid cartilage from the laryngeal prominence and you will come to its lower border. There is a slight depression immediately below, denoting the position of the **cricothyroid membrane** before the arch of the **cricoid cartilage** is felt. Now continue to run your finger down the anterior midline of the neck; as you do so, you will feel the cartilage rings in the wall of the trachea until you reach the **suprasternal notch** in the upper border of the **sternum**. You may be able to feel the **isthmus** of the **thyroid gland** crossing the front of the trachea about a finger's breadth below the arch of the cricoid at the level of the second to fourth tracheal rings. The thyroid gland has a main lobe on each side joined by the isthmus. The lobes lie deep to the **infrahyoid muscles** which are thin, strap-like muscles extending from the hyoid bone to the sternum and overlying the laryngeal cartilages. When the neck is extended, the muscles are displaced laterally, but they are too thin to prevent you feeling the underlying cartilages as described above. The thyroid gland is variable in size and position and the lobes are usually not palpable. The gland may become greatly enlarged in pathological states, a condition known as **goitre**, in which case the gland is usually visible and clearly palpable.

The surface anatomy of the larynx and trachea and their relationship to the thyroid gland is important if it becomes necessary to create an emergency opening into the lower airway, a procedure known as a **cricothyroid stab** or a tracheotomy. The cricothyroid membrane is *the* crucial landmark when performing this procedure (see Box 28.9).

There are usually two prominent skin creases running transversely across the neck. These are situated at the level of the upper and lower borders of the thyroid cartilage.

The **platysma** is a broad sheet of muscle in the subcutaneous tissues over the lower part of the face, side of the neck, and upper part of the thorax. It can be made to stand out by forcefully drawing down the lower lip and angle of the mouth; its anterior portion will be seen as a ridge under the skin below the mandible. The muscle varies in its degree of development and may be absent. Developmentally, it is part of the muscles of facial expression, but plays a comparatively minor role in this function.

One blood vessel is usually prominent in the neck under certain conditions and the positions of other important vessels are clinically important. The course of the **external jugular vein** is indicated in Figure 20.8. It is very variable in size and may be seen as a narrow elevation running from behind the angle of the mandible across the sternocleidomastoid muscle to the clavicle lateral to the lower muscular attachment. The vein is usually much more prominent when intrathoracic pressure is raised when, e.g. singing sustained notes. Raised intrathoracic pressure impedes venous return and, therefore, superficial veins become engorged as the blood is unable to flow through the vessel (see Box 20.1). The anterior jugular veins are also very variable in size and are not always present. They run subcutaneously close to the anterior midline of the neck and can also become engorged when intrathoracic pressure is raised.

Box 20.1 The external jugular vein as a clinical sign

Venous return can be impeded due to various cardiac diseases causing heart congestion. If the lower portion of a patient's external jugular vein is engorged when they are upright, this suggests a potential heart condition. If the vein becomes engorged along its visible length when they lie back, this is indicative of such a problem. The vein is acting as a biological manometer of intrathoracic pressure.

The **common carotid artery** is deep to the sternocleidomastoid muscle for most of its course in the neck. It follows a line drawn from the sternoclavicular joint to a point 1 cm below the greater cornu of the hyoid bone; here, it bifurcates into the **internal carotid** and **external carotid arteries**. Trace that line in Figure 20.8 and you will see that the common carotid artery is covered by sternocleidomastoid for most of its course until it emerges from under cover of the muscle just above and behind the superior border of the thyroid cartilage. The **internal jugular vein** runs alongside, but superficial to, the common carotid artery. The common carotid pulse is the easiest pulse to find in an emergency when assessment of cardiac function and blood circulation is required because you do not have to be too precise in locating it. The pulse may be felt as the artery emerges from the cover of the muscle.

Note that the skin is drawn quite tightly across the lower border of the mandible anteriorly, but the profile is somewhat smoother and gentler posteriorly near the angle. This is because the submandibular salivary gland lies just below the mandible at this point and bulks out the underlying tissue. Generally, the gland cannot be palpated very easily unless it is enlarged due to pathological changes.

The **submandibular lymph nodes** lie over the submandibular glands about 1 cm anterior to the angle of the mandible. The **submental nodes** are another group of superficial lymph nodes lying on the inferior border of the mandible below the position of the mental foramen. These nodes in common with other lymph nodes may be enlarged in infections of their areas of drainage which, for these two groups, include most of the oral structures (see Section 23.2.8). They then become palpable against the bone of the mandible and may be noticeable as you adjust a patient's head position. A few **superficial cervical nodes** are arranged along the external jugular vein.

Superficial lymph nodes in the head and neck all ultimately drain into the **deep cervical lymph nodes** that form a chain alongside the

internal jugular vein. The nodes are covered by the sternocleidomastoid muscle and the majority, therefore, are not normally palpable, even if enlarged. The most superior and inferior nodes are palpable in front of and behind sternocleidomastoid when they become infected or infiltrated by metastases from malignant tumours.

Two rounded ridges can be seen passing up the back of the neck either side of the midline. These are produced by the some of the extensor vertebral muscles. The spinous processes of the cervical vertebrae lie in the groove between these muscle ridges. The spinous processes of the seventh cervical and first thoracic vertebrae are easily palpated in the lower part of this groove, but the spinous processes of the upper cervical vertebrae can only be felt by deep pressure. The **external occipital protuberance** is a prominent bony lump on the occipital bone at the top of the groove and is easily palpable.

21 Embryology of the head and neck

Chapter contents

21.1 Introduction

Embryology and development have been covered after the main anatomical descriptions in the previous sections, but it is going to precede them in this section. The reason for this departure is that the embryonic development of the head and neck explains much of the mature anatomy which can seem illogical without its developmental history. The development of the head, face, and neck is an area of embryology where significant strides in our understanding have been made in the last few years.

The development of the head is intimately related to the development of the brain outlined in Chapter 19 and its effects on shaping the head will be described in Chapters 32 and 33. The major thrust of this chapter is the description of the formation of structures called the **pharyngeal** (or branchial) **arches** and the fate of the tissues that contribute to them. All four embryonic germ layers contribute to the pharyngeal arches and their derivatives, hence to further development of the head and neck.

Figure 21.1 is a cross section through the neck region of a 3-week old embryo after neurulation and folding described in Chapter 8. It shows the structures and tissues that contribute to the formation of the head and neck:

- The **neural tube** situated posteriorly and the ectomesenchymal **neural crest** cells that arise as the tube closes;
- The **paraxial mesoderm** anterolateral to the neural tube;
- The endodermal **foregut** tube anteriorly;
- The investing layer of **ectoderm**.

The development of all these tissues is intimately interrelated.

21.2 Evolutionary history of the pharyngeal arches

The pharyngeal arches are very ancient structures in the evolutionary history of vertebrates. The arches and their individual components have undergone many modifications during their long history.

In ancestral aquatic vertebrates, as in modern fishes, water was drawn in through the mouth and expelled through a series of gill slits (or branchiae, hence the term 'branchial arch') in the sides of the pharynx. Oxygen was extracted as the water was passed over a gill apparatus supported by a branchial arch skeleton moved by branchial muscles controlled by branchial nerves. Although ventilation and respiration is now a function of the lungs in land vertebrates, the pharyngeal arches persist during vertebrate development. The gill slits are represented by an external **pharyngeal cleft** and an **internal pharyngeal pouch** between each pharyngeal arch sealed by the **closing membrane;** the membrane does not rupture to form actual slits in animals that do not possess gills. As you will see, the derivatives of the arches, clefts, and pouches become incorporated into other structures of the head and neck and have been modified to serve other purposes.

21.3 Formation of the pharyngeal pouches

The key to the formation of the pharyngeal arches is the differentiation of the endoderm to form the **pharyngeal pouches** on the inner aspect of the foregut. If this does not occur, the pharyngeal arches do not form and their derivatives are, therefore, absent. Essentially, specific regions of endoderm differentiate to form pharyngeal pouches, thus defining the anterior and posterior limits of each pharyngeal

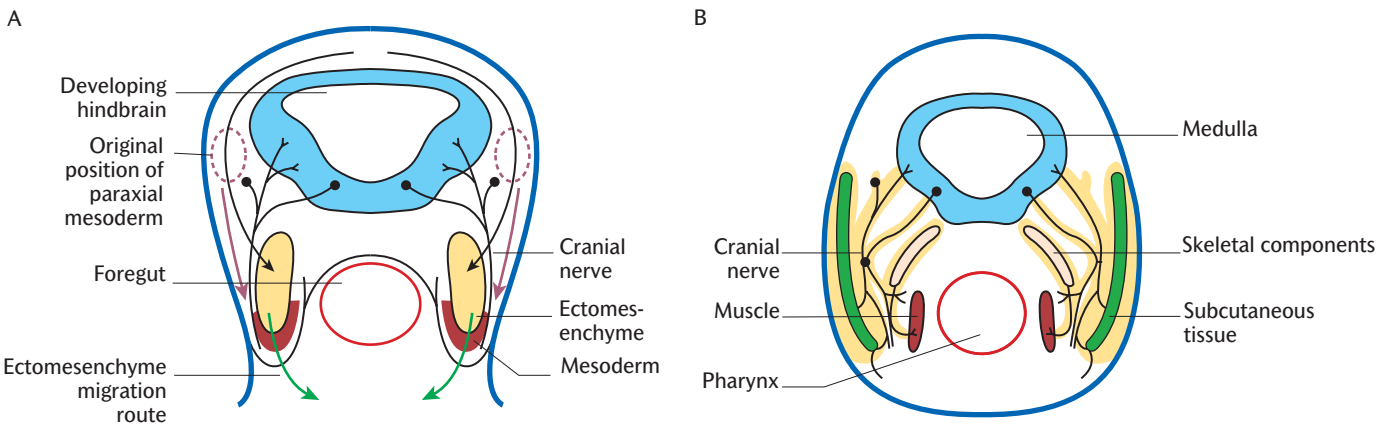


Fig. 21.1 A cross section through the head of a 3-week embryo. A) Arrows indicate the early migration routes of ectomesenchyme (black and green arrows) and paraxial mesoderm (red arrows); B) Later differentiation of tissues from ectomesenchyme (outlined in yellow) and mesoderm.

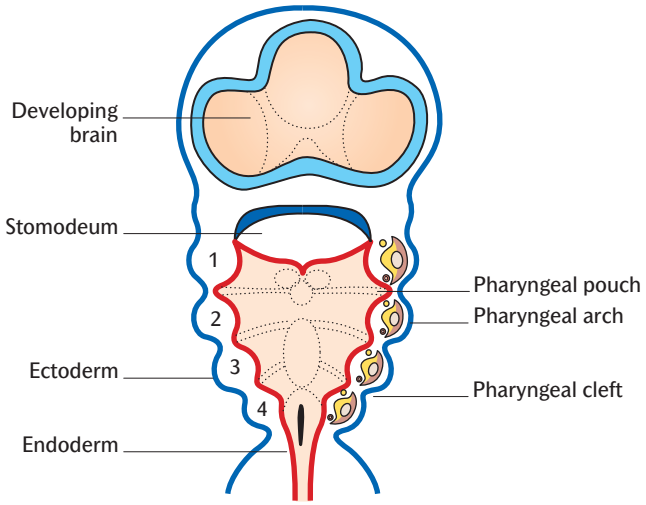


Fig. 21.2 The pharyngeal arches in the floor of the pharynx after removal of the posterior part of the embryo.

arch. There were six pairs of arches in early vertebrates; in modern ones, the fifth is a transitory structure at most if it exists at all. The fourth and sixth arches coalesce to form a single arch as the fifth arch regresses; but there is however a fourth pouch, indicating the original division between the last two extant arches. The pouches separating the arches appear in a superior to inferior sequence. As shown in Figure 21.2, the first pouch is between the first and second arches, the second between the second and third, the third between the third and fourth arch, and the fourth pouch below the fourth arch. During neural tube closure, ectomesenchymal cells migrate from the neural crest and invade the arches; the arches bulk out, deepening the intervening pouches and clefts considerably. The pouches are also shown in Figure 21.3A.

The differentiation of pharyngeal endoderm into pharyngeal pouches at specific locations is dependent on signalling by retinoic acid. If retinoic acid is absent, the first pouch and first arch will still form, but the second arch is severely reduced and the lower arches are absent.

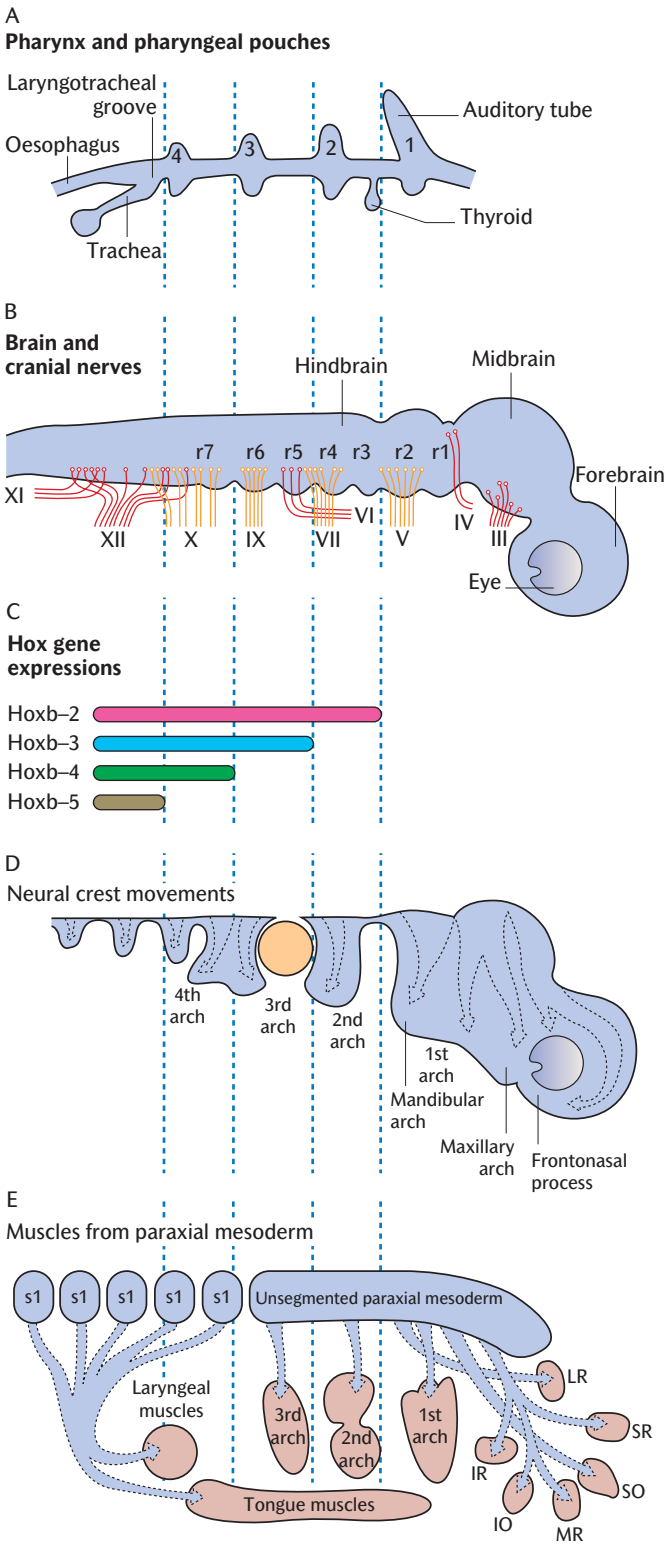


Fig. 21.3 The rhombomeres and internal tissues of the pharyngeal arches in an exploded view. The vertical dotted lines indicate how the axial levels of each frame correspond. A) The pharyngeal pouches. B) The rhombomeres and cranial nerves. C) The homeobox code. D) The migration of ectomesenchymal tissue into the pharyngeal arches. E) The paraxial mesoderm and its muscle derivatives. Redrawn after D.M. Noden and P.A. Trainor, Journal of Anatomy 207, 588 (2005).

21.4 Formation of the pharyngeal arches

As mentioned above and in Sections 8.3.4 and 19.3, ectomesenchymal cells migrate from the **neural crest**, the leading edges of the closing neural tube, into underlying tissues during neural tube closure. In the rhombencephalon, this process is somewhat more elaborate. The rhombencephalon is the only part of the developing CNS that shows true segmentation. Figure 21.3B indicates seven **rhombomeres** in the future hindbrain; these can be observed as transverse ridges across the rhombencephalon. Figure 21.3C shows that the boundaries between successive pairs of rhombomeres are demarcated biochemically by the superior expression limits of different **homeobox (Hox) genes**. The expression of these genes at succeeding superior levels produces specific marker molecules within different ectomesenchymal cell populations that give the cells within each rhombomere a unique identity. The ectomesenchymal tissue within each rhombomere proliferates, but is restricted to specific migratory pathways because cells derived from adjacent rhombomeres that carry a different Hox coding will not mix with each other. Follow the arrows in Figure 21.3D to trace which rhombomeres produce neural crest cells to populate each pharyngeal arch. Rhombomeres 1 and 2 do not carry a Hox code and neural crest cells in rhombomeres 3 and 5 do not proliferate, but undergo apoptosis *in situ*. As indicated by arrows in Figure 21.1A, the cells initially stream out laterally before turning anteriorly under the foregut tube to meet their opposite numbers from the other side. Figures 21.1B and 21.2 show the result of this migration—the formation of a series of curved sausage-shaped blocks of tissue known as the **pharyngeal** or **branchial arches** around the foregut. Observe in Figure 21.2 how the **pharyngeal pouches** and **pharyngeal clefts** are deepened considerably as the migrating ectomesenchyme invades each arch beneath the walls of the foregut. The endoderm of the pharyngeal pouches meets the surface ectoderm lining the clefts to form a **closing membrane**.

A subsidiary arch is produced on the upper posterior aspect of each first (mandibular) arch by a second wave of ectomesenchymal migration from the upper two rhombomeres around six weeks post-fertilization. This is the **maxillary arch** which grows forward between the upper aspect of the mandibular arch and the ectoderm of the frontal eminence covering the developing brain as indicated in Figure 21.3D. Although the maxillary arches lack skeletal and muscular components, they contribute to the formation of the middle third of the face, including the palate; their role in these developments is described in Chapter 32.

Only the leading ectomesenchymal cells are actively migrating by extending cell processes that adhere to the extracellular material between cells and pull the cells forwards; the subsequent cells are attached to the leading cells and are dragged behind as cords of cells. These cords carry in front of them a section of the paraxial mesoderm lying laterally to the neural tube shown in Figure 21.1. Figure 21.3E shows where the mesoderm is distributed by these movements; the development of different muscle groups from this material will be described in Section 21.6. As the ectomesenchyme and mesoderm move into the arches, the motor components of specific cranial nerves move with them (see Section 21.5.2 and Table 21.1). In parallel with the development of spinal nerves described in Section 19.2, the motor axons of cranial nerves form from the basal plate of the neural tube whereas the sensory components are formed from neural crest cells.

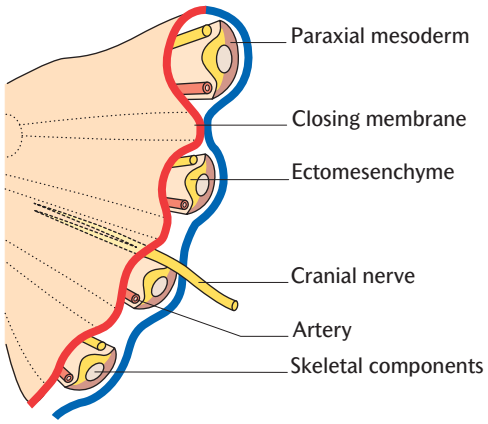


Fig. 21.4 The general arrangement of structures within the pharyngeal arches.

Figure 21.4 shows the general structure of a pharyngeal arch. Each one contains:

- A **cranial nerve**;
- **Ectomesenchyme** that will develop into skeletal elements;
- A second block of ectomesenchyme that develops into the coverings and attachments of muscles;
- A block of **paraxial mesoderm** that will differentiate into skeletal muscle;
- **Lateral mesoderm** that will develop into an aortic arch artery, linking the ventral and dorsal aorta (see Chapter 13).

Each arch is covered by ectoderm externally and in the clefts and lined by endoderm internally and in the pouches.

21.5 Derivatives of the pharyngeal arches

The fate of the skeletal, muscular, and nervous components of each pharyngeal arch are all interlinked. The skeletal structures may persist as cartilage, bone, or ligaments usually close to the original position of their arch of origin. In contrast, many muscles tend to migrate considerable distances. However, their nerve supply is *always* from the cranial nerve of that same arch so their arch of origin can be identified by. All structures derived from a particular arch retain the specific Hox gene code and they will only associate with each other; for example, nerves will only innervate structures bearing the same Hox code. Eventually, the Hox coding extends into the ectodermal and endodermal

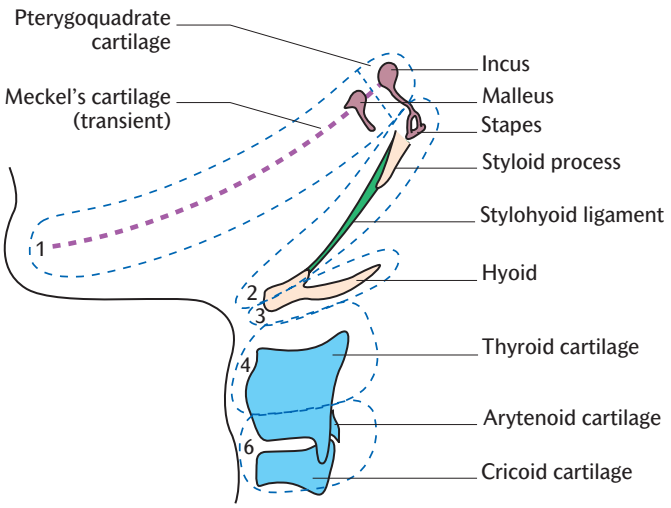


Fig. 21.5 Skeletal derivatives of the pharyngeal arches.

components on the outer and inner surfaces of the arches. The nerve of that particular arch will only innervate ectodermal or endodermal components derived from the same arch and carrying the same Hox code.

21.5.1 Skeletal elements

The skeletal elements derived from each arch are shown in Figure 21.5 which should be followed as the description is read.

The skeletal elements of the first pair of arches, **Meckel's cartilage**, extend as a temporary support from the area destined to become the middle ear cavity along the length of the arch to the midline. The proximal end of Meckel's cartilage eventually ossifies to form the **malleus**, one of the ossicles of the developing middle ear. The **mandible** develops close to Meckel's cartilage, but not from it; the distal part of the cartilage is resorbed as the mandible develops in its lateral side. Some of its covering perichondrium becomes the **sphenomandibular ligament**. The **pterygoquadrate bar** is a smaller cartilage in the proximal tip of the first arch which ossifies to form the **incus,** the second of the middle ear ossicles.

The proximal end of the second arch cartilage (Reichert's cartilage) is also destined for the future middle ear cavity; this part of the cartilage ossifies to form the **stapes**, the third ossicle. The remainder of the cartilaginous bar becomes the **styloid process** of the temporal bone, the **stylohyoid ligament**, and the most distal area forms parts of the **hyoid bone.** The rest of the hyoid bone is formed from the distal part of the third arch cartilage; the rest is resorbed. The cartilaginous elements of fourth arch form the laryngeal cartilages, the **thyroid, cricoid,** and **arytenoid cartilages.**

21.5.2 Cranial nerves and muscles

The cranial nerves and muscles derived from each arch are shown in Table 21.1. Note that the muscles are always supplied by the nerve of the same arch.

Mesoderm and ectomesenchyme migrate into the pharyngeal arches together as described in Section 21.4, but there appears to be no interaction between them until myoblasts, the precursors of muscle cells, differentiate from the mesoderm. The non-skeletal ectomesenchyme then invades the developing muscle to form the investing connective tissues and muscle attachments such as tendons.

21.5.3 Derivatives of the pharyngeal clefts and pouches

Only the first clefts produce structures that persist into maturity; the other clefts are overgrown and obliterated. All the pharyngeal pouches give rise to structures that persist in the head and neck. The derivatives of the pouches and clefts are shown schematically in Figure 21.6. Study the diagrams as you read the description.

The first pharyngeal pouch and cleft develop into components of the outer and middle ears. Each first cleft deepens with further growth to become the **external auditory meatus** and the closing membrane separating the cleft and pouch becomes the **tympanic membrane.** The part of the first pouch nearest the closing membrane widens to form the **middle ear cavity** and the part nearest the pharynx remains narrow and forms the **auditory tube** that connects the middle ear and upper pharynx, allowing equalization of pressure across the tympanic membrane and drainage of the middle ear (See Section 28.3.3).

The second pouch is infiltrated by lymphoid tissue to form the **palatine tonsil**. Parathyroid endocrine tissue differentiates from the endoderm lining the superior part of the third pouch and eventually forms the **inferior parathyroid gland;** the inferior part differentiates into the forerunner of the **thymus** which is part of the immune system involved in the maturation and function of lymphocytes. The thymus is displaced

Table 21.1 Derivatives of the pharyngeal arches

	Skeletal derivatives	Cranial nerve	Muscular derivatives
1st (mandibular) arch	Meckel's cartilage → malleus, spheno-mandibular ligament. Pterygoquadrate bar → incus	Trigeminal	Muscles of mastication, anterior belly of digastric, mylohyoid, tensor veli palatini, tensor tympani.
2nd (hyoid) arch	Stapes, styloid process, stylohyoid ligament, hyoid,	Facial	Muscles of facial expression, posterior belly of the digastric, stylohyoid, stapedius.
3rd arch	Hyoid	Glossopharyngeal	Stylopharyngeus
4th arch	Laryngeal cartilages	Vagus	Muscles of soft palate, pharyngeal constrictors, muscles of the larynx.

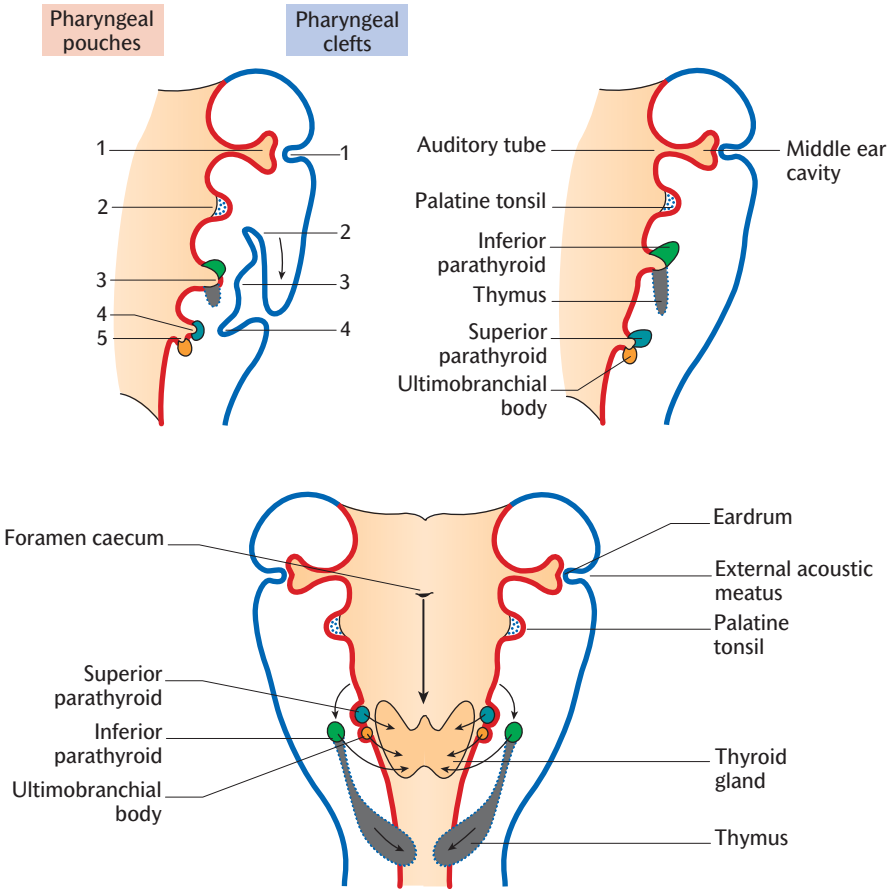


Fig. 21.6 Derivatives of the pharyngeal clefts and pouches.

into the thorax as the heart and great vessels form and move to their adult location. It is located in the anterior mediastinum (Section 12.2; Figure 12.1), but it is usually not visible as it blends with the adipose tissue in that area. The superior part of each fourth pouch forms the **superior parathyroid gland** and the inferior part develops into the **calcitonin-secreting cells** of the thyroid gland.

The thyroid gland develops from the floor of the pharynx as described in Section 21.7 and as shown in Figure 21.6B, it migrates down into its position adjacent to the trachea. It picks up the hormonal tissues forming in the third and fourth pouches and drags them with it which is how the parathyroid glands and the calcitonin-secreting cells become incorporated within the thyroid gland (see also Boxes 21.1 and 21.2).

Box 21.1 First arch syndromes

Considering the complex sequence of events that determine the formation of the pharyngeal arches and their derivatives, it is somewhat surprising that disturbances in the development of the pharyngeal arches are very rare. Most developmental defects affect the first pharyngeal (mandibular) arch and are called **first arch syndromes**. They produce a wide variety of defects that can affect not only the mandibular region, but the areas formed from the maxillary arch such as the nose and palate (see Chapter 32). The first arch contains the expected complement of nerve, artery, skeletal, connective tissue, and muscular elements; it is primarily the skeletal elements that are affected by first arch syndromes. There are several features common to all first arch syndromes:

- An abnormally small **mandible** results in a recessed chin and a Class II occlusion (a marked overjet of the maxillary teeth anterior to the mandibular teeth—see Section 26.2);
- The **malleus and incus**, formed from the proximal part of Meckel's cartilage and the pterygoquadrate bar, respectively, are also small or absent, resulting in **conductive hearing loss**.
- The **external ear** is formed from epithelial tags around the first pharyngeal cleft; these are often deformed, resulting in malformed external ears.

It is now possible to treat children affected by first arch syndromes very effectively. The small mandible and other facial defects can be corrected by bone grafts, the occlusion improved by orthodontic treatment; the external ear defects can be corrected by reconstructive surgery and the hearing loss managed by provision of appropriate hearing aids.

21.6 Embryology of other muscle groups

The segmentation of paraxial mesoderm into somites and their subsequent division into dermatome, sclerotome, and myotome has been described in Section 8.3.5. The most superior fully formed somites are four pairs of **occipital somites** in the paraxial mesoderm adjacent to the lower part of the hindbrain. As shown in Figure 21.3E, the mesoderm derived from their myotomes migrates to the floor of the mouth where it differentiates into the musculature of the **tongue**. The nerves supplying these myotomes are the equivalent of ventral roots of spinal nerves which fused to form the **hypoglossal nerve**; the course of the twelfth cranial nerve in the neck and mouth indicates the migration route of the occipital somites. The sclerotomes of the occipital somites are believed to become incorporated in the posterior part of the skull base (see Section 33.3).

As shown in Figure 21.3E, blocks of unsegmented mesoderm called somitomeres form within the paraxial mesoderm alongside the forebrain, midbrain, and upper half of the hindbrain. As described above, these form several groups of striated muscles in the face, jaws, and neck as the pharyngeal arches develop (see Table 21.1).

Prootic somites develop in the paraxial mesoderm anterior to the developing ear. As illustrated in Figure 21.3E, their myotome components give rise to the **extraocular muscles** which actually form within the prootic somites and subsequently migrate to the orbit. The muscles are supplied by the **oculomotor, trochlear,** and **abducens nerves** which are equivalent to the ventral roots of spinal nerves supplying the muscles of the trunk. The area that is derived from dermatomes of the prootic somites are supplied by the ophthalmic division of the trigeminal cranial nerves.

The tongue muscles form first, followed by the pharyngeal arch muscles, then the extraocular muscles. The precocious development of the tongue muscles means the tongue develops quite early with respect to other oral structures; this has profound effects on their development as we will see in Section 32.2.

Box 21.2 Evolution of the pharyngeal arches

In fossils of early jawed fishes that evolved about 400 million years ago, the branchial skeletal components were arranged as a series of hinged rods, each with a major anterior and posterior component. The upper and lower jaws appear to be derived from the transformed skeletal elements of an anterior gill arch, with the primitive jaw joint developing from the joint between the anterior and posterior components. The upper and lower jaw elements have persisted in all vertebrates as the mouth has expanded and the jaws have been furnished with teeth. In mammalian embryos, the precursor of the upper jaw, the pterygoquadrate bar, is reduced considerably while the lower element, Meckel's cartilage, is prominent but contributes little to the adult lower jaw. Mammalian jaws are instead composed almost entirely of dermal bones which develop independently of the cartilaginous skeleton (see Chapter 33.4).

When land vertebrates evolved, the gills were abandoned in favour of lungs. New structures developed from the arches to accommodate this fundamental change; the skeletal elements of the second to sixth arches developed into the hyoid apparatus supporting the tongue and the laryngeal cartilages guarding the entrance to the lungs. More recently, mammals have evolved a new jaw joint, the temporomandibular joint, between the mandible and the squamous temporal bone, both dermal bones. The bones, which ossify in Meckel's cartilage and the pterygoquadrate bar to form the jaw joint in all other vertebrates, have become incorporated into the middle ear as the malleus and incus. Romer succinctly summed up the evolution of these structures: 'Breathing aids have become feeding aids and finally hearing aids.' (Romer, A.S. (1949). The *vertebrate body*. Saunders, Philadelphia.)

21.7 Development of the tongue and thyroid gland

The tongue and thyroid gland develop from the floor of the pharynx. The relationship between the development of the tongue and its innervation is a very graphic example of how derivatives from each pharyngeal arch are interlinked.

21.7.1 Tongue development

If you have already read Chapter 18 on the cranial nerves, you may have been struck by the fact that the tongue is innervated by no less than *five* different cranial nerves when most other structures are innervated by a single nerve. The explanation for this apparent complexity lies in the story of the development of the tongue and its aftermath in the fully formed organ.

Figure 21.7A shows the major structures contributing to the tongue; it should be followed as the following description is read. The precursors of the tongue appear in the floor of the pharynx at four weeks. Two **lateral lingual swellings** appear first in the endoderm on each side of the first arch. A median swelling, the **tuberculum impar**, appears a little later at the junction between the first and second arches. Two further swellings develop in the midline behind the tuberculum impar, the **copula** or **hypobranchial eminence** derived from the third arch, and the **epiglottal swelling** formed from the fourth arch. The respiratory system develops immediately behind the epiglottis as the **respiratory diverticulum** or **laryngotracheal tube** (see Section 13.3).

As indicated by the arrows in Figure 21.7A, the lateral lingual swellings enlarge and fuse and overgrow the tuberculum impar as they do so. As shown in Figure 21.7B, they form the mucosa covering the anterior two-thirds of the tongue, the part of the tongue occupying the floor of the mouth. This area is derived from the first arch and so receives its somatic

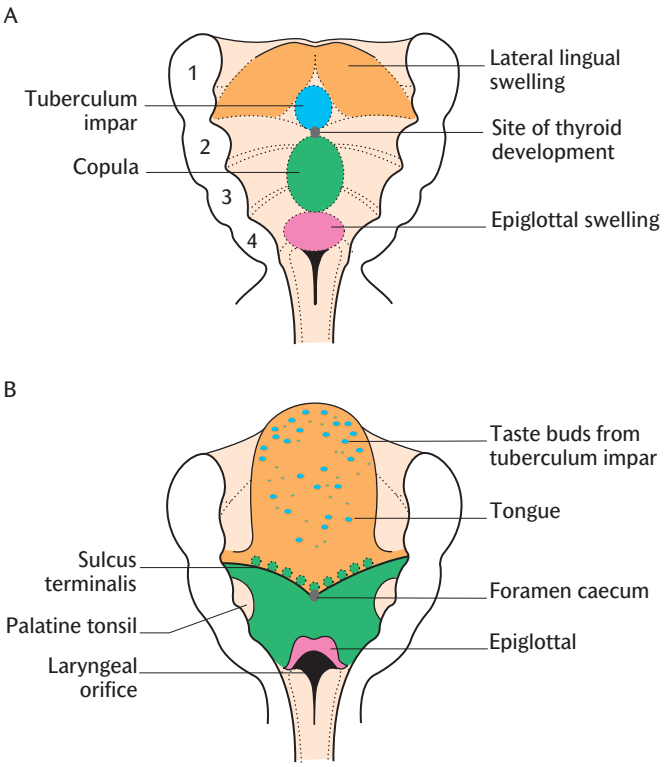


Fig. 21.7 Development of the tongue. A) The precursors of the tongue. B) Their contribution to the mature structure.

sensory innervation from the nerve of the first arch, the **mandibular division** of the **trigeminal nerve** (Table 21.1). The **taste buds** on the anterior two-thirds are derived from the cells of the tuberculum impar; this is a second arch structure which is why taste sensation from the anterior tongue is carried in the **facial nerve**, the nerve of the second arch (Table 21.1). The mucosa of the posterior one-third of the tongue which occupies the anterior part of the pharynx is formed from the third arch. It follows, therefore, that its mucosa is innervated by the nerve of the third arch, the **glossopharyngeal nerve**; the glossopharyngeal nerve conveys somatic and taste sensation. A V-shaped groove, the **sulcus terminalis**, demarcates the embryonic boundaries of the anterior two-thirds and posterior one-third of the tongue. However, some tissue migrates forwards from the posterior one-third during development, to lie just anterior to the sulcus terminalis where it gives rise to the **circumvallate papillae.** Despite being anatomically located on the anterior tongue, these papillae are derived embryologically from the posterior tongue and hence are innervated by the glossopharyngeal nerve. The mucosa on the extreme posterior part of the tongue and the oral side of the epiglottis is innervated by the **superior laryngeal nerve**, a branch of the **vagus nerve**, the nerve of the fourth arch.

You will appreciate from this brief description of the development of the tongue that tissues migrating from their original locations carry their arch-specific nerve supply with them as they do. It also demonstrates why the tongue receives sensory innervation from four different cranial nerves.

As described above, the tongue muscles are derived from the myotomes of the **occipital somites** that are supplied by the **hypoglossal vnerve**; the radically different embryological origin of the tongue muscles explains why the motor innervation of the tongue is supplied by yet another cranial nerve.

Some of the consequences of developmental disturbances on the tongue are described in Box 21.3.

Box 21.3 Abnormalities of tongue development

Occasionally, the tongue appears to be abnormally large at birth, a condition known as **macroglossia**. In most cases, this is due to undergrowth of the rest of the mouth and is gradually corrected during post-natal growth of the jaws. **True macroglossia** occurs and persists in Down syndrome and cretinism. Very rarely, the lateral lingual swellings fail to fuse, producing a bifid (forked) tongue; this is often associated with an equally rare cleft lower lip.

Box 21.4 Developmental abnormalities of the thyroid gland

In several development events, a common problem is that ectodermal or endodermal tissue remains when it should die off and is left in isolation. Certain stimuli, often pathological, stimulate the residual tissue to proliferate to form a fluid-filled spherical structures called **cysts. Thyroglossal cysts** may occur where thyroglossal duct cells persist. They may occur at any point on the path of migration of the thyroid gland; they are commonest close to the hyoid bone but may also occur at the base of the tongue. Sometimes residual cells can actually differentiate into thyroid tissue. **Ectopic** thyroid tissue may also be found anywhere along the migratory path; it occurs most commonly in the base of the tongue close to the foramen caecum, forming a **lingual thyroid**.

21.7.2 Development of the thyroid gland

Towards the end of the third week of development, the endothelium proliferates between the future tuberculum impar and copula. This tissue will develop to form the major components of the thyroid gland. This point is marked in later life by the **foramen caecum** on the dorsum of the tongue as shown in Figures 21.6 and 21.7. As the endoderm forming the tongue mucosa is invaded by mesoderm to form the muscles, the tongue enlarges considerably. This differential growth of the tongue tissue surrounding the thyroid primordium produces relative movement so that the thyroid appears to descend in front of the pharyngeal foregut. By the seventh week, the thyroid has passed down in front of the developing hyoid bone and laryngeal cartilages to lie in front of the developing trachea. The thyroid remains connected to the foramen caecum for some time by a strand of cells known as the **thyroglossal duct**. Eventually, the duct cells die off and disappear.

Developmental abnormalities of the thyroid are considered in Box 21.4

22 The skull

Chapter contents

22.1 Introduction

Dental students and practitioners require a sound knowledge of the structure, growth, and development of the skull as a whole. The structure of the skull can be examined and studied more efficiently if you have access to a dried skull or one of the very good plastic replica skulls which are now available; you can identify the structures on the diagrams accompanying the following descriptions and examine a skull at the same time to appreciate the size and relationships of individual components.

This chapter outlines the basic principles of the development and structure of the skull and includes some reference to individual bones where this makes understanding easier. The more detailed aspects of particular regions of the skull will be covered in the appropriate chapter describing the whole anatomy of that region; it is much easier to learn the parts of the skull in context of overall structure and function rather than learning a long list of bones, foramina, and muscle attachments in isolation from the related soft tissue structures. Only the maxilla and mandible which are bones of significant clinical importance are described as separate bones.

22.2 Components and subdivisions of the skull

As already demonstrated in Chapter 20, the skull is the structural basis for the anatomy of the head. The skull has many functions.

- It encloses and protects the brain.
- It provides protective capsules for the eyes and middle and inner ear.
- It forms the skeleton of the entrances to the respiratory and gastrointestinal tracts (GIT) through the nose and mouth, respectively.

Those skull components that form the entrance to the GIT also house and support the teeth and soft tissues of the oral region as part of this function.

As already outlined in Chapter 20, the skull is made up of several bones joined together to form the **cranium** which articulates with the separate **mandible** forming the lower jaw at the **temporomandibular joints**. The cranium specifically refers to the skull without the mandible; the terms 'skull' and 'cranium' are not strictly synonymous but they are frequently used as though they are.

The cranium can be subdivided into the **braincase** enclosing the brain and the **facial skeleton.** The roof and sides of the braincase, the **cranial vault,** are formed by relatively thin, smooth, curved bones. The more complex bones of the upper facial skeleton form the walls of the orbits, the nasal cavity, and the upper jaws. The **mandible** forms the lower jaw to complete the bony components around the oral cavity. A strong bar of bone, the **zygomatic arch**, runs on each side from the lower lateral part of the cranial vault to the side of the upper facial skeleton. The braincase, upper facial skeleton, and mandible can be identified very easily on a dried skull.

The **cranial base** or **chondrocranium** which forms the floor of the braincase and the roof of the nasal cavity, and houses the inner ear is much less easy to differentiate instinctively. Only the posterior part of the cranial base can be seen in an intact skull when viewed from the underside because the anterior part of the cranial base is obscured by the upper facial skeleton which is attached to its underside. Dried and model skulls usually have a detachable skull cap which can be removed for examination of the inside of the braincase. The bones forming the whole length of the cranial base can be seen in the floor of the braincase when the skull cap is removed. Recall from Chapter 15 (Figure 15.1) that the floor of the cranial cavity is arranged as three step-like hollows—the **anterior, middle**, and **posterior cranial fossae**.

22.2.1 Evolution and development of the skull

The division of the cranium into cranial vault, facial skeleton, and cranial base is based upon their developmental and evolutionary history in addition to the structural reasons. The bones that contribute to the skull can be divided on developmental grounds into the **chondrocranium,** where bones develop first as a cartilage template which is then replaced by bone and **dermal bones** which ossify directly in mesenchyme without an intervening cartilaginous stage.

In early vertebrates, the chondrocranium was a well-developed structure, forming protective boxes around the brain and organs of special sense as well as contributing to the upper and lower jaws through the pterygoquadrate and Meckel's cartilages and their replacing bones (see Section 21.5.1). The chondrocranium is greatly reduced in most modern vertebrates but still forms major skull components. In mammalian skulls, the chondrocranium and its replacement bones are restricted to the **cranial base** and the capsules around the inner ears and nasal cavities. The chondrocranial elements that form the upper and lower jaw in non-mammalian vertebrates take no real part in their formation and structure in modern mammals; the malleus and incus of the middle ear are their only derivatives. The bones that develop from cartilaginous precursors of the **chondrocranium** mineralize by a process of **endochondral ossification.** This term is not strictly accurate as the cartilage actually grows, then bone replaces the older parts of the growing cartilage to consolidate the structure. The most accurate term for bones that mineralize in cartilage is **cartilage-replacing bones**. Note that cartilage-replacing bones form the whole of the post-cranial skeleton, except the clavicles.

Dermal bones, as their name implies, were originally bony plates formed as protective armour plating within the dermis, the connective tissue component of skin; during evolution, they were added to the skull to provide further protection for the brain and sense organs and to complete the jaws. The cranial vault, the lower jaw, and the upper facial skeleton, apart from some of the bones around the nose, are made up of dermal bones. Dermal bones mineralize by a process called **intramembranous ossification.** They are also referred to as membrane bones or intramembranous bones, but dermal bone is the most accurate and reflects the evolutionary origin of these bones. The clavicles are the only other dermal bone found in the human skeleton.

The growth of the different compartments of the skull is determined to a large extent by the growth of the tissues they enclose (see also Section

33.2). The growth of the cranial vault is largely determined by growth of the brain and is also referred to as the **neurocranium** whereas growth of the facial skeleton is related to the growth rate of viscera and thus becomes the **viscerocranium**. The cranial basal grows at an intermediate rate to accommodate the different growth rates of neural tissue above and viscera below. The terms 'neurocranium' and 'viscerocranium' tend to be used more when describing the growth and development of the skull. The neurocranium grows faster than the facial skeleton because the CNS grows at a much more rapid rate than the rest of the body initially. The different growth rates account for the change in proportion of the skull throughout growth. A baby's face seems very small whereas its braincase and eyes are extremely large; the proportions change as growth proceeds until the adult proportions are achieved when the forehead occupies the upper third of the skull, the upper facial skeleton the middle third, and the mandible the lower third.

22.2.2 Joints in the skull

The joints between the dermal bones of the upper facial skeleton and of the cranial vault are fibrous **sutures** whereas the joints in the central regions of the cranial base consist of hyaline cartilage and are termed **synchondroses**. The sutures and synchondroses are named from the contributory bones in many cases, e.g. the zygomaticomaxillary suture; the spheno-occipital synchondrosis. In some instances, they are named from their shape (the lambdoid suture resembles the Greek letter lambda λ) or position (the coronal suture passes across the crown).

Sutures and synchondroses in the developing skull function as growth sites but do not allow movement (see Sections 33.3.1 and 33.4.1). However, at the time of birth, the sutures of the cranial vault are sufficiently flexible to allow some overriding of adjacent bones which enables the head, usually the first part to be born, to pass more readily through the vagina. Synchondroses are replaced by bone as growth ceases and all traces are obliterated. Sutures persist and characteristically the edges of adjacent bones are wavy in outline due to small finger-like projections from adjacent bones which interdigitate to produce a strong interlock between them. Sutures are overgrown by bone later in life, usually from the inner surface to the external surface, and may be difficult to distinguish on the skull of an elderly person; suture obliteration can give useful information about age in forensic dental examinations.

22.3 Let's build a skull

From the outline of the skull and study of surface anatomy presented in Chapter 20, you will appreciate that some bones contributing to the skull are relatively simple in shape whereas others are complex and made up of multiple parts. These complex bones often contribute to more than one of the three basic compartments of the skull. A really excellent way to understand the skull, its component bones, and the contribution of complex bones to different parts of the skull is to build a skull from scratch. We can do this by studying the figures that follow. Figures 22.2 to 22.6 illustrate the complete skull from the anterior, lateral, inferior, internal, and superior views. Figure 22.7 will illustrate the sequential build-up of the skull as bones are added. As each bone is added to the skull, identify the new addition in Figure 22.7 to gain an overall view of where it is, then study the different views of the skull in Figures 22.2 to 22.6 to identify the components of the bone in question and its contribution to different views of the skull.

As described in Section 33.2, the cranial base and the capsules housing the organs of special sense are the first parts of the skull to form. The dermal bones forming the braincase and facial skeleton are then added to the cranial base. We will build our skull following the actual developmental sequence, starting with the cranial base, then adding the facial skeleton and cranial vault.

22.3.1 The cranial base

The sphenoid bone

The **sphenoid bone** is the key bone of the cranial base and all other skull components are attached to it either directly or indirectly. It is one of the first bones to form in the developing skull. The sphenoid also contributes to the walls of the orbit and nasal cavities in the facial skeleton and to the cranial vault. Study the three views shown in Figure 21.1 and on a skull, if possible, and identify:

- The **body**;
- Two **greater wings**;
- Two **lesser wings**;
- Two **pterygoid processes**.

The centrally placed body is approximately cuboidal; it is actually hollow and contains the two **sphenoidal air sinuses.** As you can see in Figure 22.1A, the superior surface of the body has a concavity, the **pituitary fossa**, which houses the pituitary gland. Note particularly in Figures 22.1B and 22.1C, the laterally projecting lesser and greater wings of the sphenoid; each lesser wing is above the greater wing and is separated from it by the **superior orbital fissure**. This fissure transmits the **oculomotor, trochlear, abducens,** and **ophthalmic division** of the **trigeminal nerves** from the cranial cavity into the orbit on each side. Not surprisingly, the greater wing projects further laterally than the lesser wing above it. Each greater wing is curved upwards and laterally. Its inner face forms the anterior wall and part of the side wall of the middle cranial fossa (Figures 22.4 and 22.5) and its outer face contributes to the orbit (Figure 22.2) and forms part of the lower lateral wall of the cranial vault (Figure 22.3). In Figure 22.1A, identify the **foramen rotundum** and **ovale** on each side in the root of the greater wing where it joins the body. They transmit the **maxillary** and **mandibular divisions** of the **trigeminal nerve**, respectively. The lesser wings of the sphenoid form part of the anterior boundary of the middle cranial fossa. Note in Figure 22.1B, the two **optic canals** in the bases of the lesser wings; the **optic nerves** pass through these canals to the orbits. Figures 22.1B and 22.1C show the pterygoid processes hanging down from the sphenoid

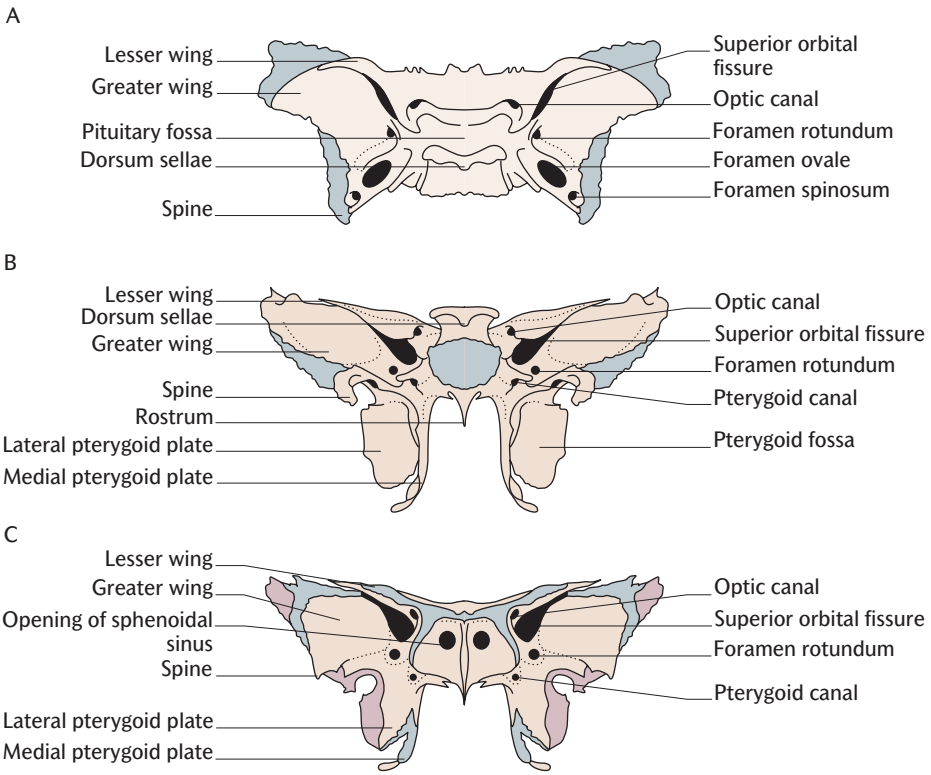


Fig. 22.1 The sphenoid bone. A) Superior view; B) Posterior view; C) Anterior view. Dark grey indicates areas of articulation with other bones.

body and roots of the greater wings. Each process consists of a **medial and lateral pterygoid plate**. These can be seen on lateral (Figure 22.3) and inferior views (Figure 22.4) of the skull. The sphenoid bone is the first bone to appear on the schematic construction of the skull shown in Figure 22.7 and is the only one present in Figure 22.7A.

The occipital bone

The cranial base is extended backwards from the body of the sphenoid by the basal part of the **occipital bone** as shown in Figure 22.7B. This bone is somewhat simpler than the sphenoid bone. Its two major components are the **basal part** (or **basiocciput**), a cartilage-replacing bone, and the **squamous part** which is a dermal bone; the two fuse during development.

The basiocciput bone is easily recognized because it is pierced by the huge midline **foramen magnum** which you can see very clearly in Figures 22.4 and 22.5. This foramen marks the continuation of the medulla and spinal cord. You are unlikely to find a joint between the body of the sphenoid and the basiocciput on most skulls. The **spheno-occipital synchondrosis** is a crucial growth site in the chondrocranium during development (see Section 33.2.3). This joint is totally overgrown when growth at this site ceases around the time of puberty and is, therefore, not visible after this age. The colouration of different bones in Figures 22.4 and 22.5 does indicate its location. In Figure 22.4, you can see two prominent hemispherical bulges on the underside of the basiocciput either side of the foramen magnum. These **occipital condyles** are the superior articulatory surfaces of the **atlanto-occipital joints** and articulate with concave surfaces on the superior aspect of the **atlas,** the first cervical vertebra; flexion and extension (nodding movements) of the head on the neck take place here.

The posterior rim of the foramen magnum is formed by the basiocciput which blends posteriorly with the squamous occipital bone; no joint is visible. The **squamous occipital** bone forms the posterior part of the floor of the posterior cranial fossa (Figures 22.4 and 22.5) and then curves backwards and upwards to form the posterior aspect of the cranial vault (Figure 22.3). It is roughly triangular in outline and its upper borders and apex meet the parietal bones at the **lambdoid suture** shown in Figures 22.3 and 22.6.

The temporal bone

The **temporal bone** is the next bone to be added to our growing skull as shown in Figure 22.7C. The temporal bone is another bone where some parts form as cartilage-replacing bone and others as dermal bones and later fuse. Figure 22.8 shows lateral, inferior, and medial views of the temporal bone. Its different parts are the **petrous, mastoid, squamous, tympanic**, and **styloid**; identify them in the figure and a skull.

The thick wedge-shaped **petrous temporal** bone that houses the middle and inner ear is the major contribution to the cranial base. It is most obvious in Figure 22.8B. Once you have identified its shape from Figure 22.8, you should be able to locate it in Figures 22.4 and 22.5 too. Note in Figures 22.7C and 22.5 how the wedge fits in between the posterior part of the greater wing of the sphenoid and anterior margin of the basiocciput on each side. The gap with a jagged (lacerated) outline

where the three bones meet is the **foramen lacerum**. The upper opening of the bony **carotid canal** opens high in its posterior wall above the level of the cartilage in life. The carotid canal is a wide S-shaped channel passing through the petrous bone which transmits the internal carotid artery. The foramen lacerum does not exist in life as it is plugged with cartilage remnants from the development of these cartilage-replacing bones.

As you can see in Figure 22.8C, there is an obvious foramen on the medial aspect of the petrous temporal bone; this is the **internal auditory meatus** through which the **vestibulocochlear nerves** travel to the inner ear accompanied by the facial nerves. The facial nerves continue on through the middle ear where they make an abrupt 90° bend to exit through the **stylomastoid foramen** on the inferior surface of the petrous bone seen in Figure 22.8B. The upper and medial surfaces of the petrous bone meet almost at right angles. This edge is the boundary between the middle and posterior cranial fossae seen in Figure 22.5; the superior surface forms the posterior floor of the middle fossa and the medial surface the anterior wall of the posterior cranial fossa.

The **tympanic plate** is a small flat area that forms the anterior wall of the external auditory meatus, the wide canal entering the petrous temporal bone from its lateral aspect as shown in Figures 22.8A and 22.3.

The **styloid process** is a thin pointed bony spike about 2 to 3 cm long and is visible on all views in Figure 22.8. It is often broken off dried skulls, especially those that have been handled by generations of dental students, but its root is usually still identifiable. This process develops from the second pharyngeal arch cartilage (see Section 21.5.1) and several muscles and ligaments are attached to it; these will be described in later chapters.

As Figure 22.8A shows, the **mastoid** part is the posterior area of the temporal bone and the **mastoid process** projects inferiorly from it. As already described in Chapter 20 (Figure 20.8), the mastoid process is the superior attachment of the sternocleidomastoid muscle. Figure 22.8B also shows the **mastoid notch,** a deep groove on the medial side of the process for the attachment of the posterior belly of the digastric muscle.

In Figure 22.8B, you can see the **squamous temporal bone** is a relatively thin, flat, vertical plate almost at right angles to the petrous part. The squamous temporal bone forms the middle part of the lateral wall of the cranial vault as shown in Figure 22.3. As seen in Figures 22.8A and 22.3, its **zygomatic process** runs forward from its root just above the external auditory meatus to meet the zygomatic bone to form the **zygomatic arch**. The superior articular surfaces of the **temporomandibular joints** are on the underside of the squamous temporal anterior to the external auditory meatus. These are best seen in Figure 22.8A. The **mandibular** (or glenoid) **fossa** is a concave depression housing the condyle of the mandible and the convex **articular eminence** lies anterior to it. When the mouth is opened wide, the mandibular condyle slides forward on to the eminence (see Chapter 26). The posterior border of the mandibular fossa is separated from the tympanic plate by the **squamotympanic fissure**; the **chorda tympani** branches of the **facial nerve**, carrying taste and parasympathetic nerves to the tongue and floor of the mouth, exit from the skull in its wider medial area.

Examine Figure 22.7C and you can see that the cranial base runs along the midline of the middle and posterior cranial fossae. These two fossae are completed laterally by the **greater wings of the sphenoid**, the **petrous temporal**, and **squamous occipital** bones. We have already started to build the lateral walls of the cranial vault with the greater wings of the sphenoid and squamous temporal bones and its posterior wall with the squamous occipital bone. We clearly lack the anterior components of the cranial base and bones to complete the floor of the anterior cranial fossa.

The ethmoid bone

In Figure 22.5 and a skull with the skull cap removed, you should have little trouble in identifying a plate of bone punctured by numerous small holes in the midline of the anterior cranial fossa just anterior to the body of the sphenoid. This is the **cribriform plate** of the **ethmoid bone** and is the site where the olfactory nerves connect the olfactory bulbs on the underside of the frontal lobes to the olfactory mucosa in the roof of the nasal cavities. The ethmoid is another complex bone and most of it is hidden in the figures of the skull and even on a skull itself.

Look at Figure 22.9A, a schematic diagram of the ethmoid bone seen in a coronal section of the skull through the orbits and nasal cavity. The real bone viewed from the front is illustrated in Figure 22.9B. The ethmoid bone resembles a letter E on its side. The cribriform plates form the 'backbone' of the E. The middle downward extension is the **perpendicular plate** that contributes to the nasal septum that divides the nasal cavity into right and left halves. The left and right downward extensions are the **ethmoid labyrinths** which contain small air-filled sacs continuous with the nasal cavity; these are the **ethmoidal air cells** (or air sinuses). Note in Figure 22.9 that the medial wall of each labyrinth has two extensions, the **superior and middle conchae;** these form part of the upper lateral wall of the nasal cavity. You can appreciate from studying Figure 22.9A that the ethmoid bone contributes to the anterior cranial fossa above, the medial walls of the orbits laterally, the lateral wall of the nose medially, and the nasal septum centrally. Identify the **crista galli** in Figures 22.5 and 22.9. This prominent crest of bone projects upwards into the anterior cranial fossa from the midline of the ethmoid bone; the anterior edge of the falx cerebri is attached to it.

The cribriform plate of the ethmoid completes the cranial base when the ethmoid bone is added to the skull in Figure 22.7D, but we still have a huge gap where the rest of the floor of the anterior cranial fossa should be.

The frontal and parietal bones

As shown in Figure 22.5, the floor of the anterior cranial fossa is completed by the horizontal orbital processes of the **frontal bone** on either side of cribriform plate; the two orbital plates join anterior to the cribriform plate. Figures 22.2 and 22.3 illustrate the frontal bone turning up at the superior margins of the orbits to form the forehead, then arching back to the vertex of the skull. The supraorbital part of the frontal bone is hollow and contains the two **frontal (paranasal) air sinuses;** these are described in Section 27.4. We only need to add the **parietal bones,** two gently curved plates, between the frontal bone and the squamous occipital bone to complete the braincase as shown in Figures 22.6 and 22.7E.

Note from Figure 22.6 that the frontal and parietal bones join at the **coronal suture**, the two parietal bones at the **sagittal suture**, and the parietal and occipital bones at the **lambdoid suture**. The frontal bone develops as two halves and a **frontal suture** is present

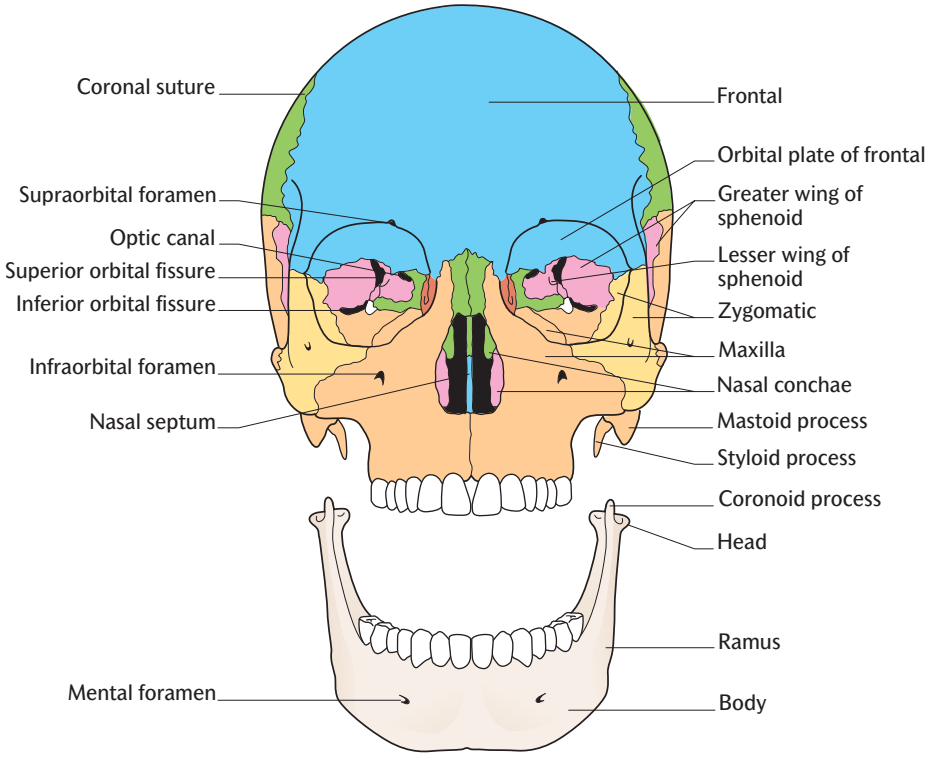


Fig. 22.2 Anterior view of the skull.

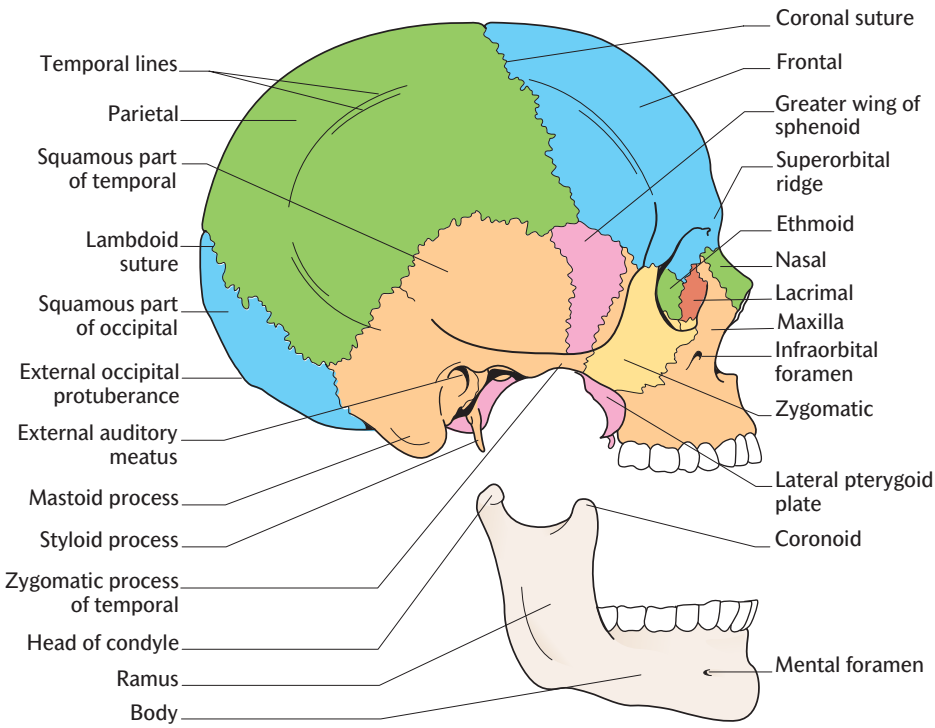


Fig. 22.3 Lateral view of the skull.

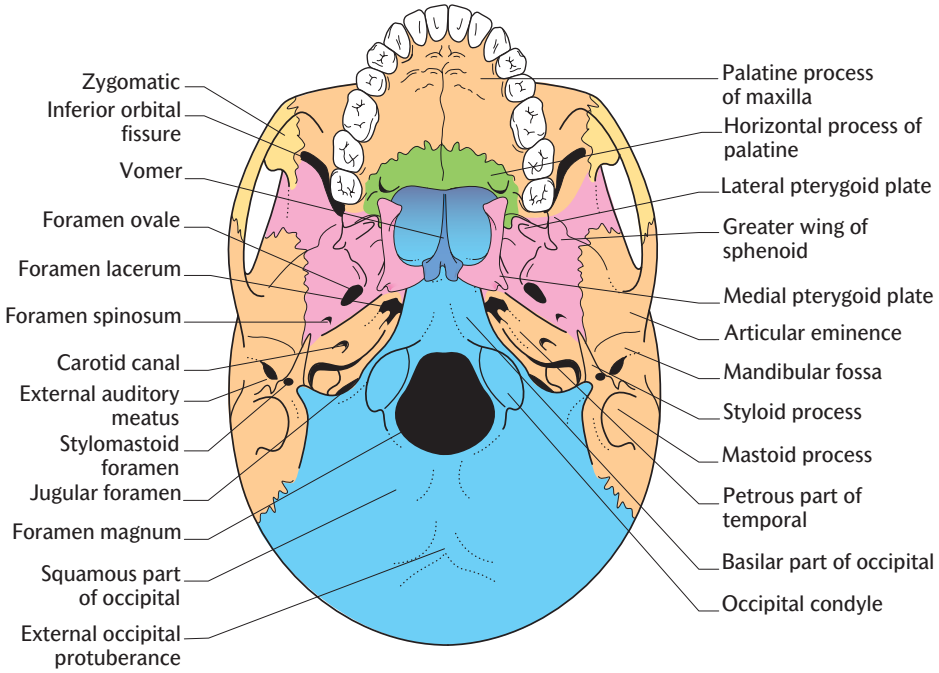


Fig. 22.4 Inferior view of the cranial base.

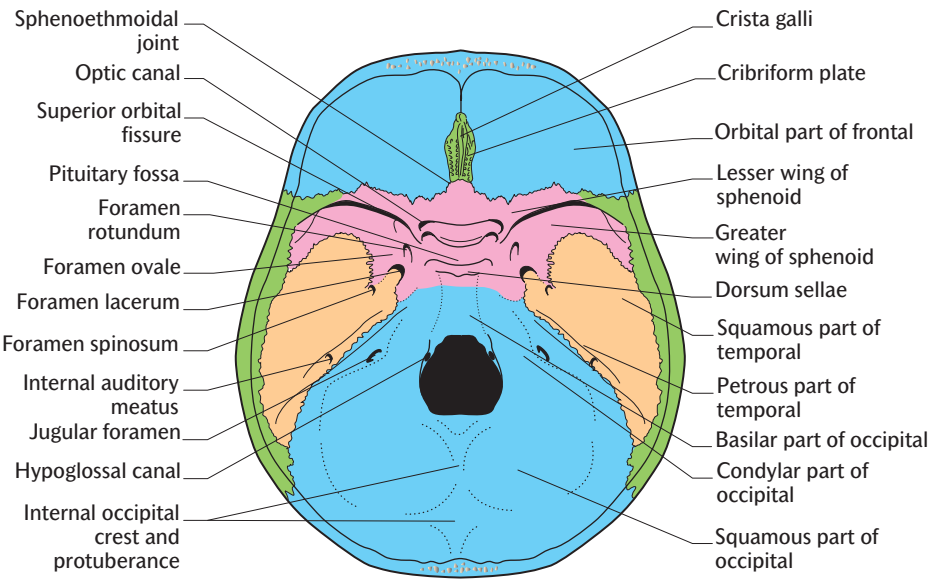


Fig. 22.5 Superior view of the cranial base.

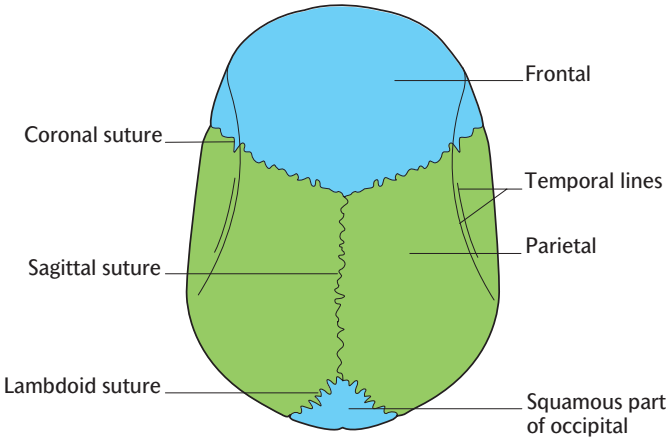


Fig. 22.6 Superior view of the skull.

in the median plane of the frontal bone in immature skulls. The frontal suture fuses during early childhood in the majority of individuals, but in a few, some of the suture may persist into adult life as the **metopic suture**. The lateral wall of the cranial vault inferior to the parietal bone is formed, as we have already seen and as illustrated in Figure 22.3, by the greater wing of the sphenoid and the squamous part of the temporal bone between the posterior orbit and parietal bones. These bones meet the frontal and parietal bones at the **pterion,** an irregular H-shaped arrangement of sutures in the temple region.

The lateral wall of the cranial vault ends inferiorly at the **infratemporal crest**, a pronounced ridge on the greater wing of the sphenoid and squamous part of the temporal where the bones turn medially to form the roof of the **infratemporal fossa** (see Section 24.5).

22.3.2 The facial skeleton

We must now add the bones that comprise the facial skeleton to complete the skull. We know where to put them because they have to fit anterior to the pterygoid processes of the sphenoid, below the orbital processes of the frontal bones and around the ethmoid bone. The facial bones complete the walls of the nasal cavity and the orbits and form the upper jaw and roof of the mouth. The position of the orbital cavities and upper nasal cavity is already defined by the ethmoid bone and the orbital plates of the frontal bones. A good place to start to build the facial skeleton is to complete the outline of the orbital and nasal cavities.

The maxilla

The two maxillae form the upper jaw and also contribute to the orbits and nasal cavity. The basic outline of each **maxilla** can be seen in Figure 22.2. Locate the point where the left and right maxillary central

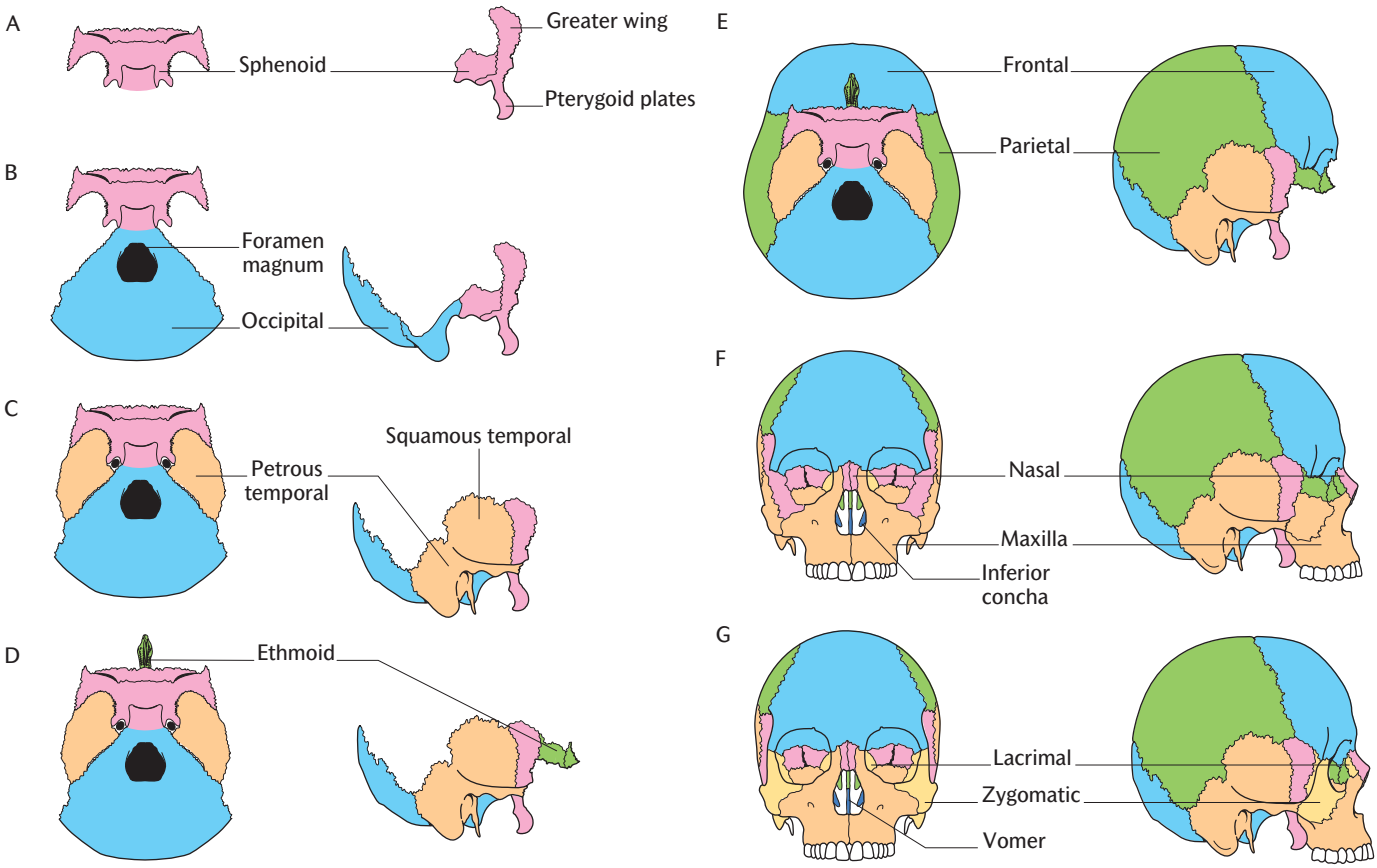


Fig. 22.7 A progressive build-up of the skull to show the contribution of different bones to the subdivisions of the skull. A to E in the left hand columns are the same superior view as Figures 22.5 and F and G are anterior views as Figure 22.2; right hand columns A to G show lateral views as Figure 22.3.

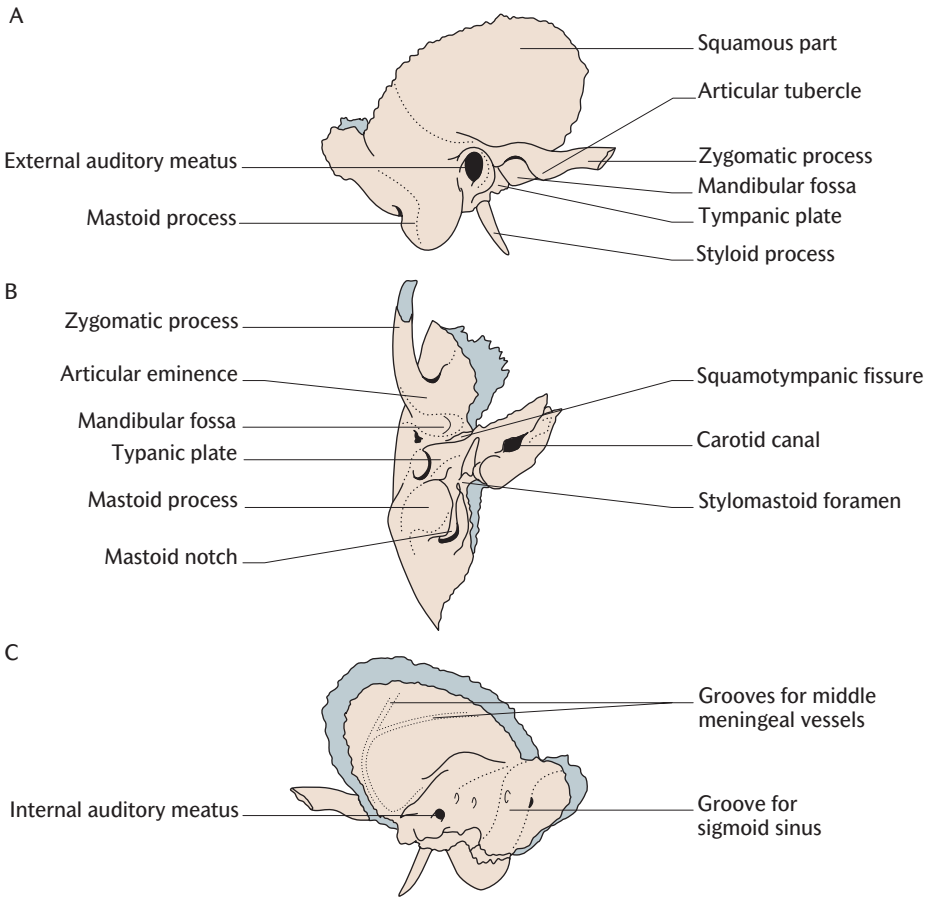


Fig. 22.8 The right temporal bone. A) Lateral view; B) Inferior view; C) Medial view. Grey shading indicates areas of articulation with other bones.

incisors meet in the midline. The **intermaxillary suture** between the two maxillary bones runs vertically above this point to the floor of the nasal cavity. On the anterior view in Figure 22.2, the outline of each bone is roughly triangular with the apex pointing laterally. The lower side is the **alveolar process** housing the upper teeth and the upper side forms the medial rim and medial part of the inferior rim of the orbit. The base runs from the intermaxillary suture and is then markedly indented where it forms the lateral border of the anterior nasal opening; it then continues upwards as the **frontal process** to meet the frontal bone above.

Each maxilla is quite extensive as shown in Figures 22.2, 22.3, and 22.4. Each consists of a large body that is approximately pyramidal and is hollowed out by the **maxillary air sinus**. The upper surface forms the floor of the orbit, the anterior surface the curved external surface of the upper jaw, and the posterior surface forms the anterior part of the infratemporal fossa (see Section 25.4). The medial surface forms the base of the pyramid and is a major component of the lateral wall of the nasal cavity. Much of the maxilla cannot be seen on external views of the skull. However, it is so important to the practice of dentistry that it will be described in more detail in Section 22.6.4.

We can now put the maxillae in place in our skull as shown in Figure 22.7F. When in position, each maxilla completes the rims of the orbits superiorly and medially, but we still have a gap inferiorly and laterally. Note that the two maxillae are not joined to each other at the top of the anterior nasal opening.

Nasal, palatine, zygomatic, lacrimal, vomer, inferior nasal concha

Several small bones contribute to the upper facial skeleton, some of which are hidden from view.

The superior gap between the maxillae is bridged by the two **nasal bones** that form the bridge of the nose as shown in Figure 22.2. Adding the nasal bones to Figure 22.7F completes the anterior nasal opening.

The inferior view of the skull in Figure 22.4 shows that the U-shaped upper dental arch encloses the bony palate. The intermaxillary suture running down the midline joins the **palatine processes** of the two maxillae. Note another suture towards the back of the bony palate running perpendicular to the intermaxillary suture; this separates the maxilla from the horizontal process of the **palatine bone** that also contributes to the bony palate on each side. You can see in Figures 22.3 and 22.4 that the maxilla posterior to the third molar tooth is in contact with the pterygoid process of the sphenoid bone.

The facial skeleton in Figure 22.7F still has a large lateral defect, but this can be filled by the addition of the **zygomatic bone** on each side, the last major bones of the upper facial skeleton. When these are placed lateral to the maxillae, the inferior and lateral parts of the orbital rim and the zygomatic arch are completed as can be seen by studying

Figures 22.2 and 22.3. The facial skeleton now appears almost complete as shown in Figure 22.7G.

There are other bones that contribute to the upper facial skeleton. The **lacrimal bones** are small rectangular bones about the size of a fingernail that form part of the medial wall of each orbit; identify them in Figures 22.2 and 22.3. The inferior part of the nasal septum is formed by the **vomer**, a single bone situated below the perpendicular plate of the ethmoid and above the palatine processes of the maxillae. Its position can be seen by looking into the nasal cavity in Figure 22.2, but the suture lines between the vomer and other bones are not visible.

Look again into the anterior nasal aperture and count the thin curved bones protruding from each lateral wall. On a good dried or model skull, you should find three **nasal conchae.** We have already met the superior and middle concha as part of the ethmoid bone in Figure 22.9; the inferior concha is a formed by a separate bone known as the **inferior nasal concha.** The skeletal components of the nasal walls will be described in Section 27.3 when the contributions of the vomer and inferior conchae to the nasal skeleton will become more obvious.

The mandible

We have now constructed the cranium comprising the cranial base, cranial vault, and upper facial skeleton. We only need to add the mandible to turn the cranium into a skull.

The major features of the mandible are shown in Figures 22.2 and 22.3. The **body** of the mandible is the robust horizontal U–shaped bar of bone carrying the lower teeth. A vertical ramus projects upwards at the posterior end of each half of the body; each ramus carries two processes, the anterior triangular **coronoid process**, and the posterior **condylar process** or **condyle**. Each condyle is expanded into a transversely running hemispherical head; the two heads articulate with the mandibular fossae on the inferior aspects of the temporal bones to form the **temporomandibular joints**. The detailed anatomy if the mandible will be described in more detail in Section 22.6.5.

The stage by stage construction of the skull will have shown you the shape of the different bones, their relationship to each other, and their contribution to the overall shape and compartments of the skull. This building exercise provides an outline of the skull and includes some obvious anatomical features. Some more features will now be covered but most of the specific anatomical detail required by dental students and practitioners will be covered with the regional anatomy described in the following chapters.

22.4 The cranial vault

22.4.1 Bones and sutures

The **frontal**, paired **parietals**, the **squamous** part of the **occipital**, the **greater wings** of the **sphenoid**, and the **squamous** parts of the paired **temporal bones** forming the cranial vault all have the same structure. They consist of an inner and outer layer of compact bone separated by a thin layer of cancellous bone (the **diploë**) that contains red marrow actively forming red and white blood cells well into old age.

Foramina are not a feature of the cranial vault. The supraorbital notch (or foramen as illustrated in Figure 22.2) is at the junction of the lateral two-thirds and medial one-third of the supraorbital margin on the frontal bone. **Emissary veins** connect the intracranial venous sinuses with extracranial veins through inconsistent small foramina in the bones of the vault (see Section 15.5.2).

As already described in Chapter 20 (Figure 20.8), the **superior** and **inferior temporal lines** are visible on the lateral aspect of the external surface of the braincase. The **temporalis fascia** is attached superiorly to the superior temporal line and inferiorly to the upper border of the zygomatic arch. As you can see in Figure 22.3, the inferior temporal line is the superior attachment of the **temporalis muscle**. They begin anteriorly as a single line which is continuous with the prominent ridge where the frontal bone articulates with the zygomatic bone. The two lines diverge as they pass posteriorly and then curve across the frontal and parietal bones about 1 cm apart. Posteriorly, the superior temporal line fades away, but the inferior line becomes more prominent and curves downwards and forwards across the squamous part of the temporal to blend with the posterior root of the zygomatic arch.

The posterior aspect of the underside of the cranial vault formed from the squamous part of the occipital bone has a number of ridges marking the attachment of the extensor muscles of the neck as shown in Figures 22.2 and 22.4. The **superior nuchal line** curves on each side from the mastoid process of the temporal bone to end at the **external occipital protuberance,** a midline prominence on the occipital bone.

22.4.2 Internal features

In addition to the sutures already described, the inner aspects of the bones of the cranial vault carry markings produced by vascular structures; the majority of these are made by the venous sinuses described in Section 15.5.2.

The **sagittal sulcus** is produced by the **superior sagittal sinus**. It begins on the inner surface of the frontal bone and runs posteriorly in the median plane, widening progressively as it does so, to end close to the **internal occipital protuberance**, a prominence on the internal aspect of the occipital bone located opposite the external occipital protuberance. Figure 22.5 shows a shallow horizontal groove curving forwards away from the protuberance on each side, marking the course of the **transverse venous sinuses**. Anteriorly, each groove runs downwards and medially and then forwards in an S shape to reach the jugular foramen. The S-shaped groove is occupied by the **sigmoid sinus**, a continuation of the transverse sinus.

The inner surface of each parietal bone and the internal surface of the pterion are marked by a system of irregular, deeply cut grooves

produced by the branches of the **middle meningeal** blood vessels running between the bones and the dura. If these markings are traced downwards to the cranial base, they converge on to the small **foramen spinosum** which can be identified in Figure 22.5, immediately posterolateral to foramen ovale. Viewed from the underside of the skull in Figure 22.4, the foramen ovale is easily recognized by its distinctive oval shape; once located, the foramen spinosum is also easy to find. The nervus spinosus, a meningeal branch of the mandibular division of the trigeminal nerve, also passes through this foramen.

22.5 The cranial base

The three subdivisions of the cranial base, the **anterior, middle**, and **posterior cranial fossae,** house the cerebral frontal lobes, temporal lobes, and the cerebellum and brainstem, respectively (see Chapter 15, Figure 15.1).

22.5.1 The anterior cranial fossa

As already described, the floor of the anterior cranial fossa is formed by the cribriform plate of the ethmoid medially and the orbital plates of the frontal bone laterally. The lesser wings and body of the sphenoid form the posterior part of the floor in the midline as shown in Figure 22.5; the posterior border of the fossa is formed by the same components of the sphenoid.

22.5.2 The middle cranial fossa

The features of this area are shown in Figure 22.5. The length of the middle cranial fossa is relatively short in the midline, but more extensive laterally. The midline area is occupied by the pituitary fossa in the upper surface of the body of the sphenoid. Laterally, the fossa is formed by the **greater wings of the sphenoid**, the upper surfaces of the **petrous temporal bone**, and the inferior part of the **squamous temporal bone** on each side. Numerous foramina described earlier pierce the floor of the fossa through the sphenoid components or the joint lines between the contributory bones. The posterior border of the middle cranial fossa is formed by the posterior ridge of the pituitary fossa and the junction of the superior and posterior surfaces of the petrous temporal bones laterally.

The inferior surface of the cranial base under the middle cranial fossa is formed by the pterygoid processes of the sphenoid, with the **pterygoid plates** projecting downwards on either side from the junction of the body and greater wing of the sphenoid. The larger **lateral pterygoid plates** and smaller **medial pterygoid plates** are illustrated in Figures 22.1B and 22.4; the plates diverge posteriorly to enclose the **pterygoid fossa**. The pterygoid plates are structurally part of the cranial base but function as component of the upper facial skeleton. The **lateral** and **medial pterygoid muscles**, the two deep muscles of the muscles of mastication, attach to the lateral pterygoid plates. The pterygoid plates and their muscle attachments are described in more detail in Section 24.3.3.

The **infratemporal fossa** is the depression lateral to the pterygoid process and under the greater wings of the sphenoid where the infratemporal crest marks its outer limit. It contains many soft tissues of real importance to the dental student that a large part of Chapter 24 is devoted to the fossa and its contents.

22.5.3 The posterior cranial fossa

This is the largest and deepest of the three cranial fossae and houses the cerebellum and lower brainstem. Its central zone is formed by the **body of the sphenoid** and the **basiocciput**. The floor of the fossa is pierced in the midline by the **foramen magnum**. The lateral parts of the fossa are formed by the posterior surfaces of the **petrous temporal bones** and the condylar and squamous parts of the **occipital bone**.

In Figure 22.4, locate the **petro-occipital fissure** running posterolaterally from the foramen lacerum between the petrous temporal bone and the basilar and condylar parts of the occipital bone. Its anteromedial part is closed in life by cartilage continuous with that plugging the foramen lacerum. The posterolateral end of each fissure is widened to form the **jugular foramen** which transmits the **glossopharyngeal, vagus,** and **accessory nerves**. The grove formed by the sigmoid sinus described above can be traced down to locate the jugular foramen; the sigmoid and inferior petrosal sinuses described in Section 15.5.2 meet on the inner side of the cranial base to form the **internal jugular vein** as they pass through the foramen.

The occipital bone is pierced by the **hypoglossal canals** which transmit the hypoglossal nerves. Their internal openings can be seen in Figure 22.5 in the anterolateral wall of the foramen magnum. The canals open on the inferior surface of the skull lateral to the occipital condyles.

22.6 The facial skeleton

22.6.1 The orbit

The orbital cavities contain the eyeballs, their associated muscles, nerves and vessels, the lacrimal apparatus, and a large amount of fat to cushion and protect the globe; some nerves and vessels pass through the orbits to reach the face. As you can see in Figure 22.2, each orbit is a pyramidal recess with a roof, floor, and medial and lateral walls converging to an apex posteriorly; its base is at the orbital opening. The long axis of the orbit is directed forwards and laterally.

The greater part of the roof of the orbit is formed by the inferior surface of the **orbital part of the frontal bone**. The lateral wall is formed by the orbital surfaces of the **zygomatic bone** and the **greater wing of the sphenoid**. The floor of the orbit is formed by the thin plate of bone forming the upper surface of the **body of the maxilla** which is also the roof of the maxillary paranasal air sinus in the maxillary body (see Section 22.6.3). The medial orbital wall is very thin and is frequently damaged in the dried skull. Several bones contribute to the medial wall and are described in more detail in Section 30.2.

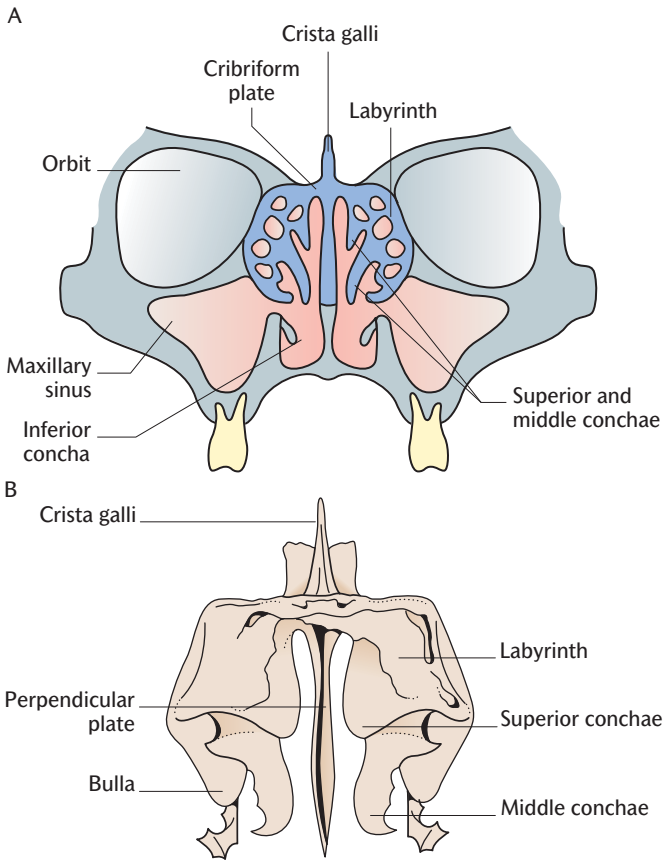


Fig. 22.9 A) A diagram of the relationships of the ethmoid bone in coronal section; B) A posterior view of the ethmoid bone.

22.6.2 The nasal cavity

This is an irregularly shaped cavity extending from the bony palate below to the floor of the anterior cranial fossa between the orbits above. Figure 22.2 shows the **nasal septum**, dividing the nasal cavity into right and left halves. In life, the septum is completed anteriorly by cartilage, but in dried skulls, the bony part of the septum does not reach as far as the **anterior nasal aperture** which is, therefore, undivided. As you can see in Figure 22.4, the **posterior nasal aperture** between the pterygoid processes of the sphenoid is divided into two by the back edge of the bony septum. The internal bony structure of the nasal cavity can only be examined on a skull sectioned in the median plane. The structure of the nasal cavity is described in more detail in Section 27.3.

As mentioned above, some of the cranial bones are hollowed out by **the paranasal air sinuses,** air-containing spaces which communicate with the nasal cavity. The ethmoidal labyrinth and sphenoid in the cranial base, the frontal bone in the cranial vault, and the maxillae in the facial skeleton all contain sinuses. These will be described in more detail in Section 27.4.

22.6.3 The maxilla

The upper jaw is made up of the two **maxillae**. Each consists of a **body** and four **processes.** As shown in Figure 22.10, the two maxillae form a median projection, the **anterior nasal spine,** at the inferior margin of the anterior nasal aperture.

As described above, the body is large and roughly pyramidal in shape and its interior is hollowed out by the **maxillary air sinus**. Figure 22.10A illustrates the continuity of the anterior surface above the molar teeth with the convex infratemporal surface which faces posterolaterally, forming the anterior wall of the infratemporal fossa. The infratemporal surface ends below at the **maxillary tuberosity,** a prominent rounded eminence behind the last molar tooth. A number of small openings are shown in Figure 22.10A about half way up the infratemporal surface; these can be quite difficult to locate on dried and plastic model skulls. The openings lead to the posterior alveolar canals through which the **posterior alveolar nerves** and arteries reach the upper molar teeth and associated structures (see Section 25.4.1). The **infraorbital groove** and canal run across the orbital surface of the maxilla, carrying the **infraorbital branch** of the **maxillary trigeminal nerve**. It emerges on to the face through the **infraorbital foramen** shown in Figures 22.2 and 22.10B.

Each maxilla has four processes:

- The zygomatic process shown in Figure 22.10A projects laterally from the junction of the anterior and infratemporal surfaces of the body to articulate with the zygomatic bone as you can see in Figure 22.3;
- The frontal process which articulates with the frontal bone above and contributes to the medial wall of the orbit, the lateral wall of the nose, and the bridge of the nose behind the nasal bone (Figures 22.2 and 22.10);
- The alveolar process which projects downwards and forms the sockets for the roots of the upper teeth and ends posteriorly at the tuberosity (Figures 22.2, 22.3, 22.4, and 22.10);
- The palatine process which projects medially to articulate with the corresponding process of the opposite maxilla to form the anterior three-quarters of the bony palate (Figures 22.4 and 22.11).

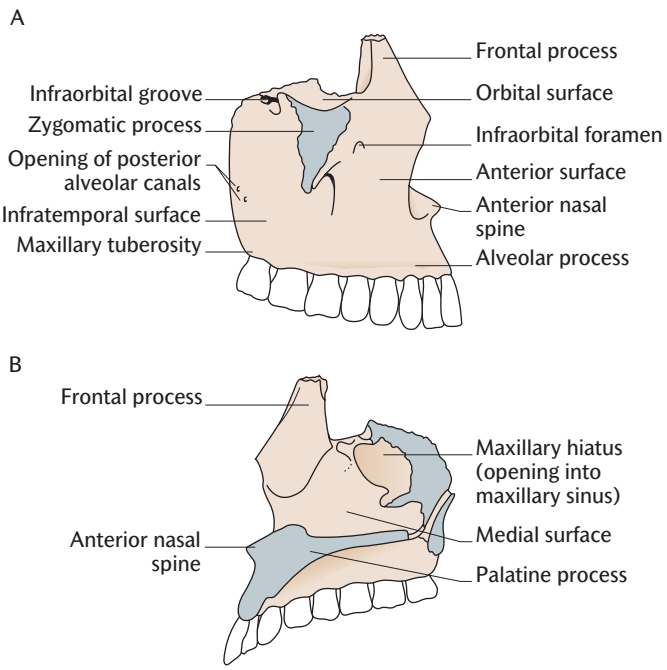


Fig. 22.10 The right maxilla. A) Lateral view; B) Medial view.

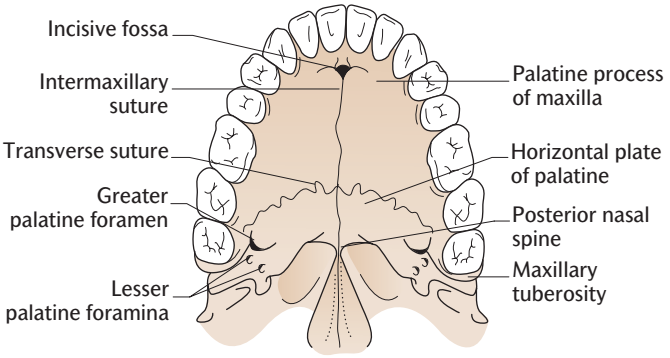


Fig. 22.11 Inferior view of the bony palate.

As shown in Figure 22.11, the **palatine processes of the maxillae** contribute to the anterior three-quarters of the skeleton of the **bony palate;** the **horizontal plates of the palatine bones** form its posterior one-quarter. Sutures join the palatine bones and maxillae and the left and right halves of the palate. The hard palate is the roof of the mouth and the floor of the nasal cavity and is described in more detail in Section 25.3.2.

22.6.4 The mandible

The features of the mandible are illustrated in Figures 22.12 and 22.13. As already outlined, the mandible consists of a **body,** which is horseshoe-shaped when viewed from above in Figure 22.13, and two vertical **rami** which project upwards from each posterior end of the body.

The mandible develops as two separate halves united in the midline by a fibrocartilaginous joint. This joint is obliterated during the first year of postnatal life and the two halves of the mandible fuse to form the single body.

The body

The body has external and internal surfaces. The external surfaces are adjacent to the lips anteriorly and the cheeks posteriorly so are termed the **buccal** and **labial** surfaces, respectively. The internal surface is adjacent to the tongue and is the **lingual** surface.

The external labial surface is marked by a vertical ridge in the midline which indicates the fusion line of the two halves of the mandible. As you can see in Figure 22.12A, this ridge runs inferiorly into a raised area termed the **mental protuberance**, which gives the human chin its characteristic shape. The **oblique line** is a faint ridge on the buccal surface; it is prominent below the molar teeth, but becomes indistinct anteriorly as you can see in Figure 22.12A. It is a continuation of the **external oblique ridge** forming the anterior border of the ramus and runs forwards and downwards towards the mental tubercle. A **mental foramen** is present on the buccal surface on each side between the root apices of the first and second premolar teeth. This foramen transmits the **mental branches** of the **inferior alveolar nerve** and blood vessels which themselves run in the **mandibular canal** within the body of the mandible.

As you can see in Figure 22.12B, the lingual surface of the body bears an oblique ridge, the **mylohyoid line,** which is the attachment of the mylohyoid muscle**;** it begins as a prominent crest a short distance below the last molar tooth and runs forwards and downwards, becoming indistinct in the region below the anterior teeth. The **submandibular fossa** is a concave area below the mylohyoid line which lodges the submandibular salivary gland. The **mylohyoid groove** runs forwards from below the mandibular foramen on the ramus into the submandibular fossa to fade out anteriorly. The groove carries the **mylohyoid branches** of the inferior alveolar nerve and vessels. The inferior part of the internal lingual surface below the incisor teeth bears small elevations close to the midline, the **superior** and **inferior genial tubercles** (or **mental spines)** although it is often difficult to distinguish them as separate entities. The genioglossus muscles of the tongue attach to the superior tubercles and the geniohyoid muscles to the inferior ones. A small, roughened **digastric fossa**, positioned to each side of the midline on the inferior border of the body, is the attachment of the anterior belly of the digastric muscle.

The **alveolar process** lies above the upper part of the body of the mandible. It contains the sockets for the roots of the lower teeth and only develops as the teeth undergo root formation and erupt into the oral cavity. If teeth are lost through trauma or disease, the supporting alveolar bone is resorbed (see Section 33.4.4).

The ramus

As you can see in Figure 22.12, each ramus is a quadrilateral plate of bone. Its flat lateral surface is roughened posteroinferiorly by the attachment of the masseter muscle. The **mandibular foramen** is approximately at the centre of the medial surface and is usually at the same level as the occlusal surfaces of the mandibular teeth. The foramen transmits the **inferior alveolar nerve** and blood vessels into the **mandibular canal** which runs downwards and forwards into the body of the mandible where it continues directly forwards just below the roots of the teeth (see Section 25.4.2). The **lingula** is a thin plate of bone which projects over the foramen from its anterior border as shown in Figure 22.12B. The **mylohyoid groove** begins at the lower border of the mandibular foramen. The area posteroinferior to the mylohyoid groove is roughened by the attachment of the medial pterygoid muscle. The inferior and posterior borders of the ramus meet at the **angle** of the mandible.

Figure 22.12 shows the **coronoid** and **condylar processes** extending from the superior border of the ramus; they are separated from each other by the curved **mandibular incisure**. The coronoid process is the anterior triangular plate of bone which projects upwards and forwards. The **temporal crest** is a faint ridge on the medial aspect of the coronoid process which becomes more prominent towards the margin of the alveolar bone medial to the last molar tooth. The **retromolar fossa** is seen most clearly in Figure 22.13. It is the area posterior to the last molar tooth between the temporal crest and the external oblique ridge marking the anterior border of the ramus. The temporalis muscle attaches from the superior and anterior margins of the coronoid process, down the anterior border of the ramus to the retromolar fossa.

The condylar process (or more simply the **condyle**) is the posterior process on the superior border of the ramus. It is expanded to form the **head of the condyle**. In life, its superior and posterior surfaces of each head are covered with fibrocartilage and articulate with the articular surfaces of the **squamous temporal** bones to form the synovial **temporomandibular joints**. When viewed from the side as in Figure 22.12,

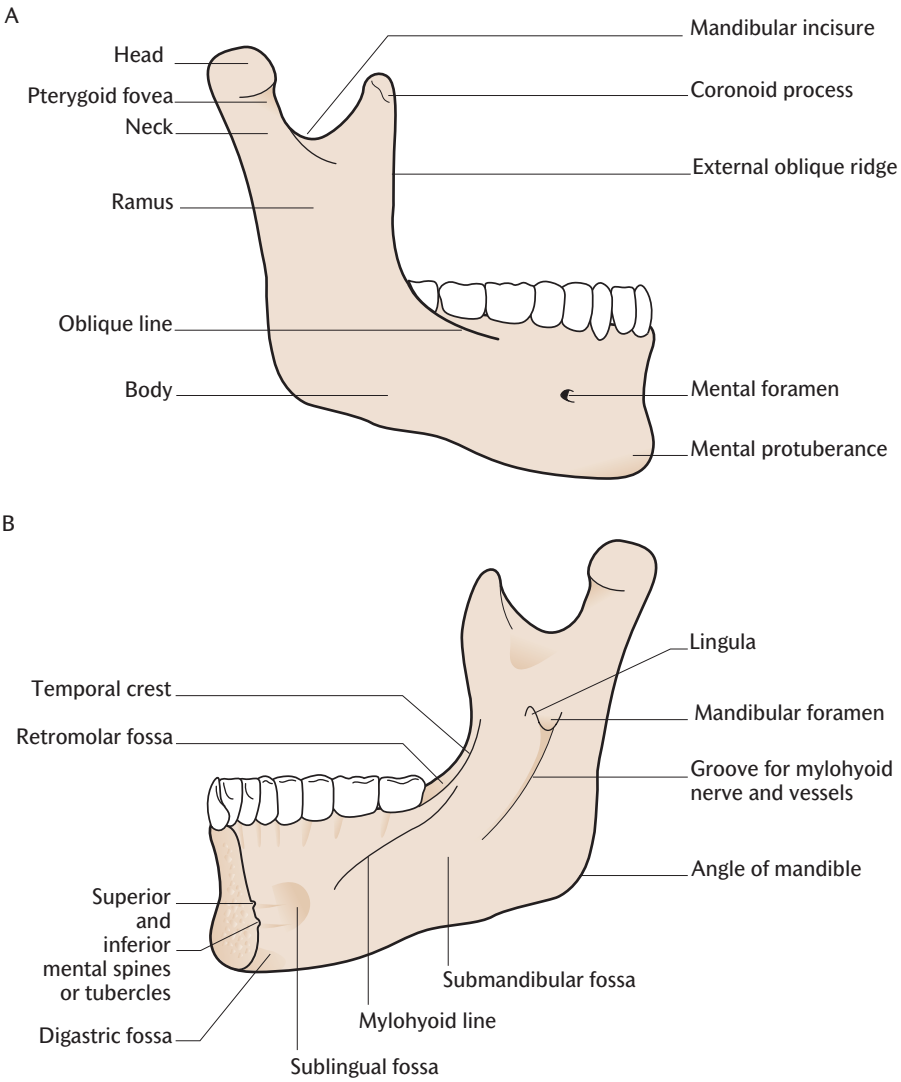


Fig. 22.12 The mandible. A) Lateral view; B) Medial view.

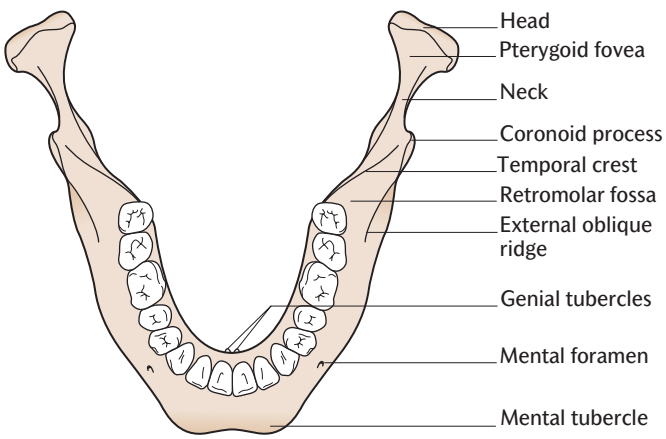


Fig. 22.13 Superior view of the mandible.

the head approximates to a narrow hemicylinder. Viewed from above (Figure 22.13), the condyle is expanded from side to side. As you can see in Figure 22.13, the long axis of each condyle is not in the transverse plane, but is at an angle of about 30° to it with the lateral pole of the head lying more anteriorly than the medial pole. The **neck** of the mandible is the constricted part of the condyle joining the head to the ramus. The **pterygoid fovea** is a shallow depression on its anterior aspect where the lateral pterygoid muscle attaches.

Head injuries are a relatively common feature of modern life usually associated with violent incidents such as road and sporting accidents and criminal assaults; skull fractures are also a common type of war injury. Although dental practitioners are most likely to be called upon to treat fractures of the upper facial skeleton and mandible, they should always bear in mind that other parts of the skull may have been fractured in all such cases. Injury may involve the cranial vault and cranial base as well as the upper facial skeleton or mandible and may be single or

Box 22.1 Fractures of the skull

Fractures of the cranial vault

Fractures of the cranial vault occur most frequently in regions where the bone is thin such as the **pterion**. The bone may split, starting at the site of the trauma and travelling along the line of least resistance or an area of bone may become detached and displaced inwards to produce a **depressed fracture**. Intracranial bleeding resulting from tearing of the blood vessels is a potential serious complication of fractures of the vault. The dural venous sinuses and their tributaries may be involved or the middle meningeal vessels could be damaged if the injury is in the region of the pterion. The resulting haemorrhage is into the extradural or subdural spaces; the effect of bleeding into these areas has already been described in Box 15.3.

The cranial base

Except for the petrous temporal bone, the bone of the cranial base is relatively thin and is frequently involved in split fractures spreading from the cranial vault. The clinical signs and symptoms depend upon the precise site of the fracture and vary according to the proximity of important structures to the injury.

Fractures of the anterior cranial fossa may involve the roof of the orbit, the roof of the nose, and the frontal, sphenoidal, and ethmoidal sinuses. Bleeding into the orbit may produce a black eye and subconjunctival haemorrhage. Fractures involving the nasal cavity and paranasal air sinuses may be accompanied by bleeding into the nose or mouth. If the meninges have been torn, cerebrospinal fluid may leak into the nose or air into the cranial cavity. **Anosmia**, the lack of sense of smell, is a possible long-term effect of anterior cranial fossa fractures because the olfactory nerves are damaged where they pass through the cribriform plate.

Fractures of the middle cranial fossa may be accompanied by bleeding or the leakage of cerebrospinal fluid into the mouth or into the middle ear and external acoustic meatus; facial paralysis and deafness due to damage to the facial and vestibulocochlear nerves may follow.

The glossopharyngeal, vagus, accessory, or hypoglossal nerves may be damaged by fractures of the posterior fossa; the specific signs and symptoms will depend upon which nerves are affected and have been described in Chapter 18.

multiple. Skull damage can also lead to damage to the contents of the skull, including the brain so this possibility has to be considered as well in such cases. Fractures of the cranial vault and base are described in Box 22.1.

Fractures of the upper facial skeleton may be relatively simple, involving a single bone or may be extensive and result in massive dislocation of the facial skeleton. Fractures of the nasal bones and nasal septum and the maxilla alveolar process are examples of the simpler injuries; these are usually produced by blows to the front of the face. Fractures of the middle third of the face involving the maxillae and zygomatic bones are relatively common as are fractures of the mandible. It is necessary to understand muscle insertions and the relationships of other tissues and structures to these bones to appreciate the consequences and potential complications of fractures to these bones. Fractures of the mandible will be, therefore, covered in Chapter 24 (see Box 24.3) where the relevant anatomy is described. Fractures of the middle third of the face and fractures of the maxilla will be included in Chapters 27 and 30 because fractures to this bone may affect structures in the nose or orbit. More complex fractures of the upper facial skeleton are known as Le Fort fractures after the surgeon who first classified them. Le Fort fractures can affect a wide variety of structures so their description is deferred until the anatomy of the head is completed in Chapter 30.

23

The face and superficial neck

Chapter contents

23.1 Introduction

The surface anatomies of the face and neck and their supporting structures that can be palpated have been described in Chapter 20. It is now time to move to the structures that lie under the skin but which cannot be identified by touch starting with the neck and moving up on to the face and scalp.

23.2 The neck

23.2.1 The cervical vertebral column

The cervical vertebral column comprises the seven cervical vertebrae and the intervening intervertebral discs. These have the same basic structure as the thoracic vertebrae described in Section 10.1.1. Examine the features of the cervical vertebra shown in Figure 23.1 and compare it with the thoracic vertebra shown in Figure 10.3. You will see that cervical vertebrae have a small body and a large vertebral foramen. They also have two distinguishing features, a bifid spinous process and a **transverse foramen**, piercing each transverse process; the vertebral vessels travel through these foramina.

The first and second vertebrae are modified. The first vertebra, the **atlas**, has no body. Instead, it has two lateral masses connected by anterior and posterior arches. The lateral masses have concave superior facets which articulate with the **occipital condyles** where nodding movements of the head take place at the **atlanto-occipital joints**. The second cervical vertebra, the **axis**, has a strong **odontoid process** (or **dens** because of its supposed resemblance to a tooth) projecting upwards from its body. This process is, in fact, the body of the first vertebra which has fused with the body of the axis instead of being incorporated into the atlas. The front of the dens articulates with the back of the anterior arch of the atlas; rotary (shaking) movements of the head occur at this joint. The seventh cervical vertebra has a very long spinous process which is easily palpable.

The primary curvature of the vertebral column is concave forwards and this persists in the thoracic and pelvic regions. In contrast, the cervical and lumbar parts of the vertebral column are convexly curved anteriorly. These anterior curvatures are secondary curvatures which appear in late fetal life. The cervical curvature becomes accentuated in early childhood as the child begins to support its own head and the lumbar curve develops as the child begins to sit up. There is only a small degree of movement between adjacent cervical vertebrae. However, the cumulative effect is considerable and gives the neck its characteristic flexibility. When the neck is fully flexed, the cervical vertebrae are in a straight line.

23.2.2 Cervical fascial layers

The layers of connective tissue that form compartments between tissues or enclose muscles, for example, are impermeable to fluids such as blood and pus. Pathological fluids spread through the spaces between the fascial layers rather than piercing the fascial planes and infected material is able to track considerable distances from its point of origin.

Knowledge of the fascial planes and spaces used to be of considerable clinical significance for correct diagnosis and treatment of infective and inflammatory conditions. The accumulation of large quantities of pus from dental infections is now an uncommon event in developed

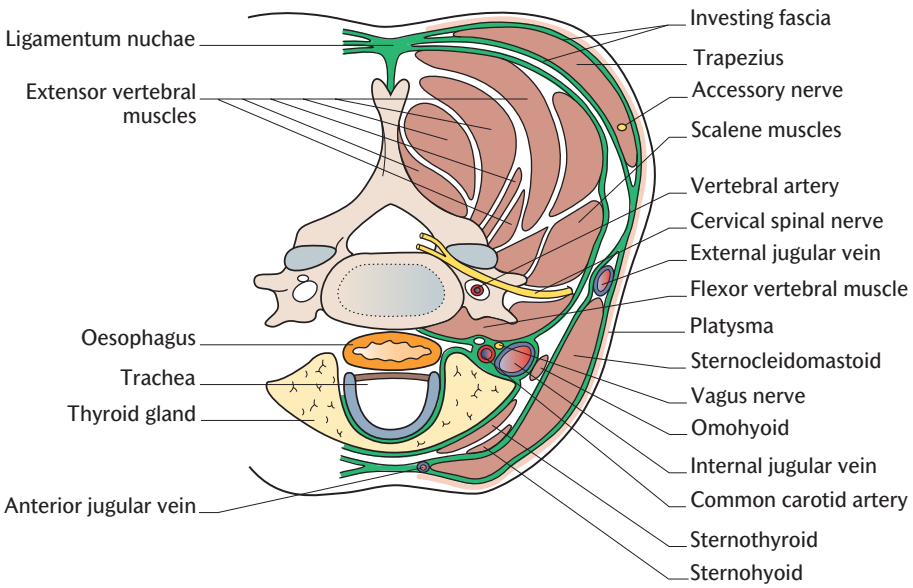


Fig. 23.1 The arrangement of the deep fascia of the neck shown on a horizontal section at the level of the sixth cervical vertebra.

Box 23.1 Fascial planes in the neck

Superficial fascia

The superficial fascia is the subcutaneous layer of loose connective tissue. It does not usually contain such large amounts of fat as the equivalent layer in the trunk but there may be as much in some individuals.

Deep fascia

As shown in Figure 23.1, the deep fascia comprises the layers of connective tissue surrounding the deeper structures of the neck. The **investing layer** of the deep cervical fascia is arranged like a cylinder around the neck, lying immediately deep to the superficial fascia. It attaches inferiorly to the scapula, clavicle, and the manubrium sterni. Posteriorly, its upper attachment is to the external occipital protuberance, superior nuchal line, and mastoid process of the skull; the fascia splits to enclose the attachments of the trapezius and sternocleidomastoid muscles. It also splits into two layers to form the **parotid capsule**, enclosing the parotid gland between the mastoid process and the posterior border of the mandibular ramus. Anteriorly, the investing layer is attached to the lower border of the mandible.

The **prevertebral fascia** is a tough membrane covering the anterior aspect of the flexor muscles of the cervical spine. It extends from the base of the skull downwards to the third thoracic vertebra. Laterally, it passes in front of the lateral vertebral (scalene) muscles and continues backwards, enveloping the extensor vertebral muscles. The **carotid sheath** of each side consists of a tube of connective tissue surrounding the common carotid artery and internal carotid artery above the bifurcation, the internal jugular vein, and vagus nerve. The sheath is attached to the base of the skull around the opening of the carotid canal and continues downwards along the common carotid artery to fuse with the connective tissue around the arch of the aorta.

countries owing to the availability of early treatment and the widespread use of antibiotics. Nevertheless, a knowledge of the fascial planes in the neck and their connections with tissue spaces in the floor of the mouth (see Section 25.3.4) is still of value to dentists practising in areas where primary treatment and antibiotics are not widely available. Fasciae are remarkably difficult to distinguish on conventionally preserved human cadavers. The fascial planes are described in Box 23.1.

23.2.3 Cervical muscle groups

There are six main groups of neck muscles, the **superficial muscles**, the **suprahyoid** and **infrahyoid muscles**, and the **flexors, extensors, and lateral vertebral muscles of the cervical spine**. Only the superficial and suprahyoid and infrahyoid muscles will be described as the other groups have no direct relevance to clinical dentistry. The arrangement of the flexor, extensor, and lateral vertebral muscles groups can be seen in Figure 23.1.

Superficial muscles

Platysma

The **platysma** is the first muscle you will encounter beneath the skin if you have the opportunity to dissect a human cadaver. Each muscle is a broad sheet arising from the fascia covering the pectoralis muscles on the front of the thorax. Their fibres sweep upwards and medially in the superficial fascia across the clavicle and into the side of the neck as shown in Figure 23.1. Figure 23.8 shows their superior attachments into the lower border of the mandible and into the skin and muscle of the lower lip, especially towards the angle of the mouth. The platysma muscles belong to the muscles of facial expression (see Section 23.3.2) and are innervated by the cervical branches of the facial nerve. The muscles vary in size and may be absent. Platysma tightens the skin of the neck. If present, the fibres inserting into the lip depress the corners of the mouth.

Sternocleidomastoid

We have already encountered the bulky strap-like sternocleidomastoid muscle when considering the surface anatomy in Chapter 20. As shown in Figure 20.8, it passes obliquely across the side of the neck, dividing the anterior and posterior triangles. Figure 23.8 illustrates its attachments to the manubrium sterni by a tendinous **sternal head** and to the upper surface of the medial third of the clavicle by a fleshy **clavicular head**; the two heads blend as they pass upwards. The muscle is attached superiorly to the lateral aspect of the mastoid process and the lateral part of the superior nuchal line. It receives its motor innervation from the spinal accessory nerve. When the muscle contracts, the mastoid process is brought towards the sternum. When the two muscles act together, they flex the neck but at the same time extend the head; the head is drawn forwards, but the face remains vertical. If only one muscle is active, the back of the head is drawn towards the shoulder of the same side, thus turning the face towards the opposite side and tilting it upwards.

Trapezius

Again this is a muscle already met in Chapter 20 (see Figure 20.8). Each muscle is a large flat triangular muscle; the muscles of the two sides form a trapezium from which the name of the muscle is derived. Each muscle is attached from the superior nuchal line and external occipital protuberance above and the ligamentum nuchae between the extensor muscles and the spinous processes of the seventh cervical and all the thoracic vertebrae. Figure 23.8 shows the superior fibres in the neck pass downwards and laterally to attach to the lateral third of the clavicle. The inferior fibres running laterally from the posterior thoracic wall converge and attach into the scapula. This muscle is also innervated by the spinal accessory nerve. The muscle mainly functions to control the position of the scapula working with other thoracic and upper limb muscles. When the scapula is fixed, the upper fibres in the neck extend the head and neck.

The accessory nerve

The origins and intracranial course of the **spinal accessory nerves** have already been described in Section 18.11. Each nerve exits through the jugular foramen and slopes downwards and backwards to the upper part

of the sternocleidomastoid muscle. It runs through the muscle to emerge in the posterior triangle which it crosses to enter the trapezius muscle.

The suprahyoid muscles

This group is made up of four pairs of muscles, the **mylohyoid, geniohyoid, stylohyoid**, and **digastric muscles**. They all attach to the hyoid bone below and to the mandible, styloid, and mastoid processes above. The muscles are illustrated in Figure 23.2 which should be examined as you read the following description.

The **mylohyoid** muscle passes medially from the **mylohyoid line** of the mandible to attach to the hyoid bone posteriorly and to meet its opposite number at the **median raphe**; the anterior components of the muscles form the floor of the mouth. Each **digastric muscle** has two muscle bellies joined by a tendon. The anterior belly is attached to the **digastric fossa** on the inside of the mandible body close to the midline and runs backwards towards the hyoid bone. The posterior belly attaches to the mastoid notch and runs forward towards the hyoid bone. As you can see in Figure 23.2, the two bellies are joined by the intermediate tendon which runs through a fibrous sling attached to the hyoid bone. The entire muscle thus forms a shallow V. The **stylohyoid** muscles are largely covered by other muscles and the **geniohyoid** muscles run deep to the mylohyoid so are not superficial structures. These muscles are described as part of the floor of the mouth in Chapter 25.

The suprahyoid muscles elevate the hyoid bone and laryngeal skeleton during swallowing; they also play a role in opening the mouth when they act from a fixed hyoid bone to pull down on the mandible (see Chapter 26). Their nerve supply is complicated because different muscles arise from different pharyngeal arches (Table 21.1). The mylohyoid and anterior digastric are first arch muscles and are, therefore, innervated by the mandibular trigeminal nerve. The stylohyoid and posterior digastric are second arch derivatives and therefore obtain their motor innervation from the facial nerve. The embryology of the geniohyoid is uncertain; it is innervated by the first cervical nerve.

The infrahyoid muscles

The **sternohyoid, sternothyroid, thyrohyoid, and omohyoid muscles** make up this group of four pairs of strap-like muscles running from the hyoid to the sternum and clavicle via the laryngeal skeleton. The muscles can be traced in Figure 23.2. The two parts of the name of each muscle tell you their attachments, e.g. sternohyoid attaches to the sternum and hyoid. They depress the hyoid bone and laryngeal skeleton on completion of swallowing. They also fix the hyoid so that the suprahyoid muscles can act on the mandible using the hyoid as a fixed attachment. They are supplied by branches of the cervical spinal nerves through the cervical plexus (see Section 23.1.4). Further details of this muscle group are provided in Box 23.2 for those that require it.

The **lateral vertebral muscles**, the anterior, medius, and posterior **scalene muscles** shown in Figure 23.1, attach to the transverse processes of the cervical vertebrae and run downwards and outwards to the upper

Box 23.2 The infrahyoid muscles

The **sternohyoid muscles** are the most superficial and run from the manubrium sterni and adjacent end of the clavicle to the body of the hyoid bone. They cover the sternothyroid and thyrohyoid muscles. The **sternothyroid** runs from the manubrium sterni to the oblique line on the thyroid cartilage. The **thyrohyoid** muscle continues this layer upwards from the oblique line to the lower border of the greater cornu and body of the hyoid bone. Each **omohyoid** muscle consists of two bellies. The inferior belly arises from the scapula and passes forwards across the lower part of the neck deep to sternocleidomastoid. The superior belly attaches to the body of the hyoid lateral to sternohyoid and runs directly downwards where it attaches to the intermediate tendon linking it to the inferior belly. The intermediate tendon is held in place by a fibrous loop attached to the clavicle and first rib.

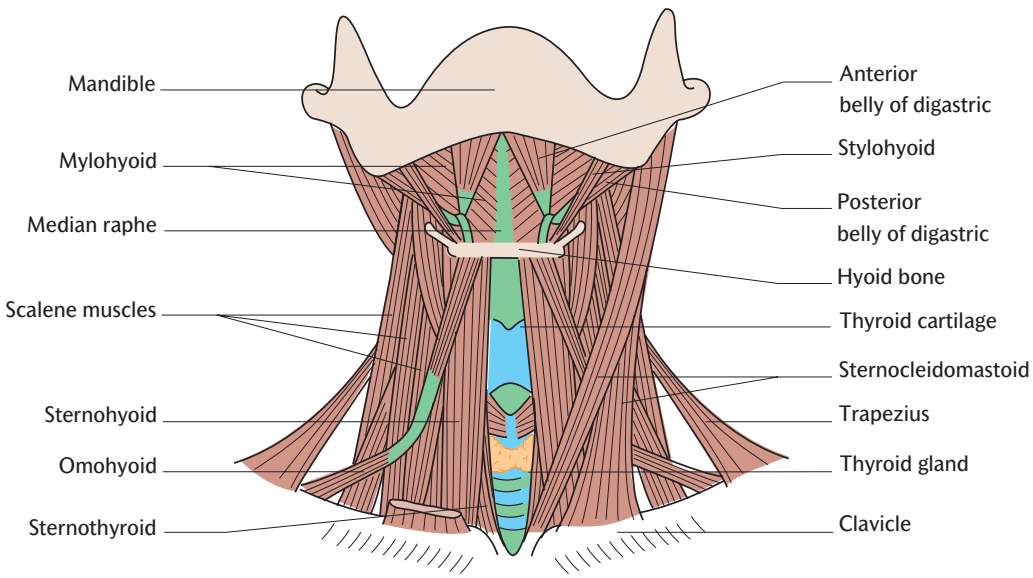


Fig. 23.2 The infrahyoid muscles and some of the suprahyoid muscles. The right sternocleidomastoid has been removed for clarity.

two ribs. They laterally flex the cervical vertebrae as their name implies, but can act as accessory muscles of inspiration (see Section 11.4.2).

The posterior triangle of the neck

The borders of **posterior triangles** have already been described in Chapter 20 and illustrated in Figure 20.8. The accessory nerve crosses the triangle almost vertically from the posterior border of sternocleidomastoid to the anterior border of trapezius. The four superficial branches of the cervical plexus (see below) emerge from the posterior triangle around the posterior edge of sternocleidomastoid.

23.2.4 Cervical spinal nerves

There are eight pairs of cervical spinal nerves. The first to seventh cervical nerves leaves the vertebral canal by passing *above* the vertebra of the same number; the first nerve emerges between the occipital bone and the atlas. The eighth nerve emerges between the seventh cervical and the first thoracic vertebrae. Each nerve, like all spinal nerves, then divides into a dorsal and ventral ramus.

The dorsal rami run posteriorly to supply the extensor muscles of the neck and the skin over the back of the neck and scalp. The dorsal ramus of each second cervical nerve forms a large cutaneous branch, the **greater occipital nerve**, which supplies the posterior aspect of the scalp as far as the vertex. The dorsal rami of the remaining cervical nerves have small cutaneous branches to the back of the neck.

The ventral rami of the first four cervical spinal nerves (C1–4) join to form the **cervical plexus**. The ventral rami of the remaining four cervical nerves and the first thoracic nerve (C5–T1) join to form the **brachial plexus**, the nerve supply to the skin and muscles of the upper limb.

The cervical plexus

The four ventral rami branch and rejoin as illustrated in Figure 23.3 to produce the cervical plexus. Deep branches from the plexus supply motor axons to the infrahyoid muscle group and some other muscles. The most important motor branch is the **phrenic nerve** to the diaphragm formed from the third, fourth, and fifth cervical nerves. Superficial branches are distributed to the skin of the front and sides of the neck and part of the side of the head. Further details of the cervical plexus are provided in Box 23.3 if your curriculum demands them.

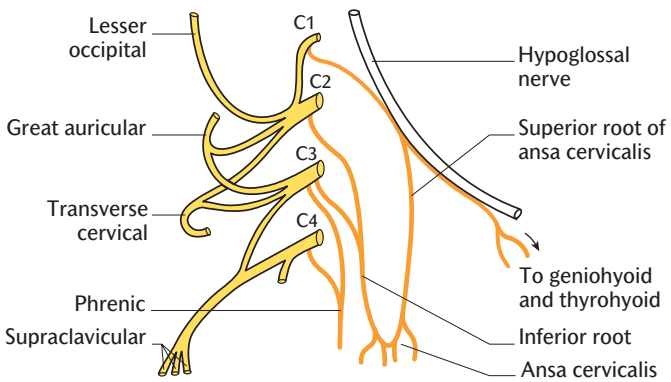


Fig. 23.3 The cervical plexus. Sensory branches are shown in yellow and motor branches in orange.

23.2.5 Arteries in the neck

The common carotid arteries and vertebral arteries pass through the neck on each side to supply the head, including the brain as already described in Section 15.5. The carotid arteries arise from the aortic arch in the thorax and the vertebral arteries are branches of the subclavian arteries. The common carotid arteries divide high in the neck into the external and internal carotid arteries. Branches from the vertebral and external carotid arteries supply the neck itself.

Box 23.3 The cervical plexus

The deep branches are:

- **A communicating branch** from the first cervical nerve to the hypoglossal nerve carrying motor axons to the geniohyoid and infrahyoid muscles. The nerves supplying the geniohyoid and thyrohyoid run to their destination in the hypoglossal nerve. Axons to the remaining infrahyoid muscles leave the hypoglossal nerve in the **superior root of the ansa cervicalis branch** (also called the **descendens hypoglossi**) which passes along the front of the carotid sheath to join the inferior root to form the **ansa cervicalis**.
- **Muscular branches** pass from all four cervical nerves to the prevertebral muscles.
- **Proprioceptive branches** pass to the sternocleidomastoid and trapezius muscles, respectively.
- The **inferior root of the ansa cervicalis** (or **descendens cervicalis**) from the second and third cervical nerves joins the superior root nerve to form the **ansa cervicalis**. Branches of the latter supply the infrahyoid muscles, except thyrohyoid.
- The **phrenic nerve**, derived mainly from the fourth cervical nerve with small additions from the third and fifth nerves, runs vertically downwards over the anterior scalene muscle and then passes behind the subclavian vein to enter the mediastinum. Its further course is described in the Section 10.2.4.

The superficial branches of the cervical plexus hook around the posterior border of sternocleidomastoid and radiate outwards to supply skin over the neck, a small area of scalp, and an area of the face. Their distribution is shown in Figure 23.12.

- The **lesser occipital nerve** derived from the second cervical nerve runs upwards along the posterior border of sternocleidomastoid to supply to skin of the scalp behind the auricle.
- The **great auricular nerve** from the second and third cervical nerves runs upwards to supply the skin of the auricle and of the face over the parotid gland and angle of the mandible.
- The **transverse cervical nerve**, another derivative from the second and third nerves, runs forwards to supply the skin of the side and front of the neck.
- The **supraclavicular nerves**, derived from the third and fourth cervical nerves, pass downwards and fan out to supply skin over the chest and shoulder.

The common carotid artery

The origin of the right and left common carotid arteries is different on each side. On the right side, the brachiocephalic trunk divides after a short course into the right common carotid and subclavian artery whereas the left common carotid and subclavian arteries are direct branches of the aortic arch (see Chapter 12, Figure 12.10).

Each common carotid artery leaves the thorax and ascends through the neck alongside the corresponding internal jugular vein and vagus nerve. These three structures are bound together by the **carotid sheath**. As illustrated in Figure 23.4, the common carotid arteries bifurcate into the internal and external carotid arteries; the level of bifurcation is usually about the level of the upper border of the thyroid cartilage or hyoid bone. The internal carotid artery continues upwards within the carotid sheath whereas the external carotid artery leaves the sheath and thus becomes external to it.

The **carotid sinus** is a dilatation on the common carotid artery at its bifurcation which continues a little way up the internal carotid branch. The carotid sinus contains baroceptors monitoring blood pressure innervated by the carotid branch of the **glossopharyngeal nerve**. The **carotid body** is usually situated behind the sinus or between the internal and external carotid arteries and is only a few millimetres in width. It contains chemoreceptors responding to oxygen and carbon dioxide levels in the blood and is also innervated by the glossopharyngeal nerve. Its carotid branch forms the afferent arc of cardiovascular and respiratory reflexes in response to altered blood chemistry or pressure.

The internal carotid artery

The internal carotid arteries are shown in Figure 23.5. They ascend on either side of the pharynx without branching to enter the base of the skull through the **carotid canals**. Each artery follows an S-shaped course through the canal, tracking superiorly at first, then turning medially before resuming its superior course to enter the middle cranial fossa just above the foramen lacerum. The internal carotid arteries terminate by branching into the anterior and middle cerebral arteries which are described in Chapter 15 and illustrated in Figure15.24.

The external carotid artery

Each **external carotid artery** ascends through the upper part of the neck supplying numerous branches to adjacent structures. They are illustrated in Figure 23.4; follow their course as you read the description. Each external carotid artery enters the parotid gland at about the level of the angle of the mandible and terminates within the substance of the parotid glands by dividing into two terminal arteries. Each external carotid artery has eight branches; some of these are of little direct importance to clinical dental practice.

The **superior thyroid arteries** branch off almost immediately after the bifurcation, then descend and spread out to supply the anterosuperior aspects of the thyroid gland. They also supply small penetrating branches that supply the larynx.

The **ascending pharyngeal arteries** arise at about the same level as the superior thyroid arteries but are hidden on the medial aspect of each external carotid artery; they are, therefore, not visible in Figure 23.4. These arteries ascend on either side of the pharynx and supply it as they do so. They terminate by contributing to the blood supply of the palatine tonsils.

The lingual and facial arteries may arise separately or from a short common stem. As their names imply, they supply the tongue and face, respectively. The **lingual arteries** pass deep to the mandible and cross the floor of the mouth to ascend into the tongue. They supply the tongue and the floor of the mouth and their course here is described in

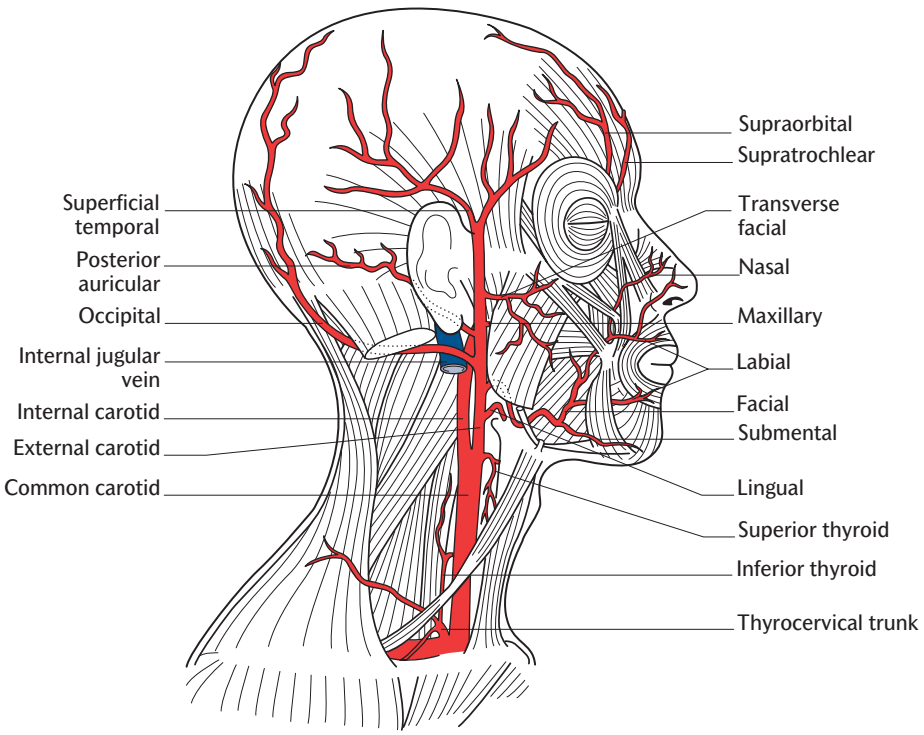


Fig. 23.4 The carotid arteries and their branches.

Section 25.3.4. The course of the **facial artery** is described in Section 23.2.1. The lingual and facial arteries also supply the bone of the mandible through periosteal branches along their course.

The **occipital arteries** supply skin over the back of the neck and scalp. The **posterior auricular arteries** supply the scalp behind the ears and have a **stylomastoid branch** that enters the stylomastoid foramen to supply the tympanic cavity.

The two terminal branches of the external carotid arteries are the superficial temporal and maxillary arteries on each side. The **superficial temporal arteries** emerge from the parotid glands and branch extensively over the scalp. The **maxillary arteries** lie deeply and about one-third of their course is within the maxillary bone. Each artery has no less than15 branches but most are tiny and trivial.

Each maxillary artery exits the parotid gland to enter the infratemporal fossa where it usually runs deep to the **lateral pterygoid muscle** before entering the maxilla through the pterygopalatine fossa. The maxillary artery is traditionally divided into three parts for descriptive purposes; the first third before it is crossed by the muscle, the second as it is crossed, and the third part after the muscle. Fifteen divided by five is three and very conveniently for those who do need to remember such things, there are five branches from each part. The middle five branches supply the muscles of mastication and the distal five branches follow the branches of the maxillary division of the trigeminal nerve. The branches of the maxillary arteries are described in Section 24.5.2.

The most important branches of the maxillary arteries are the middle meningeal and inferior alveolar arteries, both of which arise from the first part of the artery. The **middle meningeal arteries** enter the cranial cavity to supply the skull and dura and are described in Section 15.1.5. The **inferior alveolar arteries** supply the mandibular dentition and vary in size with the state of development of the dentition. The arteries are quite well-developed during childhood and early adolescence as the primary and secondary dentitions develop and erupt. They shrink considerably once the dentition is complete once only a maintenance blood supply is required. In an edentulous patient, each artery may be almost invisible as there are no teeth to supply and the inferior alveolar arteries contribute little, if anything, to the blood supply of the mandible.

Branches of the subclavian arteries

Some branches of the subclavian arteries contribute to the blood supply of the head and neck.

The **thyrocervical trunk** branches from the subclavian artery close to the medial border of the anterior scalene muscle and divides into three branches. Only one of these branches, the **inferior thyroid artery**, is important. It runs upwards to supply the lower posterior aspect of the lobe of the thyroid gland and the larynx and trachea through its inferior laryngeal branch.

The **vertebral arteries** are the most important derivatives of the subclavian arteries and have already been encountered in Chapter 15. The vertebral artery on each side arises from near the root of the subclavian artery as shown in Figure 23.5. It runs upwards through the foramina in the transverse processes of the upper six cervical vertebrae, then passes behind the lateral mass of the atlas to enter the cranial cavity through the foramen magnum. The two vertebral arteries unite within the cranial cavity to form the **basilar artery** at the lower border of the pons. The branches of the vertebrobasilar system are described and illustrated in Section 15.5.1.

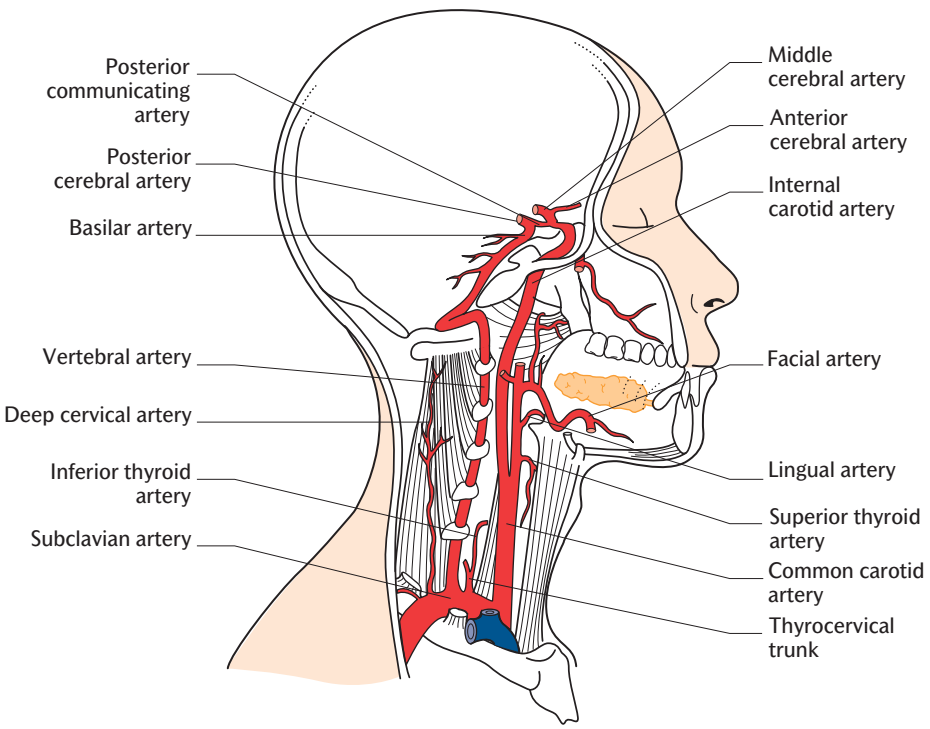


Fig. 23.5 The vertebral artery and cervical branches of the subclavian artery.

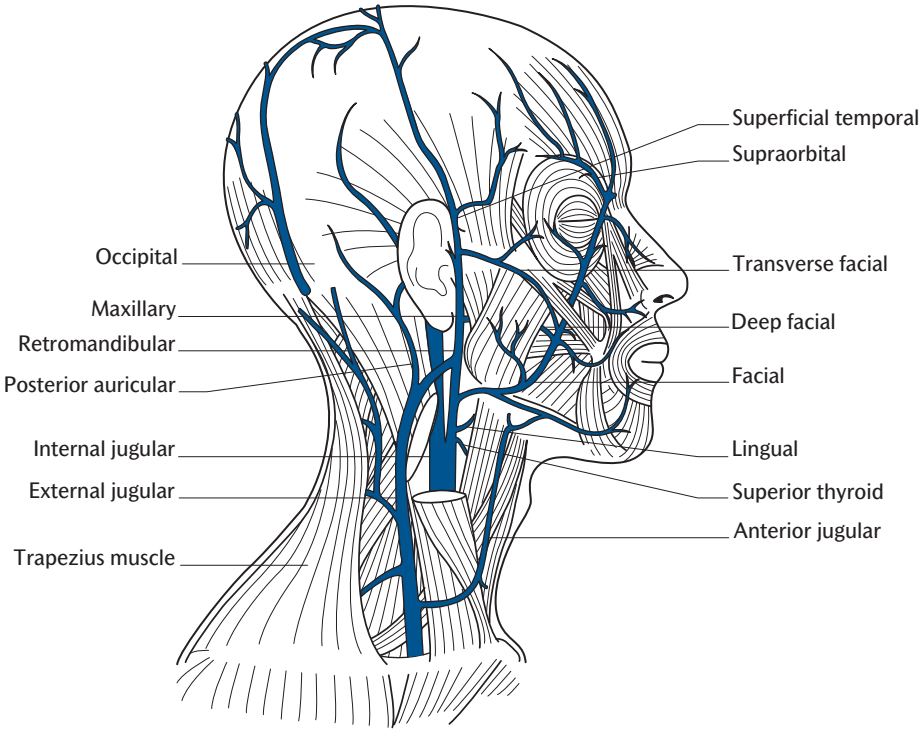


Fig. 23.6 The veins of the head and neck.

23.2.6 Veins in the neck

As in the rest of the body, the veins in the head and neck lie at two levels. The **deep veins** draining deep structures tend to follow the same course as the corresponding arteries and are consistent in position. The **superficial veins** draining the skin and subcutaneous tissues often do not correspond to arteries; they may or may not be present and are very variable in size and position. The deep and superficial veins communicate extensively with each other and with the intracranial **venous sinuses** through **emissary veins**. The internal jugular veins are the major veins of the deep system draining the head and neck and receive many other veins during their course.

The **internal jugular veins** and their branches are illustrated in Figure 23.6 which should be studied as you read the description. The internal jugular veins emerge from the jugular foramina to run at first behind, then lateral to the internal carotid arteries in the carotid sheaths. The **vagus nerve** lies between the vein and the artery on each side. In the lower part of their course, they are covered by the sternocleidomastoid muscles. The internal jugular veins join the subclavian veins to form the **brachiocephalic veins** in the root of the neck.

As you can see in Figure 23.6, the internal jugular veins receive numerous tributaries; their arrangement can show considerable individual variation. The more important tributaries are the **facial** and **lingual veins**, the **pharyngeal veins** from the pharyngeal plexus that drains the pharynx (see Section 28.2.4), and the **superior thyroid veins** from the upper part of the gland.

The superficial veins are much more variable. As shown in Figure 23.6, the **external jugular vein**s are formed by the union of the posterior division of the **retromandibular veins** and the **posterior auricular veins**. They run downwards superficial to the sternocleidomastoid muscle, pierce the deep cervical fascia above the middle of the clavicle, and drain into the subclavian veins. As noted in Chapter 20, the external jugular veins are very variable in size and may even be absent. The **anterior jugular veins** begin beneath the chin on each side of the midline and passes downwards to the suprasternal region where they empty into the external jugular veins. The anterior jugular veins are absent in many people.

23.2.7 The cervical sympathetic trunks

As mentioned in Chapter 17, the sympathetic trunks continue upwards from the thorax and ascend in the neck as the **cervical sympathetic trunks**, lying between the prevertebral fascia and the carotid sheath to end in front of the lateral mass of the atlas.

Figure 23.7 illustrates the three ganglia on each cervical trunk, the superior, middle, and inferior ganglia. The **superior ganglion** is formed by fusion of the ganglia originally associated with the upper four cervical nerves; it is the largest of the three ganglia and lies in front of the transverse process of the axis. The smallest **middle ganglion** represents the ganglia of the fifth and sixth cervical nerves. The inferior ganglion is the product of fusion of the ganglia of the remaining two cervical nerves; it is often fused with the first thoracic ganglion to form the **stellate** (cervicothoracic) **ganglion** close to the neck of the first rib. The **internal carotid nerve** is an upward continuation of the trunk from the superior ganglion, forming a plexus around the internal carotid artery to deliver a sympathetic nerve supply to intracranial vessels.

Recall from Chapter 17 that sympathetic nerves originate from the CNS as the thoracolumbar outflow; the cervical spinal nerves, therefore, do not carry sympathetic nerves. All sympathetic preganglionic axons that synapse in the three cervical ganglia of the cervical sympathetic

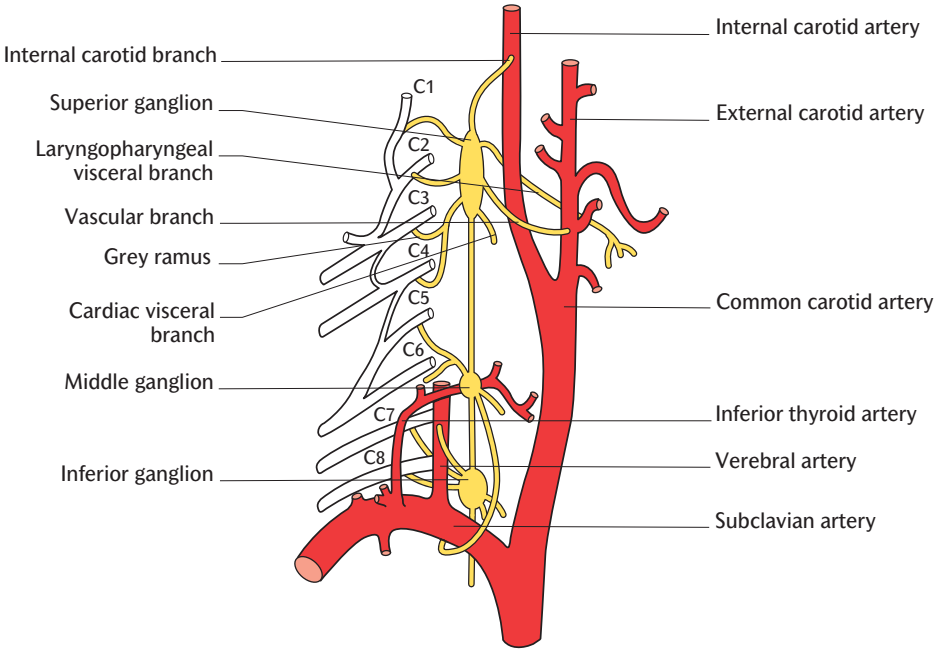


Fig. 23.7 Branches of the cervical sympathetic ganglia.

trunk originate from the upper thoracic segments (T1 to T4). The post-ganglionic axons pass from the ganglia to their destination by various routes.

- Grey rami communicantes pass to cervical spinal nerves to supply blood vessels, sweat glands, and arrector pili muscles in the skin innervated by each nerve.
- Vascular branches from the ganglia form networks which pass along neighbouring arteries to reach their destination; these nerves also have a vasoconstrictive effect on the vessels that they travel along.
- A visceral branch passes from each ganglion to the cardiac plexus to provide the sympathetic innervation of the heart.

The major vascular output is from the superior ganglion to the common and external carotid arteries and their branches. The superior ganglion also gives rise to **laryngopharyngeal visceral branches** which enter the pharyngeal plexus. Vascular branches of the middle ganglion travel along the subclavian and inferior thyroid arteries to the larynx, trachea, and oesophagus. Those of the inferior ganglion accompany the vertebral artery and its branches.

23.3 The face

As you will recall from Chapter 20, there is very little subcutaneous adipose tissue on the face. The tissue between the skin and bones of the face are made up of muscles of facial expression and the superficial muscles of mastication. There are also arteries and veins, motor nerves to the facial muscles, and cutaneous nerves to the skin and the parotid salivary glands.

23.3.1 Blood supply

The face is supplied principally by the facial and superficial temporal arteries, both branches of the external carotid artery. Other smaller branches also supply the face.

The facial artery

The course and distribution of the facial artery is illustrated in Figure 23.4. The **facial artery** runs deep to the submandibular glands, then arches over their superior aspect between the gland and the inferior border of the mandible to enter the face. This crossing point is just anterior to the anterior border of masseter; with care, a pulse can be detected as the artery crosses the mandible. Each artery ascends diagonally across the face, passing just lateral to the corner of the mouth and then the nostrils to reach the medial canthus of the eye. Several small branches run approximately horizontally from the main trunk to supply the lower and upper lips and nose. The artery is crossed by the branches of the facial nerve. The consequence of injuries to the facial arteries are described in Box 23.4. In human cadavers the facial artery often follows a tortuous course; the reasons for this are outlined in Box 23.5.

Box 23.4 Facial artery injuries

The facial arteries are relatively superficial therefore facial trauma may produce profuse bleeding. It may be difficult to compress the artery to arrest bleeding without exacerbating the injury. However, bleeding can be arrested by compressing the artery as it crosses the inferior border of the mandible, the same point used to detect its pulse.

Box 23.5 Is the facial artery really as kinky as pictures show?

Look at the facial artery in Figure 23.4 and you will see it has a tortuous course across the face. Many anatomy textbooks claim the facial artery has this wavy course to allow the artery to adapt to opening of the mouth without compromising the facial blood supply. This is nonsense. If you have the opportunity to observe the facial artery on a cadaver, it will, in all likelihood, have a tortuous course; that is why it appears that way in Figure 23.4 because it was drawn from a dissection. If you could visualize your own facial artery on an angiogram, you would see that it runs relatively straight between the mandible and eye. The majority of those who generously donate their bodies for medical study are elderly. Arteries lose some of their elasticity with age and kink as a result, which explains why the wiggly course is observed in cadavers but not on angiograms of younger people. The loss of elasticity is not unique to the facial arteries; it happens to most arteries with age.

The superficial temporal artery

The **superficial temporal arteries** are the smaller of the two terminal branches of the external carotid arteries. Follow their course in Figure 23.4. They begin in the parotid gland immediately behind the neck of the mandible and run upwards across the posterior root of the zygomatic arch. They then divide into anterior and posterior branches that supply the frontal and parietal regions of the scalp respectively. They are accompanied in the scalp by the corresponding veins and branches of the auriculotemporal branch of the mandibular trigeminal nerve. Each artery gives off several branches before dividing into its terminal branches; these are described in Box 23.6 for those who need this level of detail.

Terminal branches of arteries accompany the nerves emerging through foramina on the face to supply cutaneous innervation of the face (see Section 23.2.7); they supply blood to local areas of the face. Supraorbital and supratrochlear arteries arise from the ophthalmic artery within the orbit. They hook around the superior orbital rim to supply the forehead. Blood also reaches the face through the infraorbital and mental arteries.

Box 23.6 Branches of the superficial temporal arteries

- The **transverse facial artery** which arises in the parotid gland and runs forwards across the masseter muscle, supplying the gland and its duct and the masseter muscle.
- Branches to the external ear.
- The **zygomatic artery** which runs along the upper border of the zygomatic arch towards the lateral angle of the eye, supplying adjacent muscles.
- The **middle temporal artery**, which arises above the zygomatic arch, pierces the temporal fascia and supplies, together with the deep temporal branch of the maxillary artery, the temporalis muscle.

Facial veins

Most of the blood from the face returns by superficial veins which are illustrated in Figure 23.6.

The forehead is drained by veins which pass downwards towards the inner angle of the eye where they fuse to form the **facial vein**. This vein runs downwards and backwards just posterior to the facial artery and receives tributaries corresponding to the branches of the facial artery. The facial vein crosses the lower border of the mandible just anterior to the masseter muscle attachment, passes through the deep fascia, and enters the neck to join the anterior division of the retromandibular vein . The facial vein drains into the internal jugular vein. The **deep facial vein** draining the **pterygoid plexus** (see Section 24.5.3) also joins the facial vein.

Numerous tributaries draining a wide area of the scalp form the **superficial temporal vein** which then enters the parotid gland. It is joined by the **transverse facial vein** from the upper part of the side of the face. The superficial temporal vein unites with the **maxillary vein** within the parotid gland to form the retromandibular vein.

The **retromandibular vein** descends within the parotid gland deep to the facial nerve but superficial to the external carotid artery. The vein splits into anterior and posterior divisions, either while still within the gland or just after emerging from it. The anterior division passes forwards to join the facial vein. The posterior division continues more directly downwards and is joined by the posterior auricular vein, either within or just outside the gland to form the **external jugular vein**.

23.3.2 The muscles of facial expression

The **muscles of facial expression** produce the rapid changes in facial appearance which occur in response to alterations in mood and emotions; they play a very important role in non-verbal communication. They also regulate the size of the orifices with which they are associated. The muscles of facial expression are derived from the mesoderm of the second pharyngeal arch and, therefore, receive their motor innervation from the facial nerve (Chapter 21, Table 21.1). Many of these muscles are attached into the dermis of the skin with muscle fibres anchored to the adjacent bones of the facial skeleton.

The many muscles making up this group all have individual names and attachments, but there is no practical reason for memorizing that level of detail. It is of more value to identify *functional muscle groups*, each composed of constrictors and dilators around each of the facial openings. They will be described from above downwards, starting with those round the eye and finishing with those around the mouth. The important muscles are illustrated in Figure 23.8 which should be referred to as you read the following description.

Muscles of the eyelids

Orbicularis oculi, a circular muscle, surrounds each orbit and acts as the sphincter of the eye. As shown in Figure 23.8, each muscle has two parts. The **palpebral part** is confined to the eyelids and closes the eyes gently as in blinking. The **orbital part** of the muscle is much larger and surrounds the palpebral part; its fibres run in concentric loops and many insert into the skin of the eyebrow. The orbital part produces more forcible closure, as in 'screwing up' the eyes against a bright light. Reflex closing of the eyes usually involves just the palpebral component.

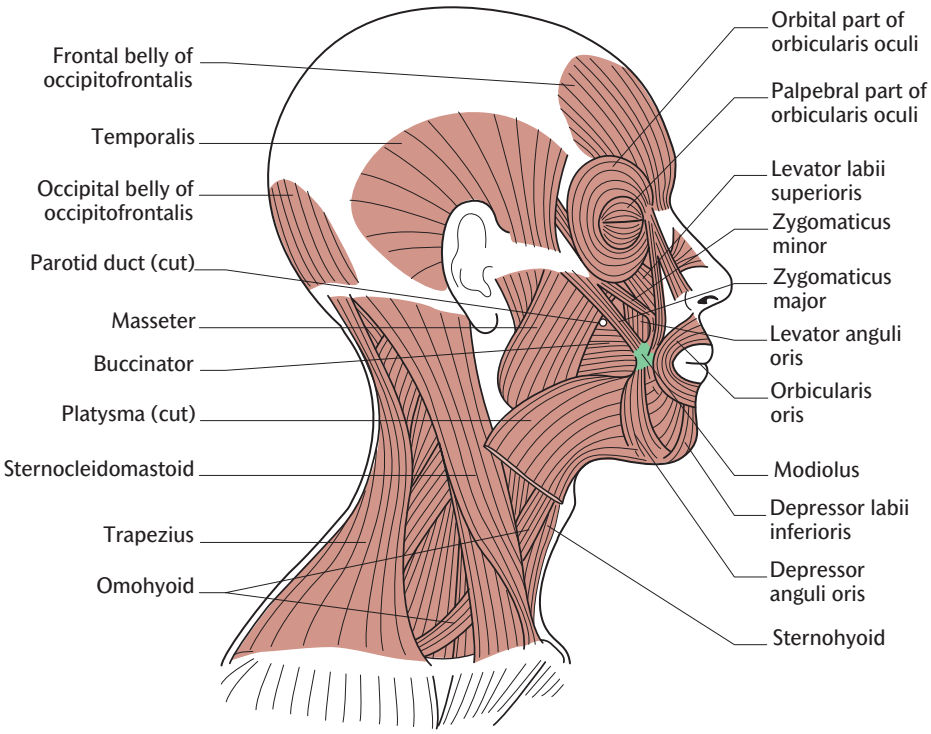


Fig. 23.8 The muscles of facial expression and other superficial muscles of the head and neck.

As you can see in Figure 23.8, the **occipitofrontalis** has two muscle bellies on each side; these are joined by the **epicranial aponeurosis**, a sheet of fibrous connective tissue. Each occipital belly is attached to the superior nuchal line of the occipital region of the cranial vault and the posterolateral border of the epicranial aponeurosis. Each frontal belly arises from the anterolateral margin of the aponeurosis and is inserted into the skin of the corresponding eyebrow and into the orbicularis oculi muscle. The occipital bellies fix the epicranial aponeurosis so the frontal bellies can raise the eyebrows. Although occipitofrontalis also plays a role in opening the eyelids, the **levator palpebrae superioris** muscles in the orbit are the major dilators (see Section 30.4.1).

Muscles of the nostrils and ears

There are weak compressors and dilators of the nostrils, some of which are illustrated, but not labelled in Figure 23.8. These muscles are of little clinical consequence; they allow some people to flare their nostrils in anger or as a party trick. Similarly, there are several small muscles associated with the external ears. They are virtually functionless in man but enable some people to waggle their ears.

Muscles of the lips

The contour of the lips is formed by circular muscle fibres surrounding the mouth to form **orbicularis oris**, the sphincter of the lips. Some of its fibres are contained entirely within the lips while others are continuous with those of the **buccinator muscles** of the cheek. Many of the fibres that are contained entirely within orbicularis oris pass obliquely through the thickness of the lips from the dermis of the skin on the outer labial surface to the oral mucosa on the oral aspect. Orbicularis oris has also been described as consisting of eight components that can operate independently; there is an outer and inner segment in each quadrant. The fibres running from orbicularis oris into buccinator decussate at the corner of the mouth so that those from the lower lip pass into the upper part of buccinator and vice versa.

Several of the dilator muscles converge at the corner of the mouth and interlace with the orbicularis oris—buccinator fibres. This produces a knot of muscle termed the **modiolus**. The range of lip movement is considerable and very fine gradations of movement are possible, especially during speech. The lips can, for example, close the mouth, thin or purse the lips, or compresses the lips against the teeth. As was stressed in Chapter 2, muscles rarely act alone; many 'lip' movements also involve contraction of the muscles that comprise the dilators of the mouth.

Several muscles dilate the mouth but functionally they divide into two groups, the **elevators** and **depressors**. Identify the elevator muscles in Figure 23.8—**levator labii superioris, levator anguli oris, zygomaticus major, and zygomaticus minor**. As you can see, these enter the upper lip from different angles, almost vertical in the case of levator labii superioris, but much more obliquely for the zygomaticus muscles. Each muscle will exert a slightly different directional pull on the upper lip and, therefore, produce subtly different movements. The depressors of the lower lip are also shown in Figure 23.8; the **depressor labii inferioris** and **depressor anguli oris** are attached to the mandible anterior to the mental foramen and insert into the muscles and skin of the lower lip. Note that several of the elevators and depressors converge on to the modiolus. What happens to the corner of your mouth is, therefore, dependent on the action of several muscles; if the elevators predominate, the corners of the mouth will turn up and smile whereas if the depressors are acting strongly, the corners will turn down and you will look sad. The **platysma** muscles are thin sheets of muscle that run subcutaneously from the lower border of the mandible to the clavicles; some of their fibres also blend with orbicularis oris and have a depressive action.

A small slip of muscle, the **mentalis**, passes from the front of the mandible near to the midline to be inserted into the skin of the chin.

Muscles of the cheek

As you can appreciate from Figure 23.8, each **buccinator muscle** is quite deep compared with the other muscles of facial expression. Its muscle fibres run horizontally. As already described, the fibres of buccinator decussate anteriorly at the modiolus and blend with orbicularis oris. In the cheek, the upper and lowermost fibres attach to the maxilla and mandible at about the level of the root apices of the molar teeth, respectively; these insertions into the maxilla and mandible lie just above the reflections of the oral mucosa in the upper and lower buccal sulci (see Section 25.2.2). Posteriorly, the fibres move medially, crossing behind the last molars and in front of the anterior border of the ramus the mandible to attach to the **pterygomandibular raphe**, a vertical line of connective tissue running downwards behind the last molar teeth from pterygoid hamulus to the posterior end of the mylohyoid line. As shown in Figure 23.10, the fibres of buccinators interdigitate with those of the **superior constrictor** muscle of the pharynx at the raphe. Buccinator compresses the cheeks against the teeth and, with the cheeks first distended with air, is used in the acts of blowing and whistling. The buccinator and orbicularis oris muscles have an important function of clearing food from the buccal and labial sulci and replacing it between the teeth during chewing.

The position and actions of the perioral muscles of facial expression must be taken into account when designing and constructing dentures as outlined in Box 23.7.

Box 23.7 The muscles of facial expression and prosthetic dentistry

An awareness of the position and actions of the muscles of facial expression is important during taking impression in the first stages of providing dentures for a patient. The position of the superior and inferior lines of attachment of buccinator must be borne in mind. If the buccinator muscle impinges on the edge of the denture during movements of the cheek, the denture will be dislodged or areas of soreness will develop where oral mucosa is trapped between the muscle and the denture. Mentalis lies just to the side of the frenulum of the lower lip and its contraction may dislodge a lower denture if its base has been extended too low.

The **modiolus**, where several of the facial muscles join together, contains a high proportion of fibrous tissue formed by the perimyseal coverings of the muscles; it, therefore, does not contract. The modiolus is slightly below the corner of the mouth. Its position should be moulded into the impression material on the lower border of a lower denture by moving the modiolus around with your finger; this creates a hollow where the modiolus can sit without dislodging the denture or causing soreness.

23.3.3 The parotid glands

Major salivary glands are those that form a distinct organ whereas minor salivary glands are small aggregates of salivary cells embedded in the submucous layer of the mucosa lining the oral cavity.

The parotid glands are the largest of the three pairs of major salivary glands. Their position is shown in Figure 23.9. Each gland is wedged between the mandibular ramus and its associated muscles in front and the mastoid process and sternocleidomastoid muscle behind. The superficial part of the gland is triangular in outline. The base of the triangle lies on the level of the zygomatic arch and the two sides overlap the masseter in front and the sternocleidomastoid behind as they converge on to the apex. The apex is usually about the level of the angle of the mandible but can extend down into the neck. The superficial surface is flattened and is covered by subcutaneous tissue and skin. The gland is enclosed within a thick **parotid capsule** of connective tissue formed by the investing layer of the deep cervical fascia.

In Figure 23.9, the **parotid duct** draining the gland can be seen emerging from the anterior border of the gland and running forwards across masseter. It turns medially at the front edge of masseter and pierces buccinator to open into the upper buccal sulcus of the oral cavity opposite the crown of the second upper molar. Cotton wool rolls placed in the sulcus will absorb parotid saliva during dental procedures. The anterior part of the gland overlying the masseter may be separated from the remainder of the gland to form the **accessory parotid** gland along the duct; it may be extensive enough to obscure the duct completely. The surface landmarks of the parotid duct have already been described in Section 20.3.1.

The parotid gland is also approximately triangular in outline when viewed in a horizontal section as shown in Figure 23.10. Its base is the superficial surface and the **glenoid lobe** projects medially between the mandible and sternocleidomastoid and may reach as far as the styloid process and its attached muscles.

Structures within the gland

Two vascular structures pass vertically through each parotid gland and a cranial nerve passes horizontally. As you can see from Figures 23.10 and 23.11, the external carotid artery is deepest and is accompanied by the retromandibular vein slightly more superficially. The facial nerve begins quite deeply, but becomes more superficial as it crosses through the gland.

As shown in Figure 23.10, the **external carotid artery** enters the gland through the lower part of its posteromedial surface. As already described in Section 23.3.1 on the facial blood supply, the artery divides into its two terminal branches, the **maxillary** and **superficial temporal artery**, within the gland. The maxillary artery is deep and leaves the gland through its anteromedial surface whereas the superficial temporal artery is superficial as soon as it leaves through the upper part of the gland.

The **retromandibular vein** is formed within the gland by the union of the two corresponding veins, the maxillary and superficial temporal veins. It runs downwards superficial to the external carotid artery and splits into anterior and posterior divisions in the lower part of the gland or after emerging from it as described above and shown in Figure 23.6.

Figure 23.11 shows the **facial nerve** entering the upper part of the posteromedial surface of the gland and running forward superficial to the retromandibular vein. It divides into five divisions within the gland; these nerves, which may have multiple branches, leave the

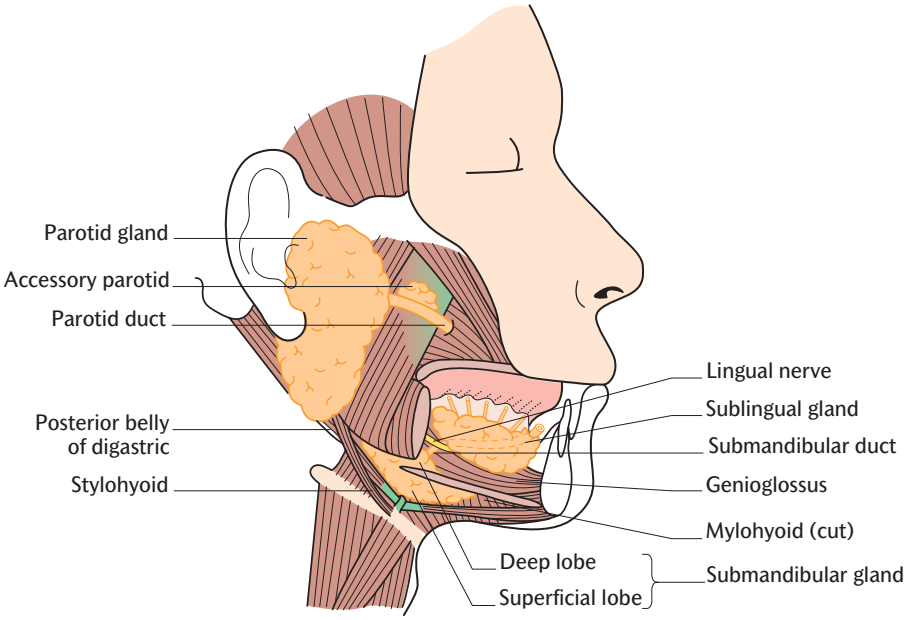


Fig. 23.9 The parotid gland and other major salivary glands.

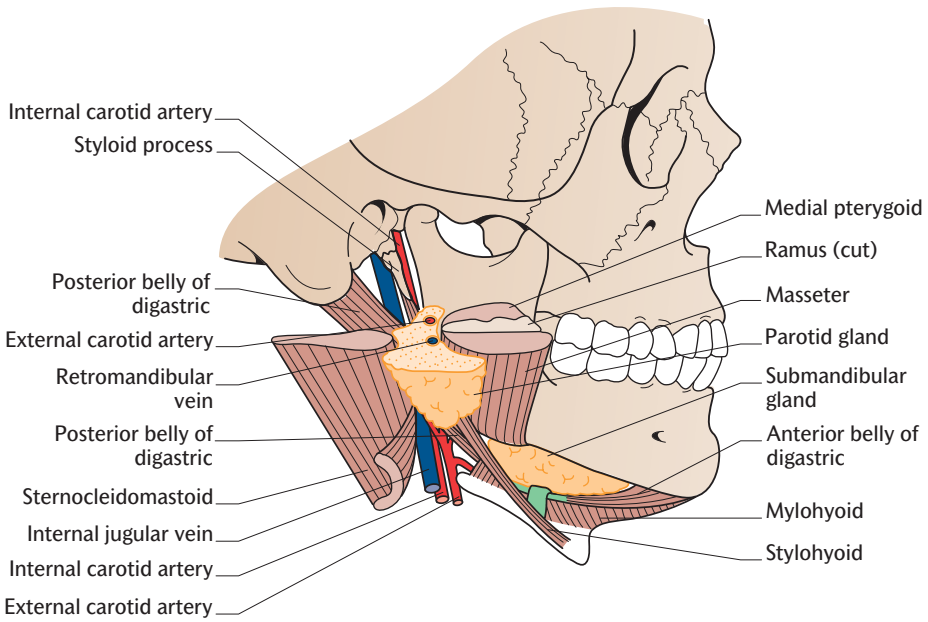


Fig. 23.10 A cross section through the parotid gland to show its contents and relationships to other structures.

anteromedial surface of the gland to supply the muscles of facial expression.

Innervation

The parotid gland receives its parasympathetic supply from the **glossopharyngeal nerves**. The course of the parasympathetic nerves to the gland is described in Box 23.8 for those who require more detail. Its sympathetic supply is from the plexus on the external carotid artery travelling from the superior cervical ganglion of the sympathetic trunk. The facial nerve does *not* supply the gland despite passing through it.

Box 23.9 describes the clinical conditions that may affect the parotid glands.

Box 23.8 Parasympathetic innervation of the parotid gland

The parasympathetic supply reaches the parotid through a complex pathway. The preganglionic axons begin in the **inferior salivatory nucleus** in the medulla and leave the brainstem in the **glossopharyngeal nerve**. They pass through its tympanic branch to the **tympanic plexus** on the tympanic membrane and then along the **lesser petrosal nerve** to the **otic ganglion** where they synapse. Post-ganglionic axons travel to the gland in the **auriculotemporal nerve**, a branch of the mandibular trigeminal nerve.

Box 23.9 Clinical anatomy of the parotid gland

The parotid gland may become inflamed like other salivary glands. The commonest cause of this is **epidemic parotitis**, more commonly known as **mumps**, a viral infection which occurs most frequently in children. Infections ascending from the mouth through the parotid duct may also cause acute and chronic parotitis. When the gland becomes inflamed, it will swell; this is very painful because of the restriction on gland enlargement due to the thick parotid capsule. In chronic cases, the flow of saliva is reduced. A swollen gland may also be due to a salivary calculus lodged in the duct. A salivary calculus is a small calcified stone formed by ectopic calcification within a gland.

Benign and malignant tumours may occur in the parotid gland. One of the commoner types of tumour is a **pleomorphic adenoma** (or mixed parotid tumour); they are locally invasive, but do not usually metastasize (see Box 4.4).

Facial paralysis may also result from a tumour invading the facial nerve (see Section 23.3.4). Extreme care must be taken to avoid damaging the facial nerve during surgery to remove a tumour. As described in Section 23.2.4, the branches of the facial nerve are small and quite difficult to distinguish from strands of connective tissue within the gland. A small current is applied to suspected nervous tissue through a hook electrode; if the tissue is a nerve, then one or more of the facial muscles will twitch whereas there will be no effect if the material tested is connective tissue.

23.3.4 The facial nerve

The course of the facial nerves within the petrous temporal bones has already been described in Section 18.7. Their peripheral course and distribution on the face are illustrated in Figure 23.12; study this picture as you read the description.

Each facial nerve leaves the skull through the **stylomastoid foramen** and enters the parotid gland. Before the nerve enters the parotid gland, it gives off:

- A posterior auricular branch to the occipital belly of occipitofrontalis and auricular muscles;
- A muscular branch to the posterior belly of digastric and the stylohyoid muscle arise.

Once within the gland, the nerve divides into five or more components. These components are usually called branches, but the term division is better since each division may have multiple branches.

As you can see in Figure 23.12, the five divisions emerge from the anteromedial surface of the gland and then run across the face to supply the muscles of facial expression. They are, from above downwards:

- The **temporal division** which leaves the upper part of the parotid gland, crosses the zygomatic arch, and supplies muscles of the external ear and part of frontalis;
- The zygomatic division which runs forwards to supply orbicularis oculi and the remainder of frontalis;
- The buccal division which runs forwards very close to the parotid duct to supply buccinator, the muscles of the upper lip upper;

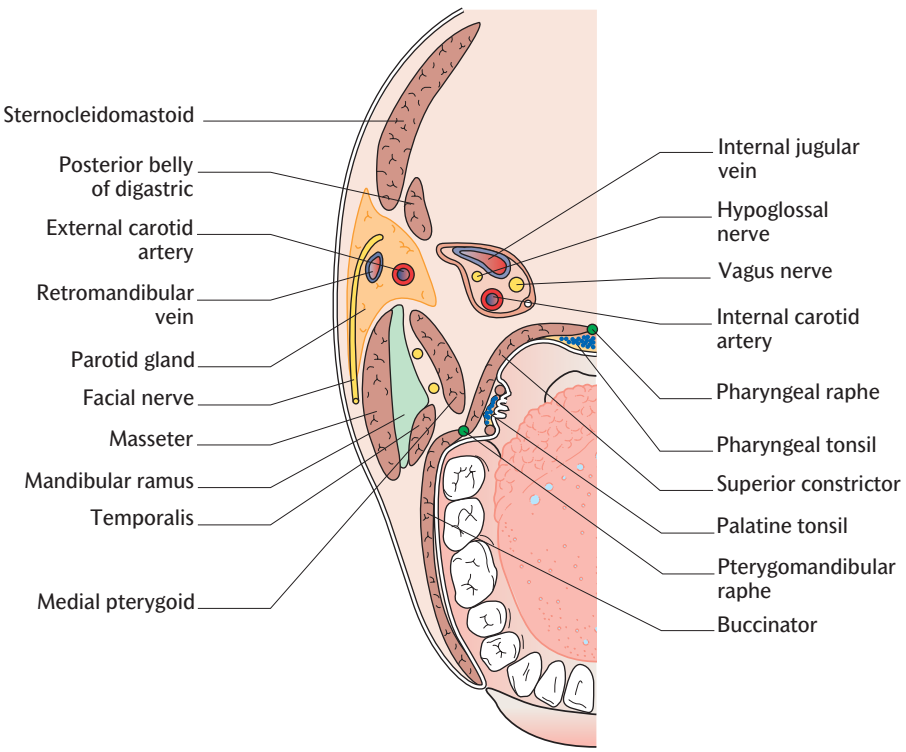


Fig. 23.11 A horizontal section at the level of the mandibular occlusal plane to show the deep parts of the parotid gland.

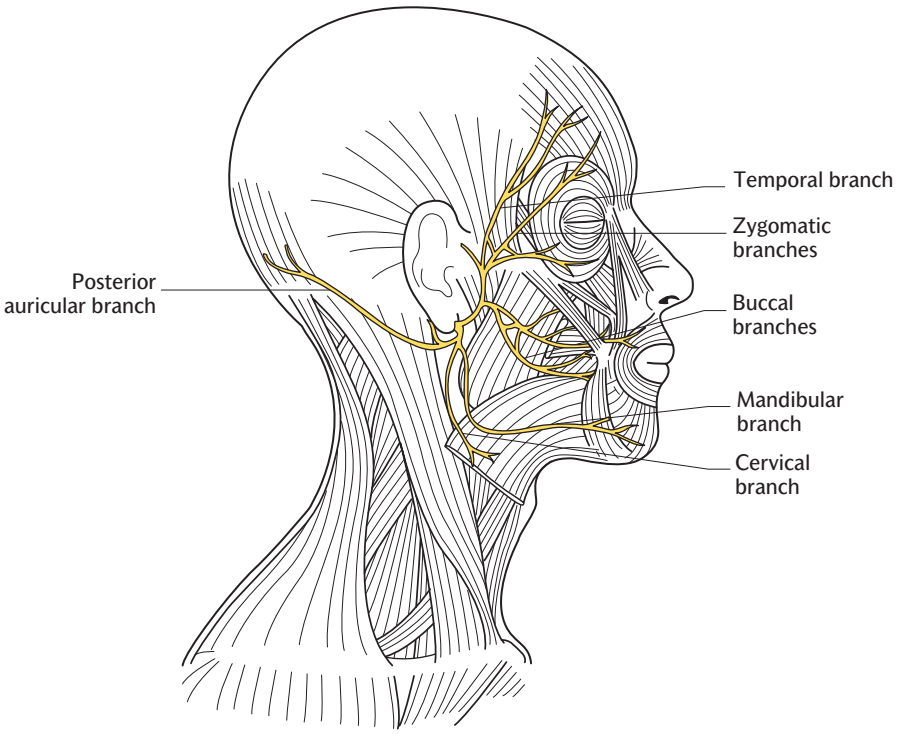


Fig. 23.12 The facial nerve and its branches on the face. The position of the parotid gland is indicated.

- The mandibular division which emerges from the lower anterior border of the gland down into the neck for a variable distance before turning up to cross the lower border of the mandible. It runs forwards to supply the muscles of the lower lip;
- The cervical division which runs vertically downwards behind the angle of the mandible to supply platysma.

The posterior auricular branch usually arises from the cervical division and passes backwards beneath the ear to the occipital part of occipitofrontalis.

The need to avoid damage to the facial nerves influences surgical access routes to underlying structures; possible access routes are described in Box 23.10.

Box 23.10 Surgical access to the face and underlying structures

The facial nerves run very superficially in the face and are, therefore, at risk of damage as a result of traumatic or malicious incisions of the face. Cuts running across the direction of the nerves are more likely to cause damage than those parallel to the nerve branches. The results of such damage will be flaccid paralysis of the muscles supplied by the cut branch. As emphasized in Chapter 18, flaccid paralysis will cause functional and aesthetic defects if, as is usually the face with incisive injuries, the nerves fail to regenerate.

As you will appreciate from Figure 23.12, the facial nerve divisions overlie most of the deep structures of the face that maxillofacial surgeons may need to access such as the mandible, upper facial skeleton and orbit, temporomandibular joints, parotid and submandibular glands, to name a few. No-one is going to make an unnecessary surgical incision on the face because the potential disfigurement it may cause and the potential danger to the facial nerve. Access to deep structures may be gained by several routes that obviate or minimize scarring and avoid the facial nerve:

- If the face is already traumatically cut, the surgeon may access the operative field through the wound if access is sufficient for the intended procedure;
- Access to the zygomatic and maxillary bones for fracture repair is often made through an incision into the lower eyelid (see Box 30.1);
- Access to the upper facial skeleton may be achieved through an incision running up from the ear and curving forward across the temple within the hairline. Skin is remarkably pliable and very wide access may be achieved by dissecting along the plane just deep to the nerve. The patient should be warned that there may be temporary facial paralysis due to stretching of the facial nerve. Hair will grow back and hide the scar.
- Access to the mandible and related structures is often made using one of the transverse skin creases in the neck described in Section 20.3.2. Once again, excellent access may be achieved albeit with the possibility of stretching the lower divisions of the facial nerve as a skin flap is raised; again, the patient should be warned of the possibility of temporary facial palsy. The skin creases hide the scar to some extent and it can be further disguised by wearing a scarf or high-necked clothing.

23.3.5 The scalp

The scalp is the hair-bearing skin and underlying tissues that cover the braincase from the nape of the neck to the forehead and down on the temples laterally. The scalp does not correspond exactly to the bones of the braincase; the scalp does not extend down the full extent of the forehead overlying the frontal bone.

Layers of the scalp

The scalp consists of five layers of tissue. These are, from superficial to deep:

- **Skin** with a high density of hair follicles;
- Dense subcutaneous **Connective tissue**;
- **Epicranial Aponeurosis** linking the **occipital** and **frontalis muscles**;
- Loose adipose **subaponeurotic layer;**
- **Periosteum.**

The underlined letters spell out SCALP—a simple aide memoire to the five scalp layers.

The **epicranial aponeurosis** is a sheet of tough fibrous tissue connecting two occipital and two frontal muscles bellies over the cranial vault; the four muscle bellies make up the **occipitofrontalis muscle**. The aponeurosis is firmly attached to the skin of the scalp by the dense subcutaneous layer; any movement of the aponeurosis will be mirrored by movement of the skin of the scalp.

The arterial supply to the scalp is extremely rich to support the thousands of hair follicles in the scalp. It is derived from the **occipital, posterior auricular**, and **superficial temporal** branches of the **external carotid arteries** which anastomose freely with each other in the subcutaneous layer. The venous drainage follows the arterial supply. The consequences of scalp wounds are outlined in Box 23.11.

23.3.6 The temporomandibular joint and superficial muscles of mastication

As you will realize from your examination of the surface anatomy of the neck and face in Chapter 20, the **temporomandibular joints** are superficial structures. Their movements can be felt quite easily below the posterior root of the zygomatic arch. Two of the four pairs of muscles of mastication are also superficial and can be palpated when they are made to contract. The **masseter muscle** overlies most of the superficial surface of the ramus of the mandible. The **temporalis muscle** covers an extensive area on the side of the cranial vault. It may seem logical to describe these three relatively superficial structures in this chapter, but their description will be left until Chapter 24 when their anatomy can be seen in the context of the anatomy of the jaws and other muscles of mastication.

Box 23.11 Scalp wounds

The skin, subcutaneous tissue, and epicranial aponeurosis are so firmly bound to each other that they are always torn away as a single layer—**a scalping injury**. If the aponeurosis is cut by deeply penetrating wounds, it is pulled apart by the occipitofrontalis muscle to create a gaping wound. The blood vessels in the subcutaneous tissue are pulled apart and tend not to contract so bleeding from the scalp is profuse.

23.3.7 Cutaneous innervation of the head and neck

Figure 23.13A indicates the areas of skin of the face, scalp, and neck supplied by different nerves and the main illustration of Figure 23.13B shows the actual nerve branches. The skin of the face and anterior and lateral scalp is supplied by the terminal branches of the three divisions of the **trigeminal nerve**. The skin behind the ear and the side and front of the neck is supplied by branches of the **cervical plexus** and the skin on the back of the neck and scalp by the upper four dorsal rami of the cervical nerves.

Cutaneous branches of ophthalmic division of the trigeminal nerve

These branches supply the forehead region as far back as the highest point of the skull (the vertex) down to the upper eyelid and a strip down the midline of the external nose. The **supraorbital nerve** to the forehead and frontal part of the scalp is the biggest branch. Box 23.12 describes the minor branches of this division.

Cutaneous branches of maxillary division of the trigeminal nerve

The large **infraorbital nerve** is the continuation of the main trunk of the maxillary nerve (see Section 24.5.3). It emerges from the **infraorbital foramen** and supplies the skin between and including the upper lip and the lower eyelid, the upper part of the cheek, and adjacent oral mucosa in the mouth. The small **zygomaticofacial** and **zygomaticotemporal** nerves emerging through tiny foramina on the surface of the zygomatic bone supply an area of skin of the temporal region behind the orbit.

Cutaneous branches of mandibular division of the trigeminal nerve

As indicated in Figure 23.13A, the cutaneous branches of the mandibular division of the trigeminal nerve supply a large area extending from the chin and lower lip anteriorly, across the skin over much of the mandibular ramus and lower part of the cheek, up to the lower part of the

Box 23.12 Minor cutaneous branches of the ophthalmic trigeminal nerve

The other cutaneous branches of the ophthalmic trigeminal are:

- The **supratrochlear nerve** to the medial part of the forehead;
- The **lacrimal nerve** to the skin and conjunctiva of the lateral part of the upper eyelid;
- The **infratrochlear nerve** to the medial part of the upper eyelid; the **external nasal nerve** to the skin of the nose.

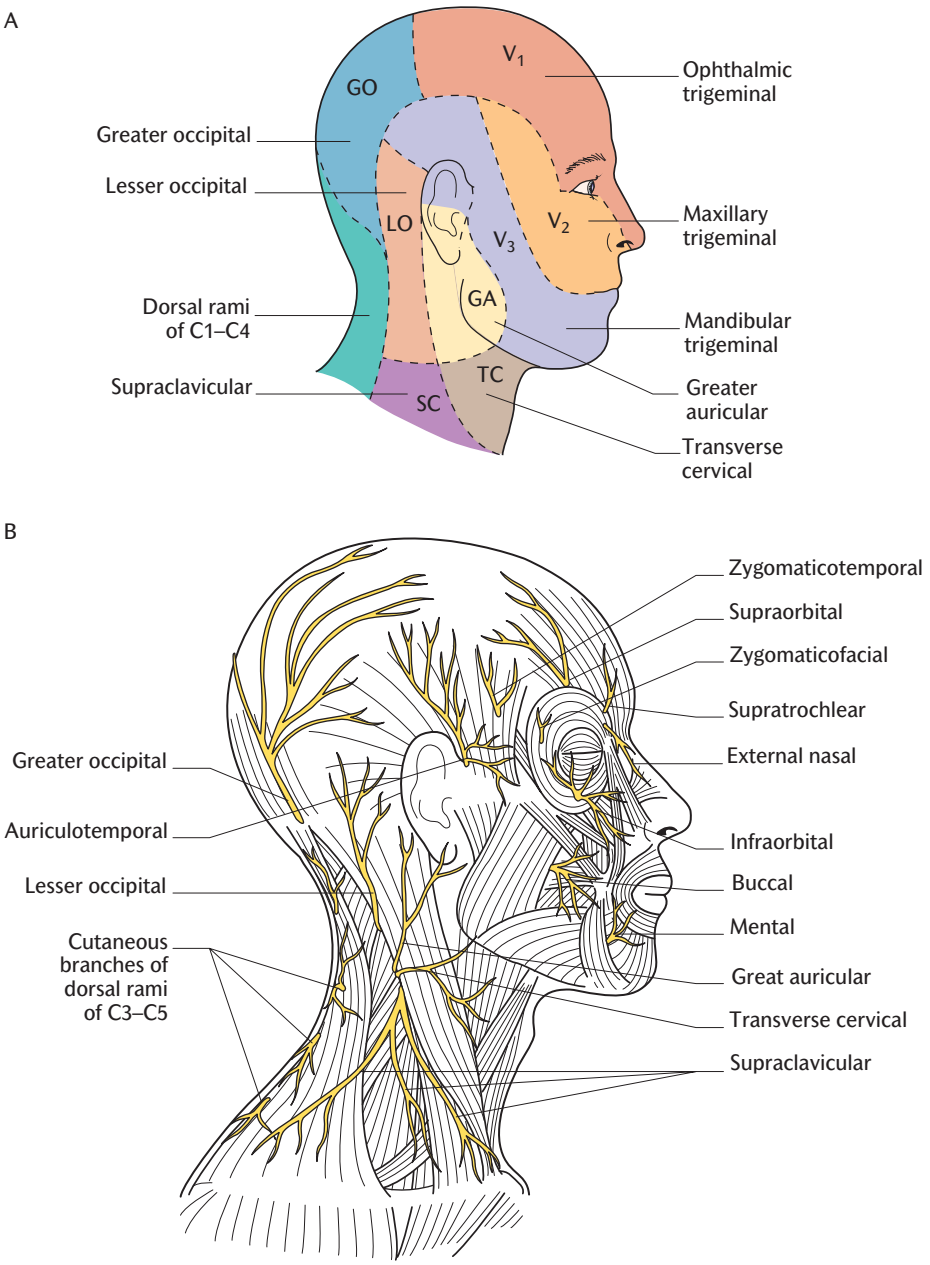


Fig. 23.13 The cutaneous innervation of the head and neck. A) The areas supplied by major nerve divisions and branches; B) Cutaneous branches of the nerves indicated in A.

temple posteriorly. The branches supplying these areas can be seen in Figure 23.13B.

The **auriculotemporal nerve** ascends from behind the neck of the mandible, crossing over the posterior root of the zygomatic process of the temporal bone to supply the temple, part of the skin lining the external acoustic meatus and superficial aspect of the ear drum, and the upper part of the auricle.

The **buccal nerve** runs forwards from under cover of the mandibular ramus to cross its anterior border at the level of the occlusal plane of the lower teeth on to the buccinator muscle which it then pierces. It supplies the skin covering and the oral mucosa lining the cheek over the lower molar teeth.

The **mental nerve** is the cutaneous branch of the **inferior alveolar nerve**. It emerges from through the **mental foramen** to supply the skin and oral mucosa of the lower lip and chin.

In Figure 23.13A, you will notice that the skin over the angle of the mandible is the only part of the facial skin that is *not* supplied by the trigeminal nerve as you may anticipate. It is actually supplied by the **greater auricular nerve** of the cervical plexus (see Section 23.1.4).

The posterior part of the scalp is supplied by the **greater occipital nerves** and the skin of the back of the neck by the posterior primary rami of cervical spinal nerves 1 to 4. The scalp behind the ear is supplied by the **lesser occipital** branch of the cervical plexus. The skin of the front of the neck is supplied by the **transverse cervical branches** of the cervical plexus.

Box 23.13 The clinical significance of regional lymphatic drainage

Lymph from a particular area drains into a particular node or group of lymph nodes. This drainage area is known as the **regional lymphatic drainage** and is the most important concept to take from this section. If you know the regions drained by a particular node, you can work out where the source of infection or location of a primary tumour might be. Other lymph nodes further down the chain of vessels returning lymph to the venous system will enlarge to help combat the infection if the regional lymph node or nodes is incapable of dealing with the attack; these nodes will also be enlarged.

The lymph nodes of the head and neck may also become involved in diseases of the lymphatic system itself such as **Hodgkin's disease** or **lymphoma**.

23.3.8 Lymph nodes of the head and neck

As outlined in Chapter 20, dental students and practitioners need to know the lymphatic drainage of the head and neck. It is important because they can track back to possible sources of infection using their knowledge of regional lymphatic drainage described in Box 23.13 when they encounter enlarged lymph nodes (**lymphadenopathy**) making an immune response to the infective organisms. More sinisterly, lymphatic vessels are one of the routes that malignant cells may spread from a primary tumour to form secondary tumours, a process known as **metastasis**.

Lymph from all parts of the head and neck eventually drains into the **deep cervical lymph nodes** situated along the internal jugular vein beneath the sternocleidomastoid muscle. Some regions drain directly into these nodes while other regions first drain through one of the outlying groups of nodes.

Superficial groups of nodes

Some of the superficial groups of nodes illustrated in Figure 23.14 are nodes we have already been met in Chapter 20. The illustration indicates that they form a discontinuous collar from the chin to the occipital region essentially along the line where the neck and head meet. They are described in this order.

The **submental nodes** are situated on the superficial surface of the mylohyoid muscle just inside the inferior border of the mandible below the mental foramen. They drain lymph from the tip of the tongue, floor of the mouth, the lower incisor teeth, including their periodontal tissues and the central part of the lower lip. They empty into the **jugulo-omohyoid node** of the deep cervical chain (see p. 240) and the submandibular nodes.

The **submandibular nodes** are on the surface of the superficial part of the submandibular gland under cover of the inferior border of the mandible. As you can see in Figure 23.14, lymph from a wide area of the face drains into them. In addition, lymph from the anterior part of the nasal cavity, most of the teeth, the floor of the mouth, and anterior two-thirds of tongue also empties into these nodes. The lymph vessels draining the anterior part of the face may have buccal nodes scattered along their course in the cheek. The submandibular nodes drain into the deep cervical nodes, especially the **jugulo-omohyoid node**.

Lymph from the forehead, temple, eyelids, and external ear drains into the **parotid** (preauricular) **nodes** situated on the lateral surface of the parotid gland. The **deep parotid nodes** within the gland receive

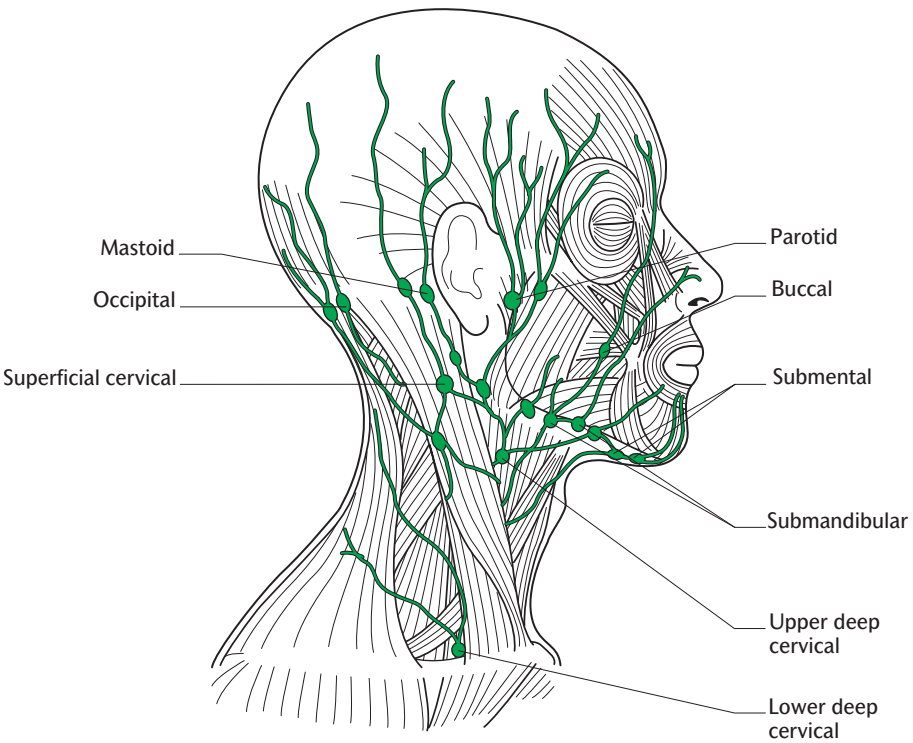


Fig. 23.14 The superficial lymph nodes of the head and neck. The dotted lines indicate the regional drainage of each group of nodes.

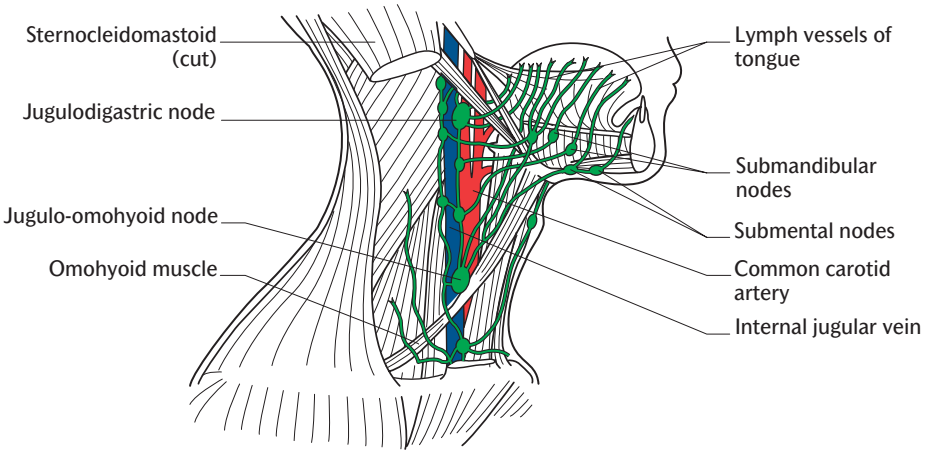


Fig. 23.15 The deep cervical lymph nodes.

lymph from the eyelids and the middle ear. The parotid nodes drain to the upper deep cervical group.

The **mastoid** (retroauricular) **nodes** are superficial to the mastoid process. They drain the scalp behind the external ear and empty into the upper deep cervical nodes. The **occipital nodes** lie on the occipital bone at the apex of the posterior triangle. They receive lymph from the posterior part of the scalp and upper neck and empty into the lower deep cervical nodes.

The superficial structures of the neck drain into the **superficial cervical nodes** located around the external jugular vein; they empty into the deep cervical nodes.

Deep cervical lymph nodes

The **deep cervical nodes** are illustrated in Figure 23.15. The **superior group** surrounds the upper part of the internal jugular vein and the **inferior group** lies on its lower course close to the subclavian vessels. You will now be aware from this chapter and Chapter 20 that sternocleidomastoid covers the carotid sheath and the major vessels and nerves it contains; the deep nodes located around the internal jugular veins cannot, therefore, be palpated very easily. However, two key nodes can be felt if enlarged as they are not covered by the muscle; the **jugulodigastric node** in the superior group is situated where the digastric muscle crosses the internal jugular vein and the **jugulo-omohyoid node** in the inferior group is close to the intermediate tendon of the omohyoid muscle. As you can see from Figure 23.15, the jugulodigastric node receives lymph from the posterior tongue whereas the jugulo-omohyoid node receives lymph from the anterior part and tip of the tongue through the submandibular and submental nodes.

Groups of nodes around the pharynx, larynx, and trachea drain lymph from these deeper structures into the deep cervical nodes. The deep cervical lymph nodes of each side drain through a jugular trunk into the venous system around the junction of the internal jugular and subclavian veins.

24

The temporomandibular joints, muscles of mastication, and the infratemporal and pterygopalatine fossae

Chapter contents

24.1 Introduction

It is essential that dental students and practitioners understand the structure and function of the **temporomandibular joints** and the **muscles of mastication** and other muscle groups that move them. The **infratemporal fossa** and **pterygopalatine fossa** are deep to the mandible and its related muscles; many of the nerves and blood vessels supplying the structures of the mouth run through or close to these areas, therefore, knowledge of the anatomy of these regions and their contents is essential for understanding the dental region.

24.2 The temporomandibular joints

The temporomandibular joints (TMJ) are the only freely movable articulations in the skull together with the joints between the ossicles of the middle ear; they are all synovial joints. The muscles of mastication move the TMJ and the suprahyoid and infrahyoid muscles also play a significant role in jaw movements.

24.2.1 Articular surfaces

The articular surfaces of the **squamous temporal bone** and of the **condylar head** (**condyle**) of the **mandible** form each **temporomandibular joint**. These surfaces have been briefly described in Chapter 22 on the skull and Figure 24.1A indicates their shape. The concave **mandibular fossa** is the posterior articulating surface of each squamous temporal bone and houses the mandibular condyle at rest. The condyle is translated forwards on to the convex **articular eminence** anterior to the mandibular fossa during jaw movements.

The articular surfaces of temporomandibular joints are atypical; they covered by **fibrocartilage** (mostly collagen with some chondrocytes) instead of hyaline cartilage found in most other synovial joints.

24.2.2. Capsule and ligaments

Figures 24.1B and 24.1C show the capsule and ligaments associated with the TMJ. The tough, fibrous capsule is attached above to the anterior lip of the squamotympanic fissure and to the squamous bone around the margin of the upper articular surface and below to the neck of the

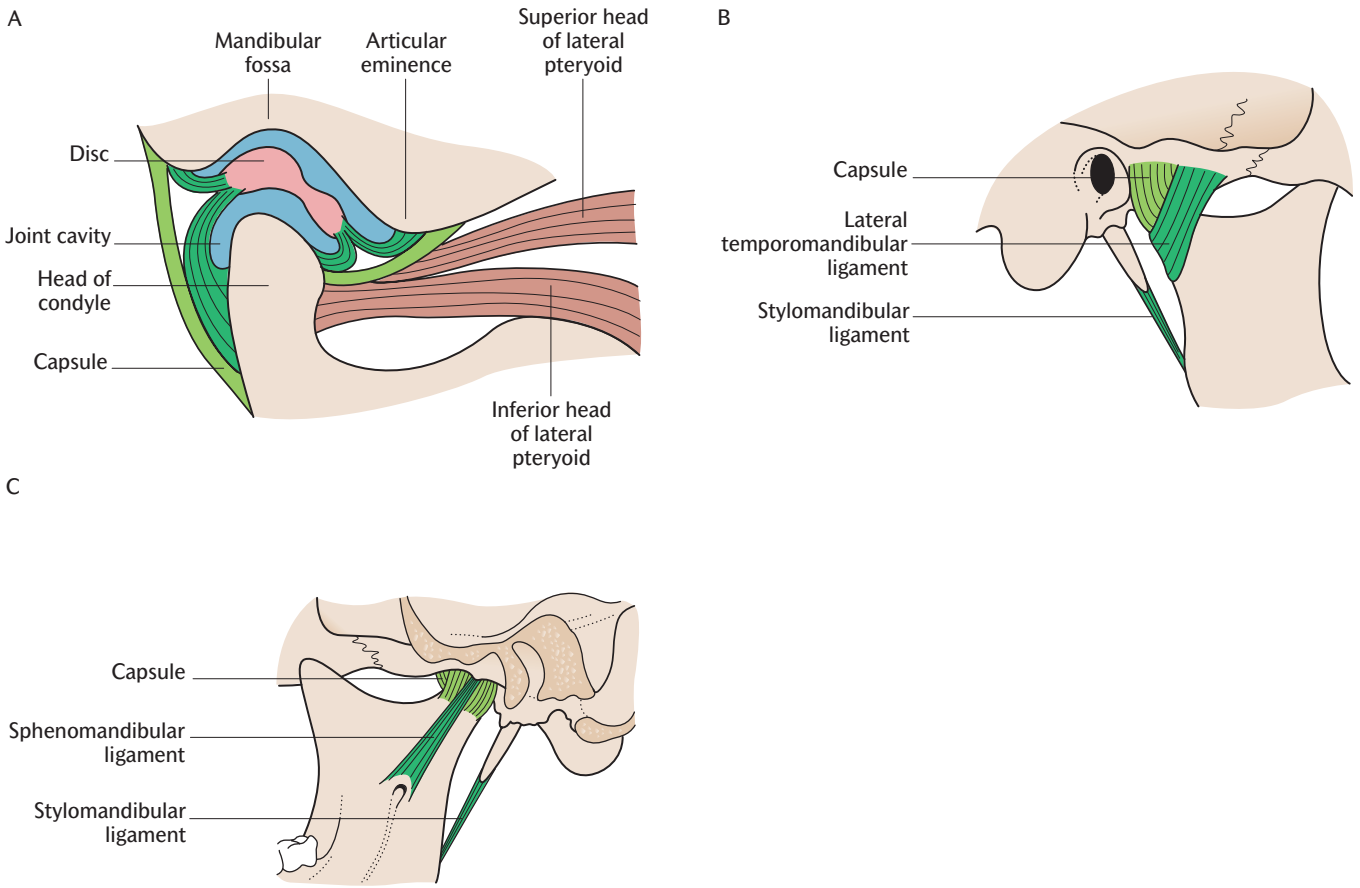


Fig. 24.1 The right temporomandibular joint. A) In sagittal section to show its internal structure; B) Lateral view; C) Medial view.

mandible a short distance below the limit of the lower articular surface. The capsule is slack between the articular disc (see Section 24.2.3) and the squamous bone, but much tighter between the disc and the neck of the mandible. Part of the lateral pterygoid muscle is inserted into the anterior surface of the capsule. As in other synovial joints, the non-load-bearing internal surfaces of the joint are covered with **synovial membrane**.

As illustrated in Figure 24.1B, the capsule is strengthened laterally by the **lateral temporomandibular ligament**, a thick band of fibrous tissue; it extends obliquely downwards and backwards from the articular tubercle to the lateral surface of the mandibular neck to prevent excess posterior movement of the TMJ.

There are two accessory ligaments of the TMJ which do not blend with or strengthen the capsule. As shown in Figure 24.1C, the **sphenomandibular** ligaments extend downwards and forwards from the spine of the sphenoid to the lingula of the mandible and are some distance medial to the joint. They act as a sling for the mandible and it rotates about an axis passing through the lower attachments of the ligaments during some mandibular movements. The **stylomandibular ligaments** extend from the styloid process to the angle of the mandible behind each joint; these are thickenings of the parotid capsule rather than true ligaments.

Recall from Section 2.4.1 that the capsule and ligaments of synovial joints have an important sensory role; they contain proprioceptive nerve endings and provide information on joint position—absolutely vital for the function of the TMJ. The sensory nerves are supplied from the **auriculotemporal** and **masseteric nerves**, both branches of the mandibular division of the trigeminal nerve (see Section 24.5.1).

24.2.3 The articular disc

As you can see in Figure 24.1A, the joint cavity of each TMJ is completely divided into an upper and lower compartment by the **articular disc** (or **meniscus**). The joint cavity is not divided in other synovial joints. The disc is made of dense fibrous tissue and is essentially a continuation of the tendon of attachment of the lateral pterygoid muscle through the joint cavity.

The disc blends laterally and medially with the capsule of the joint. As Figure 24.1A shows, it is attached in front to the anterior border of the squamous articular surface and the capsule. The disc divides into two layers posteriorly. The upper layer is attached to the anterior margin of the **squamotympanic fissure** while the lower layer is attached to the posterior surface of the neck of the mandible. The capsule on the posterior aspect of the joint blends with these two layers. The tendon of the **lateral pterygoid muscle** is inserted into the anterior margin of the disc.

Figure 24.1A shows that the disc is not uniform in thickness. The posterior part of the disc is thickest, the central part is thinnest and the front part of the disc is also thickened although not as much as the posterior part. Occasionally, the disc is perforated in the central thin region and the two compartments are then in communication. This makes no difference to joint function.

The functions of the articular disc are uncertain because surgical removal of the disc from patients with arthritic TMJs improves rather than impedes function. The disc seems unnecessary for efficient joint function. The lateral pterygoid muscles protrude the mandible by drawing the mandibular condyles forwards from the mandibular fossa to the articular eminence (see Section 24.3.3). They also draw the disc forwards wedging the thick posterior portion between the back of the condyle and articular eminence and may stabilize the joint as the mouth is opened.

The TMJ may be dislocated. The causes and consequences are described in Box 24.2.

24.3 The muscles of mastication

The muscles of mastication comprise four pairs of muscles. The temporalis and masseter muscles are superficial to the mandible whereas the medial and lateral pterygoid muscles are deep to it.

24.3.1 The temporalis muscle and temporal fascia

As shown in Figure 24.2, each temporalis muscle arises from an area on the external surface of the cranial vault between the inferior temporal line and the infratemporal crest below although the crest is hidden by the muscle in the figure. The muscle fibres converge from this wide area towards the coronoid process of the mandible. As Figure 24.2 illustrates, the anterior fibres run downwards and slightly backwards, the middle fibres pass directly downwards and the posterior fibres thus run downwards and forwards with the most posterior fibres running almost horizontally. The fibres attach to a well-developed tendon, passing deep to the zygomatic arch near the coronoid process. The tendon attaches to the apex, anterior, posterior borders, and medial aspect of the coronoid process; the attachment continues below the coronoid process down the anterior border of the ramus as far down as the retromolar fossa posterior to the mandibular third molar. The extent of this attachment must be taken into consideration during the design of lower dentures as described in Box 24.1. The fibres arising close to the infratemporal crest have a fleshy attachment into the medial surface of the coronoid process and ramus.

The **temporal fascia**, a strong sheet of fibrous tissue, covers the temporalis muscle. It is attached above to the whole length of the **superior temporal line**. Inferiorly, it splits to attach to the external and medial margins of the upper border of the zygomatic arch. The superficial temporal blood vessels and the auriculotemporal nerve cross the fascia superficially.

The temporalis is supplied by the **deep temporal nerves** from the anterior division of the **mandibular nerve**. Its principal action is elevation of the mandible to close the mouth. The disposition of the fibres within the muscle mean that some fibres are always aligned for the most efficient mechanical advantage irrespective of the position of the TMJ. The more horizontal posterior fibres retract the mandible.

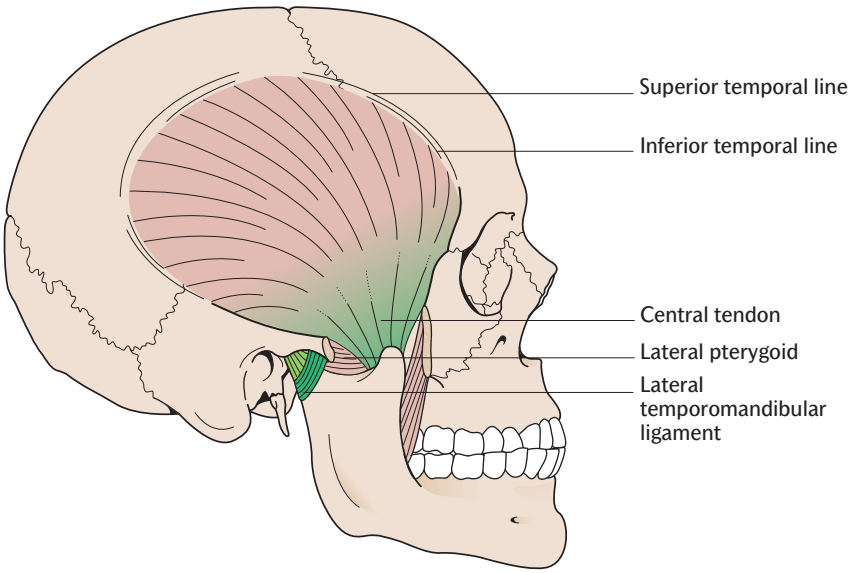


Fig. 24.2 The temporalis muscle.

Box 24.1 The attachments of temporalis and prosthetic dentistry

The attachment of the temporalis tendon into the retromolar fossa can be verified on yourself. Push your finger along between your teeth and cheek and feel the anterior border of the mandibular ramus. Now clench your teeth and you can feel the tendon. Note how far it does come down into the fossa.

This relationship is clinically important when designing dentures. If the base of a lower denture is extended too far backwards, the tendon can displace the denture when temporalis contracts or oral mucosa is trapped between the denture base and the muscle causing soreness and discomfort. Avoidance of these potential problems is implicit in denture construction; prosthetic teeth tend to be smaller than the corresponding living teeth and only seven teeth are incorporated into a full lower denture, thus the denture does not have to be extended so far back that it will impinge on the temporalis attachment.

24.3.2 The masseter muscles

As illustrated in Figure 24.3A, each masseter muscle attaches between the zygomatic arch above and the outer surface of the angle of the mandible below. Take a closer look at Figure 24.3A and a dissected specimen if you have access and you will see that the superficial layer, the largest component of the muscle, conforms to this description. It arises from a strong aponeurosis attached to the anterior two-thirds of the lower border of the zygomatic arch and pass downwards and backwards to the angle of the mandible. You will also see more vertical fibres deep to the superficial layer. This is the middle layer attached above between the medial surface of the anterior two-thirds and the lower border of the posterior one-third of the arch and to the lateral surface of the middle part of the ramus below. The deep layer arises from the whole length of the medial surface of the zygomatic arch and its fibres pass downwards to attach to the upper part of the mandibular ramus. As shown in Figure 24.3B, these three layers are quite distinct and separable posteriorly, but anteriorly, they fuse to form a single mass. The three layers of muscle provide fibres running in the line of best mechanical advantage for any position of the TMJ.

There are several tendinous septa within the masseter muscle; some are attached to the zygomatic arch and interdigitate with others attached to the lateral surface of the mandible. The muscle fibres run between these septa in a multipennate arrangement (see Section 2.5.1 and Figure 2.5), making the masseter muscles extremely powerful elevators of the mandible. They are innervated by the masseteric branches of the anterior division of the mandibular nerve. As shown in Figure 24.3B, the masseteric branch of the **transverse facial artery** runs between the middle and superficial layers and provides the principal blood supply to the muscles.

24.3.3 The pterygoid muscles

The lateral and medial pterygoid muscles are the two deep pairs of the **muscles of mastication.** The lateral and medial pterygoid muscles, the two deep muscles of the muscles of mastication, attach to the lateral and medial sides of the lateral pterygoid plates, respectively. The **pterygoid plates** are part of the pterygoid processes of the sphenoid bone in the cranial base but they function as a component of the upper facial skeleton.

The lateral pterygoid muscles

As you can see in Figure 24.4, each **lateral pterygoid muscle** arises by two heads. The upper head is attached to the infratemporal surface of the greater wing of the sphenoid and the lower head to the *lateral surface* of the **lateral pterygoid plate**. The fibres run backwards and laterally. The larger lower head of the muscle is attached by a short tendon into the **pterygoid fovea** on the anterior surface of the neck of the mandible. The smaller upper head is attached to the anterior aspect of the capsule and hence into the **articular disc** of the TMJ. The two parts of the muscle pull the head of the mandible and the disc forwards, respectively. There is good electrophysiological evidence that the two parts of the muscle act independently during movements of the mandible; these movements and the part played by the lateral pterygoids

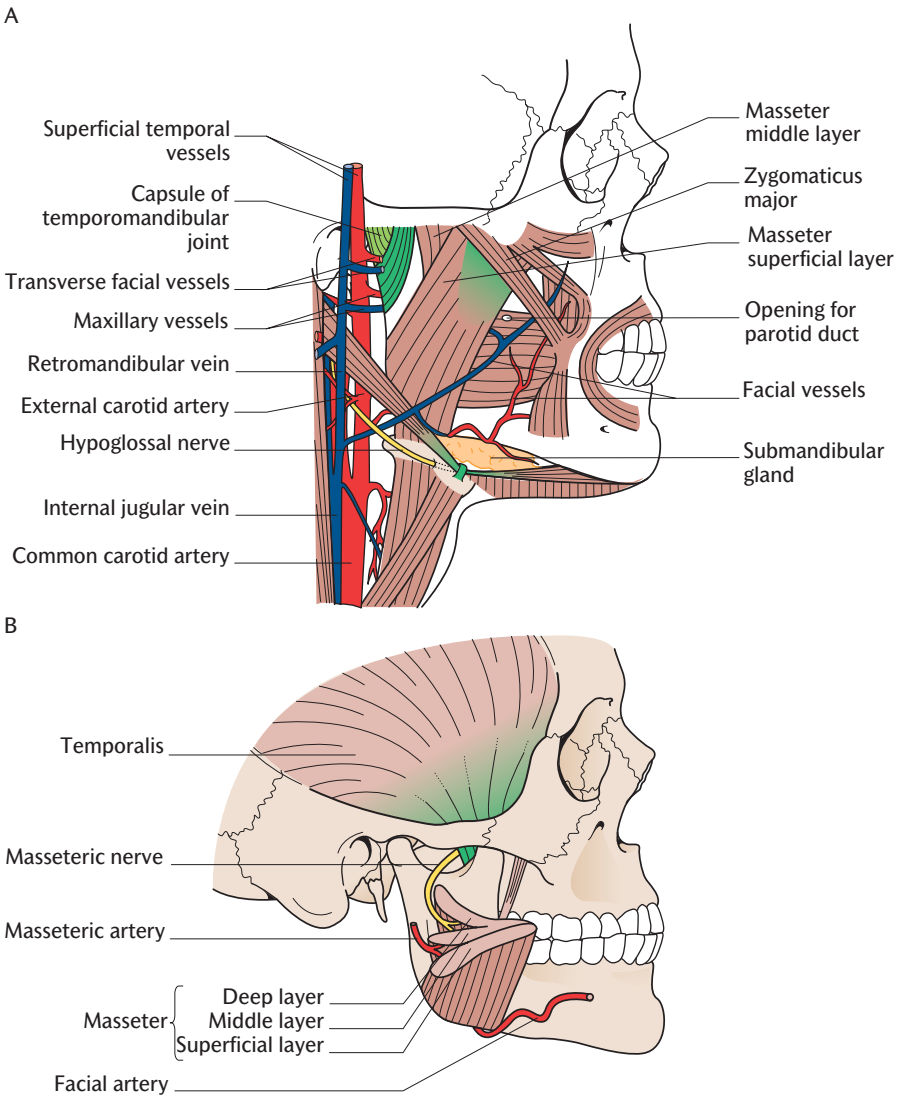


Fig. 24.3 A) The masseter muscle and related structures. The parotid gland has been removed for clarity. B) The three layers of the masseter muscle and its nerve and blood supply.

is described in Section 24.4. The muscle is innervated by a branch of the anterior trunk of the mandibular division of the trigeminal nerve.

The medial pterygoid muscles

As shown in Figure 24.4, each **medial pterygoid muscle** also arises by two heads. Most of its fibres arise from the *medial surface* of the **lateral pterygoid plate**; a small slip arises from the maxillary tuberosity and adjacent palatine bone to form the superficial head. The fibres run downwards, backwards, and laterally to insert into the roughened area on the medial surface of the ramus and **angle of the mandible**. Each medial pterygoid muscle contains a number of tendinous septa which give it a multipennate arrangement like the masseter muscle. The medial pterygoid muscle is a powerful elevator of the mandible; its role in mandibular movements is described in Section 24.4. The muscle is innervated by a branch from the main trunk of the **mandibular trigeminal nerve**.

The **pterygomandibular space** is a gap between the muscle attachment to the angle and the medial surface of the ramus above. This is the area in which inferior alveolar nerve blocks are placed for local anaesthesia of the lower teeth. The anatomy of local anaesthesia is described in Section 25.5.2.

24.4 Movements of the TMJ

A unique feature of the TMJ is that the left and right joints are joined by the very robust mandible; one joint, therefore, cannot be moved in isolation so there must be compensatory movement in the other joint. For instance, if you wish to deviate your mandible to one side so that the cusps of the posterior teeth can grind food as you chew, you will slide the condyle of the opposite side forwards using the lateral pterygoid muscle while holding back the other side back with the horizontal fibres of temporalis. Move your mandible to the left while palpating both joints. You will feel the right joint sliding forward and at the same time, the left joint making a slight backward movement to compensate.

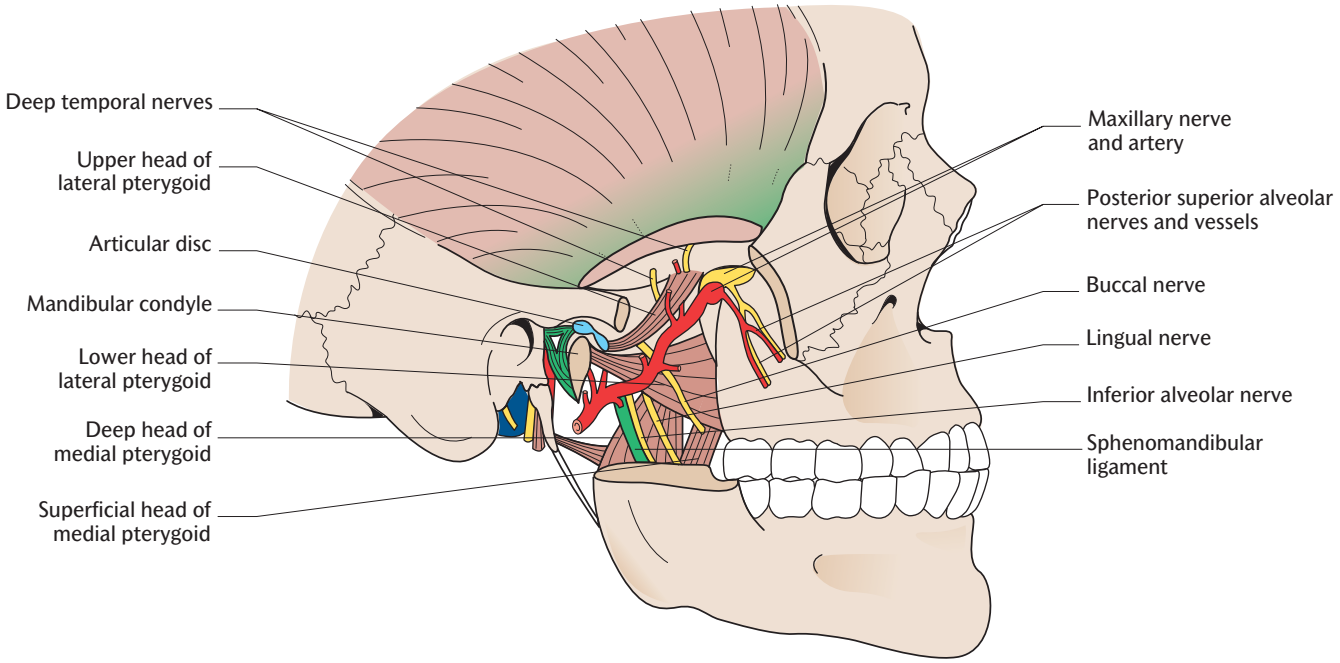


Fig. 24.4 The pterygoid muscles and other contents of the right infratemporal fossa. The ramus of the mandible has been removed at a level just above the mandibular foramen.

Both TMJs must make movements simultaneously because they cannot operate independently; however the movements do not have to be identical in both joints.

Each TMJ can rotate about an axis through the head of the condyle and can slide anteriorly from the mandibular fossa on to the articular eminence. Rotation alone produces relatively little opening movement. You can verify this by palpating your own joints as you open your mouth gradually; after the mandible has lowered a few millimetres, you will feel the forward sliding movements as you open wider. It is possible to slide the TMJ forwards (protrusion) without rotation but your teeth must be slightly apart otherwise the cusps interfere with jaw movement. In reality, sliding and rotator movements occur simultaneously. Reversing the direction of sliding will retract the jaw and rotating in the opposite direction will close the jaw.

The only possible movements at the joints are:

- Rotation of the head of the condyle within the mandibular fossa;
- Sliding of the condyle along the articular eminence.

These are combined in various ways to produce the functional movements of depression, elevation, protrusion, retraction, and lateral deviation of the mandible. They are illustrated in Figure 24.5. The functional movements are, in turn, combined to produce the complex patterns of movements during incision and mastication described in Section 26.3.

It is important to realize that the muscles of mastication are rarely completely relaxed, except when in deep sleep or unconscious. The muscles maintain a low level of activity (**muscle tone**) to counteract gravity sufficient to keep the mouth closed without the maxillary and mandibular teeth contacting. This is known as the **mandibular rest position** or postural position. Feel the space between the upper and lower teeth by running your tongue around your teeth; the 2–3 mm gap between them is the **interocclusal** (or **freeway) space. Occlusion** is the jaw position when the upper and lower teeth are in maximal contact. Most movements of the mandible start from the rest position rather than occlusion.

24.4.1 Basic movements

Figure 24.5A illustrates **rotational movement** of the head of the condyle about a *transverse axis*. This transverse axis of rotation does not coincide exactly with the long axis of the condylar head since the latter is angled posteromedially from the transverse plane (see Figure 22.13). The outcome of rotational movement is the depression of the mandible, but as already described, the mouth can only open a little without any sliding movement. If you attempt to depress the mandible by rotation alone, you will find that you can open your mouth only a short distance. The posterior border of the mandibular ramus soon impinges on the **parotid gland** behind it and further movement is impossible once because of its resistance to compression. Continued opening past this point requires that the mandibular head to translate forwards on to the articular tubercle.

The **sliding** movement is shown in Figure 24.5; the mandibular head and disc slide together forwards and downwards from the mandibular fossa on to the articular eminence. Verify this for yourself by palpating the lateral poles of the mandibular heads whilst depressing the mandible to its full extent; note how soon the rotational movement is also accompanied by sliding. During sliding, the axis of rotation of the mandible moves downwards from the condylar heads to a position approximately between the right and left mandibular foramina as shown in Figure 24.5. The steepness of the anterior surface of the mandibular

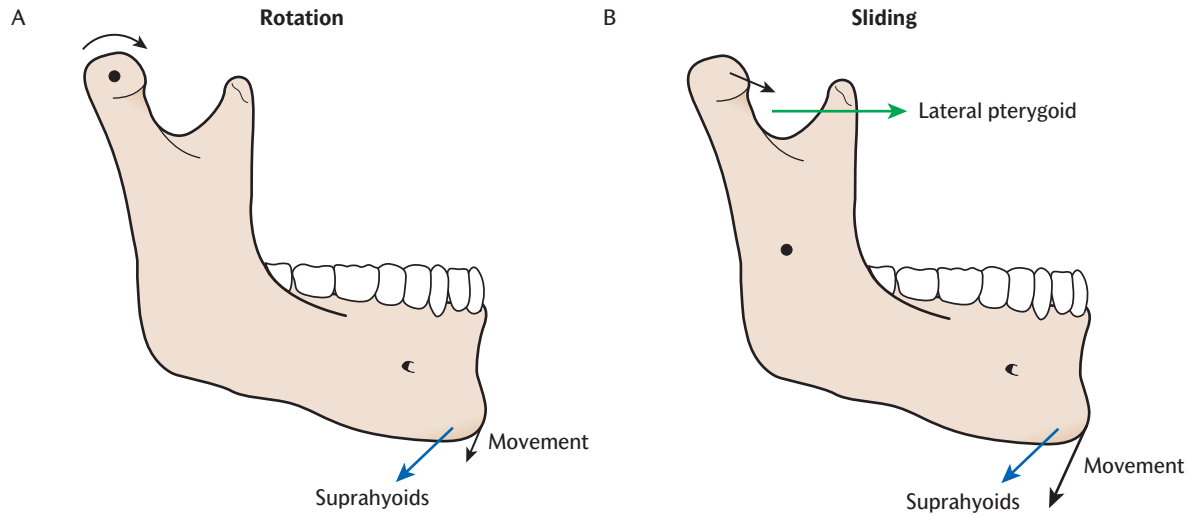


Fig. 24.5 Movement during depression of the mandible. A) Rotational movement; B) Sliding movement. The axis of rotation is marked by the small solid circle, the direction of movement by black arrows, and the direction of muscle pull by coloured arrows.

fossa determines how soon the intercuspation of teeth in occlusion is released. In prosthetics, this is the **condylar guidance angle**. Sliding movements are not naturally used in isolation, except in specific circumstances such as recording the guidance angle during denture construction.

24.4.2 Actions of the jaw muscles

The action of individual jaw muscles can be inferred from the disposition of their attachments and the general direction of their fibres as described in Section 24.3. However, the jaw muscles do not act singly, but combine in various ways to produce the complex movements used during incision and mastication. Electromyographic analysis has advanced our knowledge of the contributions made by individual muscles to specific jaw movements. Electrodes are placed in or over the muscles being investigated and their electrical activity recorded while appropriate movements are made; a non-contracting muscle is electrically quiet but produces a burst of electrical activity as it contracts. The interpretation of electromyographical data can be difficult because the electrodes tend to pick up activity from more than one muscle; this why there are some discrepancies between different analyses of jaw movements.

Functional movements

Depression

Depressing the mandible consists of a smooth, progressive combination of rotation and sliding with the mandibular heads rotating against the under surface of the discs as they slide forwards onto the articular eminences. The mandibular foramina are the parts of the mandible which move least for most of the range of depression. It may be significant that this is the point where the major nerves and blood vessels enter the mandible.

The **lateral pterygoid muscles** are bilaterally active and slide the condyles forwards during depression of the mandible. The lower head is the prime mover of the condyle and the upper head slides the articular disc of each TMJ forward at the same time. This activity is accompanied in the later stages by activity of the **digastric** and other **suprahyoid muscles** to produce a downward pull on the mandible; the suprahyoids can only act in this way if the hyoid bone is *fixed* by action of the **infrahyoids** at the same time.

Elevation

Elevation of the mandible is the reverse sequence of movements during depression. It involves the coordinated contraction of the **temporalis, masseter**, and **medial pterygoid muscles** of the two sides. The medial pterygoids initiate the closing movement. A certain amount of elevation is achieved by passive tension generated in the elevator muscles and ligaments during opening of the mouth; consequently, there may be little electrical activity in the muscles during the initial phase of closing.

Protrusion and retrusion

Protrusion is produced by symmetrical sliding forwards at the two TMJs and **retrusion** is the reverse movement. These movements are rarely made in isolation but are usually coupled with depression and elevation. Bilateral contraction of the **medial and lateral pterygoids** protrudes the mandible. Retrusion is brought about by the bilateral contraction of the *posterior fibres* of the **temporalis muscles**.

Lateral excursion

Lateral excursion to the left side is shown in Figure 24.6. As you can see by the direction of the arrows, the head of the mandible on the side to which movement is taking place is retained in the mandibular fossa while the contralateral head is translated forwards onto the articular eminence. The mandible rotates about a vertical axis just behind the ipsilateral head. In consequence, the ipsilateral head makes a small lateral compensatory movement called the **Bennett movement**. Lateral movements are produced by contraction of the *contralateral* **lateral and medial pterygoids** while the posterior fibres of the *ipsilateral*

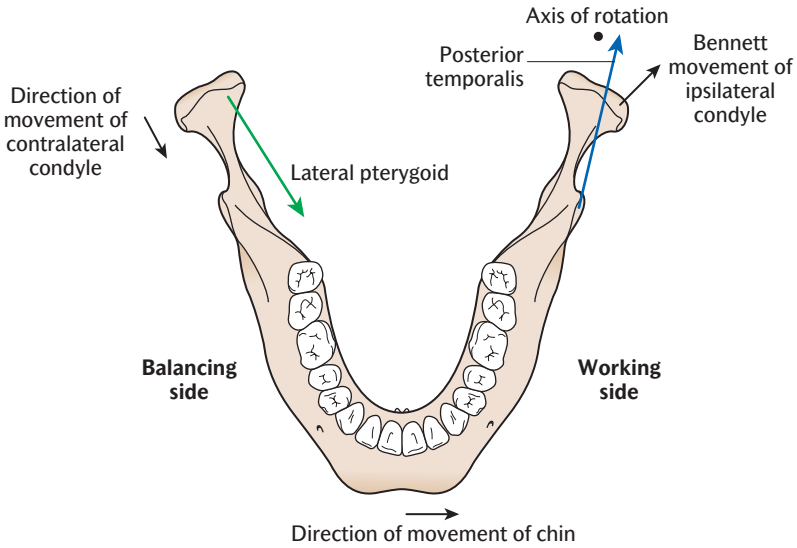


Fig. 24.6 Lateral excursion and the Bennett movement; solid circles and arrows are used the same as Figure 24.5.

temporalis muscle prevent the ipsilateral condyle from moving forwards. The side on which the teeth come into maximal contact during lateral excursion is the **working side** where mastication will take place; the other side is the **balancing side**; in Figure 24.6, the left side is the working side.

The functioning of the TMJs and their associated musculature in mastication is described in Chapter 26.

Fractures of the mandible are a relatively common injury. Box 24.3 describes the consequences of such fractures which depend on the position of the fracture and the pull of the muscles acting on the mandible.

Box 24.2 TMJ dislocation

Bony constraints prevent the TMJs from dislocating backwards because of the tympanic parts of the temporal bones posterior to the mandibular fossa or medially because of similar bony constraints. The only direction in which it can dislocate is forwards; the condyles slide too far anteriorly on to the anterior surface of the articular eminence. This can occur following blows to the mandible or if the mouth is opened very wide such as when you yawn in a boring lecture. The mouth is opened wide because the condyle overriding the eminence is pulled up into the infratemporal fossa by the elevator muscles.

A dislocation can be reduced immediately by placing your thumbs along the occlusal surfaces of the lower teeth and applying pressure downwards and backwards to push the condyle back behind the eminence into the mandibular fossa. If the dislocation is not treated very quickly, the muscles tend to go into spasm so manual reduction just described is extremely difficult. If this does occur, muscle relaxants are given to reduce the spasm before manipulation of the mandible.

Any dislocation stretches the ligaments and capsule of the joint in question and the TMJ is no exception. The likelihood of recurrence of the dislocation is increased because of the soft tissue injuries.

24.5 The infratemporal fossa

The **infratemporal fossa** is the area under the cranial base deep to the mandibular ramus. It contains many soft structures relevant to dental practice, including the pterygoid muscles and the pterygoid plexus of veins, the mandibular division of the trigeminal nerve, and the maxillary artery.

Some of the walls of the fossa are formed by bones as shown in Figure 24.8 (see also Figure 21.4). Its anterior wall is the posterior surface of the maxilla, its medial wall is the lateral pterygoid plate, and its lateral wall is the ramus of the mandible; the inferior surface of the greater wing of sphenoid is its roof. There is no bony posterior wall; the posterior boundary is marked by the carotid sheath and styloid apparatus.

Figure 24.9 is a posterior view of an isolated sphenoid bone; it is the best way to see some of the features of the pterygoid processes and should be studied in conjunction with the examination of a skull as you read the following description. The **pterygoid processes** project downwards on either side from the junction of the body and greater wing of the sphenoid. They are formed by the larger **lateral** and smaller **medial pterygoid plates** which diverge posteriorly to enclose the **pterygoid fossa**. As you can see from Figure 24.8, the inferior third of the anterior border of the lateral pterygoid plate articulates with the posterior border of the perpendicular plate of the palatine bone. A narrow gap separates the upper two-thirds of the plate from the palatine

Box 24.3 Fractures of the mandible and zygomatic arch

Fractures of the mandible and the zygomatic and maxillary bones of middle third of the face facial skeleton occur at about the same frequency.

Fractures of the mandible

The common sites of mandibular fracture are indicated in Figure 24.7; the injury is bilateral in many cases.

Fractures of the **neck of the condylar process** are usually the result of a blow to the chin; the break may be within or outside the capsule of the TMJ; the degree of bony displacement depends upon whether or not the periosteum and the capsule and ligaments of the joint are torn. If the fracture tears the periosteum below the capsular attachment, there is nothing to hold the bone fragments together. The lateral pterygoid will pull the condylar head and upper fragment of the neck anteriorly and the other muscles of mastication will pull the rest of the mandible superiorly so that the bones are displaced. If the fracture is within the capsule, the fragments are usually held in place by the periosteum and ligaments so that displacement is minimal.

As indicated in Figure 24.7, fractures of the **angle of the mandible** may run downwards and forwards or downwards and backwards. A break running downwards and forwards is a **favourable fracture** of the angle because masseter, temporalis, and medial pterygoid muscles attached to the ramus pull the upper fragment in a superior and medial direction while the suprahyoid muscles pull the body inferiorly, thus keeping the bones together. The likelihood of damage to adjacent or enclosed structures is minimized. On the other hand, when the fracture line runs downwards and backwards, the muscles acting on the ramus and the muscles acting on the body are pulling the bony fragments in opposite directions so there is considerable dislocation. The inferior alveolar nerve may be damaged with consequent loss of sensation from the lower lip, chin, and teeth; the lingual nerve, the major sensory nerve to the tongue, is also vulnerable as it passes into the floor of the mouth on the medial side of the mandible across the fracture site.

Fractures of the **body of the mandible** are frequently located in the *canine region* because the length of the root of the canine tooth weakens the bone in this position; a blow on the side of the jaw is most likely to cause a break at the maximum convexity of the mandible which is close to the canine tooth. If the body is fractured bilaterally in the canine region, the digastric and geniohyoid muscles attached to the mental region displace and dislocate the anterior fragment downwards. A lateral blow may sometimes break the body of the mandible on the side of the blow and the neck of the mandible on the opposite side.

Fractures of the zygomatic arch

Blows to the side of the face may result in a fracture of the zygomatic arch, with or without displacement. The tough temporalis fascia will often hold the bony fragment in place. A severe blow will break the arch at one point and drive the bone inwards; as the bone either side of the break is pushed, it also fractures at the arch, producing a characteristic W-shaped fracture. The depressed bone fragments impinge on the masseter and temporalis muscles and interfere with their function.

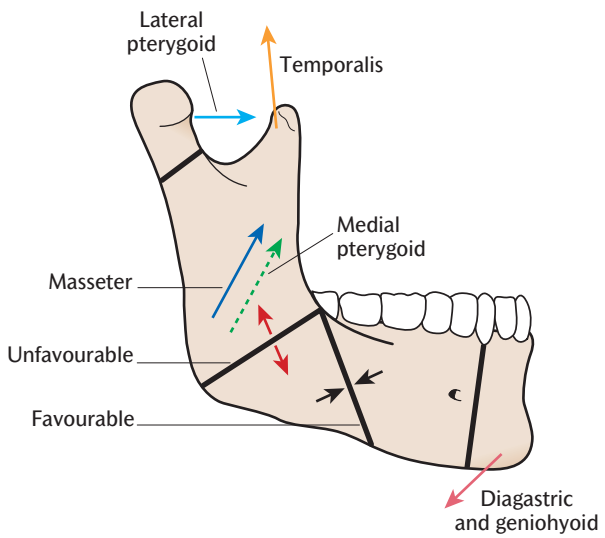


Fig. 24.7 Common sites for fractures of the mandible. Black and red arrows indicate the movement of bony fragments; other coloured arrows indicate the direction of muscle pull.

bone to form the **pterygomaxillary fissure** that leads medially into the **pterygopalatine fossa** (see Section 24.5.3). The **pterygoid canal** is also visible, running anteriorly through the root of the medial pterygoid plate.

As already mentioned, the pterygoid plates are structurally part of the cranial base but function as a component of the upper facial skeleton. The **lateral** and **medial pterygoid muscles**, as we have already seen, attach to the lateral and medial sides of the lateral pterygoid plates, respectively. The **superior constrictor muscle** forming the muscular wall of the upper pharynx is attached to the posterior border of the medial pterygoid plate. The **scaphoid fossa** is a shallow depression enclosed by the division of the posterior border of the medial plate at its upper extremity; this is the upper attachment of the **tensor veli palatini** muscle which tenses the soft palate (see Section 28.3.1). The lower end of the posterior border projects as the hook-like **pterygoid hamulus**. The tendon of the tensor veli palatini curves around its lower surface and the **pterygomandibular raphe** also attaches to this process. The hamulus is often broken off from dried skulls, especially if they have passed through several generations of dental students. The right and left medial pterygoid plates enclose the posterior nasal aperture.

The **foramen ovale** and **foramen spinosum,** already described in Section 22.5.2, connect the infratemporal fossa with the cranial cavity.

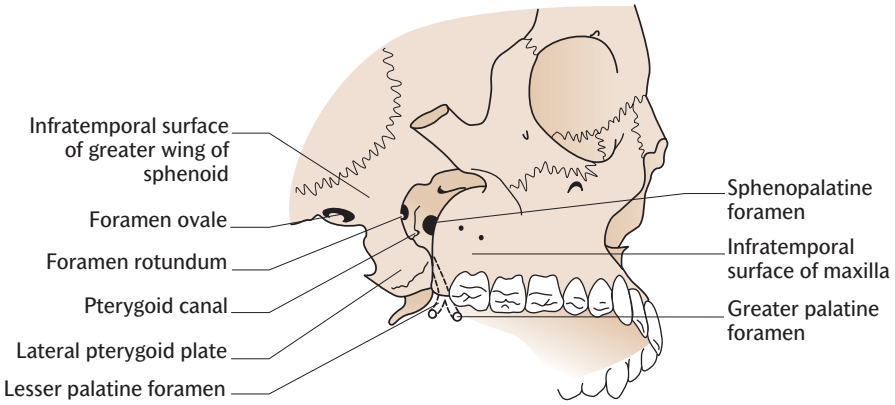


Fig. 24.8 The right pterygopalatine fossa seen through the pterygomaxillary fissure. The skull is tilted so that the fossa is viewed from below; the width of the pterygomaxillary fissure is exaggerated so that the foramina can be seen.

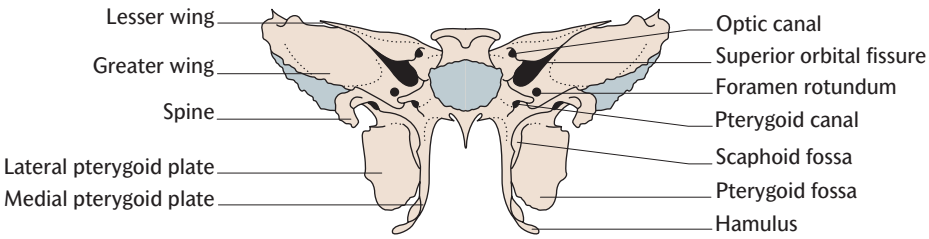


Fig. 24.9 A posterior view of the sphenoid bone.

The pterygomaxillary fissure links the fossa to the pterygopalatine fossa (see Section 24.5.3). The **inferior orbital fissure** located between the upper margin of the posterior wall of the maxilla and the greater wing of the sphenoid connects the orbit with the infratemporal fossa laterally and with the pterygopalatine fossa medially. The **maxillary trigeminal nerve** which becomes the **infraorbital nerve** once in the orbit and veins connecting the inferior ophthalmic vein and the pterygoid plexus of veins pass through the **infraorbital fissure**.

Apart from the pterygoid muscles, the mandibular division of the trigeminal nerve and its branches and the first and second parts of the maxillary artery are the major contents of the infratemporal fossa.

24.5.1 The mandibular division of the trigeminal nerve

The general plan of the mandibular trigeminal nerve is illustrated in Figure 24.10. Study this figure as you as read the description You will also be guided to other figures that give an indication of the relationship between specific branches and other tissues.

The sensory neuronal processes forming each mandibular division of the trigeminal nerve exit from the lateral part of each **trigeminal ganglion** through the **foramen ovale** into the infratemporal fossa. The axons forming the trigeminal motor root run beneath the ganglion and unite with the mandibular nerve within or just below the foramen ovale to form a mixed motor and sensory nerve. Two branches arise at this point. The **nervus spinosus** runs back into the middle cranial fossa through the **foramen spinosum** to supply the meninges. The **nerve to medial pterygoid** passes forwards to supply that muscle and also **tensor veli palatini** and **tensor tympani**. The latter muscle is a small muscle inserted into the malleus of the middle ear; it acts with stapedius to damp down oscillations of the ossicles in response to loud sounds.

As Figure 24.10 shows, each nerve divides into an **anterior** and **posterior trunk** shortly after it emerges from the foramen ovale. The anterior trunk is the smaller and consists mainly of motor axons; its only sensory branch is the buccal nerve. The larger posterior trunk is mainly sensory with motor axons only distributed through the mylohyoid nerve.

Branches from the anterior trunk

As illustrated in Figure 24.10, the **buccal nerve** runs laterally between the two heads of the lateral pterygoid to emerge deep to the temporalis muscle; it then continues downwards and forwards onto the lateral surface of buccinator. It gives branches to the skin of the cheek before piercing buccinator to supply the oral mucosa of the cheek, buccal sulcus, and buccal gingivae of the lower molars. The buccal nerve also contains a few post-ganglionic parasympathetic axons from the otic ganglion (see p. 252), which supply minor salivary glands in the buccal oral mucosa.

The **masseteric nerve** passes through the mandibular incisure to enter the deep surface of masseter; it gives a small sensory branch to the TMJ. The **deep temporal nerves** and the **nerve to lateral pterygoid** supply the muscles of the same names.

Branches from the posterior trunk

As you can see from Figure 24.10, each posterior trunk divides into three large branches, the auriculotemporal, lingual, and inferior alveolar nerves just after it splits from the anterior trunk.

The **auriculotemporal nerve** arises by two roots which run posteriorly, one on either side of the middle meningeal artery before uniting behind the artery. The arrangement of the nerve and artery is illustrated in Figure 24.11. The nerve then continues posteriorly deep to the lateral pterygoid muscle and the neck of the mandible. It curves laterally around the posterior surface of the mandibular neck, passing through

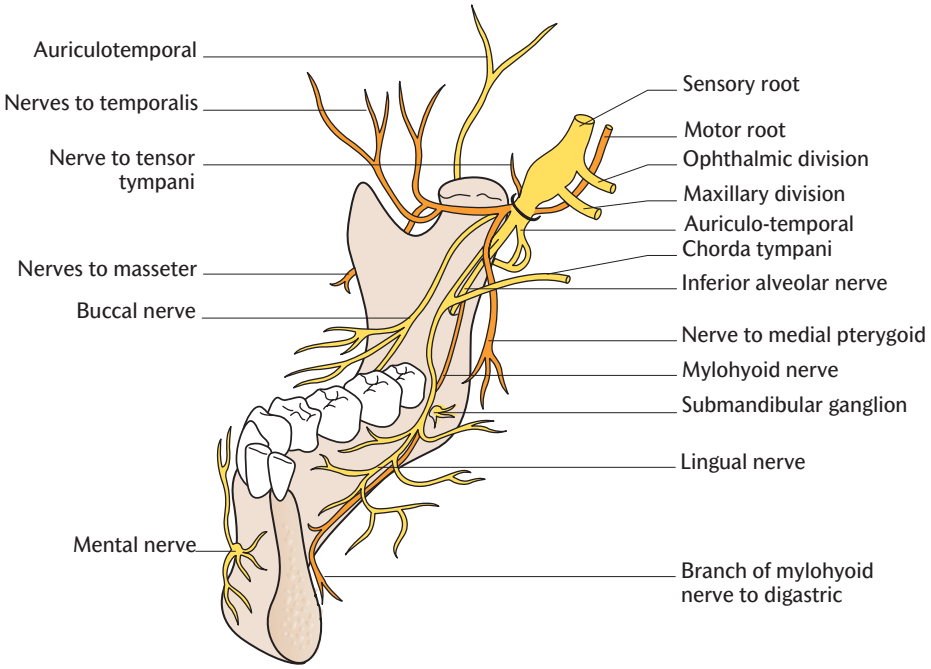


Fig. 24.10 The mandibular division of the trigeminal nerve. Sensory nerves are indicated in yellow and motor nerves in orange.

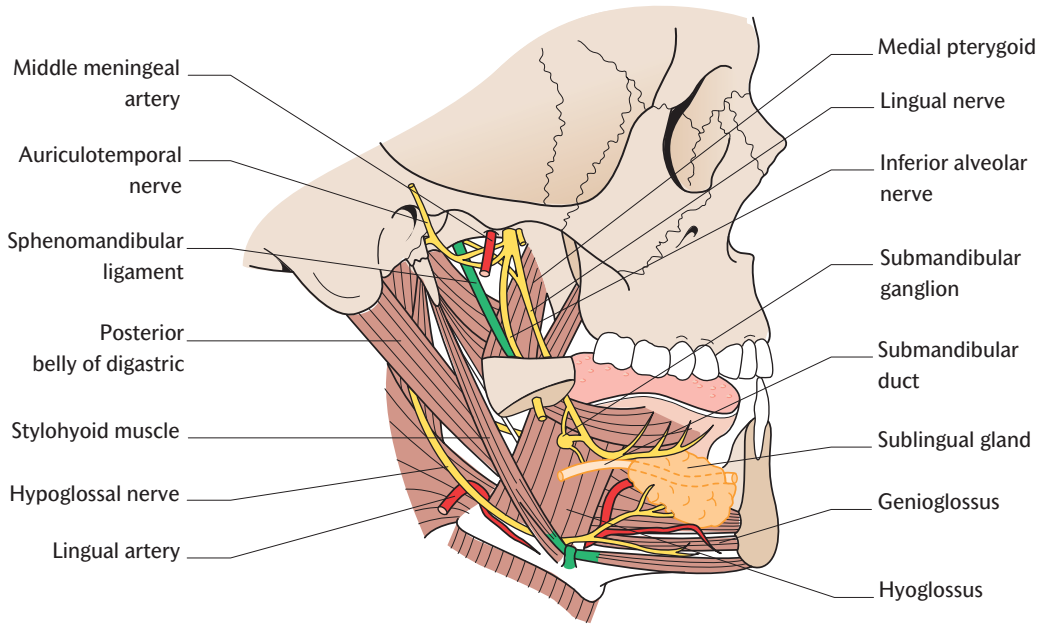


Fig. 24.11 The oral course of branches of the mandibular trigeminal seen in a lateral view of the floor of the mouth and its contents.

the parotid gland or within its capsule, to emerge laterally from behind the TMJ to which it gives a small branch. It then ascends over the posterior root of the zygomatic process of the temporal bone posterior to the superficial temporal vessels to supply the skin of the temple (see Figure 23.13). The nerve also gives sensory branches to the external acoustic meatus, the outer surface of the tympanic membrane, and to the skin of the auricle. Branches to the parotid gland carry post-ganglionic parasympathetic secretomotor axons from the otic ganglion.

The **lingual nerve** runs downwards between the lateral pterygoid and tensor veli palatini muscles. The **chorda tympani nerve**, a branch of the facial nerve arising from the main nerve in the middle ear cavity, joins the lingual nerve about 2 cm below the skull. The chorda tympani carries taste processes from the anterior two-thirds of the tongue and preganglionic parasympathetic axons to salivary glands in the floor of the mouth. As shown in Figure 24.10, the lingual nerve emerges from beneath the lateral pterygoid muscle to curve downwards and

forwards into the pterygomandibular space between the medial pterygoid muscle and the ramus of the mandible where it lies anterior to the inferior alveolar nerve (see Figure 24.11). It runs forwards immediately adjacent to the medial surface of the mandible in the region of the *third molar*, then turns medially across the floor of the mouth, supplying the mucosa as it does so, to supply the anterior two-thirds of the tongue with somatic sensation; those from the chorda tympani of the facial nerve supply taste buds in the same area. The relationship of the lingual nerve to the mandibular third molars is clinically significant during the surgical extraction of impacted wisdom teeth as explained in Box 24.4.

The chorda tympani components travelling with the lingual nerve connect with the **submandibular ganglion** as they pass across the floor of the mouth. The location of the ganglion and its relationship to the mandibular nerve is illustrated in Figure 24.11. These preganglionic parasympathetic axons synapse with post-ganglionic neurons innervating the submandibular, sublingual, and minor salivary glands. The course of the lingual nerve in the floor of the mouth and the connections of the submandibular ganglion are described in detail in Section 25.3.4.

Figure 24.10 shows the **inferior alveolar nerve** running downwards deep to the lateral pterygoid muscle about 1 cm posterior and lateral to the lingual nerve. After emerging from beneath the muscle, the nerve continues downwards between the ramus of the mandible and the sphenomandibular ligament into the pterygomandibular space. It enters the **mandibular canal** through the mandibular foramen. The **mylohyoid nerve** arises just before the nerve enters the mandibular foramen.

Once in the mandibular canal, the inferior alveolar nerve runs forwards below the roots of the lower posterior teeth to supply them. When the canal reaches the premolar teeth, the nerve divides. The **mental branch** emerges through the **mental foramen** to supply the skin and labial oral mucosa membrane of the lower lip (see Figure 23.13B). The forward continuation of the inferior alveolar nerve is sometimes referred to as the **incisive branch;** this branch usually stops at the midline but may continue into the opposite side to supply the contralateral incisor teeth. The inferior alveolar nerve carries a few post-ganglionic neurons from the otic ganglion; they pass in the mental branch to supply the minor salivary glands in the lower lip.

The **mylohyoid nerve** is the only motor branch of the posterior division of the mandibular nerve. It runs downwards and forwards in the **mylohyoid groove** on the medial surface of the ramus to pass below the mylohyoid muscle which it supplies; the nerve also supplies the anterior belly of the digastric.

Box 24.4 The lingual nerve and extraction of impacted wisdom teeth

As described in the main text and illustrated in Figure 24.10, the lingual nerve lies very close to the roots of the third mandibular molar (wisdom) tooth. It is at risk during surgical removal of these teeth when they are impacted. An impacted wisdom tooth arises when the lower third molar erupts obliquely or horizontally instead of vertically forwards, thus impacting against the second molar. There is usually insufficient crown of the wisdom tooth showing through the gingivae to enable the operator to place extraction forceps. Under local, or more frequently, general anaesthesia, a flap is made in the mucosa on the lingual side of the mandible over the roots of the third molar. The periosteum over the mandible is carefully reflected then a window is created in the bone. With luck, the whole impacted tooth may be removed intact, but frequently the tooth needs to be cut up and removed piece by piece.

The lingual nerve may be damaged during creation of the mucosal flap and reflection of periosteum in as many as one in ten operations. There will be loss of somatic sensation over the ipsilateral half of the anterior tongue. This is debilitating as the patient has no idea where their tongue is; they, therefore, have difficulty with oral function such as eating and speaking and risk biting their tongue. Despite the chorda tympani also being damaged, taste loss is not usually noticed; the molecules giving food its characteristic taste dissolve in saliva and reach taste buds in areas that are still innervated before the patient would notice. In the vast majority of cases, the lingual nerve is stretched, therefore, sensory loss will return within weeks; the nerve is only actually severed in about one in 1000 cases where the nerve is damaged or 1 in 10 000 operations on lower wisdom teeth.

The otic ganglion

This ganglion is situated immediately below the foramen ovale between the main trunk of the mandibular nerve and tensor veli palatini. It is remarkably small and is unlikely to be found during dissection of this area.

The connections of the ganglion are shown schematically in Figure 24.12. It receives preganglionic parasympathetic axons from the inferior salivatory nucleus via the lesser petrosal branch of the **glossopharyngeal nerve**. These synapse in the ganglion. Post-ganglionic axons pass by a communicating branch to the **auriculotemporal nerve** to form the secretomotor nerves to the parotid gland. A few post-ganglionic axons pass to the main trunk of the mandibular nerve to be distributed through the inferior alveolar and buccal nerves to the mucous glands in the lower lip and cheek. The nerve to medial pterygoid actually passes

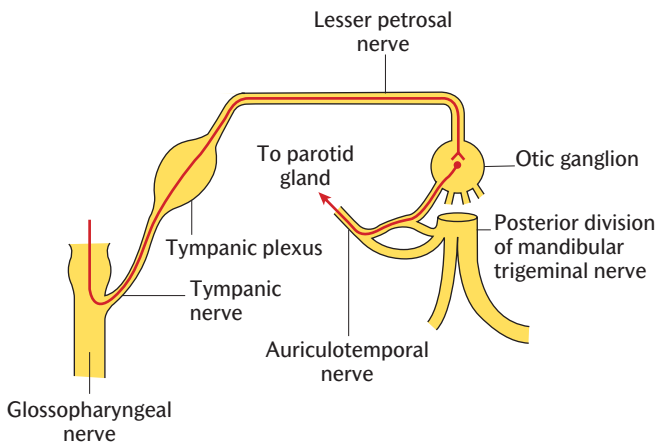


Fig. 24.12 A schematic diagram of the parasympathetic pathway to the parotid gland via the optic ganglion.

through the ganglion without synapsing on its way to supply the tensor veli palatini and tensor tympani.

24.5.2 The maxillary artery

The origin of the maxillary artery as one of the terminal branches of the external carotid artery has already been described in Chapter 23 and illustrated in Figure 23.4. As shown in Figure 24.4, the maxillary artery begins in the parotid gland posterior to the neck of the mandible. It enters the infratemporal fossa by curving forwards deep to the mandibular neck. It then crosses the lower head of the lateral pterygoid to enter the pterygopalatine fossa through the pterygomaxillary fissure; it usually lies superficial, but occasionally deep to the muscle. Recall from Section 23.1.5 the descriptive division of the artery into three parts—before, across, and beyond the lateral pterygoid, each part having five branches. The branches of the maxillary are shown in Figure 24.13.

Branches of the first part

The branches of the first part are the:

1. Deep auricular artery;
2. Anterior tympanic artery;
3. Middle meningeal artery;
4. Accessory meningeal artery;
5. Inferior alveolar artery.

The last branch is really pertinent to dental practice and the third branch may have some significance in oral and maxillofacial surgery. The other branches are trivial; they are summarized in Box 24.5 for those who require this information.

The **inferior alveolar artery** runs downwards in close relationship posterior to the inferior alveolar nerve to supply the lower dentition.

Box 24.5 Minor branches of the first part of the maxillary artery

The first two branches arise from the maxillary artery while it is still in the parotid gland; the **deep auricular artery** supplies the external acoustic meatus lining and the **anterior tympanic artery** the lining of the tympanic cavity. The **accessory meningeal art**ery ascends in front of the middle meningeal artery to enter the skull through the foramen ovale to supply the meninges; it may arise from the middle meningeal artery.

It has identical branches to the nerve; a mylohyoid branch to the muscle before entering the mandibular foramen and a mental branch to the lower lip. As mentioned in Chapter 23, the inferior alveolar artery supplies the teeth and its size changes with age and state of the dentition. It does not contribute significantly to the blood supply of the mandible.

The **middle meningeal artery** provides the principal source of blood to the meninges. It ascends deep to lateral pterygoid, passes between the two roots of the auriculotemporal nerve, and enters the cranial cavity through the **foramen spinosum**. The middle meningeal artery is the artery that is most vulnerable when a blow to the temple occurs. An extradural haemorrhage may result; its effects have already been described in Box 15.3.

Branches of the second part

The second part of the artery has five branches supplying the muscles of mastication and cheek. Two **deep temporal branches** supply the temporalis muscle, the **pterygoid branches** the pterygoid muscles, the **masseteric branch** the masseter muscle; the **buccal branch** accompanies the buccal nerve to supply the structures of the cheek.

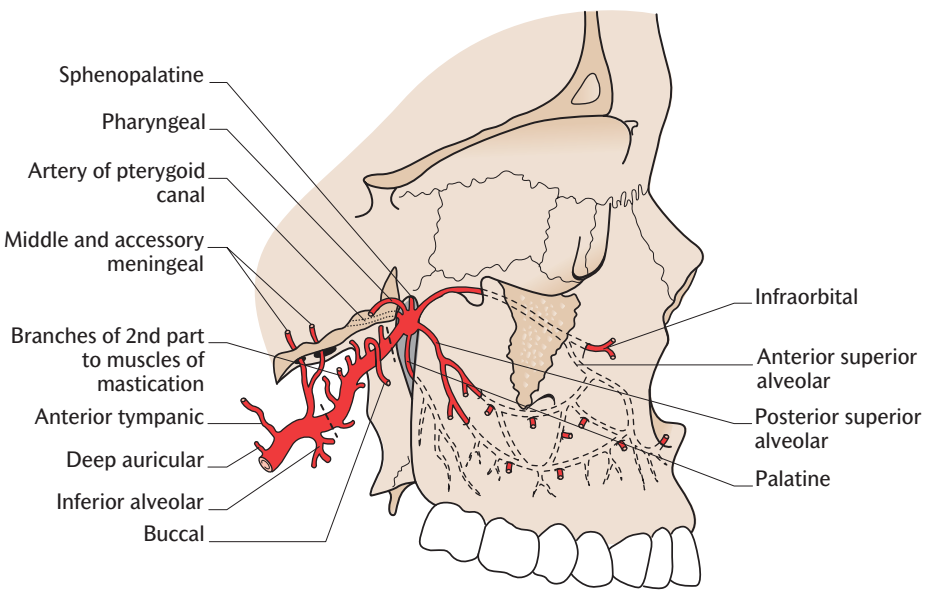


Fig. 24.13 The maxillary artery and its branches.

Branches of the third part

The branches of the third part of the artery arise within the pterygopalatine fossa; they are described in the next section.

24.5.3 The pterygopalatine fossa

As shown in Figure 24.4, the infratemporal fossa connects medially with the pterygopalatine fossa through a narrow triangular gap called the **pterygomaxillary fissure**. The pterygopalatine fossa is a small pyramidal space located just below the apex of the orbit at the junction of the anterior and medial walls of the infratemporal fossa. The fossa opens above through the posteromedial end of the inferior orbital fissure into the orbit. The principal contents of each pterygopalatine fossa are the:

- Maxillary division of the trigeminal nerve;
- Third part of the maxillary artery and its branches;
- Pterygopalatine ganglion.

These structures contribute to the sensory nerve supply, blood supply, and parasympathetic innervation of the upper jaw and associated structures.

Figure 24.4 shows that there are several openings for the passage of nerves and blood vessels through the bones forming the pterygopalatine fossa. The maxillary nerve enters the fossa posteriorly through the **foramen rotundum** shown in Figure 24.8. The nerve passes forwards and laterally across the upper part of the fossa and through the **inferior orbital fissure** to enter the orbit, where it becomes the **infraorbital nerve**. Nasal branches of the maxillary artery and nerve pass through the **sphenopalatine foramen** into the nasal cavity. The **greater palatine canal** opens inferiorly at the junction of the anterior and posterior walls of the pterygopalatine fossa. It runs downwards between the nasal surface of the maxilla and the lateral surface of the palatine bone to open on the palate through the **greater** and **lesser palatine foramina** (see Figure 25.4). The greater and lesser palatine nerves and vessels supplying the mucosa covering the hard and soft palates respectively exit from these foramina as shown in Figure 24.8.

The **pterygopalatine ganglia** are important synaptic sites between parasympathetic preganglionic and post-ganglionic neurons from the facial nerves. The position of the ganglion in relation to the bones and the maxillary division of the trigeminal nerve is shown in Figure 24.11. Its connections are described on p. 255.

The maxillary division of the trigeminal nerve

The maxillary division of the trigeminal nerve is illustrated in Figure 24.14; follow the diagram as you read the description. As you can see, the maxillary nerve enters the pterygopalatine fossa through the **foramen rotundum**. It runs forwards and somewhat laterally across the fossa and enters the orbit through the inferior orbital fissure. It then crosses the floor of the orbit, where it is known as the **infraorbital nerve**, to supply the skin of the face already described in Chapter 23 and illustrated in Figure 23.13. The infraorbital nerve gives off anterior, and sometimes middle, superior alveolar nerves that supply the upper anterior teeth and lining of the maxillary air sinus. Their distribution is shown in Figure 24.14 but is described in more detail in Sections 25.4.1 and 27.4.1.

As you can see in Figure 24.14, two **ganglionic branches** connect the pterygopalatine ganglion to the maxillary nerve within the pterygopalatine fossa. These branches contain post-ganglionic parasympathetic secretomotor neurons that are distributed to their target organs by the maxillary nerve, but most of them are somatic sensory processes of the maxillary nerve that pass through the ganglion without interruption. The principal sensory branches passing through the pterygopalatine ganglion supply the mucosa of the hard and soft palate, the nasal cavity, and upper part of the pharynx.

The **greater palatine nerve** leaves the inferior aspect of the ganglion and descends through the greater palatine canal to emerge through the **greater palatine foramen** on to the oral surface of the hard palate (see Figure 24.8). It then runs forwards in a groove located approximately halfway between the midline of the palate and the palatal surface of the posterior teeth to supply the mucous membrane of the palate as far forwards as the lateral incisor region (see Figure 25.4). **Nasal branches** arise within the greater palatine canal to supply the mucous membrane of the posteroinferior quadrant of the lateral wall of the nose (see also Section 27.3.2).

The **lesser palatine nerve** also descends in the greater palatine canal with the greater palatine nerve but emerges on to the oral surface of the bony palate through the **lesser palatine foramen**. They supply the mucosa covering the soft palate and an area around the palatine tonsil.

The **nasal and nasopalatine nerves** enter the nasal cavity through the sphenopalatine foramen on the medial wall of the pterygopalatine fossa. As their name suggests, they supply the mucosa of the lateral nasal walls, the nasal septum, and a small area of the palate. They are described in Section 27.3.2.

The **pharyngeal nerve** leaves the posterior aspect of the pterygopalatine ganglion and passes through a small canal to be distributed to the mucosa of the **nasopharynx**.

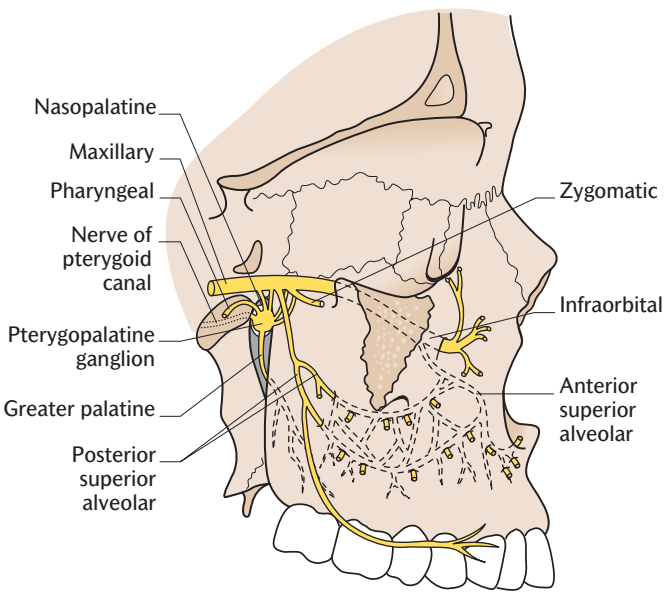


Fig. 24.14 The maxillary nerve and its branches.

As Figure 24.14 illustrates, two or three **posterior superior alveolar nerves** branch off the main nerve more anteriorly. These pass downwards and laterally through the pterygomaxillary fissure on to the anterior wall of the infratemporal fossa formed by the posterior surface of the maxilla. They enter the maxilla through small posterior alveolar foramina and supply the upper molar teeth and the lining of the maxillary sinus. As illustrated by dotted lines in Figure 24.14, some of the branches do not enter the bone, but track down to supply the buccal oral mucosa of the posterior maxillary teeth.

The small cutaneous **zygomaticotemporal** and **zygomaticofacial** branches leave the main nerve trunk as it is about to enter the inferior orbital fissure. Their distribution has already been described in Chapter 23 and illustrated in Figure 23.13.

The pterygopalatine ganglion

This is located deep within the pterygopalatine fossa close to the sphenopalatine foramen as shown in Figure 24.14. It is the synaptic site of parasympathetic secretomotor neurons derived from the facial nerve which are destined for the **lacrimal gland** and the mucous glands in the nasal cavity, nasopharynx, paranasal sinuses, palate, and upper lip.

The connections of the pterygopalatine ganglion are shown schematically in Figure 24.15. Preganglionic parasympathetic fibres from the **superior salivatory nucleus** reach the ganglion through the greater petrosal branch of the facial nerve which fuses with the deep petrosal nerve from the carotid sympathetic plexus to form the **nerve of the pterygoid canal.** It enters the pterygopalatine fossa through the **pterygoid canal.**

Post-synaptic neurons are distributed to the glands associated with the mucosa of the nose, nasopharynx, and palate through the nasal, pharyngeal, and palatine branches described on p. 254. Post-ganglionic neurons pass to the minor salivary glands in the upper lip, upper part of the cheek, and mucous membrane of the maxillary sinus through the maxillary nerve and its infraorbital continuation.

Post-ganglionic parasympathetic neurons to the **lacrimal gland** reach it through a complicated pathway. This passes via the ganglionic branches of the maxillary nerve, the maxillary nerve itself, its zygomatic and zygomaticotemporal branches, a communicating branch to the lacrimal nerve and then via the lacrimal nerve to the gland. Post-ganglionic neurons to the glands in the sphenoidal, ethmoidal, and frontal sinuses run in small orbital branches from the pterygopalatine ganglion.

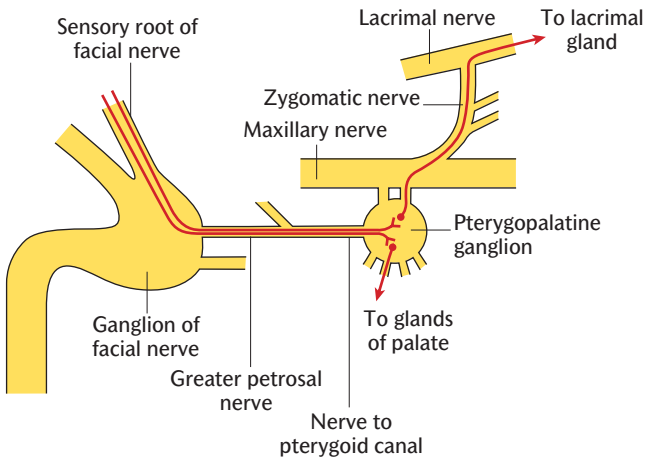


Fig. 24.15 A schematic diagram of the parasympathetic pathways through the pterygopalatine ganglion.

The **deep petrosal nerve** carries vasoconstrictor post-ganglionic sympathetic neurons which pass through the ganglion without interruption to be distributed in its branches.

The third part of the maxillary artery

Figure 24.13 shows the third part of the maxillary artery entering the pterygopalatine fossa through the pterygomaxillary fissure. It passes through the fossa into the inferior orbital fissure to enter the infraorbital fissure in the floor of the orbit, where it is known as the **infraorbital artery**. Its branches are mainly distributed alongside the branches of the maxillary trigeminal nerve covering this area.

As Figure 24.13 illustrates, the **posterior superior alveolar branches** arise from the maxillary artery as it enters the pterygopalatine fossa; they run with the corresponding branches of the maxillary nerve to supply the upper posterior teeth and adjacent structures.

The part of the maxillary artery within the pterygopalatine fossa gives off **palatine, nasal,** and **pharyngeal branches** which accompany the corresponding branches of the maxillary nerve. The **artery of the pterygoid canal** runs posteriorly through the canal to supply the nasopharynx and tympanic cavity.

The veins accompanying these branches of the maxillary artery emerge from the pterygopalatine fossa through the pterygomaxillary fissure and drain into the pterygoid venous plexus.

24.5.4 The pterygoid venous plexus

The infratemporal fossa and most of the structures it contains are drained by a series of veins that form the **pterygoid venous plexus**. As shown in Figure 24.16, this venous plexus lies around and partly within the lateral pterygoid muscle. The tributary veins correspond to the arterial branches of the maxillary artery, although the area drained is somewhat smaller than that supplied by the artery because blood from peripheral cutaneous areas supplied by the artery also drain via other routes such as the facial veins. As you can see in Figure 24.16, the plexus drains through the short wide **maxillary vein** which unites with the superficial temporal vein to form the **retromandibular vein**.

The pterygoid plexus which is part of the deep venous drainage of the head has extensive connections with other components of its venous drainage.

- It communicates with the **facial vein**, part of the superficial venous drainage, through the **deep facial vein** as shown in Figure 23.6.
- Blood from the cavernous sinus enters the plexus via emissary veins passing through the **foramen ovale**.
- **The inferior ophthalmic veins** enter the pterygoid plexus through the inferior orbital fissure.

These interconnections form potential routes for the spread of infection because the veins of the head lack valves so blood can backflow when squeezed by muscular pumps; these are described in Box 24.6.

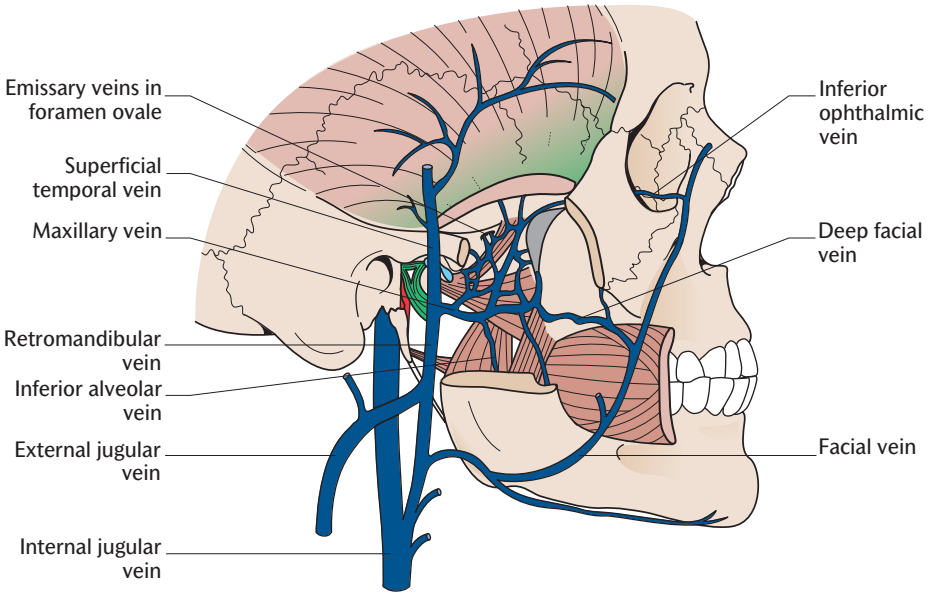


Fig. 24.16 The pterygoid venous plexus.

Box 24.6 Spread of infection from the pterygoid plexus

Potential spread of infection through the venous drainage of the head and neck. The extensive drainage area of the pterygoid venous plexus, including the jaws and teeth, the face and orbit and its numerous connections, forms a potential route whereby infection may spread from any of these sources into the **cavernous sinus**; the consequences of **cavernous sinus thrombosis** have already been covered in Box 15.2.

The plexus is frequently a prominent structure in the living subject but is usually difficult to find in cadavers because it is empty of blood. The plexus may be punctured by the needle delivering local anaesthetic solution during inferior dental nerve blocks (see also Section 25.5.2). Copious bleeding into the surrounding tissues occurs; blood may pass forwards through the inferior orbital fissure into the orbit with production of a 'black eye'.

24.5.5 The styloid apparatus

The **styloid process** is a curved bony spine of variable length which projects downwards and forwards from the inferior surface of the temporal bone lateral to the internal carotid artery. It can be seen on anterior, lateral, and inferior views of the skull as shown in Figures 22.2, 22.3, and 22.4. Its upper part is fused with the tympanic plate and two ligaments and three muscles attach to its lower part. The bone and attached muscles and ligaments form the posterior boundary of the infratemporal fossa.

The **stylohyoid ligament** is a cord of fibrous tissue which runs from the tip of the styloid process to the apex of the lesser cornu of the hyoid bone. The **stylomandibular ligament** is a thickening of the deep layer of the parotid capsule considered to be an accessory ligament of the temporomandibular joint (see Section 24.2.2). It passes from the base of the styloid process and adjacent part of the tympanic plate to the angle of the mandible.

The three muscles attached to the styloid—stylopharyngeus, stylohyoid, and styloglossus—are all members of larger muscle groups involved in swallowing (see Section 29.1.2) and other oral functions. The stylohyoid and styloglossus muscles are described in Chapter 25 and the stylopharyngeus in Chapter 28.

25

The oral cavity and related structures

Chapter contents

25.1 Introduction

Technically, the oral cavity consists of the **vestibule** between the lips and cheeks externally and the teeth and alveolar processes internally and the larger **oral cavity proper** located internal to the dental arches. In clinical practice, the whole mouth is simply referred to as the oral cavity, but 'vestibule' is used for the specific area defined above. For charting of teeth and similar dental procedures, the mouth is divided into quadrants—upper right and left and lower right and left with the midline and occlusal surfaces of the teeth forming the dividing lines.

25.2 Examination of the living mouth

It is a crucial skill for dental students and practitioners to recognize the naked eye appearance of the structures in a normal healthy mouth and variations that occur; abnormal appearances can then be recognized, diagnosed, and treated successfully. Much of the macroscopical appearance is determined by the underlying gross anatomy so this must be understood too. The best way to examine the interior of the mouth is on a subject seated in a dental chair with clinical lighting and the use of a tongue spatula and a dental mirror where necessary. However, you will be able to see most of the important features by examining the inside of your own mouth in a well-lit household mirror.

The following description and illustrations apply to an adult mouth with a full **secondary (permanent) dentition** of two incisors, one canine, two premolars, and three molars in each quadrant, making 32 teeth in total. Apart from size, the major differences in childrens' mouths are in the dentition. The **primary (deciduous) dentition** erupts into the oral cavity between the age of 6 months and 2 years. It comprises two incisors, one canine, and two molars in each quadrant, giving a total of 20 teeth. Most of the teeth of the secondary dentition erupt between the ages of 6 to 12, replacing the primary teeth; a combination of primary and secondary teeth, **a mixed dentition,** is found between these ages. Primary incisors and canines are replaced by their permanent successors, but the deciduous molars are succeeded by the permanent premolars; the three permanent molars in each quadrant are additional teeth.

Teeth may be lost from one cause or another throughout life and many older people have an incomplete dentition. The proportion of elderly subjects who are completely **edentulous** (without teeth) is decreasing as more effective preventative and conservative treatment take effect, at least in developed countries. In edentulous patients, the alveolar processes of both jaws are resorbed to some degree. Resorption may be so great that virtually no alveolar ridge can be seen.

25.2.1 The oral mucosa

Before embarking on the examination of the oral cavity, it is important to realize that the appearance of the oral mucosa is not uniform throughout the oral cavity; there are structural and regional variations. The oral mucosa may be divided into three major types.

Lining mucosa is non-keratinizing stratified squamous epithelium supported by connective tissue, the lamina propria, and submucosa containing minor salivary glands and some fat cells. This type of mucosa is generally reddish pink in colour; small blood vessels in the lamina propria are visible through the epithelium in areas where it is particularly thin. Lining mucosa covers the internal surfaces of the lips and cheeks, the ventral surface of the tongue, floor of the mouth, the soft palate, and alveolar processes up to the gingivae.

Masticatory mucosa, as its name implies, is found in areas subject to masticatory wear and tear, the gingivae, and hard palate. It has an external layer of keratinized stratified squamous epithelium supported by the lamina propria. There is no submucosa in the gingivae; it is also absent from the central regions of the hard palate but present under the rest of its surface. In the gingivae and hard palate lacking a submucosa, the lamina propria is firmly attached to the underlying periosteum by a meshwork of collagen fibres to form a composite structure called **mucoperiosteum**. Masticatory mucosa is thicker than lining mucosa. It is very pale pink in colour and appears almost white in areas of mucoperiosteum due to a greater thickness of epithelium and keratin masking underlying vessels.

Gustatory mucosa covers the dorsal surface of the tongue; its name indicates that it is involved in taste reception. The dorsal surface of the anterior two-thirds of the tongue is also involved in mastication and is, therefore, subject to abrasion. Gustatory mucosa has features in common with masticatory mucosa as well as specializations for taste reception. The epithelium and lamina propria form small projections called papillae; there are four types. The lamina propria is bound by collagen fibres to the underlying muscle and there is no submucosa. The appearance of gustatory mucosa on the tongue is described in Section 25.3.3.

The mucosa covering the posterior third of the tongue is non-keratinized and covers nodules of lymphoid tissue which make up the **lingual tonsil**.

25.2.2 The vestibule

The vestibule is little more than a potential space when the mouth is closed since the lips and cheeks are normally closely applied to the labial and buccal aspects of the teeth and gingivae.

The floor and roof of the vestibule are formed by the reflections of the oral mucosa from the alveolar processes on to the cheeks and lips. The gutters formed by these reflections are called the **sulci** and are shown in Figure 25.1A. They are designated as the upper and lower **buccal sulci** in the region of the cheeks and upper and lower **labial sulci** in the region of the lips.

Pull your upper and lower lips away from your teeth and you will be able to see the **frenulum,** a narrow midline fold of oral mucosa connecting each lip to the corresponding alveolar ridge; the upper

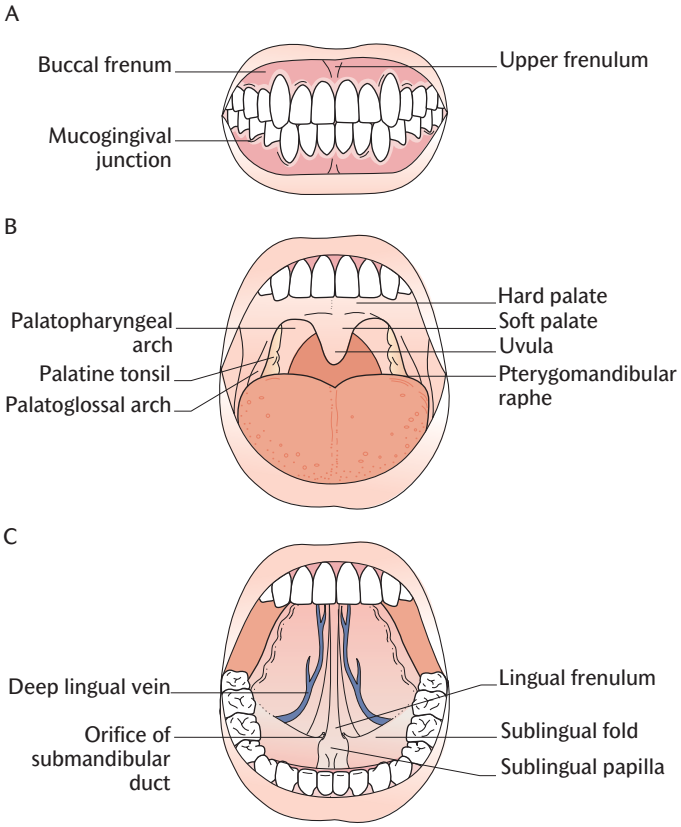


Fig. 25.1 The naked eye appearance of the oral cavity. A) The vestibule; B) The oral cavity with the tongue lowered; C) The oral cavity proper with the tongue raised.

frenulum is usually larger than the lower and both are variable in size. The upper and lower sulci in the premolar regions are usually shallower than elsewhere because of the attachment of the fibres of the lip elevator and depressor muscles to the upper and lower jaw in this area. If you move the lips around, you will see that the folds of mucosa (the **buccal frena**) over these muscle attachments are very mobile. Care has to be taken during denture construction to ensure the margins of the denture base do not impinge on the frenula or muscle attachments, otherwise lip and cheek movements will dislodge it.

The mucosa lining the lips and cheeks and the sulci has a smooth shiny appearance, is reddish pink in colour, and is loosely attached to underlying structures. A short distance from its reflection on to the alveolar processes the mucosa changes to masticatory mucosa which is tightly attached to the underlying periosteum to form a mucoperiosteum. The gingival mucosa is non-mobile and pinkish in colour. The bundles of fibrous tissue connecting the lamina propria to the bone give it a stippled appearance, somewhat like the texture of orange peel. As shown in Figure 25.1A, the distinct **mucogingival junction** demarcates the transition from reddish lining mucosa to pink masticatory mucosa; it is a scalloped line running horizontally 2–3 mm above or below the scalloped gingival margins. It is an important landmark when injecting local anaesthetic solution for local infiltration of maxillary teeth in particular (see Section 25.5.1 below).

Box 25.1 Clinical anatomy of the vestibule

During the reduction of mandibular fractures, it is often necessary to fix the upper and lower teeth together, effectively splinting the mandible. With the teeth in occlusion, the narrow spaces behind the last molars are the only communications between the vestibule and oral cavity proper. Oral feeding can be achieved by passing liquid or semi-solid nutrients through the retromolar spaces using a vessel with a specially designed spout.

A cotton wool roll is usually placed into the upper buccal sulcus during dental procedures; it will lie against the opening of the parotid duct and absorb salivary secretions from the gland.

The duct of one of the buccal or labial glands may be blocked or damaged by minor trauma such as biting the inside of the cheeks or lips; saliva is retained within the gland causing it to swell into a buccal or labial cyst.

A small papilla marks the opening of the **parotid duct** into the vestibule in the upper buccal sulcus opposite the crown of the upper second molar tooth, but this is not always visible. The **buccal** and **labial minor salivary glands** in the submucosa of the lips and cheeks open into the vestibule through ducts which are too small to see. You can feel these glands by running your tongue across the inside of the lower lip.

Other clinical aspects of the vestibule are covered in Box 25.1.

25.2.2 The oral cavity (proper)

With your mouth widely open as illustrated in Figure 25.1B, you will be able to see the roof of the oral cavity formed by the hard and soft palate, but the floor is largely obscured by the tongue. If you now raise your tongue as shown in Figure 25.1C, you will see the floor of the mouth formed by oral mucosa covering the mylohyoid muscles. The **oropharyngeal isthmus** is the junction of the oral cavity with the oropharynx behind. As you can see in Figure 25.1B and hopefully yourself or a willing subject, this is formed on each side by the **palatoglossal arch**, a fold of mucosa running from the side of the soft palate to the side of the tongue and covering the palatoglossus muscle. This fold is also known as the **anterior pillar of the fauces**.

The palate

Structurally the palate has two parts that differ in structure and function. Its features are illustrated in Figures 25.1B and 25.2. The **hard palate** is the bony partition between the nasal and oral cavities and forms roughly the anteriorly two-thirds. The muscular **soft palate** is attached to the posterior border of the hard palate and projects posteriorly, separating the nasopharynx and oropharynx posterior to the nasal and oral cavities, respectively. Four pairs of muscles attach to the fibrous palatine aponeurosis. The oral surface of the soft palate is concave at rest, conforming to the concavity of the bony hard palate. As shown in Figure 25.1B, a small conical process projects downwards from its free posterior margin; this is the **uvula**. The soft palate is highly

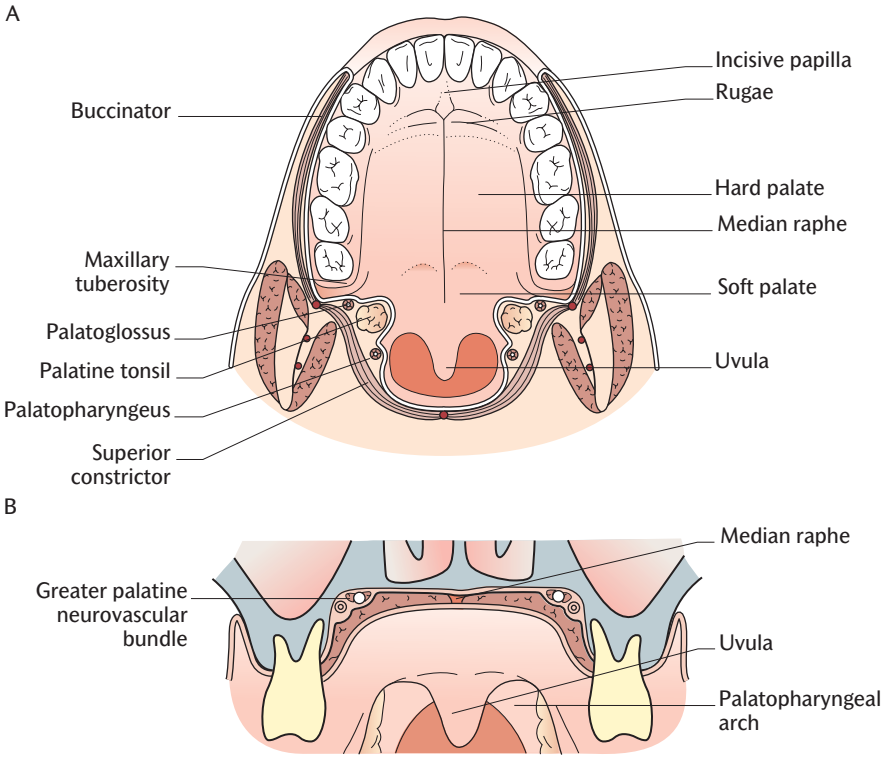


Fig. 25.2 A) An Inferior view of the palate; B) A cross section through the palate.

mobile which you can verify watching the soft palate as you or the subject say 'Aaaa'. When raised, it prevents food and drink entering the nasopharynx and nose during swallowing and also occludes air from the nose during speech; when lowered, the soft palate directs air through the nasal cavity.

As illustrated in Figure 25.2B, the submucous layer in the hard palate varies in thickness from one region to another and is absent altogether in some areas. This variation produces the different appearances in different areas which are easily recognizable in the living mouth. In the **gingival region** adjacent to the teeth and in the midline raphe the submucous layer is absent and the lamina propria of the mucosa and the periosteum blend to form a mucoperiosteum. The mucosa is light pink in colour in these regions and may be almost white in the midline. The palatine raphe ends anteriorly at a small elevation, the **incisive papilla**, overlying the incisive fossa. The submucosa is relatively well developed between the midline area and the gingival region on each side but the lamina propria is still tightly bound down to the periosteum by bands of collagen. The spaces between these bands are filled with adipose tissue in the hard palate anterior to the canine teeth; the mucosa is raised into a number of transverse ridges called the **palatine rugae**. These provide a rough surface against which the upper surface of the tongue can press during preparation of food for swallowing and in speech. Further posteriorly mucous glands fill the spaces between the fibrous bands and the mucosa is thinner and pink in colour.

The visible junction of the hard and soft palate does not coincide exactly with the anatomical boundary between them. There is a narrow non-mobile part of the soft palate where the palatine aponeurosis is attached to the posterior margin of the bone. The **vibrating line** is the junction between the fixed and mobile parts of the palate and may sometimes be seen as a faint transverse groove at rest; the vibrating line is visible when saying 'Aaaa'. This junction is an important landmark during upper denture construction as described in Box 25.2.

The upper alveolar process ends at the **maxillary tuberosity**, a rounded prominence posterior to the last molar tooth.

Box 25.2 How far back should an upper denture be extended?

Ideally, the posterior edge of a full upper denture should be positioned in the narrow non-mobile part of the soft palate between the bone–muscle junction and the vibrating line. If it is placed in front of the junction, it will sit on an area of thin submucosa and will consequently lack peripheral seal and cause discomfort. If it is extended behind the vibrating line, it may cause nausea and is likely to be dislodged by movements of the soft palate.

The tongue

The tongue is much larger than can be appreciated by intraoral examination because it extends backwards into the oropharynx. Even with the tongue fully protruded, it is not possible to see its full extent although careful use of a dental mirror can reveal some additional features. The appearance of the full extent of the tongue is shown in

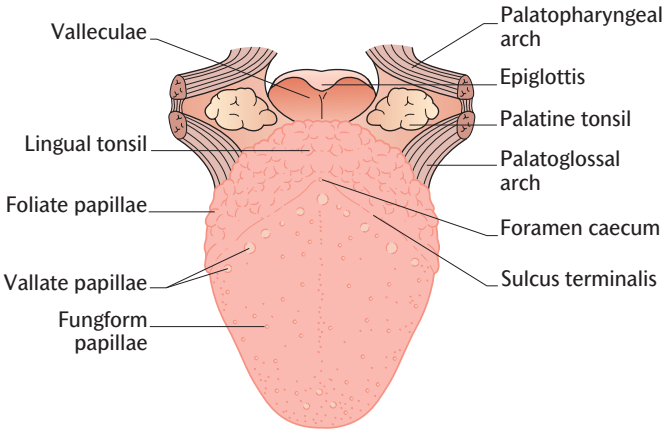


Fig. 25.3 The dorsal surface of the tongue.

Figure 25.3. The dorsum or upper surface is divided by the V-shaped **sulcus terminalis** into anterior two-thirds and posterior one-third although this is not visible in the living mouth without the aid of a dental mirror. As already described in Section 21.7, the parts on either side of the sulcus have different embryological origins and, therefore, nerve supply. As you can appreciate from Figure 25.3, the mucosa coverings of the two areas also differ.

The anterior two-thirds are covered by gustatory mucosa formed into numerous papillae and tightly bound down to the underlying muscle. There are four types of papillae. The most numerous **filiform papillae** give the dorsum of the tongue a pink velvet-like texture. They are 2–3 mm long with pointed ends; secondary papillae point downwards and laterally so that the whole structure resembles a minute fir tree. They are keratinized and provide a rough surface for gripping and manipulating food. The outer layers of epithelium are continuously shed. In illness, this shedding may be delayed and the accumulated tissue mixed with bacteria is seen as a grey coating (fur) on the tongue.

The three other types contain taste buds in their lateral surfaces and the upper surface is keratinized. **Fungiform papillae** have a slightly constricted stalk and a hemispherical upper part which vaguely resembles a mushroom. They appear on the anterior tongue as bright red or sometimes pale spots that are most easily seen towards the sides and the tip. Taste buds are present on fungiform papillae.

As seen in Figure 25.3, there are about twelve large (circum)**vallate papillae** about 2 mm in diameter parallel and immediately anterior to the sulcus terminalis. They are recessed into the oral mucosa and surrounded by a deep circular furrow. The lateral surface of each papilla bears a large number of taste buds. You will not see vallate papillae on a routine oral examination unless you use a dental mirror to examine the back of the tongue. Minor serous salivary glands open into the sulci surrounding the vallate papillae. Their thin watery secretion probably efficiently removes taste molecules from the taste buds on the papillae in readiness for the next mouthful. Similar mechanism is not required for taste buds on the fungiform and foliate papillae as they are more accessible to saliva.

There are several vertical folds on the posterior part of the lateral margins of the tongue termed **foliate papillae** which cannot be observed during a standard intraoral examination. These are rudimentary in humans but well developed in other mammals.

Papillae are absent from the posterior third of the tongue where the covering mucosa membrane is raised into numerous low elevations due to the presence of nodules of lymphoid tissue in the submucous layer. These are known collectively as the **lingual tonsil**.

If you now raise your tongue as shown in Figure 25.1C, you will see that its under surface is covered by thin smooth lining mucosa which is pinkish red in colour and firmly attached to the underlying muscles. A **deep lingual vein** can be seen on each side through the thin mucosa. A median fold of mucous membrane, the **lingual frenulum**, connects the ventral surface of the tongue to the floor of the mouth.

The floor of the mouth

The floor of the mouth is also lined with thin smooth lining oral mucosa similar to that found on the under surface of the tongue. As shown in Figure 25.1C, the **sublingual papillae** are two small elevations located on either side of the attachment of the lingual frenulum to the floor of the mouth. These are the openings of the submandibular ducts. The **sublingual folds** extend posterolaterally from each papilla as a ridge produced by the underlying **sublingual gland**. The gland drains through several tiny ducts opened on to the crest of the fold which are too small to see with the naked eye.

The oropharyngeal isthmus and neighbouring region

As illustrated in Figure 25.1B, the **palatoglossal arches** can be seen most clearly if the tongue is depressed and the soft palate raised by saying 'Aaaa'. The arches cover the palatoglossus muscles which arise from the soft palate and descend to the lateral side of the tongue. You may be able to see a second pair of folds, the **palatopharyngeal arches** (or **posterior pillars of the fauces**), a short distance behind the palatoglossal arches; these overlie the palatopharyngeal muscles extending from each side of the soft palate into the lateral walls of the pharynx. As shown in Figures 25.1B and 25.2, the **palatine tonsils** are submucosal masses of lymphoid tissue between the palatoglossal and palatopharyngeal arches on each side of the isthmus. The size of the palatine tonsils is extremely variable. Generally, they just protrude beyond the cover of the palatoglossal arch but can project much further or remain under cover of the muscle. They may have been removed by tonsillectomy. The surface of the palatine tonsils is pitted by **tonsillar crypts** but these are not always easy to see.

Open your mouth as wide as possible, then palpate lateral to the palatoglossal arch. You will feel the sharp anterior border of the ramus of the mandible. Trace this down towards the last lower molar tooth to the small **retromolar pad** of soft tissue. As illustrated in Figure 25.1B, you should see and feel a shallow groove medial to the ramus, marking the position of the **pterygomandibular raphe**. The raphe begins from the body of the mandible just behind the last lower molar tooth and runs upwards and medially to the hamulus of the medial pterygoid plate. The raphe is an important landmark for administration of inferior alveolar nerve blocks (see Section 25.5.2).

25.3 The anatomy of oral structures

25.3.1 The lips and cheeks

The lips are formed by the **orbicularis muscle** and loose connective tissue; they are covered by hairy skin externally and lining mucosa internally containing mucus-secreting minor salivary glands. The structure of the cheeks is similar with the muscle layer formed mainly by the **buccinator** muscle.

The sensory innervation of the skin and mucosa of the upper lip is from the **infraorbital nerve** and the upper cheek from the **posterior superior alveolar nerves**. The lower lip is innervated by the **mental nerve** and the cheek overlying the lower posterior teeth is supplied by the **buccal nerve** (see Figure 25.17). Secretomotor neurons to the glands in the upper lip and upper cheek pass from the pterygopalatine ganglion in the infraorbital branch of the maxillary nerve. The parasympathetic supply to the glands in the lower lip and lower cheek pass from the otic ganglion via inferior alveolar and buccal branches of the mandibular nerve.

The lips and cheeks are supplied with blood through the **mental artery** and the labial and buccal branches of the **facial artery** and their venous drainage is through tributaries of the **facial vein**. Lymph from the central region of the lower lip drains to the submental nodes; the rest of the lips and cheeks drain to the submandibular nodes.

25.3.2 The hard palate

The skeleton of the hard palate is formed by the palatine processes of the maxillae and the horizontal processes of the palatine bones as shown in Figure 25.4. The **greater palatine nerve** emerges from the greater palatine foramen and runs as far forwards as the canine tooth to supply the palatine mucosa, *including the palatal gingivae* with sensory innervation. As illustrated in Figure 25.4, the nerve runs forwards along a curved line situated in a groove in the bone just medial to where the alveolar processes curve up from the bony plate; the groove is often quite pronounced. The greater palatine nerve also carries parasympathetic post-ganglionic axons from the pterygopalatine ganglion to the palatine glands. The mucosa in the incisor region of the hard palate is innervated by the **nasopalatine nerves** which emerge on to the hard palate through the incisive foramen as illustrated in Figure 25.4. There is usually considerable overlap of the two sensory supplies. The sensory nerves supplying the palatine gingivae require local anaesthesia prior to certain dental procedures that may traumatize them, such as extractions, gingival surgery, and some restorative procedures. Anaesthesia of the palate is covered in Box 25.3.

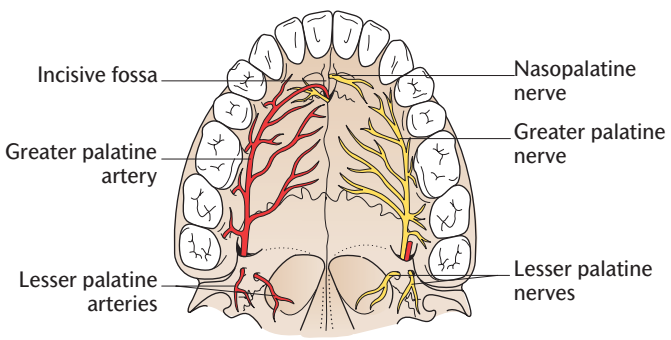


Fig. 25.4 The nerve supply (right) and blood supply (left) of the palate.

The **greater palatine artery** emerging from the greater palatine foramen lateral to the nerve provides the blood supply of the whole of the hard palate; its course is shown in Figure 25.4. Observe how the artery passes into the incisive foramen in the incisor region where it supplies a small area of nasal mucosa. Blood drains from the hard palate through veins accompanying the artery into the pterygoid plexus. The lymph drainage is to the deep cervical nodes.

25.3.3 The tongue

The tongue is a highly mobile, muscular organ which plays a major part in mastication, swallowing, and speech. Its root is attached to the mobile hyoid bone.

The mucosal coverings of the tongue have already been described on p. 261. A few mucous glands are present on the under surface of the tongue near its tip. The glands in the posterior third of the dorsum are mucus-secreting.

The bulk of the tongue is made up of striated muscles. These can be divided into two groups—the **intrinsic muscles** with attachments entirely within the tongue and the **extrinsic muscles** which have one of their attachments outside the tongue. It is often claimed that the principle action of the intrinsic muscles is to alter the shape of the tongue whereas the extrinsic muscles change its position. However, it is virtually impossible to alter tongue shape without altering its position relative to other structures or to alter its position without affecting its shape. The actions of the two groups of tongue muscles are an example par excellence of the group action of muscles.

Extrinsic muscles

There are four pairs of extrinsic muscles—**genioglossus, hyoglossus, styloglossus**, and **palatoglossus**; their general arrangement is shown in Figures 25.5 and 25.6. Palatoglossus is better considered as a muscle

Box 25.3 Local anaesthesia of the palatine nerves

As you can see from Figure 25.2B, the greater palatine nerve runs close to the bone and the overlying mucosa is tightly bound down as mucoperiosteum as described on p. 261. Local anaesthesia in the region of the nerve is, therefore, painful for two reasons. It is difficult to avoid scraping the very sensitive periosteum and the tight mucosa leaves no room for spread of the comparatively small quantity of anaesthetic required. When palatal nerve block is required, the area is first painted or sprayed with topical anaesthetic. Once this has taken effect, an injection may be given if the operative procedure is likely to take time. It is important to make sure that the point of bevel of the needle is on the side away from the bone to minimize the risk of periosteal trauma.

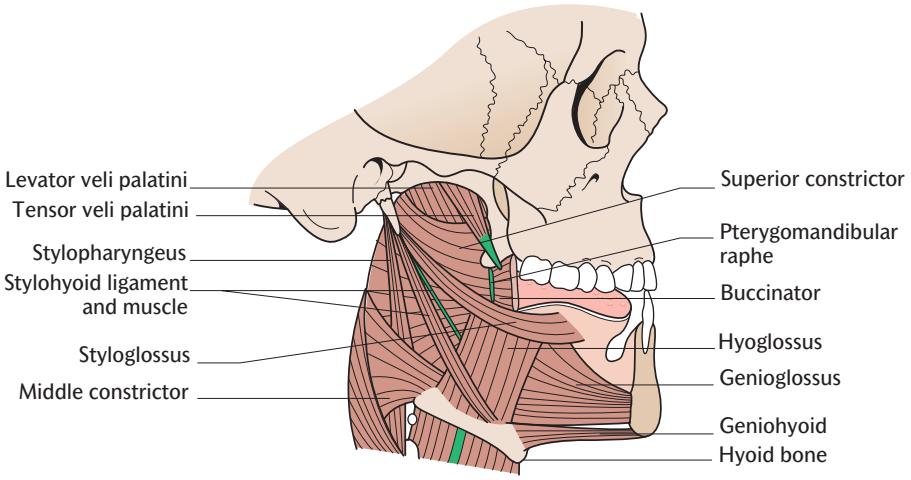


Fig. 25.5 The extrinsic muscles of the tongue and styloid apparatus.

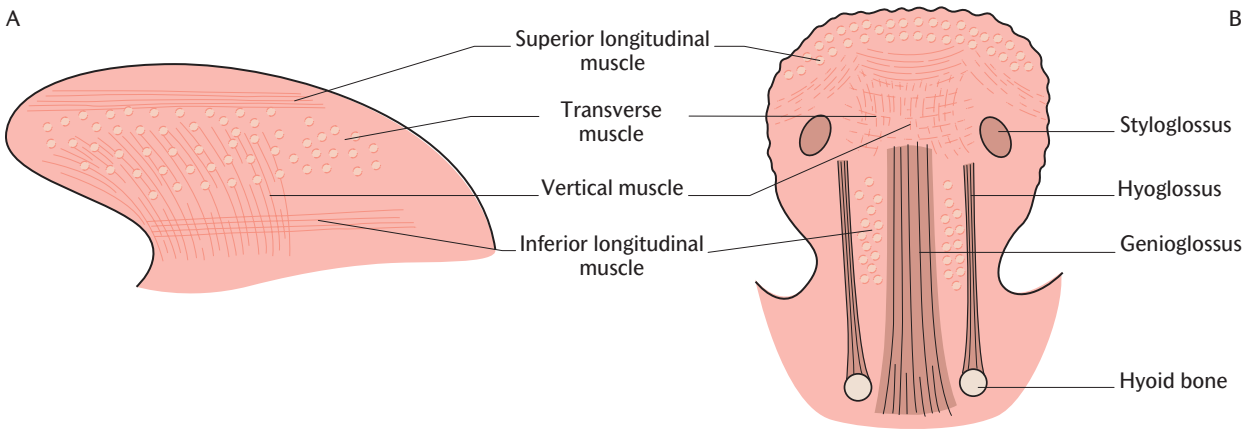


Fig. 25.6 The intrinsic muscles of the tongue. A) Lateral section; B) Transverse section

of the soft palate on the basis of its development, nerve supply, and actions and is described in Section 28.3.1.

The genioglossus muscles form the bulk of the tongue. They lie adjacent to the midline separated from each other by a midline raphe of connective tissue formed from the perimysium of the two muscles. Each one is attached to the upper genial tubercle of the mandible immediately above geniohyoid which arises from the lower genial tubercle. Their fibres run posteriorly from the mandibular attachment, fanning outwards along the whole length of the ventral surface of the tongue from root to tip. From the attachments of the muscles, it is easy to appreciate that they draw the tongue forwards and pull down the central part of the dorsum; in reality, they produce a wide range of tongue movements by interacting with the intrinsic muscles.

As shown in Figure 25.5, each **hyoglossus** muscle is a quadrilateral sheet which arises from the upper surface of the whole length of the hyoid bone; its fibres ascend vertically to insert into the side of the tongue. Its action is to depress the tongue.

Figure 25.5 shows the posterior attachment of each **styloglossus** muscle into the lower end of the styloid process and adjacent part of the stylohyoid ligament. It runs downwards and forwards across the lateral surface of the superior constrictor muscle of the pharynx into the side of the tongue, interdigitating with the fibres of hyoglossus. It draws the tongue upwards and backwards.

Intrinsic muscles

The intrinsic muscles are arranged in four groups on each side of the median fibrous septum as shown in Figure 25.6. Their fibres run in all three dimensions and interweave with the extrinsic muscles; unlike the extrinsic muscles, they cannot be distinguished clearly on the dissected tongue.

The **superior longitudinal fibres** form a band immediately beneath the mucosa of the dorsum of the tongue. They are attached to the mucosa over the posterior part of the tongue and run forwards to the mucosa at its edges and tip. They shorten the tongue and turn its tip and edges upwards, thus making the dorsum concave. The **inferior longitudinal fibres** are in the lower part of the tongue lateral to genioglossus. They shorten the tongue and turn its tip and edges downwards, giving the dorsum a convex shape. The **transverse fibres** arise from the median septum and pass laterally on each side to the sides of the tongue. They narrow and elongate the tongue. The **vertical fibres** run from the dorsum to the ventral surface and flatten and broaden the tongue.

Nerve and blood supply

The multiple nerve supply of different areas of the tongue is a reflection of their developmental origins from the four pharyngeal arches described in Section 21.7.1.

The mucosa of the posterior one-third of the dorsum of the tongue is derived from the third pharyngeal arch and is, therefore, supplied with both taste and somatic sensation by the **glossopharyngeal nerve**. A small area immediately in front of the epiglottis developed from the fourth arch is supplied by the **internal laryngeal branch** of the **vagus nerve**. The glossopharyngeal nerve also supplies taste neurons to the vallate papillae; the vallate papillae are anterior to the sulcus terminalis as shown in Figure 25.3, but they are actually derived from the third arch and migrate across the sulcus during fusion of the anterior and posterior parts of the tongue. The majority of the anterior two-thirds which develops from the first pharyngeal arch receives somatic sensory nerves via the **lingual nerve**, a branch of the **mandibular trigeminal nerve**; neurons conveyed taste reach the CNS via the lingual nerve and chorda tympani branch of the **facial nerve**.

All the intrinsic and extrinsic muscles of the tongue, except palatoglossus, are derived from occipital myotomes and are innervated by the **hypoglossal nerve**. Palatoglossus is a fourth pharyngeal arch derivative and is, therefore, supplied by the vagus nerve.

The course of the lingual, glossopharyngeal, and hypoglossal nerves in the floor of the mouth are described in Section 25.3.4.

The **lingual artery**, a branch of the external carotid artery, is the main source of blood to the tongue. Small lingual branches are also branch off the ascending pharyngeal arteries. The lingual artery is very low in the floor of the mouth near its origin and runs forwards above the greater cornu of the hyoid to turn upwards at the anterior border of the hyoglossus muscle into the ventral surface of the tongue. The venous drainage is via the **lingual** and **deep lingual veins**.

Lymphatic drainage

The lymph drainage of the tongue is complex, but is important because of the tendency for malignant neoplasms to spread through this route. A primary carcinoma in the anterior two-thirds should be obvious to the patient although they are unlikely to make a correct diagnosis. On the other hand, a primary lesion in the posterior one-third of the tongue is not usually apparent to the patient until it has enlarged sufficiently to cause obstruction. It is unlikely to be detected even by their dentist until metastasis causes enlarged regional lymph nodes. The lymphatic drainage is illustrated in Figure 25.7.

Marginal vessels drain lymph bilaterally from the tip of the tongue. They descend under the mucosa covering the ventral surface of the tongue and pierce mylohyoid to drain firstly into the **submental nodes** and then onwards into the **jugulo-omohyoid node** of the deep cervical lymphatic chain. Some of the marginal vessels drain via the submandibular nodes. Central vessels drain the rest of the tongue anterior to the vallate papillae. They descend on each side between the genioglossus and the median fibrous septum. Some pass posteriorly to the **jugulodigastric nodes** while others pierce mylohyoid to enter the **submandibular nodes** and eventually the jugulo-omohyoid nodes. D**orsal vessels** drain the area posterior to the vallate papillae bilaterally into the jugulodigastric nodes.

The pattern of lymphatic drainage has important diagnostic and prognostic implications which are outlined in Box 25.4.

Box 25.4 The lymphatic drainage of the tongue

The fibrous **median septum** of the tongue, like other fascial sheets, is impermeable to fluids; lymph from one side of the tongue will not cross to the other side. However, the septum is thin enough at the tongue tip and in the posterior tongue to permit fluids to move through it. In addition, lymph vessels arising on one side of the tongue tip can cross on its under surface to drain into nodes of the opposite side.

A lingual carcinoma on the tongue tip is, therefore, likely to drain *bilaterally* into the submental nodes and beyond so can quickly metastasize through several nodes. A cancer further back on the anterior two thirds usually only drains unilaterally so likely spread is more confined. Carcinoma on the anterior tongue should be easy to detect by oral examination; like all cancers, the prognosis is good if the lesion in the epithelial layer is detected in its early stages before it has invaded underlying connective tissue to gain access to the lymph vessels. As outlined above, carcinoma on the posterior tongue often goes undetected until it has invaded deeper tissues and metastasized to lymph nodes; once again, lymph from the posterior tongue drains *bilaterally* to lymph nodes that are not so easy to palpate; potential spread is wide and detection may be late making the prognosis relatively poor.

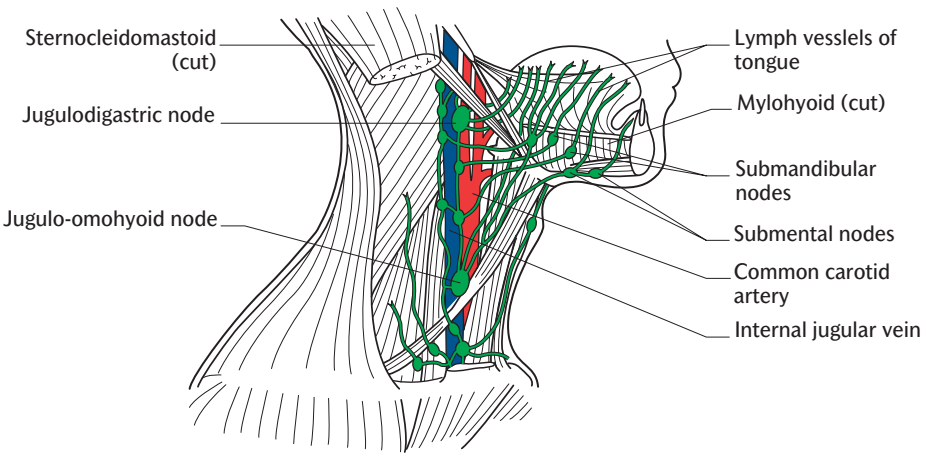


Fig. 25.7 The lymphatic drainage of the tongue into superficial and deep cervical lymph nodes.

25.3.4 The floor of the mouth

The **mylohyoid muscles** form the floor of the mouth as illustrated in Figure 25.9. They are one of the members of the **suprahyoid** group of muscles along with the geniohyoid, stylohyoid, and digastric muscles. They are attached to the **hyoid bone** which is the only skeletal component of the floor of the mouth.

The hyoid bone

Recall from Section 20.3.2 and Figure 20.8 the location of the hyoid in the anterior part of the upper neck, a short distance below the inferior border of the mandible and above the larynx. It has no articulations with other skeletal elements, but is connected to the cranium, mandible, and laryngeal cartilages by ligaments and muscles. The hyoid consists of a **body** and two **greater cornua** (or horns) and two **lesser cornua**.

When viewed from above in Figure 25.8A, the hyoid is U-shaped, with the body forming its base and the greater cornua forming the sides of the U. The body constitutes the anterior part of the hyoid bone and is the part of the hyoid bone that is felt when palpating the anterior midline of the neck (see Section 20.3.2). The greater cornua can also be palpated if you move your fingers laterally; press very gently because pressure on the cornua is quite painful. The small lesser cornua can be seen more clearly in a side view in Figure 25.8B.

The anterior surface of the body is slightly roughened by the attachments of the **geniohyoid** and **mylohyoid** muscles of the suprahyoid group and some of the infrahyoid muscles. The **stylohyoid** muscle and the fibrous sling for the **digastric muscle** attach near the junction of the body and greater cornua. The greater cornua project backwards from each end of the body. The **middle constrictors** of the pharynx and **hyoglossus** muscles are attached to their upper border and the thyrohyoid muscle to the lateral border. The stylohyoid ligaments attach to the lesser cornua.

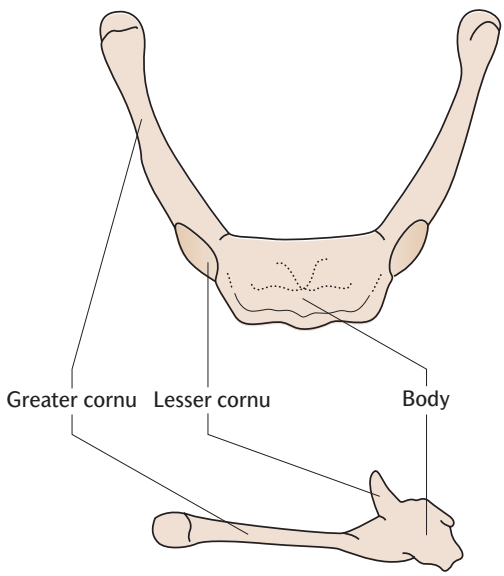


Fig. 25.8 A) Superior; B) lateral views of the hyoid bone.

The suprahyoid muscles

As can be seen in Figure 25.9, each **mylohyoid muscle** is a thin sheet attached along the whole length of the mylohyoid line on the medial aspect of the mandible. It has a free posterior border. The posterior fibres run medially and downwards to the anterior surface of the body of the hyoid bone. The majority of its fibres pass downwards and medially in direction to meet those of the opposite side at a median raphe. The muscles are innervated by the **mylohyoid branch** of the **inferior alveolar nerve**.

Figure 25.9A also illustrates the position of the **geniohyoid muscles** deep to the mylohyoid muscles. Each muscle is a narrow band of muscle fibres attached to the inferior genial tubercle (mental spine) of the mandible anteriorly and the anterior surface of the hyoid bone posteriorly. It lies on the medial part of the upper surface of the mylohyoid muscle in contact with its opposite number. The genioglossus muscles are immediately above them (see Figure 25.5). Each geniohyoid is supplied by the first cervical spinal nerve through a branch of the hypoglossal nerve.

Each **digastric muscle** has two bellies as its name suggests. Follow the location of the muscle in Figure 25.10 as you read the description. The posterior belly arises from the **mastoid notch** of the temporal bone. It passes downwards and forwards towards the hyoid bone where it becomes the intermediate tendon which passes through a perforation in the stylohyoid muscle just above the latter's insertion into the hyoid bone. A fibrous loop is attached to the junction of the body and the greater cornu of the hyoid holds the tendon in place. The intermediate tendon becomes the anterior belly as it passes upwards and

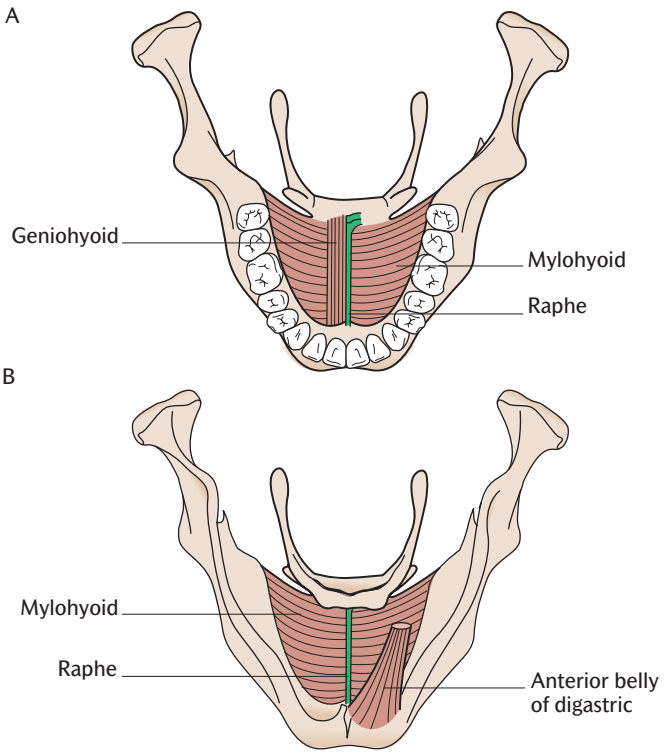


Fig. 25.9 The mylohyoid muscles. A) A superior view, including the geniohyoid muscle; B) A view from below and behind, including the anterior belly of the digastric.

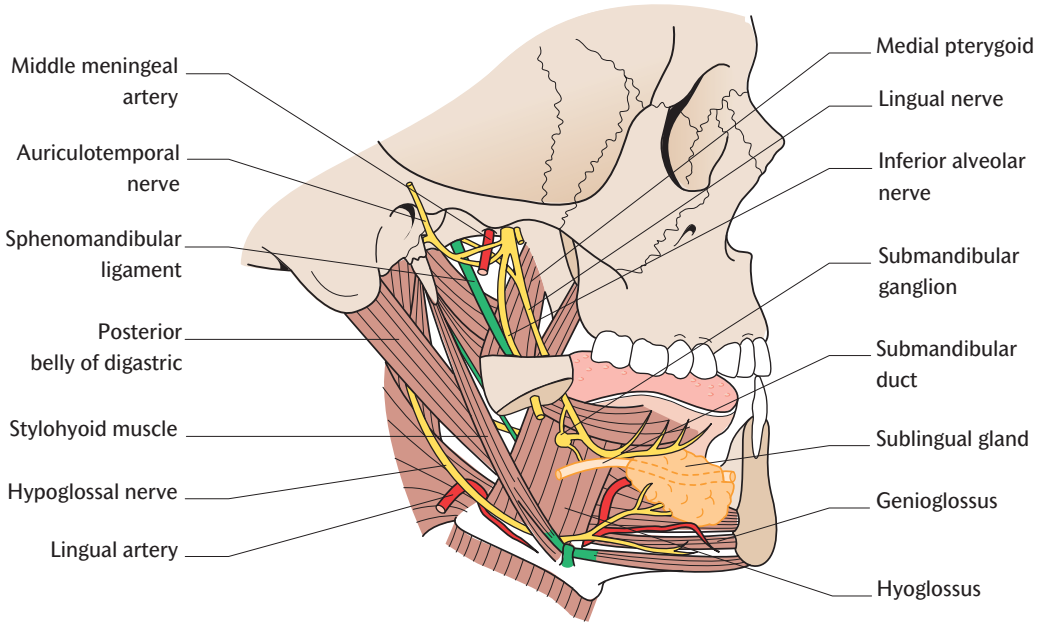


Fig. 25.10 Lateral view of the floor of the mouth and its contents. The mylohyoid muscle and submandibular gland have been removed for clarity.

forwards beneath the mylohyoid. Its attachment to the **digastric fossa** on the lower border of the mandible is clearly shown in Figure 25.9B. The posterior belly is derived from the second pharyngeal arch and is therefore innervated by a branch of the **facial nerve**; the origin of the anterior belly is the first arch so it is supplied by the mylohyoid branch of the inferior alveolar nerve from the mandibular trigeminal nerve.

Figure 25.10 shows the **stylohyoid muscle** attached superiorly to the **styloid process**. The muscle divides into two slips passing either side of the intermediate tendon of the digastric to attach to the base of the greater cornu of the hyoid bone.

The suprahyoid muscles elevate the floor of the mouth and hyoid and with it the laryngeal skeleton, a very important movement during swallowing (see Section 29.1.2). The hyoid bone can be fixed when the suprahyoids contract in concert with the infrahyoid muscles already described in Section 23.1.3; the suprahyoids can then act from the fixed hyoid to pull down on the mandible to open the mouth working with the lateral pterygoid muscles (see Section 24.4.3).

Salivary glands in the floor of the mouth

Two of the three pairs of major salivary glands are in the floor of the mouth. The **sublingual gland** is entirely in the floor of the mouth whereas the **submandibular gland** lies partly in the floor of the mouth and partly in the neck. The sublingual gland secretes mucous saliva whereas the submandibular gland is a mixed gland secreting both mucous and serous saliva. The parotid glands, the third pair of major glands, have already been described in Section 23.2.3.

The submandibular glands

As you can see in Figure 25.11, each submandibular gland consists of a large **superficial lobe** situated in the neck superficial to the mylohyoid muscle and a small **deep lobe** lying in the floor of the mouth between mylohyoid and the hyoglossus muscle on the lateral aspect of the tongue. Figure 25.11 shows the two lobes are continuous with each other around the posterior free border of the mylohyoid muscles.

The lateral surface of the **superficial lobe** is in the shallow submandibular fossa below the mylohyoid line on the medial surface of the mandible. The facial artery runs downwards between the superficial lobe and the medial surface of the mandible before turning upwards across the face at the lower border of the mandible. The facial vein also crosses the gland.

The smaller **deep lobe** lies in the narrow gap between the mylohyoid and hyoglossus muscles. It extends as far forwards as the second molar tooth to meet the posterior pole of the sublingual gland. The **lingual nerve** is above and the **hypoglossal nerve** below it.

The **submandibular duct** begins in the superficial lobe and curves around the posterior border of mylohyoid through the deep lobe to emerge from its anterior surface. It then runs forwards firstly medial to the lingual nerve and then lateral to it as the nerve arches up into the tongue. It continues anteriorly between the sublingual gland laterally and the genioglossus muscle medially to open into the mouth through the **sublingual papilla**.

The sublingual glands

These are the smallest of the three major salivary glands. As you can see in Figure 25.11, each one is situated between the mylohyoid laterally and the genioglossus medially in front of the deep lobe of the submandibular gland. The gland is quite variable in size; it extends from approximately opposite the second molar to the premolar region. Its upper surface is directly under the lining mucosa of the floor of the mouth which is raised as the **sublingual fold**. The **lingual nerve** and **submandibular duct** cross the medial surface of the gland. The gland opens onto the surface of the sublingual fold through about 15 small ducts; other ducts probably join the submandibular duct.

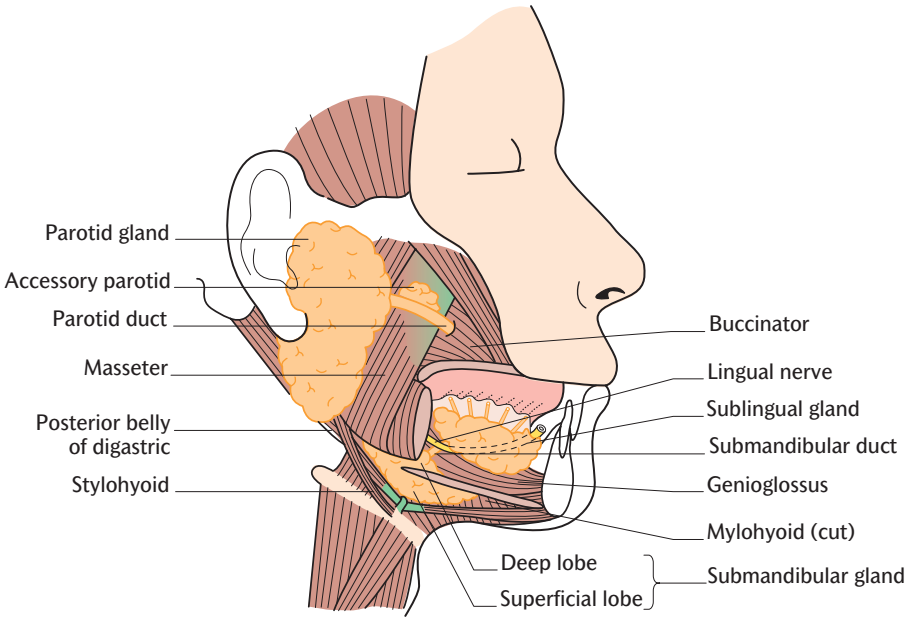


Fig. 25.11 The major salivary glands.

Branches of adjacent arteries supply blood—the facial and lingual arteries to the submandibular gland and the sublingual branch of the lingual artery to the sublingual gland. Their venous drainage is through corresponding veins.

As illustrated in Figure 25.12, the innervation of the submandibular and sublingual glands is through the **submandibular ganglion**. Each ganglion is suspended from the lingual nerve as it crosses hyoglossus by short anterior and posterior ganglionic branches. Preganglionic parasympathetic neurons begin in the **superior salivatory nucleus** in the brainstem and travel via the **chorda tympani** of the facial nerve and **lingual nerve** to synapse in the ganglion. Post-ganglionic neurons pass to the submandibular gland through direct glandular branches from the ganglion. Those supplying the sublingual gland and the small glands of the floor of the mouth and tongue rejoin the lingual nerve to be distributed through its branches. Sympathetic nerves from the superior cervical ganglion enter the ganglion from the sympathetic plexus around the facial artery. They pass through the ganglion without synapsing again to run to the glands with the parasympathetic nerves.

The clinical anatomy of the submandibular and sublingual glands are covered in Box 25.5.

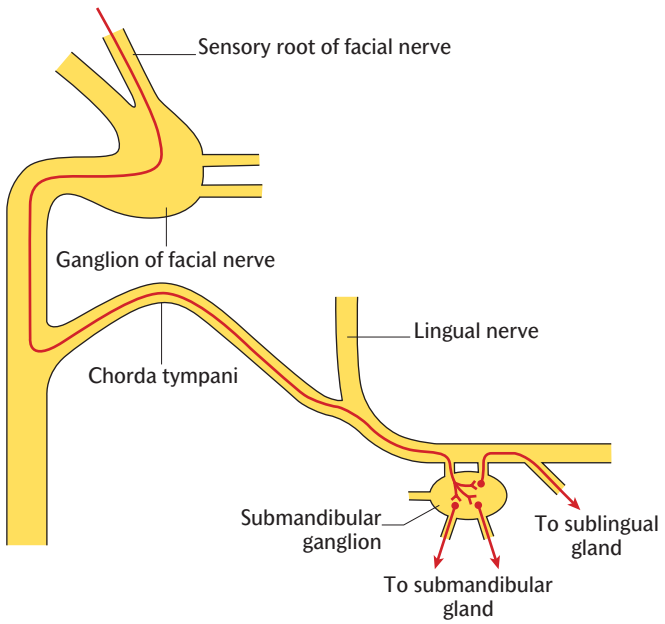


Fig. 25.12 The parasympathetic pathways through the submandibular ganglion.

Box 25.5 Clinical anatomy of the submandibular and sublingual glands

The submandibular and sublingual glands may be involved in viral or bacterial infections and in stone formation as described for the parotid glands (see Box 23.8). For unknown reasons, about 80% of all **salivary calculi** form in the submandibular gland and duct. Calculi block salivary flow which leads to enlargement and tenderness of the gland which are usually worse at mealtimes because of increased salivary secretion. The most common sites for calculi to lodge within the submandibular gland are where the duct passes from deep to superficial lobe round the free border of the mylohyoid and at the duct opening in the sublingual papilla. Calculi can often be expelled from the papilla by gentle palpation along the duct; surgical removal may be necessary if manipulation fails.

Radiotherapy applied to malignant cancers in the head and neck is liable to damage all the major salivary glands; improved radiotherapy technology does minimize collateral damage but it still occurs. If the damage is severe enough, patients will experience **xerostomia** already described in Box 3.5.

Nerves in the floor of mouth

Three important nerves—the **lingual, glossopharyngeal**, and **hypoglossal nerves**—pass through the floor of the mouth to innervate the tongue and other structures.

The lingual nerve

The early course of the lingual nerve has already been described in Section 24.5.1. Recall that the lingual nerve contains somatic sensory trigeminal processes and also carries taste and preganglionic parasympathetic processes from the **chorda tympani** of the facial nerve which join it in the infratemporal fossa. The lingual nerve enters the mouth by passing beneath the lower margin of the superior constrictor muscle close to the mandibular attachment of the pterygomandibular raphe. It lies on the medial surface of the mandible overlying the roots of the third molar (wisdom) tooth. It then runs forwards, downwards, and medially in a smooth curve across the gap between mylohyoid and hyoglossus to reach the lateral surface of the hyoglossus. As shown in Figure 25.12, the **submandibular ganglion** is suspended from it by its ganglionic branches as it continues forwards lateral to the submandibular duct. At the front edge of hyoglossus, it crosses beneath the submandibular duct from its lateral to its medial side. It then runs forwards and upwards between the sublingual gland and genioglossus and divides into its terminal branches which supply somatic sensation and taste processes to the anterior two-thirds of the tongue. The lingual nerve also carries sensation from the lining mucosa of the floor of the mouth and the lingual gingivae of the mandibular teeth through branches given off as the nerve traverses the floor of the mouth.

Glossopharyngeal nerve

Each glossopharyngeal nerve leaves the cranial cavity through the anterior compartment of the **jugular foramen**. The **tympanic branch** leaves the nerve within the jugular foramen and passes through the petrous temporal bone to reach the tympanic cavity where it forms the **tympanic plexus** and supplies the lining mucosa. This branch also contains parasympathetic preganglionic neurons which continue from the tympanic plexus in the **lesser petrosal nerve** to the **otic ganglion** to innervate the parotid gland (see Section 23.2.3).

The course of the glossopharyngeal nerve is outlined in Figure 25.13. It passes downwards and forwards between the internal and external carotid arteries, closely applied to the **stylopharyngeus muscle**—the only one which it supplies—and enters the pharynx by passing between the superior and middle constrictor muscles. It then runs forwards deep to the upper part of hyoglossus and the medial side of styloglossus to supply the posterior part of the tongue through somatic sensory, taste, and secretomotor nerves. The **pharyngeal branches** conveying sensory and preganglionic parasympathetic neurons to the pharynx will be described in Section 28.2.3. Its **carotid branch** supplies the carotid sinus and body as already described in Section 18.9.

The hypoglossal nerve

The hypoglossal nerve exits the **hypoglossal canal** and follows a curving high in the neck as shown in Figure 25.13 It passes between the internal jugular vein and the external and internal carotid arteries to enter the mouth above the posterior border of mylohyoid. It runs forwards on the lateral surface of hyoglossus deep down in the floor of the mouth just a short distance above the greater cornu of the hyoid. At the anterior margin of the muscle, it divides into several branches which run into the individual tongue muscles. The deep lingual vein accompanies the nerve across hyoglossus.

Blood vessels in the floor of the mouth

The lingual artery

The lingual artery is the main blood supply to the tongue and floor of the mouth. It is visible in Figure 25.10. It leaves the anterior surface of the

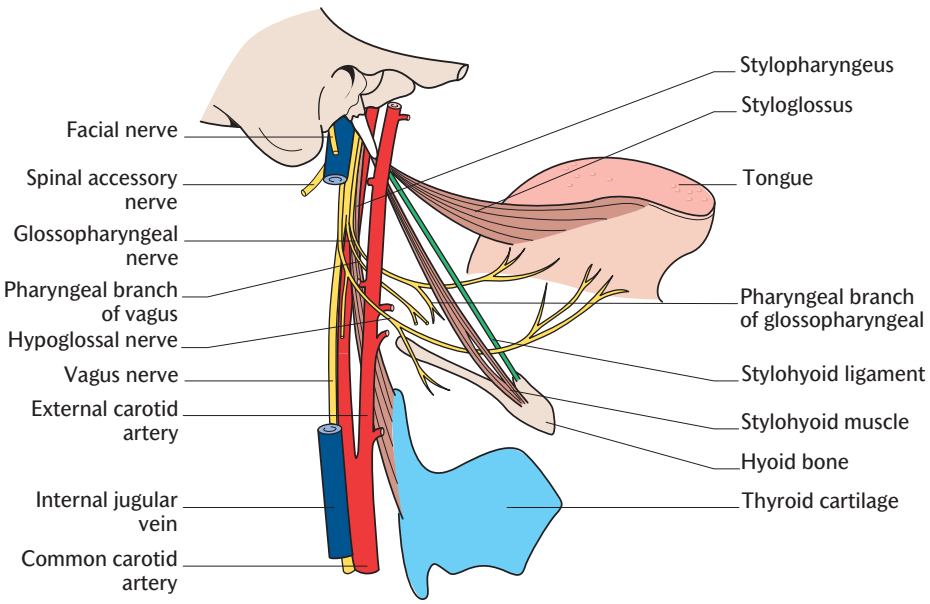


Fig. 25.13 The glossopharyngeal and hypoglossal nerves.

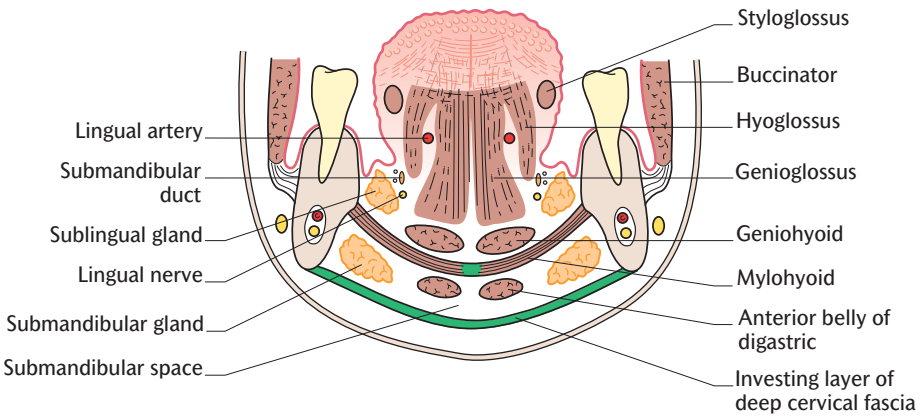


Fig. 25.14 The tissue spaces in the floor of the mouth in transverse section.

external carotid opposite the tip of the greater cornu of the hyoid. The artery forms a characteristic loop running upwards and then descending again to the level of the hyoid. The artery then enters the floor of the mouth by running deep to hyoglossus. The **sublingual branch** leaves the main artery at the anterior border of hyoglossus and runs forwards to supply the sublingual gland and the muscles of the floor of the mouth. The main trunk turns upwards and then runs forwards just beneath the mucosa on the inferior surface of the tongue close to the frenulum accompanied by the lingual nerve and deep lingual vein. The artery supplies branches which penetrate and supply the substance of the tongue.

Veins

There are two main veins in the floor of the mouth. The **deep lingual veins** begin near the tip of the tongue. Theses are the veins that can be seen through the mucosa of the ventral side of the tongue. They accompany the lingual arteries on the inferior surface of the tongue. At the anterior border of hyoglossus, they are joined by the sublingual veins from the sublingual gland and the floor of the mouth, then run lateral to the muscle close to the hypoglossal nerve. They end by draining into the facial, lingual, or internal jugular vein. Several dorsal lingual veins drain the bulk of the tongue; they join to form the **lingual veins** which run with the lingual arteries deep to hyoglossus and drain directly into the internal jugular vein.

Tissue spaces in the floor of the mouth

Untreated carious lesions will eventually infect the dental pulp of the affected tooth and a dental abscess will form. The abscess will exude from the root apex into the periodontal tissues. Pus will usually track through the alveolar bone of the tooth socket and appear as a 'gum boil' on the gingivae. If pus tracks laterally, the abscess will usually point into the buccal sulcus. If pus moves medially, its exit depends upon whether the pus leaves the bone above or below the attachment of the mylohyoid muscle to the mandible as you can see from Figure 25.14. Pus above the attachment may enter the floor of the mouth or the **submandibular space** if below it. Recall from Figure 22.12 the mylohyoid line slopes downwards as it passes forwards; an abscess from the posterior teeth is more likely to open below mylohyoid whereas one originating in the anterior teeth will point above mylohyoid in the floor of the mouth. The tissue space above mylohyoid is enclosed by the muscle below, by the lining mucosa of the floor of the mouth above, and the body of the mandible laterally and anteriorly. Posteriorly, it communicates with the tissue spaces in the neck which were described in Section 23.1.2. As you can see from Figure 25.14, the genioglossus and geniohyoid muscles and the median septum of the tongue form an effective barrier to the spread of infection across the midline from one side to the other. Mylohyoid roofs the submandibular space, the investing layer of the deep cervical fascia forms its floor, and the mandibular body forms the lateral and anterior walls. This space too communicates posteriorly with the tissue spaces in the neck. The potential consequences of spread of infection from the tissue spaces of the floor of the mouth is outlined in Box 25.6.

Box 25.6 Infections in the submandibular space

The submandibular space is surprisingly distensible and can hold a considerable volume of pus or other fluids. It is equally surprising that patients will tolerate severe discomfort and disfigurement, not to mention pain, to allow the submandibular space to expand so grossly when dental treatment and antibiotics are available. In developing countries, access to treatment or suitable antibiotics may not be available and the patient may have no alternative but to endure the infection and pain. As indicated in the main text, the submandibular space communicates with the fascial spaces in the neck and potentially with the pericardial space around the heart. This is the big danger; in cases where treatment is not readily available, infection arising from tissues within the oral cavity can spread into the pericardium which will adversely affect heart function with potentially fatal consequences.

25.4 The nerve supply, blood supply, and lymphatic drainage of the teeth and their supporting tissues

The maxillary teeth, their supporting structures (the alveolar bone of the tooth socket and the periodontal ligament), and the covering gingivae are supplied by branches of the maxillary nerve and artery. The mandibular teeth and supporting structures are innervated by branches of the mandibular nerve whereas their blood supply is still derived mainly from the maxillary artery.

25.4.1 The maxillary dentition

Nerve supply

The **maxillary nerve** and its major branches has been described and illustrated in Section 24.5.3 and Figure 24.14. Recall that the nerve crosses the pterygopalatine fossa and enters the orbit through the inferior orbital fissure when it becomes the **infraorbital nerve.** Figure 25.15 illustrates the specific branches which innervate the teeth and supporting tissues; follow the diagram as you read the description.

Two or three **posterior superior alveolar nerves** leave the maxillary nerve while it is still in the pterygopalatine fossa. They pass through the pterygomaxillary fissure on to the posterior surface of the maxilla where they enter the bone through posterior alveolar foramina. The upper branches pass along the posterolateral walls of the **maxillary sinus** to supply its respiratory mucosal lining. The lower branches run through bony canals in the posterior part of the maxilla just above the tooth root apices and send branches to supply them. Some branches do not enter the bone but continue downwards to supply the buccal gingivae of the upper posterior teeth.

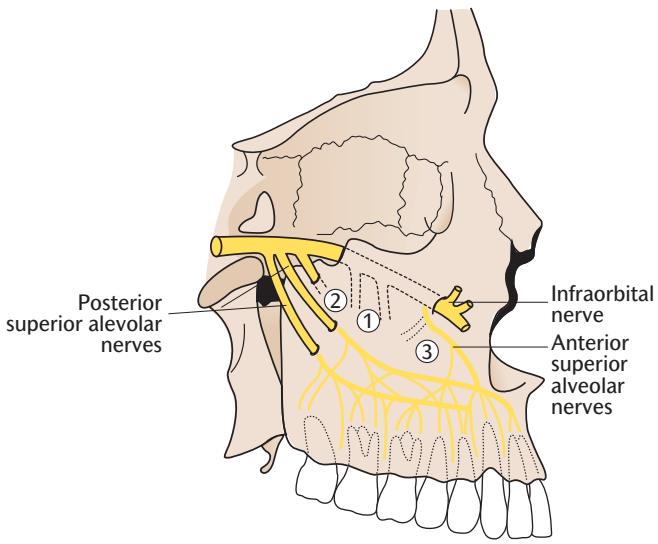


Fig. 25.15 The nerve supply of the upper teeth and their variations. In 100 specimens studied, the middle superior alveolar nerve was a branch of the infraorbital nerve (position 1) in 54, the maxillary nerve (position 2) in 11, and the anterior superior alveolar nerve (position 3) in 17; the nerve was missing in the remaining 18 specimens. Based on FitzGerald 1956, *J. Anat*. 90, 520–2.

Each **infraorbital nerve** runs forwards in the floor of the orbit in the infraorbital groove and canal to emerge into the face through the infraorbital foramen. **Middle** and **anterior superior alveolar nerves** branch from the infraorbital nerve in its course through the floor of the orbit which also forms the roof the maxillary sinus. As shown in Figure 25.15, the **anterior superior alveolar nerve** branches from the infraorbital nerve just before it emerges from the infraorbital foramen; the anterior superior alveolar nerve travels down in a canal on the anterior wall of the maxillary sinus in the maxilla to supply the upper anterior teeth.

There is considerable variation in the origin of the **middle superior alveolar nerves** which is shown in Figure 25.15; it may branch from the main maxillary nerve, the infraorbital nerve, or anterior superior alveolar nerve. It runs downwards and forwards in the posterior, lateral, or anterior wall of the maxillary sinus, depending upon its point of origin from the infraorbital nerve to innervate the premolars and first molar. The middle superior alveolar nerve is absent in about 18% of subjects.

The superior alveolar nerves form the **superior alveolar plexus** within the maxillary alveolar process a short distance above the root apices of the upper teeth. The part of the plexus formed by the **posterior superior alveolar nerves** supplies the molar and premolar regions while that formed by the **anterior superior alveolar nerve** supplies the canine and incisor regions. The **middle superior alveolar nerve** when present innervates the premolar teeth and usually the mesiobuccal root of the first molar; this tooth then is innervated by the middle and posterior alveolar nerves, but this is not clinically significant (see Section 25.6). Note that *all* the superior alveolar nerves supply the mucosa lining the **maxillary sinus** as they pass down to the teeth.

The buccal gingivae are supplied by branches of the **posterior superior alveolar nerves** as mentioned already and the labial gingivae receive their sensory innervation from the superior labial branches of the **infraorbital nerve**. As already described in Section 25.3.2, the gingival mucosa covering the palatal aspect of the upper alveolar process is innervated by the **greater palatine** and **nasopalatine nerves**.

Blood supply

The upper teeth receive their blood supply from **posterior and anterior superior alveolar branches** of the **maxillary artery** which follow the course of the corresponding nerves. The blood supply to the gingivae is also through branches of the maxillary artery corresponding to their nerve supply.

The venous drainage of the upper dentition is by veins which drain into the **pterygoid plexus**.

25.4.2 Mandibular dentition and supporting tissues

Nerve supply

The lower teeth and periodontal tissues are innervated by the **inferior alveolar nerves**. To recapitulate Section 24.5.1, they are branches of the posterior trunk of the mandibular nerves which enter

the mandible through the mandibular foramina. As illustrated in Figure 25.16A, each nerve runs within the mandibular canal below the roots of the posterior teeth as far forward as the mental foramen. The **incisive nerve** is the forward continuation within the mandible which supplies the canine and incisor teeth. The **mental nerve** emerges through the foramen to supply the skin of the lower lip and chin, the labial mucosa, and labial gingivae of the lower anterior teeth. The lingual gingivae of the mandibular teeth are innervated by the **lingual nerve**. The buccal gingivae of the posterior teeth are supplied by the **buccal nerve**, the only sensory branch of the anterior division of the mandibular nerve.

The inferior alveolar nerve has several variations which are described and illustrated in Box 25.7.

Blood supply

The inferior alveolar nerve and its branches are accompanied by the **inferior alveolar artery**, a branch of the first part of the maxillary artery. The lingual gingivae receive their blood supply from the **lingual artery**.

Venous drainage from the lower dentition and associated structures is through vessels which accompany the arteries and drain into the **pterygoid plexus**.

Box 25.7 Variations in the inferior alveolar nerve

There are variations in the arrangement of the inferior alveolar nerve as shown in Figure 25.16. Most frequently, it is distributed as described in the main text. It runs forwards as a single trunk immediately below the apices of the roots of the molar teeth; branches to the posterior teeth are then short and direct. Less commonly, the main trunk of the nerve is situated more inferiorly in the body of the mandible, close to its lower border and some distance below the roots of the teeth as illustrated in Figure 24.16B. In these cases, the dental branches are much longer and slope obliquely upwards and forwards to unite and form an alveolar plexus supplying the molar and premolar teeth. In other cases (see Figure 24.16C), the inferior alveolar nerve gives off large branches soon after entering the mandible that form an alveolar plexus below the posterior teeth; the main trunk of the nerve continues forwards in a more inferior plane to give off the mental nerve and supply the anterior teeth.

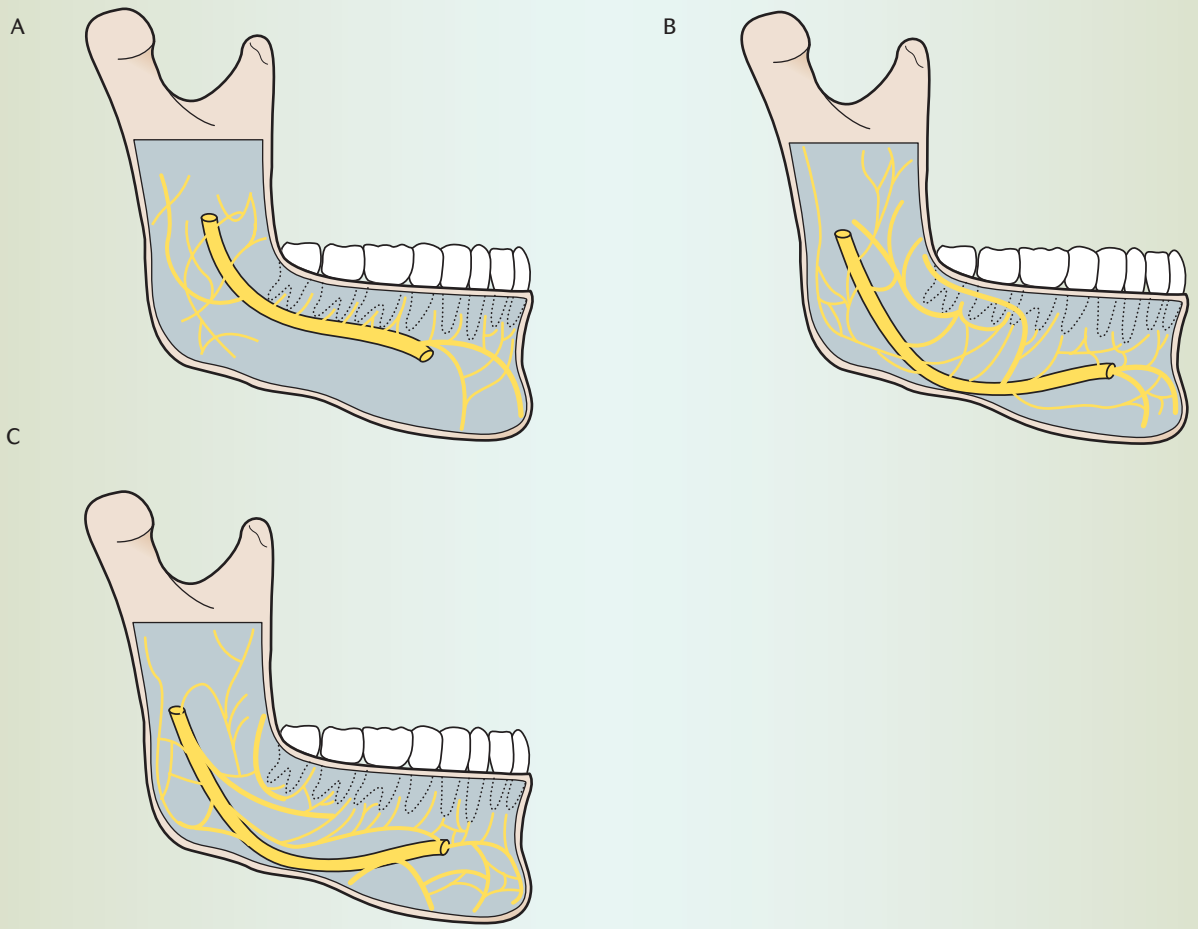


Fig. 25.16 The nerve supply of the lower teeth and its variations. (Based on Carter and Keen 1971, *J. Anat.* 108, 433–40).

Box 25.8 The inferior alveolar nerve and extraction of lower molars

As you can see in Figure 25.16A, the mandibular canal is relatively close to the root apices of the mandibular molar teeth. In some cases, the roots of the second and third molar teeth actually project into or below the mandibular canal. If either of these teeth requires extraction, the relationship of the roots to the mandibular canal should be checked on a panoramic radiograph (see Section 31.2). If the roots do project into or below the canal, surgical extraction is usually carried out to avoid potential damage to the inferior alveolar nerve within the canal that might be caused by forceps exodontia.

25.4.3 Lymphatic drainage of oral cavity

As described in Chapter 4, infections and malignant neoplasms tend to spread through the lymphatic system. Knowledge of the regional lymph drainage of the structures of the oral cavity is essential for dental students and practitioners. The oral cavity drains mainly to the **submental, submandibular**, and **deep cervical nodes.** Remind yourself of where these nodes are and where they may be palpated by rereading the relevant parts of Sections 20.3.2 and 23.2.8; look at Figures 23.14 and 23.15 and Figure 25.7 as well.

The **submental nodes** drain a wedge of tissue comprising the lower anterior teeth, the central parts of the lower lip, a small area of the floor of the mouth behind the incisor teeth, and the tip of tongue. The submental nodes usually drain directly into the **jugulo-omohyoid nodes** of the deep cervical chain but may drain through the submandibular nodes into the deep cervical chain.

The **submandibular** nodes drain extensive areas of the oral cavity. The rest of the mandibular dentition and supporting tissues, the rest of the floor of the mouth, the lateral part of the lower lip, cheek, upper lip and upper anterior teeth, and the anterior two thirds of the tongue drain through these nodes. The submandibular nodes drain into **deep cervical nodes** at various levels; more posterior structures tend to drain into higher nodes such as the **jugulodigastric nodes** and more anterior tissues into the **jugulo-omohyoid nodes**.

Lymph from the upper posterior teeth, the hard and soft palates drain to the **retropharyngeal** nodes and finally into the upper **deep cervical nodes**.

25.5 The anatomy of dental local anaesthesia

There are two techniques used for administration of local anaesthesia which are entirely dependent on the accessibility or otherwise of the nerves which the dentist wishes to anaesthetize prior to operating on a given tooth. If the nerves are accessible close to the target tooth, then **infiltration anaesthesia** can be used. If the nerves are not accessible, anaesthetic must be administered to block neurotransmission at a point where the nerves are accessible; this is a **nerve block**. The point at which a nerve is blocked may be considerably more proximally on the

Box 25.9 Why has my patient's cheek gone white?

Maxillary infiltration is usually uneventful once you have mastered the technique of placing and angling the needle so that its tip is near the root apices of the target teeth; the needle can be aimed straight where the lips can be retracted for access, but must be aimed obliquely upwards and backwards to anaesthetize the posterior teeth where direct access is restricted. Minor branches of the maxillary artery which vary in occurrence and size run across the maxilla. Local anaesthetic containing noradrenalin is usually administered unless medical condition of the patient dictates otherwise). If anaesthetic is injected into one of these vessels, it will go into immediate vasoconstriction. If the vessel supplies an area of skin, blanching of the area will be apparent. The patient will feel **ischaemic pain** due the build up of metabolites within the tissues which cannot be flushed out effectively because there is little or no vascular perfusion of the tissue. Fortunately, the pain and blanching wear off within a few minutes. It is impossible to anticipate such an occurrence as you cannot see the underlying vessels.

Box 25.10 The anatomy of infraorbital nerve block

The infraorbital foramen is palpated through the skin (see Section 20.3.1) and the syringe needle introduced through the lining mucosa of the vestibule aimed towards the foramen. Anaesthetic is deposited at the foramen and diffuses back into the infraorbital canal. As described in Section 25.4.1 the **anterior superior alveolar nerve** branches from the infraorbital nerve just before its exit from the foramen and is anaesthetized by anaesthetic flowing into the infraorbital canal; the terminal cutaneous branches of the infraorbital nerve supplying the skin of the lower eyelid, cheek, and upper lip will also be anaesthetized. It is important to keep pressure on the lower orbital rim for a couple of minutes when anaesthetic is released from the syringe to prevent anaesthetic diffusing into the orbit. Should it reach the orbit, the inferior branch of the oculomotor nerve is the most frequent structure affected with consequent diplopia.

major nerve trunk or one of its branches than the target tissue; in this case, any branches distal to the point of anaesthesia will be anaesthetized as well as those supplying the target tissue.

The bone of the body of the mandible is so dense that it acts as a barrier to diffusion of local anaesthetic solution deposited near the tooth. To anaesthetize the pulp of most lower teeth, it is necessary to administer an **inferior dental nerve block** to the main trunk of the inferior alveolar nerve before it enters the mandibular canal. The alveolar bone forming the sockets of the maxilla is comparatively thin; anaesthetic delivered close to the bone can diffuse into the sockets and stop nerve transmission where the alveolar nerves enter the roots of the teeth. **Maxillary infiltration** can thus be used to anaesthetize upper teeth.

Another general point about dental local anaesthesia is that it is usually sufficient to anaesthetize the tooth itself for restorative procedures. Procedures such as gingival surgery, extraction of teeth, or preparation of teeth for crowns and other advanced restorative techniques will traumatize the gingivae; therefore, they also need to be anaesthetized.

25.5.1 Local anaesthesia of maxillary teeth

For maxillary infiltration, the anaesthetic is deposited at the level of the apices of the tooth roots into the connective tissue overlying the buccal aspect of tooth socket of the tooth or teeth to be operated on. The anaesthetic will then diffuse through the bone into the socket and block the alveolar nerves as they cross the gap between bone and the apical foramen at the tip of the tooth root.

The needle is gently inserted into the mobile lining mucosa above the mucogingival junction. The patient would experience pain and discomfort pain if the needle pierced the mucoperiosteum of the attached gingivae because the periosteum is likely to be stripped from the bone. As pointed out at the beginning of this chapter, retraction of the lip defines the mucogingival junction very clearly and is used to determine the entry point of the needle.

Anaesthetic introduced into the labial or buccal connective tissue will also anaesthetize the nerves supplying the labial or buccal gingivae as it diffuses through the bone into the tooth socket; additional anaesthetic for advanced procedures is, therefore, not required. A palatine nerve block may be required and the techniques used for this have been outlined in Box 25.8.

The possible complications of maxillary infiltration anaesthesia are covered in Box 25.9.

Local anaesthetics do not penetrate well through pus. If there is a periapical abscess at the root of a maxillary tooth, it can be quite difficult to obtain a satisfactory level of anaesthesia through standard infiltration techniques. In such circumstances some practitioners use an **infraorbital nerve block** for anterior teeth described in Box 25.10.

25.5.2 The anatomy of inferior alveolar nerve block

The only practical place where a nerve block can be administered to the inferior alveolar nerve is in the **pterygomandibular space** as it enters the mandibular canal; this an **inferior dental** (or alveolar) **nerve block**, usually abbreviate to **ID block**. Knowledge of the anatomy of the pterygomandibular space is essential to administer an ID block safely and efficiently.

A horizontal section through the infratemporal fossa located a short distance above the mandibular foramen is presented in Figure 25.17. This is the best view to appreciate and remember the arrangement and contents of the pterygomandibular space and their relationship to each other. Study the figure as you read the following description.

As you can see in Figure 25.17, the **pterygomandibular space** is a narrow gap between the bulky **medial pterygoid muscle** and the

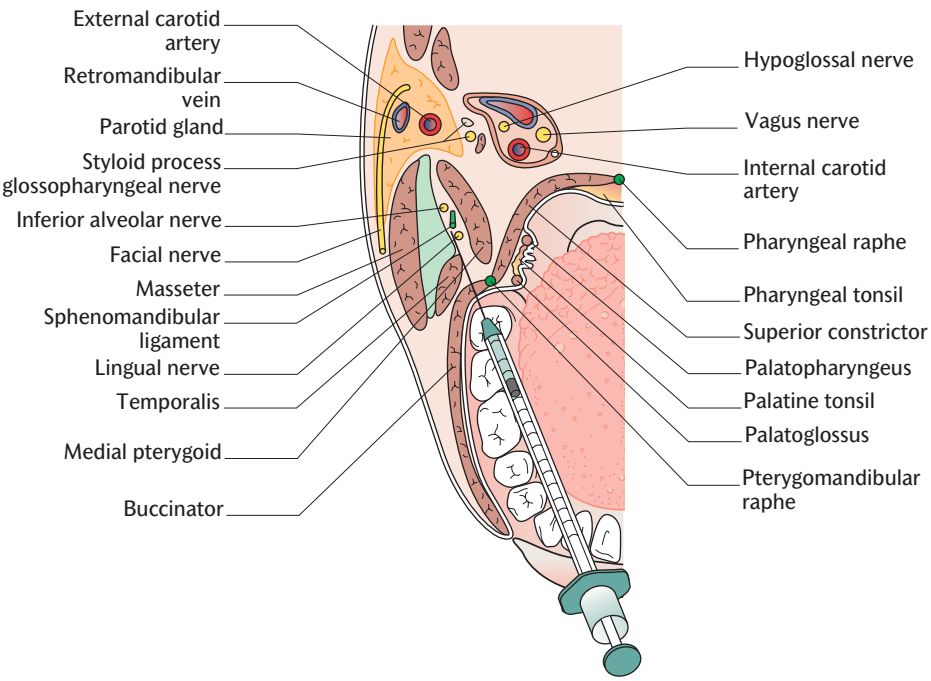


Fig. 25.17 The anatomy of the pterygomandibular space at a level above the mandibular foramen and inferior alveolar nerve block.

ramus of the mandible laterally at the level of injection; loose connective tissue fills the space. Note that the medial surface of the ramus is not flat; there is a shallow concavity behind its anterior ridge, the **temporal crest**. The **temporalis muscle** is inserted into the ramus between its anterior border and the temporal crest. The **lingual nerve** is approximately opposite the temporal crest while the **inferior alveolar** neurovascular bundle lies further back within the concavity of the ramus. Notice also that the **sphenomandibular ligament** lies close on the medial side of the inferior alveolar nerve.

The posterior boundary of the pterygomandibular space is formed by the anteromedial surface of the **parotid gland**. Figure 25.17 also shows the facial nerve, retromandibular vein, and external carotid artery within the gland in that order from superficial to deep. Although the gland is enclosed within the tough **parotid capsule,** it is easily penetrated by the sharp tip of a syringe needle (see Box 25.12).

Access to the pterygomandibular space from within the mouth is gained by inserting the needle through the buccinator and the covering lining mucosa just *lateral* to the **pterygomandibular raphe** and then advancing it into the anterior opening of the space between the temporal crest and the anterior border of the medial pterygoid muscle. Recall that the buccinator muscle is attached to the lateral aspect of the raphe and the superior constrictor to its medial aspect.

There are various clinical techniques that are used to achieve inferior alveolar block which will not be described here. They all share the following aims.

1. To gain access to the pterygomandibular space without passing the needle into the temporalis or medial pterygoid muscles. It is important to identify and use the pterygomandibular raphe as a guide landmark; the needle is inserted lateral to the raphe as shown in Figure 25.17.
2. To advance the needle far enough into the space so that it lies close to the inferior alveolar nerve; the depth of penetration of the needle is approximately 2 to 2.5 cm.
3. To advance the needle at the correct vertical level such that the local anaesthetic solution is deposited around the inferior alveolar nerve just before it enters the mandibular foramen. The correct height is found by palpating the external oblique ridge of the ramus with the thumb of your non-injecting hand to feel for the deepest concavity; the needle should be introduced along an imaginary line bisecting your thumb nail.

Box 25.11 Anatomical variations

The commonest anatomical variation is that the mandibular foramen may be higher than the occlusal plane. Your routine ID block technique may, therefore, prove unsuccessful. Placing the needle a few millimetres higher than usual will usually solve the problem.

Your ID block may appear to be successful because the patient says their lip is numb and there is no reaction when you gently squeeze it. However, when you begin to drill into the patient's tooth, usually a posterior tooth, they experience pain and react accordingly. What could possibly have gone wrong? Neurovascular bundles have been demonstrated entering the mandible through foramina situated in the areas of insertion of the muscles of mastication, especially those of the medial pterygoid and temporalis. These form a fine network within the ramus and body of the mandible lateral or medial to the roots of the molar teeth. This network makes numerous connections with the inferior alveolar nerve and its dental branches. It is possible, therefore, that in some cases, aberrant sensory nerves reach the posterior teeth from nerves supplying the muscles of mastication. Similar connecting branches have been demonstrated further forwards, passing through foramina on the medial surface of the mandible from the mylohyoid nerve to dental branches to the anterior teeth opposite the premolar teeth. It may seem peculiar that supposed motor branches carry sensory nerves, but the proof comes from clinical experience. Local infiltration of anaesthetic is introduced into the tissues on the medial side of the mandible adjacent to the tooth producing the unexpected pain, and it usually relieves it, indicating that sensory branches are gaining access to the teeth through peculiar routes.

A similar scenario may arise when an ID block is carried prior to procedures on lower anterior teeth. Once again, the signs and symptoms indicate that the block has been successful, but the patient reacts to painful stimuli once work has started. In this case, there is sometimes '**cross-over' innervation** of the central and possibly lateral incisor teeth. The inferior alveolar nerve from the opposite side does not stop at the midline continuing across to the contralateral incisor teeth which, therefore, receive bilateral innervation. The bone forming the sockets of the incisor teeth is usually thin enough to allow infiltration anaesthesia to be carried out on the painful tooth to alleviate the problem.

When placing the needle for an ID block, it is usual to feel gently for resistance from contact with bone close to the mandibular foramen when you think you have advanced the needle to the correct depth; the needle is then withdrawn slightly so that it is near the inferior alveolar nerve. As can see in Figure 25.17, the **sphenomandibular ligament** lies very close to the inferior alveolar nerve. Like many other ligaments, it may calcify in older age groups. It can then feel that the needle has contacted bone when in fact, the needle has contacted the calcified ligament; anaesthetic may not diffuse around the ligament to the nerve, therefore, anaesthesia will not be achieved. Repositioning of the needle slightly more laterally at a second attempt will usually place the needle between the ligament and bone so that successful anaesthesia can be achieved.

Box 25.12 Operator error

It is important to advance the needle the correct distance into the pterygomandibular space. If it does not enter far enough, satisfactory anaesthesia of the inferior alveolar nerve may not be achieved. The tip of the needle may pierce the **parotid capsule** if advanced too far. Local anaesthetic solution will then enter the parotid gland and spread rapidly through it to reach the **facial nerve** branches, producing some degree of temporary facial paralysis; this is uncomfortable for the patient and embarrassing to the operator. The mandibular foramen is located approximately halfway between the anterior and posterior borders of the ramus; both borders can be readily palpated through the skin which helps avoid advancing the needle too far. In some patients, a lobule of the parotid gland projects forwards into the pterygomandibular space and in these cases, it is difficult to avoid local anaesthetic solution entering the gland.

If the approach to the mandibular foramen is too far medial, the needle passes *through* medial pterygoid muscle rather than into the pterygomandibular space. When muscles are traumatized, they usually contract to immobilize their attachments and prevent further damage. This is **trismus** and the medial pterygoid will react in this way if damaged by the needle; the patient will find that they suffer limited mouth opening the following day. The problem can be overcome by slowly stretching the muscle. This is best done by inserting several lollypop sticks or tongue spatulae between the teeth for 10 minutes. The process is repeated every few hours, increasing the number of sticks at each application until mouth opening returns to normal; this may take a few days.

Sometimes, you will be too accurate and achieve a direct hit on the inferior alveolar nerve rather than placing the needle near it. The patient will react as if they have received a strong electric shock which is exactly what it feels like if the nerve is hit.

Failure to use an **aspirating syringe** or to observe the colour of aspirated fluid within the syringe could mean that the anaesthetic is injected into an artery or vein instead of the connective tissue of the pterygomandibular space. The vessels most at risk are the first and second parts of the maxillary artery and their branches (see Section 24.5.2) and the pterygoid venous plexus (see Section 24.5.3). Even if you have the best clinical and interpersonal skills in the world, most dental patients are nervous and, therefore, have some level of circulating adrenalin—the start of the 'flight or fight reaction' described in Chapters 3 and 17. Injection of anaesthetic containing noradrenalin will increase blood monoamine levels to the point where the patient may experience heart palpitations or even faint. For a patient with increased medical risks due to cardiovascular and respiratory disease, the consequences may be more severe.

Box 25.13 Other considerations

In most cases, the dentist will carry out a combined ID and lingual nerve block as routine as mentioned above. The lower teeth and lingual gingivae will, therefore, be anaesthetized. An ID block will also stop nerve conduction in the mental branch of the inferior alveolar nerve so the labial gingivae will also be insensitive. The only areas, therefore, that are not anaesthetized by a combined ID and lingual nerve block are the buccal gingivae of the posterior teeth which are supplied through the **buccal nerve**. If anaesthesia of the buccal gingivae is required, this can be achieved by infiltrating on the buccal side of the tooth to be operated on. The buccal nerve crosses the ramus of the mandible at the level of the occlusal plane of the lower teeth; a buccal block can be achieved by infiltrating anaesthetic into the soft tissue anterior to the anterior border of the ramus at this level if work is required on multiple posterior teeth.

When dental procedures are required on multiple anterior teeth, some dental surgeons prefer to use a **mental nerve block** rather than an inferior dental nerve block. As pointed out in Chapter 20, the mental foramen can usually be palpated on the lateral side of the mandible between the first and second premolars just below their root apices. An important point to bear in mind is that the mental foramen points backwards because of differential growth of the bone and nerve during development (see Chapter 33). The foramen is palpated and the needle introduced below the mucogingival junction into the foramen from behind. If it is introduced anteriorly, anaesthetic may diffuse away into adjacent soft tissue without entering the foramen and the desired effect will not be achieved.

4. To avoid injecting anaesthetic solution into vascular structures by *always* using an aspirating syringe to carry out an ID block. An **aspirating syringe** is one where the plunger can be withdrawn so that liquid is drawn into the barrel. Clear aspirated fluid is tissue fluid and delivery of anaesthetic may proceed. Bloody fluid indicates that the needle tip has entered a blood vessel; the syringe should be withdrawn and the procedure tried again. Think which vessels might be in danger and what the consequence might be if you were to inject anaesthetic into a vessel; the answers will be found in Box 25.12.
5. Lingual nerve anaesthesia is often carried out at the same time as an ID block, **a combined lingual–ID block**; if the needle is withdrawn by 1 cm after the ID block, the anaesthetic solution remaining in the syringe will be injected around the lingual nerve.

If these criteria are met by correct positioning of the needle, you should achieve a successful ID block on most occasions. After a couple of minutes, the skin of patient's lower lip and chin should feel numb and it should be safe to proceed with the procedure.

Several things can militate against a successful ID block. Some of these are due to anatomical variations which are described in Box 25.11 and some are due to operator's error considered in Box 25.12. Other factors which you may have to take into account during local anaesthesia of mandibular teeth are outlined in Box 25.13.

25.6 A summary of sensory innervation of the oral cavity

You must know the sensory innervation of the structures of the oral cavity because of its importance to dental practice. It is summarized below and diagrammatically in Figure 25.18.

1. The maxillary teeth and supporting structures and the lining of the maxillary sinus are supplied by the **anterior** and **posterior superior alveolar nerves** plus the **middle alveolar nerve** when present.
2. The upper buccal gingivae are innervated by the **superior alveolar nerves**. The labial gingivae and mucosa and skin of the upper lip receive their sensory supply from the **infraorbital nerve**.
3. The mucosa of the hard palate, including the palatal gingivae, is supplied by the **greater palatine nerve** and the **nasopalatine nerve** in the incisor region. The mucosa of the soft palate is innervated by the lesser palatine nerve.
4. The lower teeth and supporting structures receive their sensory innervation through the **inferior alveolar nerve** and its **incisive branch**.
5. The buccal gingivae are supplied by the **buccal nerve** in the molar and premolar region and the labial gingivae of the canine and incisors by the **mental nerve** which also supplies the mucosa and skin of the lower lip.
6. The lingual gingivae are innervated by the **lingual nerve**; this nerve also supplies the floor of the mouth and the anterior two-thirds of the tongue with somatic sensation.
7. Taste from the anterior two-thirds of the tongue is conveyed by processes travelling in the lingual nerve which reach the brain through the **chorda tympani** branch of the **facial nerve**.
8. The posterior one-third of the tongue (including the vallate papillae) receives both taste and general sensation from the **glossopharyngeal nerve** which also innervates the mucosa over the palatoglossal folds.
9. The mucosa and skin of the cheek is innervated by the **buccal nerve**.

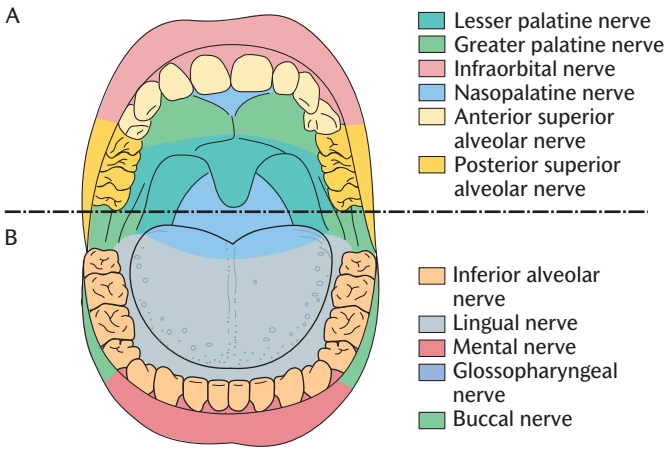


Fig. 25.18 The sensory innervation of the oral cavity. A) The maxillary dentition and supporting tissues; B) The mandibular dentition and supporting tissues.

26 Mastication

Chapter contents

26.1 Introduction

Now you have an understanding of the anatomy of the maxilla and mandible, the TMJs, and jaw musculature, we can examine how these structures work together to produce the complex actions involved in the biting and chewing of food. Technically, **incision** is biting a piece from a larger chunk of food and **mastication** is the grinding down of that piece into smaller components and mixing them with saliva. Mastication is often used to cover both actions. Box 26.1 briefly compares the anatomy of the human dentition to that of other mammals.

Box 26.1 The comparative anatomy of the human dentition

Many characteristics distinguish mammals from other vertebrates, including the possession of a single lower jaw bone articulated with the cranium by TMJs, a well-developed jaw musculature, and a **heterodont dentition** in which the teeth have different shapes to carry out different functions. The anterior teeth have sharp edges for cutting through food and the post-canine teeth bear multiple cusps for crushing food. Most placental mammals have evolved into specialized feeders with a masticatory apparatus designed to deal efficiently with a particular diet but lacking the ability to utilize all foodstuffs. The masticatory apparatus of primates has remained relatively unspecialized, both in its structure and function; the jaws retain a wide range of functions suitable for an omnivorous diet, which means we have few dietary limitations. We have the same dental formula as our nearest living relatives, the Old World monkeys and apes, but human teeth are much smaller in size relative to body mass and the jaw-closing muscles are also reduced in relative size. Despite these apparent structural disadvantages, the human masticatory apparatus can cope with a remarkable range of foodstuffs.

26.2 Dental occlusion

As well as knowledge of the TMJ, muscles of mastication, and other muscles used in jaw movements, it is necessary to appreciate some aspects of the static and dynamic relationships of the teeth to understand chewing movements.

The first thing to notice is the bigger width of the upper dental arch compared to the lower arch, a condition known as **anisognathy**. In Figure 26.1A, you can see that the maxillary molars overhang the mandibular teeth by half a cusp width so the buccal cusps of the lower molars and premolars occlude between the buccal and palatal cusps of the maxillary teeth. Observe also that the long axis of the maxillary molars and premolars incline buccally while the corresponding axis of the mandibular teeth incline lingually; the occlusal plane of the posterior teeth is thus curved transversely as illustrated in Figure 26.1A. It would be possible to chew food simply by moving the teeth up and down without any side-to-side movement, but this would be inefficient and not make full use of the cusps on the occlusal surfaces of posterior teeth. However, we can only chew on one side at a time because of the anisognathy of the upper and lower teeth.

Due to anisognathic jaw positions, the maxillary anterior teeth are also going to protrude in front of the mandibular anterior teeth. Figure 26.1B illustrates the normal relationships of the anterior teeth. The maxillary incisors overhang the mandibular incisors by about 2–3 mm in the horizontal plane; this is called the **overjet.** The upper incisors usually have a vertical overhang, the **overbite,** of about the same amount.

As mentioned in Chapter 24, the mouth at rest is closed by tonic contraction of the muscles of mastication and facial expression. The teeth are *not* in contact; they are separated by a 2–4 mm gap called the **intercuspal space** (or **freeway space**) between the teeth. This is known as the **mandibular** or **postural rest position**; clinically, it is usually simply called the rest position. When the gap between the teeth is closed and they are in maximal contact, they are in **intercuspal occlusion.** The relationships and contacts between the maxillary and mandibular dentitions should not only form an efficient functional unit, but also produce a harmonious aesthetic

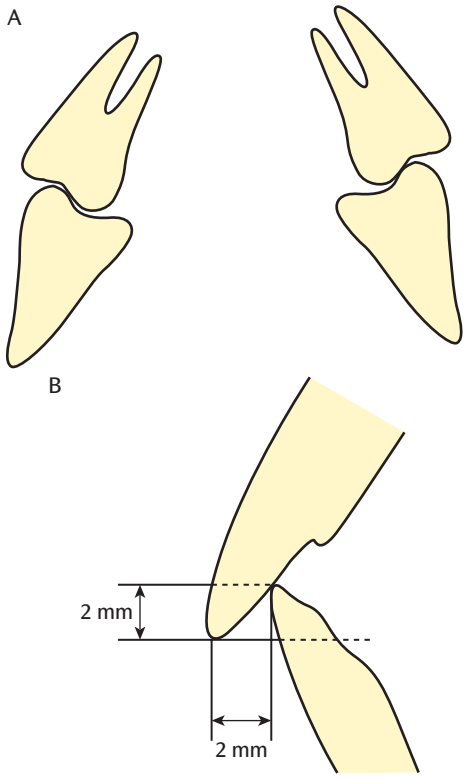


Fig. 26.1 A) Anisognathy and lateral curvature of the posterior teeth; B) Overbite and overjet of anterior teeth.

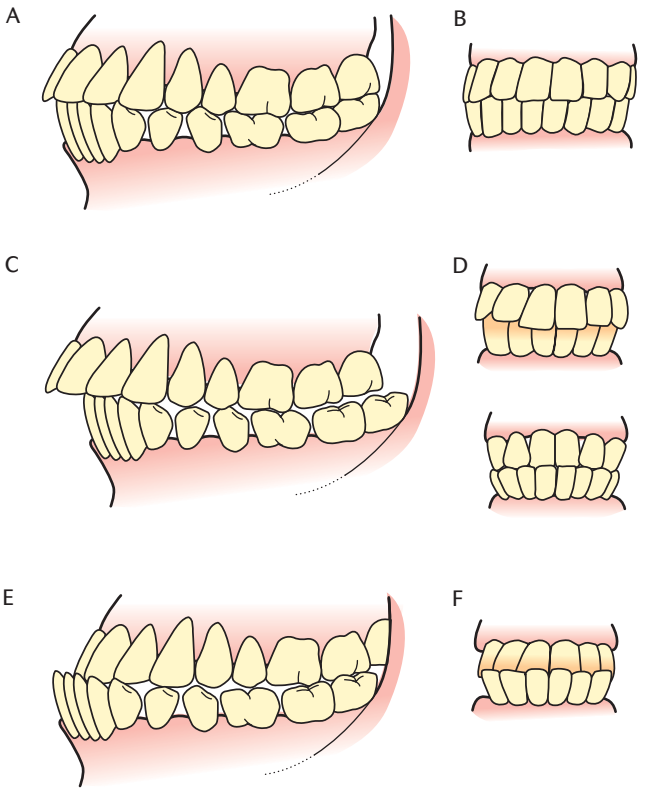


Fig. 26.2 Occlusion of teeth. A, B) Angle's class I; C, D) Angle's class II; E, F) Angle's class III.

appearance of the face. The most commonly used classification of occlusion is **Angle's classification** where the key feature defining the occlusion is the relationship of the *first permanent molars*; these are the first permanent teeth to erupt and guide the other permanent teeth into position.

Angle's Class I or **normal occlusion** is illustrated in Figure 26.2A. Find the first molars on the diagram and observe that the mesial cusps of the upper first permanent molar occlude in the groove between the mesial and distal cusps of the lower first permanent molar. It follows that there will be the same relationships between the second and third molars posteriorly; each mandibular tooth anterior to the first molars will be mesial to its maxillary equivalent; at the midline the mesial edges of the upper and lower central incisors coincide as shown in Figure 26.1B.

Figures 26.2C and 26.2D illustrate Angle's Class II and Figures 26.2E and 26.2F show Angle's Class III **occlusion**. Compare the position of the first molars in C and E with the position in the Class I occlusion shown in A. In a Class **II occlusion**, the buccal cusps of the first maxillary molar occlude between the distal surface of the lower second premolar and the mesial cusps of the first molar. The whole mandibular dentition is further back than in a Class 1 so it is also called **distocclusion**. The consequence on the anterior teeth can be seen in Figure 26.2C; the upper anterior teeth have a pronounced overjet. Figure 26.2D shows the two common variations in the incisor relationships in this occlusion. In a Class II division 1 occlusion, all the central and lateral incisors have a large overjet. In a Class II division 2, the lateral incisors are proclined with an increased overjet, but override the central incisors so pushing them back and reducing their overjet.

Figure 26.2E shows a **Class III occlusion** where the mesial cusps of the upper first molar occlude between the distal cusps of the mandibular first and mesial cusps of the second incisor. You can see that the whole mandibular dentition is echeloned further forwards than in a Class I, giving its alternative name of **mesiocclusion**. As shown in Figures 26.2E and 26.2F, the mandibular teeth are anterior to the maxillary anterior teeth so they have a *negative* overjet.

26.3 Mandibular movements during chewing activities

Human masticatory movements can be analysed stage by stage using techniques such as videophotography and cineradiography. It is clear from these studies that each individual possesses a characteristic chewing pattern which is established early in life. The chewing pattern varies within individuals with the consistency of food being consumed. Incision is the biting off of a portion of food from a larger piece by the anterior teeth although the use of cutlery or consistency of food frequently obviates the need for incision. The portion is then transferred to the posterior teeth where it is masticated; the food is finely divided and thoroughly mixed with saliva during a number of masticatory cycles. The chewing pattern is so finely balanced that dental procedures which change the shape of the occlusal surfaces, such as a dental restoration left too high or malocclusion, can temporarily or permanently modify the chewing pattern.

26.3.1 Incision

The movements of the mandible during incision are shown in Figure 26.3; follow the sequence of diagrams as you read the description. For this movement, the mandible must be depressed sufficiently to accommodate the food and protruded forwards enough so that the incisor teeth will come into edge-to-edge contact when the mouth is closed. The lateral pterygoid muscles on both sides initiate sliding movements of the mandible, then the suprahyoid muscles act to produce depression of the mandible to the desired distance, operating from a hyoid bone fixed by the infrahyoid muscles. The lateral pterygoids continue to protrude the mandible so that the incisal edges of the lower incisor teeth are positioned below the corresponding surfaces of the upper incisors. The mandible is then elevated; the medial pterygoid muscles initiate elevation and the temporalis and masseter muscles quickly become active. The incisors cut through the food until they meet edge-to-edge or nearly so. The mandible is then retracted by the action of the horizontal fibres of the temporalis muscles so that the edges of the lower incisors slide upwards across the palatal surfaces of the upper incisors to complete the incision by shearing or tearing off the portion. The tongue transfers the portion of food to the posterior teeth on one side or the other.

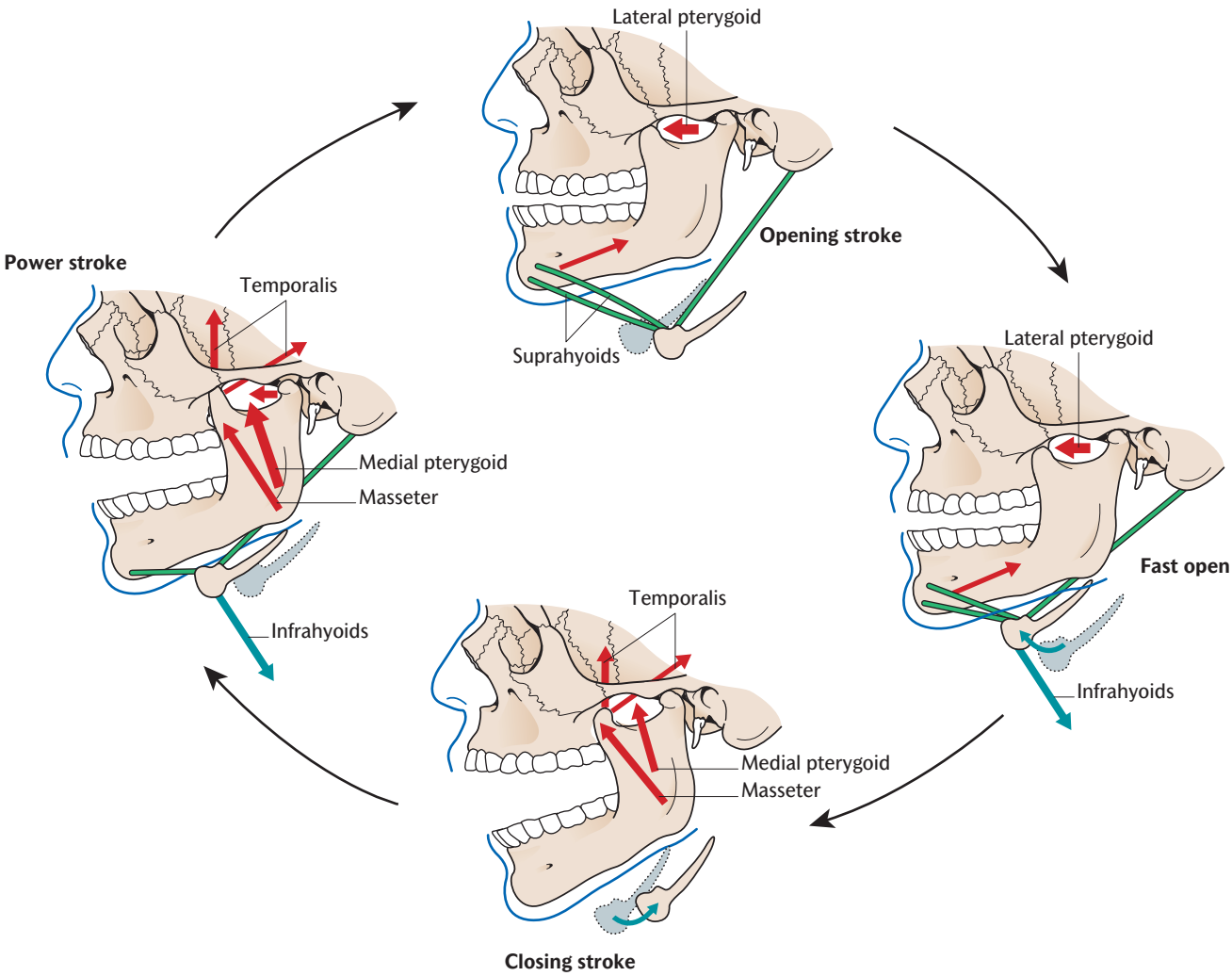


Fig. 26.3 Mandibular movements during incision. (Redrawn from Rowe, A.M.R and Johns, R.B. (Eds) *A companion to dental studies* Vol 1 part 1; Blackwell Scientific, Oxford.)

26.3.2 Mastication

Once the food is in place between the posterior teeth, a series of masticatory cycles is initiated. The sequence of events is illustrated in Figure 26.4 which should be consulted as you read the description. Each cycle consists of an **opening stroke** in which the mandible is lowered and moved laterally to the working side; this may be preceded by a brief lateral movement towards the balancing side. Food is positioned between the teeth by the action of the tongue and cheeks. The mandible is elevated in the **closing stroke** but lateral excursion to the working side is maintained. In the final stages of elevation, the mandible is moved back towards the midline so that the occlusal surfaces of the lower teeth glide medially close to, or on, the corresponding surfaces of the upper teeth. This late phase of closing is often referred to as the **power stroke.** Powerful isometric contraction of the jaw-elevating muscles keeps the teeth closely approximated so that food is ground between the opposing occlusal surfaces. The proportion of masticatory cycles in which the teeth make contact is still uncertain; the proportion increases as the food is broken down with contact probably occurring in greater than 50% of cycles towards the end of a sequence.

Each masticatory cycle is unilateral because the human dentition is anisognathic. In some individuals, a whole masticatory sequence takes place on one side. In others, the food is switched in rapid, often irregular, succession between the two sides. The duration of each masticatory cycle varies from about half a second to just under one second with the power stroke occupying approximately one-quarter of the total time. Some individuals do masticate exclusively on one side but if chewing becomes unilateral, you should suspect and investigate possible dental problems as outlined in Box 26.2.

During a masticatory cycle, muscle activities combine in a complex manner. In the opening stroke, the two **lateral pterygoid muscles** are active but the balancing side muscle predominates

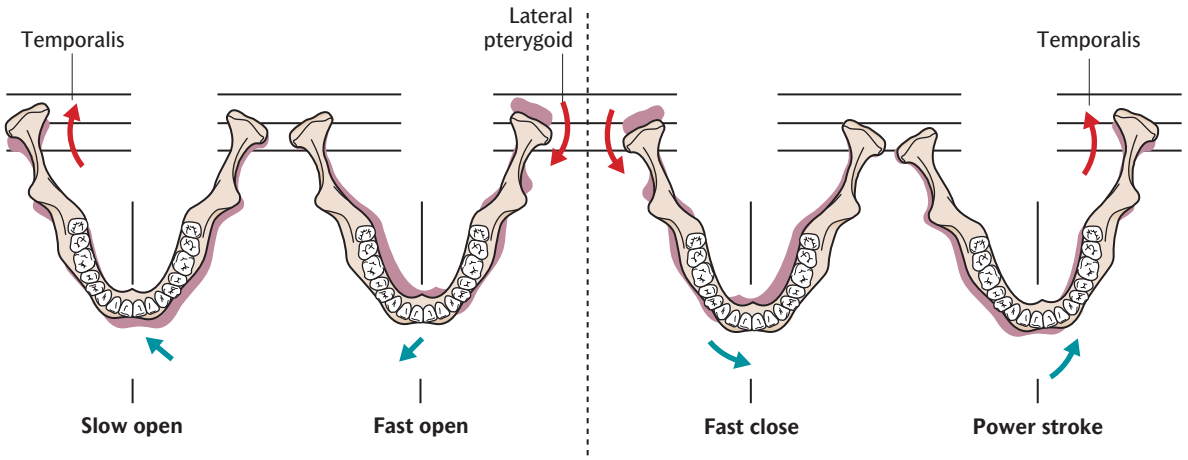


Fig. 26.4 Mandibular movements during mastication.

Box 26.2 Is chewing on just one side normal?

Mastication does take place exclusively on the left or right side in a few rare individuals. In the majority of cases, unilateral mastication is indicative of the patient avoiding pain and discomfort caused by underlying dental disease. The most common reasons for avoidance of one side are a dental abscess or high filling.

Dental abscesses are usually the result of the patient neglecting to attend the dental surgery when toothache first appears. Dental pain only occurs when dentine is exposed because enamel is insensitive; the carious lesion is, therefore, well advanced by the time it attacks dentine. Eventually, the caries will enter the dental pulp, stimulating an inflammatory reaction. The inflammatory material can only exit the tooth through its apical foramen or foramina into the periodontal tissues where a **periapical abscess** will form. Teeth are vertically displaced along their long axes by an imperceptible amount during chewing. However, if a periapical abscess is present, this tiny movement is sufficient to push the root apex into the abscess causing severe pain, hence the avoidance pattern of jaw movement adopted by the patient. A firm tap on the suspected tooth with a hard object such as the handle of a dental mirror is used for initial diagnosis of a suspected dental abscess. The tap intrudes the tooth and the patient will complain of pain; adjacent teeth are also percussed to verify the offending tooth. The tooth under which the abscess appears to be located is noted in dental records by the wonderful understatement 'tender to percussion'. The presence of a dental abscess is confirmed by dental radiographs.

so that the mandible is swung laterally towards the working side. The horizontal fibres of the temporalis of the working side prevent that condyle from sliding forwards; the Bennett movement occurs passively as the opposite condyle slides forwards (see Figure 24.4). The digastric and mylohyoid muscles are also active during the opening stroke, acting in a similar way to their action in incision; they work off the hyoid bone fixed by the infrahyoid muscles. The medial pterygoid muscles initiate closing. Activity begins first in the muscle of the balancing side but eventually reaching a higher level in the working side muscle. The masseter muscles show similar differential activity between the two sides although their activity begins later than the corresponding medial pterygoids. The degree of activity in the temporalis muscles is about the same on the two sides but begins earlier on the working side. These patterns of activity correlate with the observed jaw movements during the closing stroke.

As you might anticipate, the activity in the jaw closing muscles is greatest when tough food is chewed and least when the food is soft; activity tends to decrease with successive masticatory cycles as the food is progressively broken down and softened.

26.3.3 Factors influencing jaw movements

As outlined in Section 2.4.1, the range and direction of the movements at most joints are controlled to a large extent by the size, shape, and congruency of the articular surfaces and the disposition and tightness of its capsule and ligaments. It has been argued that this is not the case in the TMJ because there is a distinct incongruency when the convex mandibular condyle articulates with the convex articular eminence. The capsule and ligaments are also supposedly rather weak so the structure of the jaw joints cannot be the prime factor controlling the position and movements of the mandible.

The structure of the TMJ joint surfaces and the surrounding bones certainly determines the limits of mandibular movement, but it is likely that other factors are more important during actual movements within these limits. The interplay of the jaw musculature is probably the principal factor when the teeth separated. Jaw movements over most of their range are the outcome of learned patterns of muscle activity, which explains why the character of the chewing cycle is established at such an early age and there is so little alteration despite growth in the jaws and neighbouring soft tissues and eruption of teeth (see Chapter 33). It would also explain why there is so much variation in masticatory movements between individuals.

Box 26.3 Temporomandibular joint dysfunction and other disruptions of mastication

The individual learned patterns of jaw movement are easily disturbed. For example, an isolated filling left 'high' above the occlusal surface of a tooth will soon lead to pain in the TMJ and masticatory apparatus as the accustomed movement path of jaws is disrupted. High fillings cannot be detected even by the patient's exquisitively sensitive tongue, dental probes, or visual inspection. Usually the patient will feel 'uncomfortable' when asked to close gently on to the new restoration. Facets may be visible in the setting filling material where the opposing teeth have intruded into the high filling; it can then be carved down to the correct level. Disclosing paper leaves marks when chewed lightly between the teeth and is also a valuable aid for finding high spots and removing them.

The **rest position** of the mandible is the default position of the jaws. The masticatory apparatus is *not* designed to be open for long periods; otherwise the muscles are unduly stretched and become painful. It is, therefore, necessary to ensure that your patients have frequent breaks during treatment when they can close their mouth and take the strain off the joints and muscles—and also rinse out debris and clear excess saliva.

The tonus in the muscles of mastication maintains the mandible in the rest position. If the interocclusal space is not maintained, the outcome is **temporomandibular joint (or mandibular pain dysfunction) syndrome.** It presents with one or more of the following symptoms: dull pain in the region over the TMJ, pain on opening or during chewing, audible cracking noise (**crepitus**) during jaw movement, or fixation of the mandible. Pain is usually unilateral but may be bilateral. The most common cause of TMJ syndrome is stress; when patients respond to stress by clenching their jaw, the interocclusal space is obliterated for often long periods of time, producing some of the signs and symptoms listed above. Treatment usually involves making the patient aware of their habits and encouraging them to think of times when they clench or grind their teeth so they can consciously try to relax their jaws in these circumstances. Relaxation can be aided by prescription of mild tranquilizers. Patients who grind their teeth while asleep, to their own detriment and that of their partner, are often aided by the provision of bite raisers. These are like a miniature denture plate with blocks that fit on to the occlusal surface of the posterior teeth to maintain the interocclusal space.

When the teeth are within intercuspal range, the shape of their occluding surfaces and the direction of the gliding contacts between opposed cusps are major factors determining jaw movements. As would be expected, the small, but powerful, movements taking place during the power stroke can be influenced by changes in the shape of the dentition resulting from dental procedures or from eruption or loss of teeth. Box 26.3 covers some of the problems which may arise.

26.4 Nervous control of mastication

Mastication is usually carried out automatically with little conscious effort; we suddenly become acutely aware of what we are doing when we bite our tongue or cheek. A precise neural control is clearly required to execute the complex, rapid movements of the jaws described in Section 26.3, during which the teeth are in close contact and the elevator muscles can exert very powerful forces. When we consider that during masticatory movements, in particular, we have to coordinate jaw movements with those of the tongue, cheeks, and lips to keep replacing the food squeezed out during each phase back in the dental arches as it is. As with any other complex learned activity as described in Section 16.3, the brain constantly receives information about the state of the participating muscles and joints, the position of the mandible in space, and the degree of force being exerted on the teeth and their supporting tissues in order to exert such control.

Information from numerous muscle spindles in the muscles of mastication and associated musculature appears to terminate in the **trigeminal mesencephalic nucleus**, irrespective of the cranial nerve supplying the muscle (see Section 18.6.1).

Nerve receptors are present in large numbers in the capsule and ligaments of the TMJ. Numerous unencapsulated endings in the capsule may provide information about the perception of mandibular position. Encapsulated endings in the capsule are high threshold, rapidly adapting receptors that are thought to respond briefly to movements of the joint. The sensory information from these various types of receptors passes to the trigeminal mesencephalic nucleus via the **auriculotemporal** and **masseteric** branches of the mandibular trigeminal nerve. Free nerve endings are the most abundant type of endings in the capsule and are believed to be pain receptors. The nociceptive neurons send information to the **trigeminal spinal nucleus**.

If the mouth is opened an arbitrary distance before full opening, the same movement can be reproduced very accurately even after a delay of a minute or two. When the proprioceptive mechanisms in the TMJ are interrupted by injecting local anaesthetic into the joint cavities in human volunteers, jaw position can no longer be reproduced with any accuracy. The errors are compounded if the proprioceptive input from the lateral pterygoid muscles is also inhibited in a similar way, indicating that knowledge of jaw position through proprioceptive information derived from the TMJ and the muscles, especially lateral pterygoid, is crucial to fluent and accurate jaw movements.

The periodontal tissues also contain numerous receptors which respond to mechanical forces acting on the teeth. They are of several different types with different physiological responses. Many are specialized to respond maximally to forces acting in one particular direction and therefore, possess directional sensitivity whereas others respond

to speed of loading. The sensory processes of these receptors pass to the brainstem in the mandibular and maxillary trigeminal nerves and terminate in the mesencephalic nucleus. These receptors are probably important in regulating the force of muscular contraction during chewing and also play a critical role in some jaw reflexes (see Section 26.4.1).

The masticatory sequence proceeds rapidly and regularly once it is initiated. Mastication, therefore, resembles other cyclical and semiautomatic activities such as walking in this respect and involves central pattern generators (see Section 16.3.1). It is now widely accepted that there is a **central pattern generator** in the brainstem which is driven by information descending from the cerebral areas, including the motor cortex, or from nerve endings in the mouth and adjacent tissues. Recent investigations suggest that the **insula** plays an important role in the control and coordination of oral activities such as transport of food within the mouth, swallowing, and mastication; it is possible that this centre controls the whole feeding mechanism. Brainstem mechanisms are probably involved in initiating and maintaining mastication and the cortical areas then refine each cycle so that muscle range, rate, and force can be matched to different foodstuffs by inputting information about texture derived from taste, aroma, and previous experience of that particular food.

26.4.1 Jaw reflexes

A sudden downward movement of the lower jaw evokes the **jaw–jerk reflex** with a brief burst of activity in the elevator muscles. This is similar to the well-known knee–jerk reflex elicited by tapping the patellar ligament. The afferent limb of the jaw–jerk is from the elevator neuromuscular spindles and the efferent limb is via the motor neurons in the mandibular nerve.

The jaw–jerk reflex may be tested during examination of patients to investigate the integrity of the mandibular trigeminal nerve. It is elicited by placing a finger on the patient's chin and then tapping the finger sharply with the fingers of the other hand or with a patellar hammer. If you try this on yourself, you will find that the tap is followed by a small upward jerk of the chin.

The **unloading reflex** occurs in response to sudden unloading or loading of the teeth; it is a very important protective reflex that prevents damage to the teeth and their supporting tissues from excess or sudden loading by inhibiting the jaw elevator muscles. An example of sudden unloading is when a brittle substance suddenly gives way; the unexpected removal of resistance could produce very rapid closure of the jaw with tooth contact and damage, but the closure of the jaw is arrested before the teeth make contact. Loading occurs when sudden resistance is met unexpectedly, for example, when you encounter a fruit stone in a jam sandwich; the reflex will inhibit closure to prevent dental damage. Sudden changes to loading are detected by periodontal receptors, which produce swift cessation of contraction of the elevator muscles. The elevator neuromuscular spindles also play a role by stopping firing when the muscle fibres suddenly shorten during the rapid unloading or when their rapid movement is stopped by contact with a hard object. This reflex clearly safeguards the teeth and their supporting tissues from sudden violent contact or excess load.

27

The nasal cavity and paranasal sinuses

Chapter contents

27.1 Introduction

The nasal cavity is the entrance to the respiratory tract. Its functions are to clean, warm, and humidify air as it is inhaled. **Respiratory mucosa** covered by pseudostratified ciliated epithelium and goblet cells, as described in Chapter 5 and illustrated in Figure 5.2B, lines the majority of the nasal cavity. The cilia and mucus trap particles, thus cleaning the air; the mucus also humidifies the air and warming is achieved through heat exchange from blood in the very vascular mucosa. The efficiency of all these processes is increased by expanding the surface of the nasal cavity by folds of bone. The nasal cavity also houses the **olfactory mucosa** for the special sense of olfaction although the olfactory mucosa occupies a very small proportion of the surface of the nasal cavity.

The nasal cavity extends from the **nostrils** on the lower aspect of the external nose to the two **posterior nasal apertures** between the medial pterygoid plates where it is in continuation with the nasopharynx. Bear in mind that in dried or model skulls, the nasal cavity is smaller from front to back and the anterior nasal apertures seem extremely large because the cartilaginous skeleton of the external nose is lost during preparation of dried skulls.

As you can see in Figure 27.1, the nasal cavity extends vertically from the cribriform plate of the ethmoid at about the level of the orbital roof above to the palate, separating it from the oral cavity below. Figure 27.1 also shows that the nasal cavity is relatively narrow from side to side, especially in its upper part between the two orbits and widens where it sits between the right and left sides of the upper jaw below the orbits. The nasal cavity is completely divided into right and left compartments by the **nasal septum**. From the anterior view seen in Figure 27.1, you can see that the surface area of lateral walls of the nasal cavity are extended by the three folds of bone, the **nasal conchae**.

27.2 The external nose

The skeleton of the external nose shown in Figure 27.2 comprises the **nasal bones**, the upper and lower **nasal cartilages**, the **septal cartilage**, and the cartilaginous part of the nasal septum. The nasal bones and adjacent parts of the frontal processes of the maxillae form the bridge of the nose. The upper and lower nasal cartilages with a variable number of minor cartilages form the skeleton of the lower pliable part of the external nose. As seen in Figure 27.1B, the septal cartilage is quadrilateral and is continuous posteriorly with the anterior edge of the perpendicular plate of the ethmoid and the superior border of the vomer, the bones that form the bony nasal septum (see p. 287). The various cartilages are unmineralized parts of the nasal capsule from which they develop (see Section 33.3).

The lateral border of each external nostril is thickened and rounded by the presence of fatty fibrous tissue. The nasal cavity is dilated just inside the external nostrils to form the **nasal vestibule** which is lined by hairy skin with coarse hairs that filter large particles.

The sensory innervation of the external nose skin is by cutaneous branches of the ophthalmic and maxillary trigeminal nerves and its arterial supply is through branches of the facial, ophthalmic, and infraorbital arteries. The venous drainage is into corresponding veins and the lymphatic drainage is into the submandibular nodes.

27.3 The nasal cavity

The skeletal structure of the nasal cavity is illustrated in Figure 27.3. You will need to examine a skull sectioned in the sagittal plane to see clearly the internal structure of the nasal cavity and the bones that form it, but you may be able to see some features on an intact skull. Be aware that many of the bones forming the nasal walls are quite delicate and may be broken off dried skulls.

Figure 27.3 illustrates that the roof of the nasal cavity is formed by the **nasal bones** anteriorly, the **cribriform plate** of the **ethmoid** in its intermediate part, and by the underside of the **body of the sphenoid** posteriorly. Its floor is the **bony palate** made up of the **palatine processes of the maxillae** in front and **horizontal plates of the palatine bones** behind. Each palatine process is pierced anteriorly, close to the

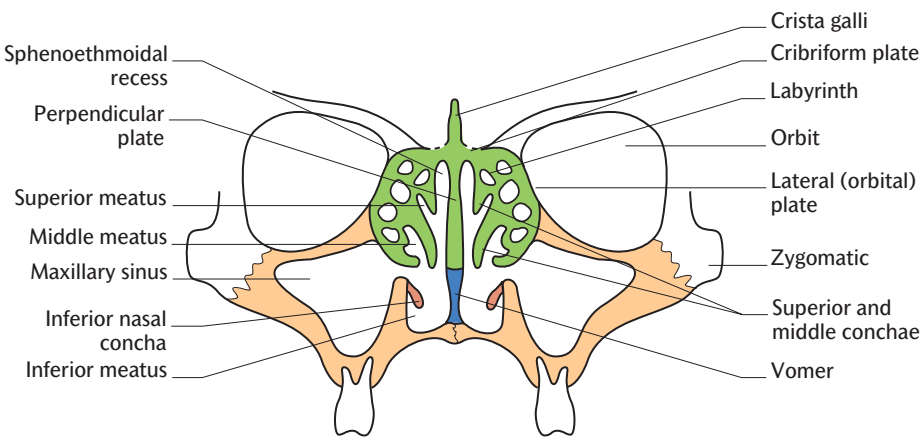


Fig. 27.1 Diagrammatic representation of the contributions of the ethmoid and maxilla bone to the lateral nasal walls.

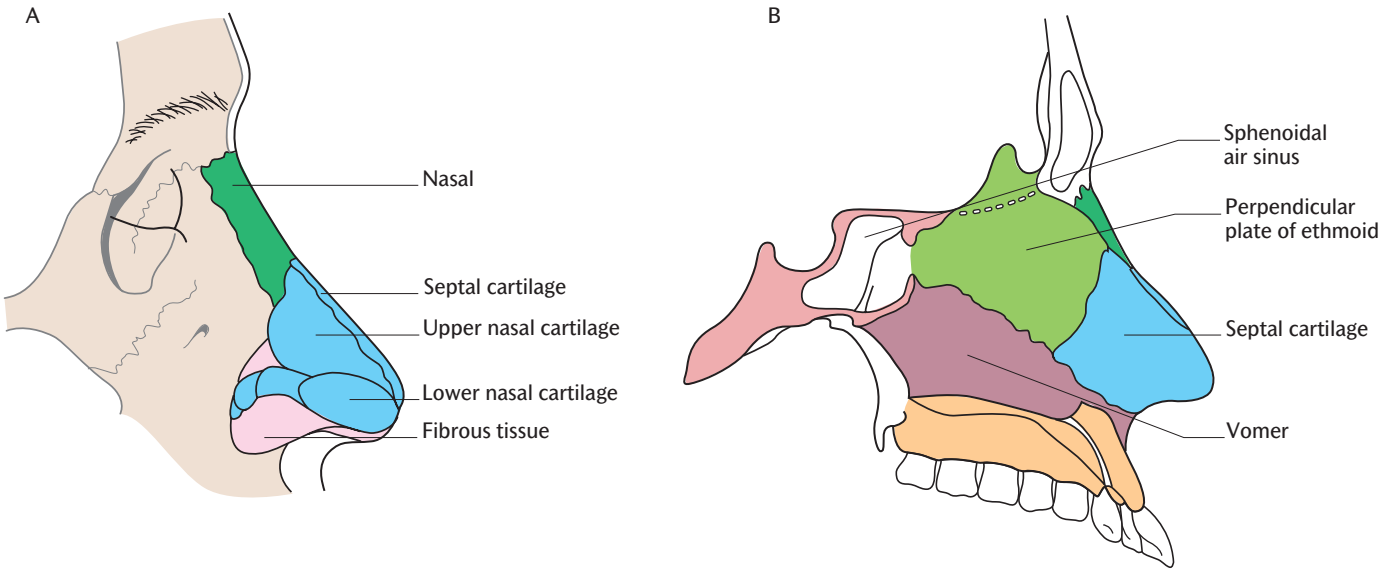


Fig. 27.2 The skeleton of the external nose. A) External view; B) View of the nasal septum.

septum, by an **incisive canal** which transmits the **nasopalatine nerves** and the terminal branch of the greater palatine artery. The two canals open into the **incisive fossa**, a single funnel-shaped depression on the oral surface of the palate (see Figure 25.4).

The **maxilla** and the **labyrinth** of the **ethmoid** are the main contributors to the lateral wall which is irregular due to three medially projecting folds of bone, the **nasal conchae**. As Figure 27.1 illustrates, the ethmoid forms the superior portion down to, and including, the middle concha and the maxilla forms the lower part. The maxillary contribution to the nasal cavity is described below. The palatine bone forms the posterior part of the lateral wall. The **palatine bone** consists of **horizontal and perpendicular plates** arranged at right angles to each other like a letter L in frontal view. The horizontal plate forms the posterior part of the bony palate. As shown in Figure 27.3, the perpendicular plate articulates with the roughened posterior area of the medial surface of the maxilla to form the posterior part of the lateral nasal wall and covers over the posterior part of the maxillary hiatus, a large defect in the lateral nasal wall leading into the maxillary air sinus. In Figure 27.3, you can see that some of the inferior wall is overlain by the separate **inferior nasal concha**, a scroll-like plate of bone projecting into the nasal cavity. Figure 27.3 also shows the minor contributions of the **nasal** and **lacrimal bones**.

As shown in Figure 27.1, the conchae incompletely divide the nasal cavity into three horizontal passages—the **superior meatus** between the superior and middle conchae, the **middle meatus** between the middle and inferior conchae, and the **inferior meatus** between the inferior concha and the palate. The area of the lateral wall above the superior concha is termed the **sphenoethmoidal recess** because it forms a recess under those two bones.

The nasal surface of an isolated maxilla is shown in Figure 27.4; the shaded areas indicate where the maxilla articulates with other bones. Identify the prominent **frontal process** and follow it down to the smoothly curved medial surface. The upper posterior area is roughened where it articulates in a complex manner with the perpendicular plate of the palatine, ethmoid labyrinth, inferior concha, and lacrimal bones. The **maxillary hiatus** is the large opening into the maxillary sinus in this area, but is only visible on the maxilla in isolation. When the maxilla is articulated with the bones listed above, the maxillary hiatus is reduced to a single opening on the lateral wall between the superior and inferior conchae; sometimes there are a number of small openings.

The formation of the nasolacrimal groove in the lateral nasal wall is described in Box 27.1 for those who require this information.

The superior part of the lateral nasal wall posterior to the lacrimal bone is formed by the medial plate of the **ethmoidal labyrinth** as shown in Figures 27.1 and 27.3A. The lateral plate of the labyrinth forms the majority of the medial wall of the orbit. The **ethmoidal air cells** are between the two plates of the labyrinth; these open into the nasal cavity into the superior and middle meatus. The **superior** and **middle nasal conchae** project into the nasal cavity from the medial

Box 27.1 The nasolacrimal groove

The deep **nasolacrimal groove** is immediately behind the frontal process in Figure 27.4. The small lacrimal bone fills the gap in the lateral wall of the nasal cavity between the frontal process of the maxilla and the ethmoidal labyrinth and its orbital surface forms a small part of the medial wall of the orbit. An inferior process medial to the nasolacrimal groove converts it into the **nasolacrimal canal**. The inferior concha articulates with the edges of the nasolacrimal groove to complete the canal. The nasolacrimal canal begins at the lacrimal groove in the medial wall of the orbit and opens into the lateral wall of the nose below the inferior concha. The nasolacrimal canal drains tears from the eyes into the nasal cavity (see Section 30.3.4).

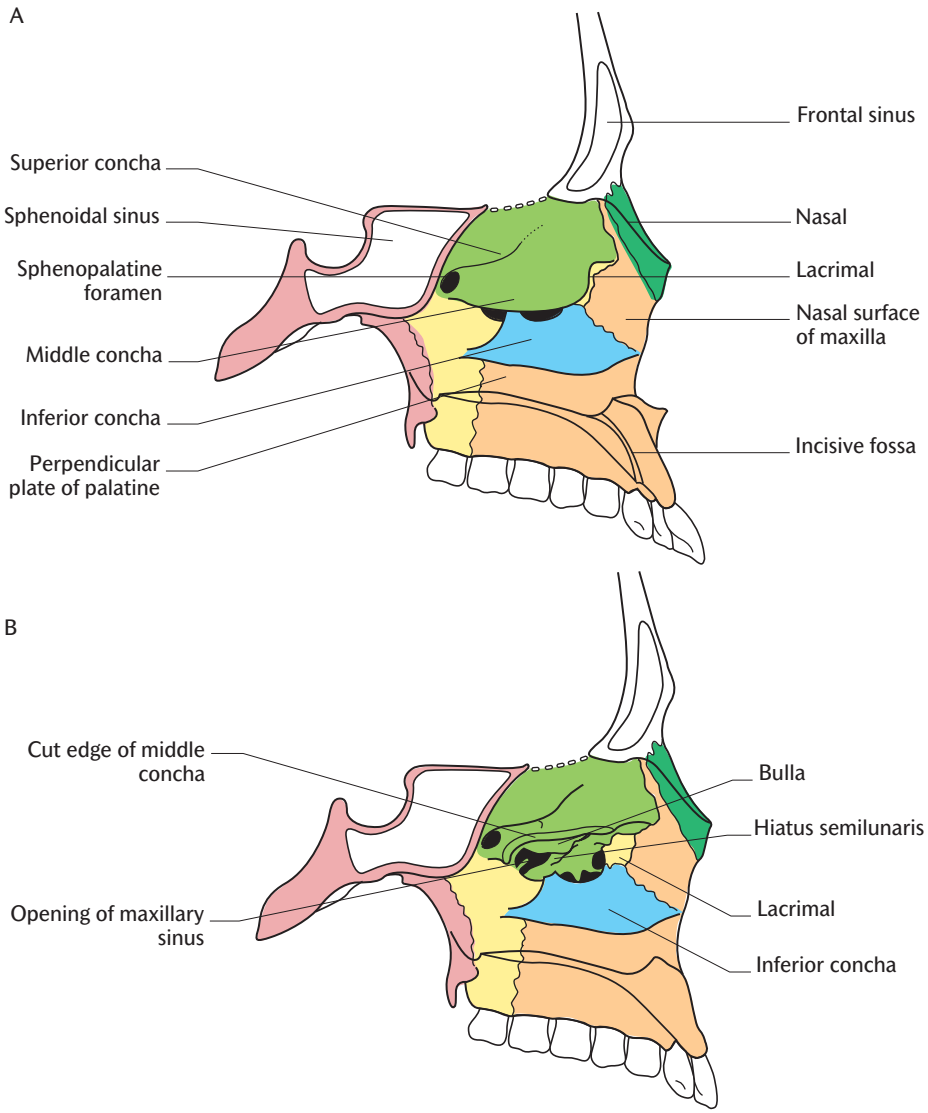


Fig. 27.3 A) The skeleton of the lateral wall of the nasal cavity; B) The lateral wall of the nasal cavity with the middle concha removed to show the structures of the middle meatus.

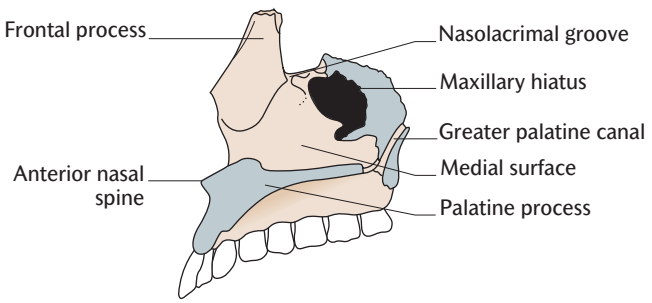


Fig. 27.4 A medial view of the maxilla.

plate. Figure 27.1 also shows the rounded swelling termed the **bulla ethmoidalis** below the middle concha. The **hiatus semilunaris** is a curved gap beneath the bulla. These are not just bony structures; as Figure 27.8 shows, the covering mucosa conforms to the bones so the bulla and hiatus are visible on the lateral wall of the nasal cavity under the cover of the middle concha. The opening of the maxillary sinus is in the posterior part of the hiatus semilunaris and the **frontal sinus** opens through the frontonasal canal into the anterior part of the hiatus semilunaris.

The nasal septum is illustrated in Figure 27.5. It is a thin sheet of bone extending from the roof of the nasal cavity to its floor formed by the **perpendicular plate of the ethmoid above** and the **vomer** below. It frequently deviates considerably from the median plane.

27.3.1 Nasal mucosa

The whole of the nasal cavity is lined with **respiratory mucosa**, except for part of the roof and adjacent walls which are lined with olfactory epithelium; the vestibule is lined by hairy skin.

Olfactory epithelium lines the roof and upper parts of the septum and lateral walls down to, and including, the superior conchae. It contains olfactory receptor cells which are the equivalent of primary sensory neurons. Unmyelinated axons run from the basal aspect of these cells through the **cribriform plate** to the **olfactory bulb** where they

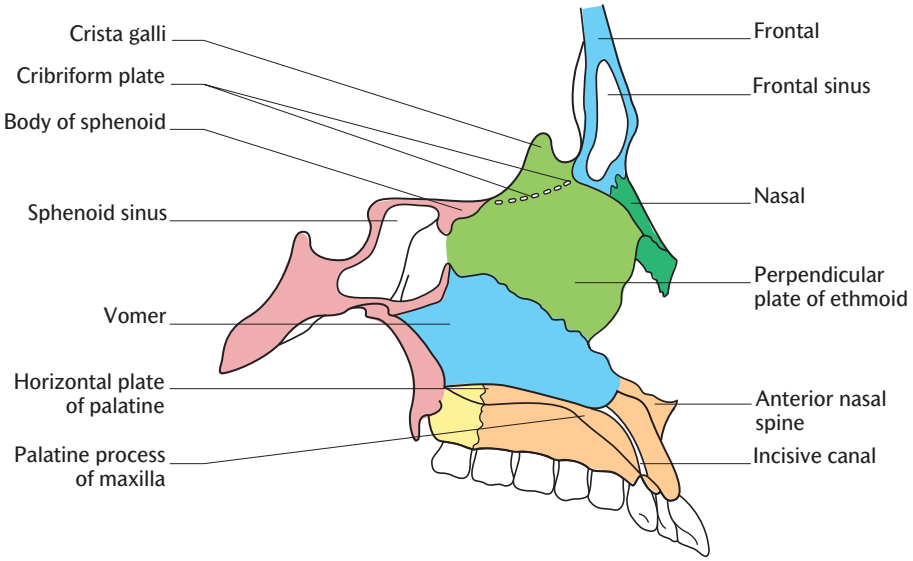


Fig. 27.5 The skeleton of the nasal septum.

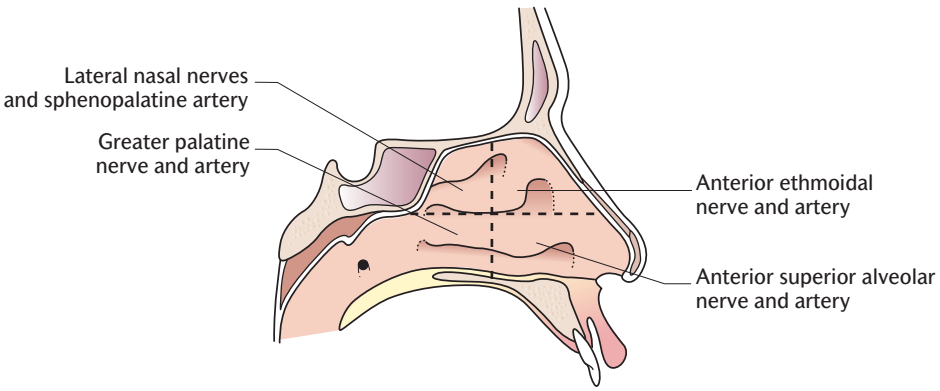


Fig. 27.6 The nerve and blood supply of the lateral wall of the nasal cavity.

synapse with neurons forming the olfactory nerves. The olfactory mucosa also contains numerous serous glands.

Respiratory mucosa lines the remainder of the nasal cavity. It is covered by ciliated columnar epithelium with mucus-secreting goblet cells. The connective tissue under the epithelium contains numerous minor mucous and serous glands. Extensive areas of vascular cavernous tissue lie beneath the mucosa; these are best developed over the conchae and warm inspired air. The mucosa is thick over the bony part of the septum, but is thinner and is tightly bound down to the underlying perichondrium over the cartilaginous part. The functions of respiratory mucosa in the nose have been outlined in the introduction to this chapter.

27.3.2 Nerve supply, blood supply, and lymphatic drainage of the nasal cavity

The innervation and arterial supply of the nasal cavity have a similar arrangement and can be described together.

The lateral wall

The lateral wall may be divided into quadrants as shown in Figure 27.6, on the basis of their different nerve and blood supply. The arteries and nerves supplying each quadrant are indicated in the figure.

The venous drainage of the posterior part of the lateral wall of the nasal cavity is to the **pharyngeal** and **pterygoid venous plexuses** while that of the anterior part is to the **facial vein**. The lymph drainage from the posterior part of the lateral wall is to the **retropharyngeal lymph nodes** and from the anterior part to the **submandibular nodes**.

The septum

Most of the septum is innervated by the medial nasal branches of the **maxillary nerve** and receives blood through the sphenopalatine branch of the **maxillary artery**. These structures enter the nasal cavity through the sphenopalatine foramen, pass across the roof to the upper border of the septum where they turn to run forwards and downwards towards the incisive fossa. The **nasopalatine nerve** passes through the incisive fossa to supply the lining mucosa of the anterior part of the hard palate and only supplies filaments to the septum en route. The sphenopalatine artery finishes short of the incisive fossa, usually anastomosing with an ascending branch of the greater palatine artery which runs upwards through the fossa.

Venous blood from the posterior half of the septum drains into the pterygoid plexus and lymph into the retropharyngeal nodes. Venous blood passes into the facial vein from the anterior half of the septum and lymph into the submandibular nodes.

The mucous glands receive parasympathetic secretomotor neurons from the facial nerve which synapse in the pterygopalatine ganglion.

There are extensive **anastomoses** between the **facial** arterial branches supplying the external nose and the **maxillary** arterial branches supplying the nasal cavity situated about where the nasal cartilages join the bones. These anastomotic vessels are fragile and bleed profusely after blows to the nose.

27.4 The paranasal air sinuses

Four of the cranial bones around the nasal cavity are hollowed out to contain air sinuses which communicate with the nasal cavity, hence the name paranasal air sinuses. Sinuses are found in the **frontal**, **sphenoid** and **maxillary** bones, and in the **ethmoidal labyrinth**. They are irregularly shaped cavities and vary greatly in size and shape from one individual to another and even between sides in the same individual. They enlarge considerably during adolescence (see Section 33.4.1) and continue to expand very slowly throughout adult life. These cavities are lined by respiratory mucosa which is often involved in upper respiratory tract infections; **sinusitis**, inflammation of their linings, is a common experience following a head cold. Sinusitis is often accompanied by general malaise and sometimes tenderness over the infected sinus. The maxillary sinuses may also be involved in dental infections and in dental surgical procedures (see Box 27.2).

Respiratory mucosa lines the sinuses. The movement of the cilia sweeps the mucus produced by the goblet cells and numerous mucous glands towards the opening of the sinus and so aids drainage. The number of cilia is frequently reduced or lost altogether following chronic sinus infection, thus reducing drainage and initiating a vicious circle in which continued infection is more likely. This is particularly likely to occur in the maxillary sinus because the opening is sited high up on the medial wall and drainage by gravity alone is impossible (see Section 27.4.1).

The function of the paranasal sinuses is uncertain. They certainly act as resonators modifying the voice; excess mucus in the sinuses during sinusitis can adversely affect the character of voice. It has been suggested that they reduce the weight of the head but the saving is so small that wearing a pair of spectacles would negate the effect. The most likely explanation for the sinuses is that they provide a considerable reservoir of mucus to add to that produced by the mucosa lining the nasal cavity itself. Furthermore, the sinuses are out of the direct airstream through the nose; when the mucus in the nose is dried under extremes of temperature, a supply will still be available from the sinuses.

27.4.1 The maxillary sinus

The **maxillary sinus** used to be called the **maxillary antrum** and you are likely meet this name in clinical contexts. These large sinuses in the body of each maxilla are roughly pyramidal in shape with the base adjacent to the lateral wall of the nose and the apex extending laterally into the zygomatic process as shown in Figure 27.7. The nasal, orbital, anterior, and infratemporal surfaces of the maxilla form the very thin walls of the sinus. The inferior wall of the sinus forms the base of the sockets of the premolars and first and second molars but is quite variable. It may be reasonably thick or very thin or may even be absent with only the mucosa lining the sinus covering the tooth roots. The floor of the sinus may extend forwards or backwards to contact the roots of the upper canine or third molar (see Figure 31.1).

Box 27.2 Clinical anatomy of the maxillary sinus

Because the maxillary teeth and maxillary sinuses share a common nerve supply, it is not uncommon for pain to be *referred* from one structure to the other. Patients may visit the dentist complaining of toothache but clinical investigation shows no dental caries or infection. The maxillary sinus should be investigated by tapping the cheek for signs of tenderness and by taking a panoramic radiograph in which the floor of the sinus is visible (see Chapter 31 and Figure 31.1). The sinus shows as a black radiolucency when healthy but will be white and opaque if excess mucus or pus is collecting in its floor. The converse may also occur. Patients may visit their doctor complaining of sinusitis, but do not show the usual symptoms of maxillary sinusitis such as tenderness of the cheek and their voice sounds normal. In such cases, there is a strong likelihood that the patient has a dental problem and should be referred to their dentist. Maxillary sinusitis is usually treated with antibiotics but surgical drainage is sometimes required.

It is easy to appreciate that dental infections can spread from an infected tooth pulp into the sinus, especially if the bone of the floor separating the tooth roots and sinus is thin or absent. When extraction of any tooth is carried out, the tooth should always be inspected to make sure the roots have not broken during the extraction. If it is a maxillary tooth with roots close to the floor of the sinus, it should also be inspected for the presence of bone on the roots to check whether the floor of the sinus has been fractured. If this has occurred, it is likely that a passage has been created between the tooth socket and the sinus called an **oroantral fistula**. A fistula is a conduit for infection and must be closed off. If an oroantral fistula is suspected the patient can be asked to close their mouth and try to blow out their cheeks. If a fistula is present, they will not succeed as air will escape from the mouth into the nose. A fistula may also be detected by asking the patient to blow through their nose while pinching their nostrils; air will fill the sinus and bubble through blood in the socket.

Occasionally, during extraction of an upper second or third molar, the alveolar process and tuberosity may break, sometimes exposing the maxillary sinus.

The orbital plate of the maxilla forms the floor of the orbit and roof of the sinus. The **infraorbital nerve** crosses the orbital plate to the infraorbital foramen and the canal frequently produces a ridge which can be seen from the inside of the sinus. The canal transmitting the **anterior superior alveolar nerves** is also within a ridge on the anterior wall of the sinus. The posterior superior alveolar nerves and blood vessels passing down the posterior wall of the sinus to the molar teeth beneath the mucosa lining the sinus do not usually mark the bone; in some cases, they are contained within canals in the wall of the sinus. The superior alveolar nerves supply the mucosa lining the maxillary sinus as well as the maxillary teeth. This common nerve supply is clinically important as described in Box 27.2. Corresponding arteries supply blood and the venous drainage is into the pterygoid plexus. Lymph drainage is to the submandibular nodes.

As described above and shown in Figure 27.8, the sinus opens into the posterior part of the **hiatus semilunaris** in the middle meatus of the nose by one or more small apertures; these may not be clearly visible as they are more or less closed by mucosa in life. The sinus openings are about two thirds of the way up the medial wall of the sinus from its floor; mucus drainage cannot be achieved by gravity so is dependent upon the action of the cilia of the lining epithelium which beat in a spiral pattern towards the openings.

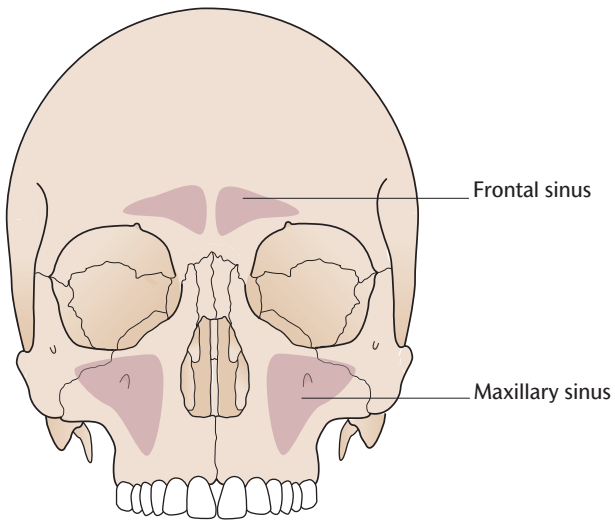


Fig. 27.7 The outlines of the maxillary and frontal sinuses. The maxillary sinus does *not* extend into the zygomatic bone. This is an optical illusion resulting from the fact that the zygomaticomaxillary suture runs obliquely outwards from the surface line; the upper posterior maxilla, therefore, extends beyond the surface suture line.

27.4.2 The frontal sinus

The frontal bone above the orbits is hollowed out by two frontal sinuses as shown in Figure 27.7. These are irregularly shaped cavities which extend to variable degrees backwards into the roof of the orbit and upwards into the frontal region as you can see in Figure 27.3. They are separated from each other by a septum which often deviates considerably from the midline so the two sinuses are frequently of unequal size.

The lining of the frontal sinus is innervated by branches of the **supraorbital nerve**. Its blood supply is through the supraorbital and anterior ethmoidal arteries and the venous drainage is into the supraorbital and superior ophthalmic veins. Lymph drainage is into the submandibular nodes.

As shown in Figure 27.8, the frontal sinus usually opens through the frontonasal canal into the anterior part of the **hiatus semilunaris** of the **middle meatus** of the corresponding side via the infundibulum in the ethmoidal labyrinth. Sometimes, the canal may open directly into the anterosuperior part of the middle meatus. Infected material draining from the frontal sinus often infects the maxillary sinus by tracking down the hiatus semilunaris which provides a natural channel into the maxillary opening.

Frontal sinusitis may present as a dull ache in the frontal region over the sinuses which may be tender.

27.4.3 The sphenoidal sinus

The sphenoidal sinuses are visible in Figures 27.3 and 27.4. The body of the sphenoid bone is hollowed out by two large irregular cavities separated from each other by a septum. The septum is usually deflected to one side or other so the left and right sinuses are asymmetrical. As shown in Figure 27.8, each sinus opens into the **sphenoethmoidal recess** of the same side.

The sphenoidal sinus receives its nerve and blood supply through the posterior ethmoidal nerve and vessels. Lymph drainage is into the retropharyngeal nodes.

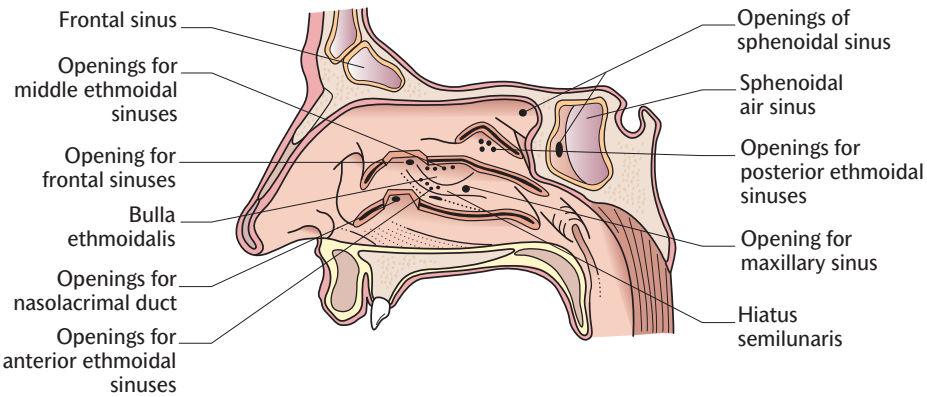


Fig. 27.8 The lateral wall of the nose showing the openings of the paranasal sinuses.

Table 27.1 Structures opening into the lateral wall of the nose

Spheno-ethmoidal recess	Sphenoidal air sinus
Superior meatus	Posterior ethmoidal air cells
Middle meatus	Frontal and maxillary sinuses; anterior and middle ethmoidal air cells
Inferior meatus	Nasolacrimal canal

27.4.4 The ethmoidal air cells

Each ethmoidal labyrinth contains a variable number of interconnected thin-walled air cells as shown diagrammatically in Figure 27.1. They are divided into three groups on the basis of their openings into the nasal cavity shown in Figure 27.8:

- The **posterior group** opens into the superior meatus, usually by a single orifice;
- The **middle group** occupies the bulla and opens by one or more orifices into the middle meatus above the bulla;
- The **anterior group** opens by one or more orifices into the frontonasal canal in the middle meatus.

The ethmoidal sinuses are innervated by the anterior and posterior ethmoidal nerves and their blood supply and venous drainage are through the corresponding arteries and veins. Lymph from these sinuses passes into the submandibular and retropharyngeal nodes.

The parasympathetic nerve supply to the mucous glands in the sinus linings is from the facial nerve via the pterygopalatine ganglion.

The structures opening into the lateral wall of the nose are summarized in Table 27.1.

28

The pharynx, soft palate, and larynx

Chapter contents

28.1 Introduction

The **pharynx** is a muscular tube beginning behind the posterior nasal apertures and extending down to the cricoid cartilage in the neck where it becomes continuous below with the oesophagus. The pharynx is incomplete anteriorly where the nasal and oral cavities and laryngeal entrance open into it. The pharynx can thus be divided for descriptive purposes into three regions. Identify the three regions and their landmarks in Figure 28.1:

- The **nasopharynx** behind the posterior nasal apertures, extending down to the soft palate;
- The **oropharynx** posterior to the oral cavity, extending from the soft palate to the tip of the epiglottis;
- The **laryngopharynx** posterior to the laryngeal entrance between the epiglottis and level of the cricoid cartilage.

Air enters the nasopharynx and passes through the oropharynx to enter the larynx at the laryngopharynx. Food and drink enter the oropharynx from the mouth, then travel through the laryngopharynx to the oesophagus and stomach. The pharynx is thus part of both the gastrointestinal and respiratory tracts. Essentially, food and air cross each other's paths in the laryngopharynx. This anatomical arrangement requires precise control of swallowing to ensure that food and drink enter the oesophagus and not the larynx.

As you can see in Figure 28.1, the **larynx,** the last part of the upper respiratory tract, is situated superficially in the midline of the neck in front of the pharynx and is only covered anteriorly by skin, fascia, and the infrahyoid muscles. The laryngeal opening is just behind and below the root of the tongue. The walls of the larynx are reinforced by cartilage like the lower respiratory tract. However, instead of simple cartilage rings or plates in the walls attached to each other by fibrous tissue, the elaborately shaped laryngeal cartilages articulate with each other through synovial joints and can be moved with precision by the laryngeal muscles. The vocal folds stretch anteroposteriorly across the larynx; they can be brought together to close the larynx and protect the lower respiratory tract or tensed to produce noise, the phonation component of speech.

The **soft palate** is a flap of muscular tissue attached to the posterior edge of the hard palate. As illustrated in Figure 28.1, it hangs down into the pharynx when relaxed. It is raised during swallowing to close off the nasopharynx to prevent food and drink entering the nasal cavity. It is also used during articulation of speech to direct air through the mouth or nose.

28.2 The pharynx

The pharynx extends from the base of the skull to the lower border of the cricoid cartilage where it is continuous with the oesophagus; it is about 12 cm long.

The **nasopharynx** lies behind the nasal cavity and above the soft palate. As you can see in Figure 28.1, the **auditory tube** opens into the lateral wall of the nasopharynx a short distance behind the nasal

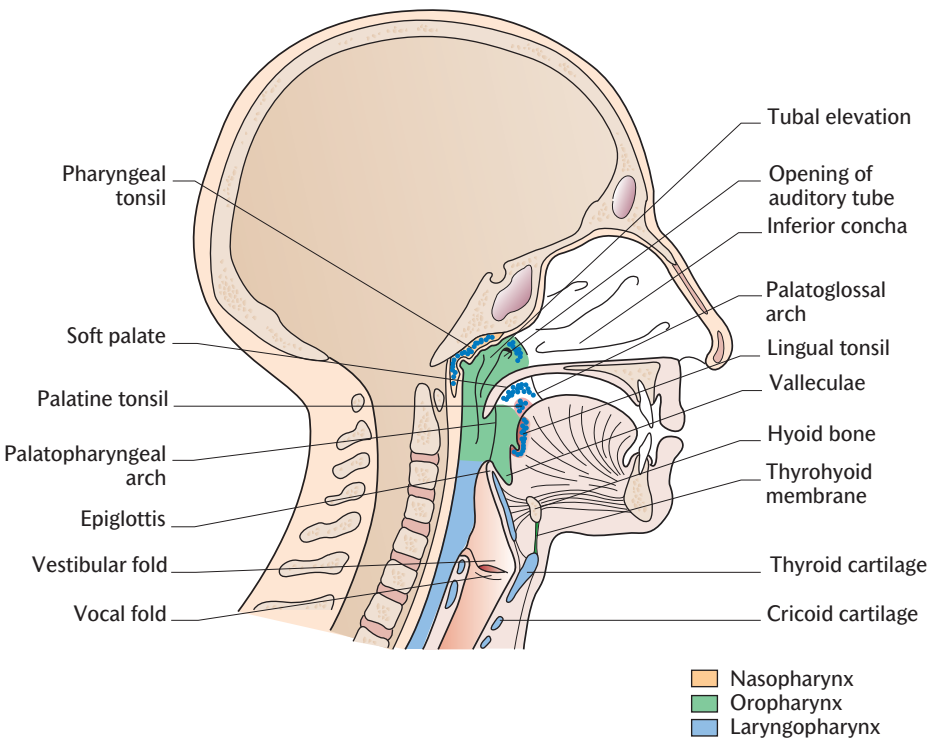


Fig. 28.1 Sagittal section of the head and neck to show the pharynx and larynx.

Box 28.1 Help! My child has swallowed part of his toy

The valleculae are about the same size as the sort of object that young children may put in their mouth such as a dried pea, a small pebble, or the head of a Lego® figure. They can become stuck in the valleculae, causing pain and minor obstruction. They cannot be removed by coughing as the airstream is deflected by the epiglottis, therefore, have to be removed manually.

aperture. This opening is deepened above and behind by the **tubal elevation** produced by the underlying cartilage of the auditory tube. Nodules of lymphoid tissue forming the **tubal tonsils** are located in the mucosa around the opening of the auditory tube. The roof and posterior wall of the nasopharynx are under the body of the sphenoid and the basiocciput in the central part of the cranial base. Figure 28.1 also shows the position of the **pharyngeal tonsil** or **adenoid** which is a collection of lymphoid tissue beneath the mucosa lining the upper part of the posterior wall. Respiratory mucosa lines the nasopharynx and receives its sensory nerve supply from the **maxillary trigeminal nerve** and secretomotor nerves through the facial nerve via the pterygopalatine ganglion. The nasopharynx, unlike the oro- and laryngopharynx, is always open because its walls are kept patent by their attachments to the base of the skull behind and the pterygoid plates in front (see Section 28.2.1).

The **oropharynx** extends from the soft palate to the tip of the epiglottis. Identify the features described in Figure 28.1. The oral cavity opens into the oropharynx anteriorly through the **oropharyngeal isthmus** formed by the **palatoglossal arches** overlying the **palatoglossus muscles**, descending from the soft palate to the side of the tongue. The **valleculae** are two shallow pits between the posterior surface of the tongue and the epiglottis separated from each other by the median glossoepiglottic fold of mucosa. The **palatopharyngeal arches** produced by the underlying **palatopharyngeus muscles** produce vertical mucosal ridges on the lateral wall of the oropharynx behind the palatoglossal arches. The **tonsillar fossa** is a depression between the palatoglossal and palatopharyngeal arches which contains the **palatine tonsil** on each side; a fibrous capsule encloses the tonsil laterally (see Figure 25.2A). The appearance of the palatine tonsils to the naked eye has already been described in Section 25.2.3. The oropharynx is lined by stratified squamous epithelium and its nerve supply is from the **pharyngeal plexus** (see Section 28.2.2).

The **laryngopharynx** extends from the epiglottis to the lower border of the cricoid cartilage. The **laryngeal inlet** is in its anterior wall below the epiglottis. There is a small recess termed the **piriform fossa** either side of the inlet (see Figure 28.11). The laryngopharynx is lined by stratified squamous epithelium and is innervated from the vagus nerve through its internal and recurrent laryngeal branches. When there is no transit of food or liquid, the laryngopharynx and oropharynx are flattened against the cervical vertebrae narrowing their lumen.

The **pharyngeal**, **tubal**, and **palatine tonsils** in the pharynx, together with the **lingual tonsils** in the posterior tongue, form an incomplete ring of lymphoid tissue around the upper parts of the respiratory and alimentary passages. The tonsils together with accumulations of lymphoid tissue in the small intestine and appendix are collectively known as mucosa-associated lymphoid tissue. They have the same functions as lymph nodes and respond to potentially harmful foreign organisms and/or their toxins entering the oropharynx through the nose or mouth by producing immune responses. The consequences of tonsillar enlargement are outlined in Box 28.2

Box 28.2 Tonsillitis

The tonsil may become enlarged in chronic infections of the upper respiratory tract. The palatine tonsils are variable in size and frequently become infected and become enlarged and painful (**tonsillitis**). **Adenoids**, enlargement of the pharyngeal tonsil, may block the airway and lead to mouth breathing. Children are subject to novel exposure to infective organisms when they are born or start school, for instance. Their tonsils are about twice as large as adult tonsils, but the pharynx is relatively narrow so blockage is much more likely in children than adults. Infections may be treated with antibiotics, but (palatine) tonsillectomy and/or adenoidectomy may be considered after repeated chronic infections.

28.2.1 Pharyngeal muscles

The posterior and lateral pharyngeal walls are formed by three pairs of **pharyngeal constrictor muscles**. The **pharyngeal raphe** is a narrow band of connective tissue running vertically in the posterior midline of the pharynx; as shown in Figure 28.2, it is attached above to the pharyngeal tubercle, a slight elevation on the underside of the body of the sphenoid, and extends downwards to blend with the connective tissue surrounding the oesophagus. Figure 28.2 illustrates each pair of muscles sweeping around the side of the pharynx to a variety of anterior structures from their posterior attachment to the raphe. They are arranged in a superior–inferior sequence rather like stacked plant pots or ice cream cones with the muscle below partially overlapping the muscle above.

The **stylopharyngeus** and **palatopharyngeus** are more vertically orientated muscles inserted into the pharyngeal wall. Palatopharyngeus is described with the soft palate in Section 28.3.1. The muscles of the pharynx are illustrated in Figures 28.2 and 28.3; examine them as you read the following description.

The superior constrictors

The uppermost fibres of these muscles are inserted into the **pharyngeal tubercle**; below this, they attach to the pharyngeal raphe as far as the lower limit of the nasopharynx. The muscles form a thin sheet around the posterior and side walls of the pharynx. Each muscle attaches to the lower two-thirds of the posterior border of the **medial pterygoid plate**, including the hamulus and the **pterygomandibular raphe** below. This raphe extends between the pterygoid hamulus to the posterior end of the mylohyoid line on the medial surface of the mandible. The superior constrictors attach to it posteriorly and the buccinators anteriorly.

You can see in Figures 28.2 and 28.3 that there is a gap between the upper border of each superior constrictor muscle and the base of the skull. This is closed by the strong **pharyngobasilar fascia** which is pierced by the auditory tube and the levator veli palatini muscle (see Section 28.3.1).

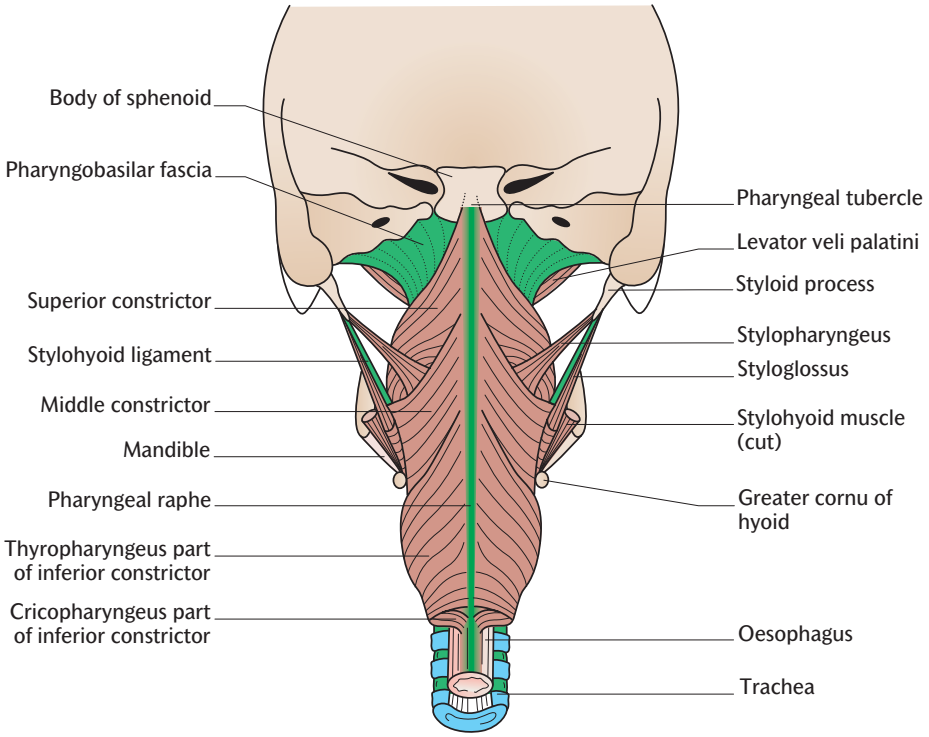


Fig. 28.2 Posterior view of the constrictor muscles of the pharynx.

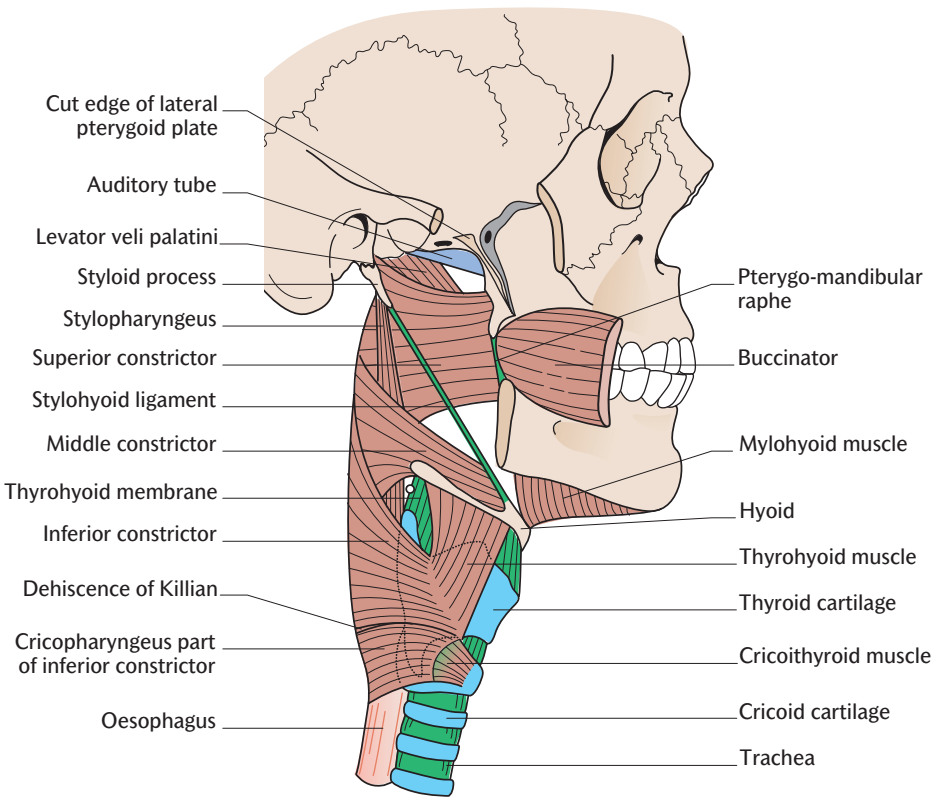


Fig. 28.3 Lateral view of the constrictor muscles of the pharynx.

The middle constrictors

The middle constrictors pass anteriorly around the pharynx from their posterior attachment to the pharyngeal raphe. The upper fibres run downwards, superficial to the lower part of the superior constrictor whereas the lower fibres run more horizontally, passing deep to the upper part of the inferior constrictor. They attach to the lower part of the **stylohyoid ligament** and between the lesser and greater cornua of the hyoid bone.

The inferior constrictors

Like the superior and middle constrictors, the inferior constrictor muscles attach posteriorly to the pharyngeal raphe. Each inferior constrictor consists of two parts. The upper part of the inferior constrictor runs anteriorly and downwards to the oblique line on the thyroid cartilage: it is sometimes called the thyropharyngeus and its uppermost fibres cover the inferior part of the middle constrictor. The lower part encircles the lowermost part of the pharynx in a thick band as the **cricopharyngeus muscle** which is sometimes regarded as a separate muscle. It is continuous inferiorly with the circular fibres of the upper part of the oesophagus and is attached anteriorly to the side of the arch of the cricoid cartilage. The inferior constrictor is attached to a tendinous band between the thyroid and cricoid cartilages which arches over the cricothyroid muscle. **Killian's dehiscence** is a posterior gap between the two parts of the inferior constrictor. The mucosal lining of the pharynx may protrude through this weak area, creating a pharyngeal pouch, a potential food trap.

Their name indicates precisely the action of the pharyngeal constrictor muscles. They constrict the pharynx in a wave of contraction from above downwards, moving food and liquids through the pharynx into the oesophagus. Their action in swallowing is described in Section 29.1.2.

The stylopharyngeus muscles

Each stylopharyngeus muscle arises from the medial surface of the **styloid process** and runs almost vertically downwards between the internal and external carotid arteries. As you can see in Figures 28.2 and 28.3, it crosses the lower border of the superior constrictor muscle and then continues downwards inside the middle constrictor, beneath the pharyngeal mucosa, to attach to the side wall of the pharynx and the posterior border of the thyroid cartilage. Some muscle slips arise from the lower part of the auditory tube close to its opening and pass downwards to blend with the constrictor muscles; these are sometimes called the **salpingopharyngeus muscle**.

Stylopharyngeus is an elevator of the pharynx and larynx. It is the only muscle derived from the third pharyngeal arch and is, therefore, innervated by the glossopharyngeal nerve.

28.2.2 Nerve supply of the pharynx

The **pharyngeal plexus** of nerves is in the connective tissue on the superficial surface of the constrictor muscles, especially the middle constrictors. The plexus is formed from the **pharyngeal branches** of the **glossopharyngeal** and **vagus nerves** and sympathetic vasoconstrictor nerves from the laryngopharyngeal branch from the superior cervical sympathetic ganglion.

The glossopharyngeal branches supply sensory nerves for the mucosa of the oropharynx and parasympathetic neurons to its glands. The preganglionic parasympathetic neurons synapse with post-ganglionic neurons within the pharyngeal mucosa. The pharyngeal branch of the vagus carries motor axons to the pharyngeal constrictor muscles and most of the muscles of the soft palate.

The lining mucosa of the nasopharynx is supplied by the pharyngeal branch of the **maxillary trigeminal nerve** which also carries facial post-ganglionic parasympathetic neurons from the pterygopalatine ganglion. The laryngopharynx receives its sensory and parasympathetic innervation through the **internal** and **recurrent laryngeal branches** of the **vagus**. The internal laryngeal nerve also supplies taste buds in the mucosa of the epiglottis and valleculae.

The vagus nerves

The vagus nerves leave the cranial cavity through the middle compartment of the jugular foramina with the spinal accessory nerves. They continue vertically downwards within the carotid sheath, running between the internal jugular vein and the internal carotid artery at first, then between the internal jugular vein and the common carotid artery.

There are several branches of the vagus supplying structures in the head and neck; the more important ones are illustrated in Figure 28.4; trace them as you read the description.

The **pharyngeal branch** passes forwards between the internal and external carotid arteries below and parallel to the glossopharyngeal nerve to the middle constrictor where it joins with the pharyngeal branch of the glossopharyngeal nerve to form the **pharyngeal plexus.** The vagus supplies motor axons to the muscles of the pharynx and soft palate through this plexus.

The **superior laryngeal nerve** leaves each vagus below the pharyngeal branch. It descends on the side of the pharynx deep to the internal carotid artery and divides into internal and external laryngeal nerves. The **internal laryngeal nerve** pierces the posterior part of the thyrohyoid membrane to supply somatosensory nerves to the pharynx around the laryngeal inlet and the lining of the larynx as far down as the vocal folds. The **external laryngeal nerve** runs downwards on the inferior constrictor to supply the **cricothyroid muscle**.

Superior and inferior **cardiac branches** run downwards into the thorax to join the **cardiac plexuses;** they convey the parasympathetic innervation of the heart.

The **recurrent laryngeal nerve** is the lowest branch supplying structures in the neck. The course differs between the two sides. Figure 12.11A shows the right nerve leaving the vagus within the neck as it crosses in front of the subclavian artery; the nerve hooks under the artery and then ascends through the neck in the groove between the trachea and oesophagus. On the left side, the recurrent laryngeal nerve arises from the vagus at the lower border of the arch of the aorta within the thorax. As shown in Figure 12.11B, it winds beneath the arch and then ascends in the neck in the groove between the trachea and oesophagus. The recurrent laryngeal nerves on both sides are closely related to the terminal branches of the **inferior thyroid artery** near the lower pole of the lobe of the thyroid gland; the nerve usually passing behind but, not infrequently, between or in front of these vessels. The relationships of the recurrent laryngeal nerve to the thyroid gland and its arteries are of clinical significance in surgery of

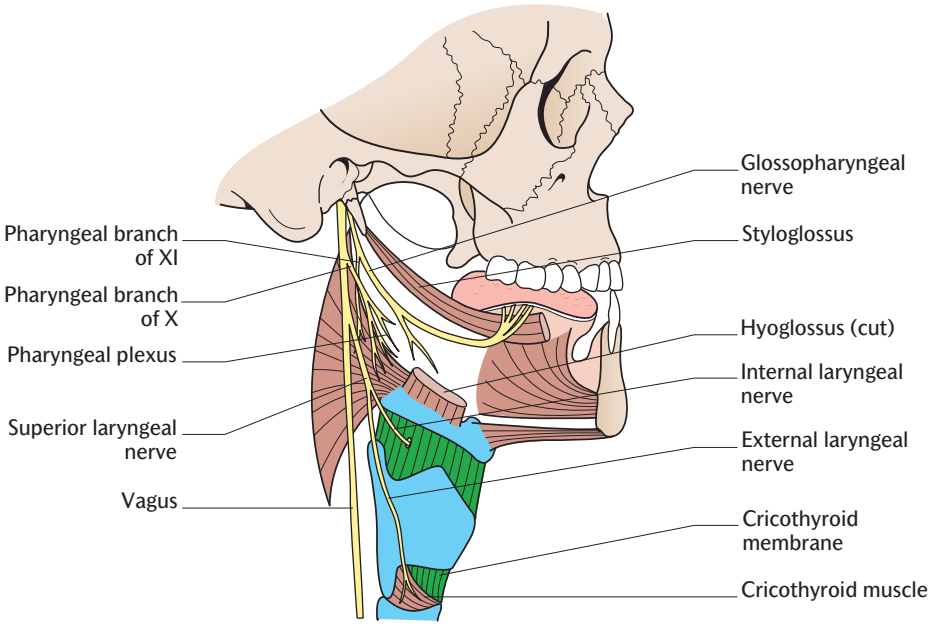


Fig. 28.4 The vagus and glossopharyngeal nerves and the formation of the pharyngeal plexus.

the thyroid gland (see Box 28.7). The nerve then enters the larynx deep to the lower border of the inferior constrictor behind the cricothyroid articulation. It supplies all the muscles of the larynx, except cricothyroid, the laryngeal mucosa below the vocal folds, and branches to the mucosa and muscular coats of the oesophagus and trachea.

Two small branches not illustrated in Figure 28.4 are the meningeal branch which supplies the dura mater of the posterior cranial fossa and the auricular branch which passes through the temporal bone to supply the skin of the auricle, the lining of the external acoustic meatus, and outer surface of the tympanic membrane.

The bulk of each vagus nerve continues downwards into the thoracic and abdominal regions and carries the parasympathetic and visceral sensory supply of the viscera of the trunk.

28.2.3 Blood supply and lymph drainage

The pharynx receives blood from various branches of the external carotid arteries. The details are given in Box 28.3 for those who require this information.

Box 28.3 Blood supply of the pharynx

The arterial branches of the external carotid artery supplying the pharynx are, from above downwards: the pharyngeal and greater palatine branches of the maxillary artery, the ascending palatine and tonsillar branches of the facial artery, ascending pharyngeal artery, branches of the lingual artery, and superior laryngeal branches of the superior thyroid arteries. The inferior laryngeal branches of the inferior thyroid arteries from the subclavian arteries complete this formidable list.

The veins draining the pharynx form a **pharyngeal venous plexus** on the superficial surface of the constrictor muscles. The plexus drains into the internal jugular vein and has connections to the pterygoid venous plexus.

The lymphatic channels from the pharynx pass to the deep cervical nodes, either directly or through the retropharyngeal nodes.

28.3 The soft palate

The soft palate (also called the **velum)** is a mobile muscular flap, consisting of four pairs of muscles attached to a fibrous aponeurosis suspended from the posterior border of the hard palate. It projects downwards and backwards, separating the nasopharynx and oropharynx. It is continuous on each side with the lateral wall of the pharynx through the palatoglossal and palatopharyngeal arches, running downwards to the tongue and side wall of the pharynx, respectively. The **uvula** is a conical projection of variable length, hanging from the lower free margin of the soft palate. The mucosa of the soft palate is lining oral mucosa on its oral side and respiratory mucosa on the nasopharyngeal surface which receives their sensory nerve supply from the lesser palatine branches of the maxillary trigeminal nerve.

28.3.1 Palatine musculature

Two pairs of muscles raise and tense the soft palate and two pairs lower it. They are illustrated in Figure 28.5.

Tensor veli palatini

Each of these thin triangular muscles arises from the **scaphoid fossa** located at the upper end of the posterior border of the medial pterygoid

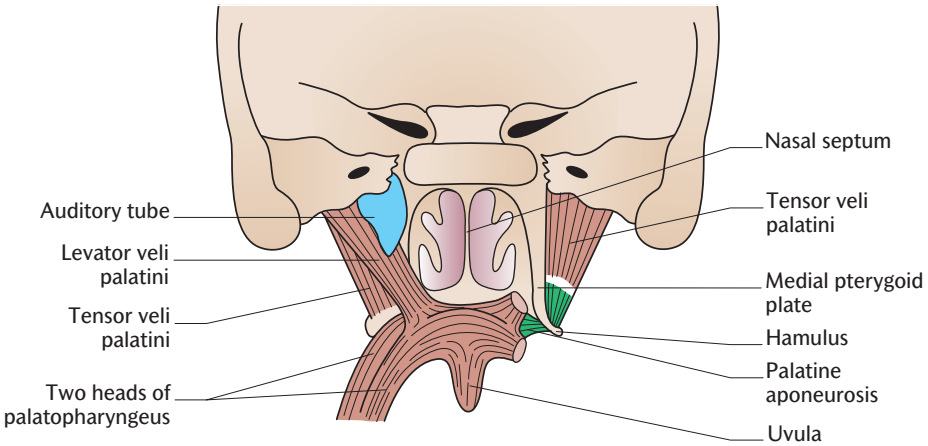


Fig. 28.5 Posterior view of the muscles of the soft palate. The levator veli palatini and auditory tube have been removed from the right side to show the underlying muscle.

plate (see Figure 24.9) and the lateral side of the cartilaginous part of the auditory tube. The fibres converge to a tendon which turns medially around the pterygoid hamulus into the soft palate. The tendon of each muscle broadens out and joins its opposite number to form the **palatine aponeurosis**. This thin strong sheet of fibrous tissue is attached to the posterior border of the bony palate and the other palatine muscles are attached to it.

The tensor muscle does what its name implies; it tenses the palatine aponeurosis so that the other palatine muscles can act. Tensing the soft palate also produces a rigid barrier between the nasopharynx and oropharynx. It is derived from the first pharyngeal arch and is, therefore, supplied by the mandibular division of the trigeminal nerve through its medial pterygoid branch.

Levator veli palatini

This muscle arises from the inferior surface of the petrous temporal bone anterior and medial to the carotid canal and from the medial side of the cartilaginous part of the auditory tube. It passes into the pharynx above the upper border of the superior constrictor and inserts into the posterior surface of the palatine aponeurosis between the two heads of palatopharyngeus. It elevates the soft palate when the palatine aponeurosis is tensed and is supplied by the vagus from the pharyngeal plexus.

Because they are attached to the cartilaginous part of the auditory tube, the tensor and levator veli palatini muscles open it as they tense and raise the soft palate; this enables air pressure on either side of the tympanic membrane to be equalized and the middle ear to drain (see Section 28.3.3). The consequences of poor drainage of the middle ear are described in Box 28.4.

Palatoglossus

This muscle arises from the oral surface of the palatine aponeurosis and runs downwards to insert into the side of the tongue, forming the palatoglossal arch beneath the mucosa of the oropharyngeal isthmus. When the two muscles contract, they pull up the posterior part of the tongue and approximate the palatoglossal arches, thus constricting the oropharyngeal isthmus. They can also depress the soft palate, depending on the action of other tongue muscles. The motor nerve supply of the muscle is from the pharyngeal plexus.

Palatopharyngeus

Palatopharyngeus arises by two heads separated from each other by the attachment of levator veli palatini as illustrated in Figure 28.5. The two heads pass laterally and downwards over the lateral border of the aponeurosis and unite to continue downwards beneath the pharyngeal mucosa, producing the palatopharyngeal arch. It inserts into the posterior border of the thyroid cartilage and the inferior constrictor muscle.

The palatopharyngeus muscles elevate the larynx and pharynx or depress the soft palate, depending on the action of other muscles. They are innervated by the pharyngeal plexus.

Separate groups of palatopharyngeal fibres attach to the lateral end of the posterior border of the bony palate and run horizontally backwards, encircling the pharynx within the superior constrictor raising an elevation known as **Passavant's ridge** on the interior aspect of the pharyngeal wall. These fibres are also referred to as the **palatopharyngeal sphincter** or 'Passavant's muscle'. The soft palate meets the pharyngeal wall at this ridge when it is elevated. It is innervated by the pharyngeal plexus.

28.3.2 Blood supply and lymph drainage

The soft palate receives blood from the lesser palatine branches of the maxillary artery with contributions from palatine branches of the ascending pharyngeal artery. Venous blood drains into the pharyngeal and pterygoid plexuses. Lymph drains into the retropharyngeal and upper deep cervical nodes.

28.3.3 The auditory tube

The **auditory tube** (also known as the **Eustachian** or **pharyngotympanic** tube) is the communication channel running downwards, forwards, and medially from the tympanic cavity of the middle ear to the nasopharynx. In the adult it is about 35 mm in length; the lateral one-third is within a bony canal whereas the medial two-thirds are enclosed in cartilage. The tube is a narrow cone which narrows from the nasopharyngeal opening to the bony part. It equalizes air pressure on the two sides of the ear drum and drains the middle ear cavity. It is illustrated in Figure 28.6.

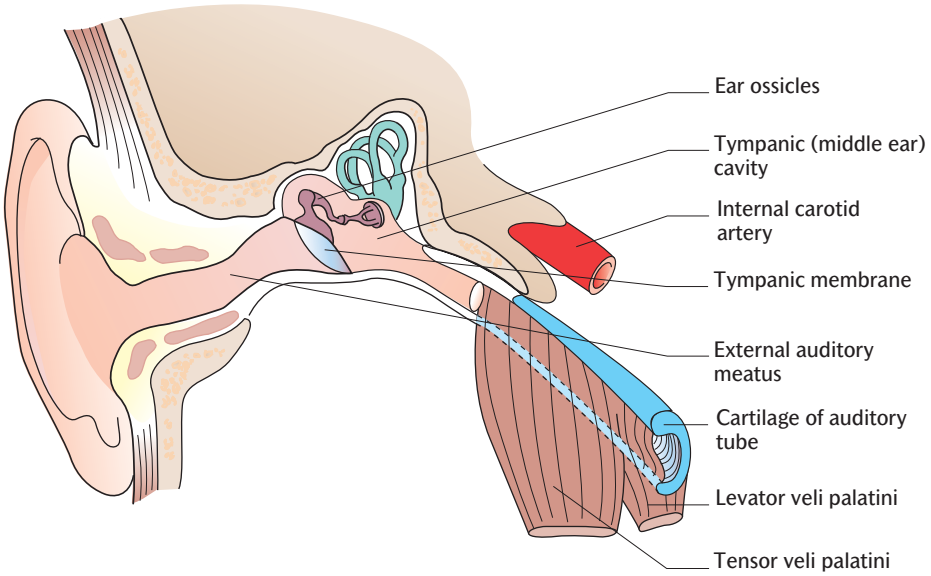


Fig. 28.6 The structure of the auditory tube viewed in coronal section.

Box 28.4 Middle ear infections

The tympanic or middle ear cavity should only contain air but as air pressure changes within the cavity, fluid is drawn from the lining mucosa. If drainage is compromised, the stagnating fluid may become infected, leading to **otitis media**; repeated ear infections are often called **'glue ear'**. These conditions are prevalent in young children because the auditory tube is more or less horizontal at birth so there is no natural drainage pathway; the auditory tube begins to incline downwards as the skull grows post-natally to reach its adult angulation by about the age of five. The presence of infected fluid within the tympanic cavity interferes with movement of the ear ossicles so conductive hearing loss is usually a problem of glue ear. Apart from the pain and discomfort, the hearing loss can interfere with language acquisition. Children with otitis media often grow out of it in early school years.

The dilating actions of the tensor and levator veli palatini on the auditory tube can be adversely affected in cleft palate; the weakened lower attachments of these muscles means that their opening on the auditory tube is not as efficient so drainage may be impaired. A small tube known as a grommet may be surgically placed in a small hole created in the lower part of the tympanic membrane to significantly improved drainage and equalization of pressure. Episodes of infection are reduced and hearing is improved.

The bony part of the tube begins at the anterior wall of the tympanic cavity within the petrous temporal bone and ends on the under surface of the cranial base in the angle between the squamous and petrous parts of the temporal bone just behind the spine of the sphenoid. A groove on the inferior surface of the cranial base continues medially from this opening in the angle between the petrous and squamous parts of the temporal bone. The cartilaginous part of the **auditory tube** occupies this groove. The tensor veli palatini attaches to the cartilaginous part of to the tube anterolaterally and levator veli palatini is posteromedial. Both muscles dilate the auditory tube during swallowing.

The tube is lined with respiratory mucosa. It receives its sensory nerve supply from the tympanic plexus and from the pharyngeal branch of the pterygopalatine ganglion. Its blood supply is from the ascending pharyngeal and middle meningeal arteries.

28.4 The larynx

The larynx has two main functions. It closes the larynx during swallowing, thus preventing accidental intake of food or liquids into the lower respiratory tract. Alterations in the position of the larynx during swallowing aid in this function. The cough reflex is elicited when foreign material enters the larynx and is also protective to the lower airway. The larynx is also the organ that generates sound; this is **phonation** and is an important component of speech in humans. It is easier to understand the larynx if you keep these major functions in mind as you study it.

As mentioned at the beginning of this chapter, the larynx shares a common structure with the rest of the conducting portion of the respiratory tract. It is a tube reinforced by cartilage. The crucial difference is that the laryngeal cartilages articulate with each other by synovial joints and can be moved with precision by the laryngeal muscles. We will build up the structure of the larynx by examining its skeletal structure, followed by the membranes and ligaments that link the cartilages, and then put in the muscles to move the larynx appropriately during swallowing and speech.

28.4.1 The laryngeal skeleton

The laryngeal skeleton is made up of cartilages suspended from the hyoid bone linked together by joints, membranes, ligaments, and muscles.

The laryngeal cartilages

Five major cartilages form the laryngeal skeleton. The **cricoid**, **thyroid**, and paired **arytenoids** cartilages consist of hyaline cartilage and tend to calcify with age. The **epiglottis** is composed of elastic cartilage. The cartilages are shown in an exploded view in Figure 28.7 so that the detail can be seen. The articulated cartilages are shown in Figures 28.8 and 28.10.

The thyroid cartilage

This is the largest of the laryngeal cartilages. It consists of two **laminae** joined anteriorly. The two laminae meet each other in the midline at an angle to form the **laryngeal prominence**, a projection at the upper part of the laminae. The angle between the two laminae is about 90° in men and about 120° in women, accentuating the laryngeal prominence in males, hence to its more usual name of the 'Adam's apple'. The larynx is generally larger in post-pubertal males than females as well. The V-shaped **superior thyroid notch** is immediately above the laryngeal prominence.

The stylopharyngeus and palatopharyngeus muscles attach to the thickened posterior border of each lamina. Figure 28.7 shows that the posterior border of each lamina also has upward and downward extension, the **superior** and **inferior horns**. As you can see in Figure 28.8, the superior horn is close to the posterior end of the greater cornu of the hyoid. The inferior horn articulates with the cricoid cartilage through a facet on each side as shown in Figure 28.8. The superior border is convex and attached to the hyoid bone above by the thyrohyoid membrane (see p. 301). The **superior tubercle** is just in front of the root of the superior cornu. The inferior border has a much straighter outline.

As shown in Figure 28.7, an **oblique line** runs downwards and forwards across the external surface of each lamina where the sternothyroid, thyrohyoid, and inferior constrictor muscles are attached. The internal surface of each lamina is smooth and forms the floor of the piriform recess.

The cricoid cartilage

This is the only complete ring of cartilage in the walls of the respiratory system. It is traditionally described as being shaped like a signet ring. As Figure 28.7 illustrates, it consists of a broad **lamina** facing posteriorly (equivalent to the part of a signet ring that bears the signature) and a narrow **arch** anteriorly. There are two articular facets on each side which form synovial joints with other laryngeal cartilages as you can see in Figure 28.7. The facet for the inferior cornu of the thyroid cartilage is located laterally at the junction of arch and lamina. The second facet is situated on the superior border of the lamina close to its lateral corner and articulates with the base of the corresponding arytenoid cartilage. The posterior surface of the lamina has two shallow depressions on either side of a median ridge where the posterior cricoarytenoid muscles attach. The cricopharyngeus part of the inferior constrictor arises from each lateral surface of the arch.

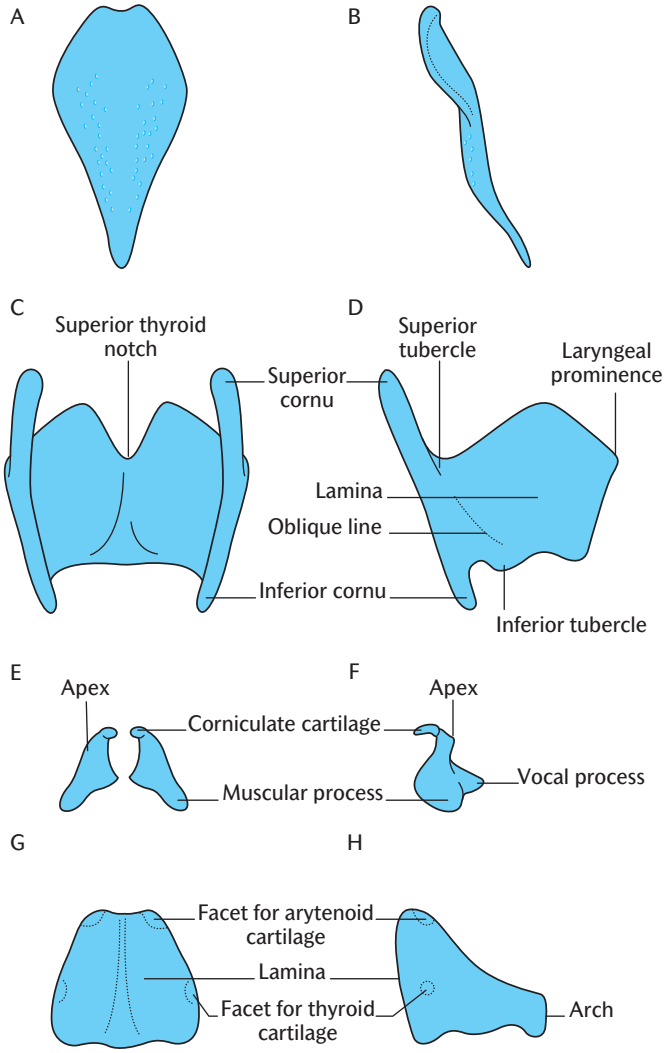


Fig. 28.7 An exploded view of the laryngeal cartilages; posterior view on the left and lateral view on the right. A, B) The epiglottis; C, D) The thyroid; E, F) The arytenoids; G, H) The cricoid cartilages.

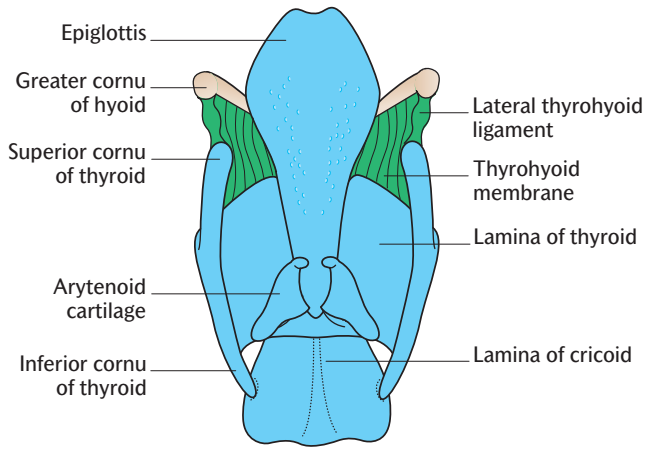


Fig. 28.8 Posterior view of the articulated cartilages of the larynx.

The arytenoid cartilages

The two small arytenoid cartilages are each shaped like a three-sided pyramid most clearly depicted in Figure 28.7. Each cartilage has a base, a posterior surface, a medial surface, an anterolateral surface, and an apex. The concave base articulates with the facet on the superolateral corners of the cricoid lamina. The posterior surface is also concave and is the area of attachment of the transverse interarytenoid muscle. The anterolateral surface is convex; the vestibular ligament is attached to its upper part and the lateral cricoarytenoid to its lower part (see p. 302). The **muscular process** is a rounded projection extending from the lateral angle of the arytenoid between the posterior and anterolateral faces. Not surprisingly, it is a site of muscle attachment—specifically the lateral and posterior cricoarytenoid muscles. The **vocal process** is a thin pointed projection from the anterior angle between the medial and anterolateral faces; the vocal ligament and vocalis muscle attach to it.

Articulatory movements

The thyroid can be stabilized by the action of the infrahyoid muscles attached to it; it is the only cartilage within the larynx that can be fixed and is, therefore, the base from which articulatory movements of other cartilages take place.

The cricoid cartilage pivots between the two inferior horns of the thyroid cartilage as you can see in Figures 28.8 and 28.9. The cricothyroid muscles bring the arch of the cricoids upwards towards the inferior border of the thyroid laminae but, at the same time, as Figure 28.9 shows, the upper part of the lamina moves backwards, carrying the arytenoids cartilages with it. Figure 28.9 also shows the attachments of the vocal folds between the vocal processes of the arytenoids and the inner aspect of the thyroid laminae near the midline; movement at the cricothyroid joints will, therefore, lengthen the vocal folds.

The arytenoids cartilages can slide medially and laterally on the upper rim of the cricoids lamina. Lateral movement as shown in Figure 28.14A open the vocal folds whereas medial movement closes the vocal folds as Figure 28.14B shows. The concavity on the underside of the arytenoids cartilages is flatter than the convexity of the cricoids lamina; this lack of congruity means that some rotation of the arytenoids about a vertical axis is possible so that they twist clockwise or anticlockwise on the cricoid, also producing opening or strong closure shown in Figure 28.14C. Finally, the arytenoids can tilt forwards or backwards on the cricoid lamina; backward movement lengthens the vocal folds or whereas forward movement shortens them as indicated by the arrows on Fig 28.14.D.

The epiglottis

This is a thin, curled leaf-shaped cartilage as Figure 28.7 indicates. It is attached by its stalk to the inner aspect of the angle between the thyroid laminae just below the thyroid notch as you can see in Figure 28.10. Its broad superior part projects upwards into the pharynx. As you can see in Figure 28.10, the convex anterior surface lies behind the hyoid bone and the posterior surface of the tongue above and forms the anterior wall of the laryngeal inlet. The aryepiglottic folds run on either side of the inlet to the arytenoid cartilages (see Figure 28.12).

The **corniculate cartilages** are two tiny cones of cartilage on the apex of each arytenoid cartilage within the aryepiglottic fold. The

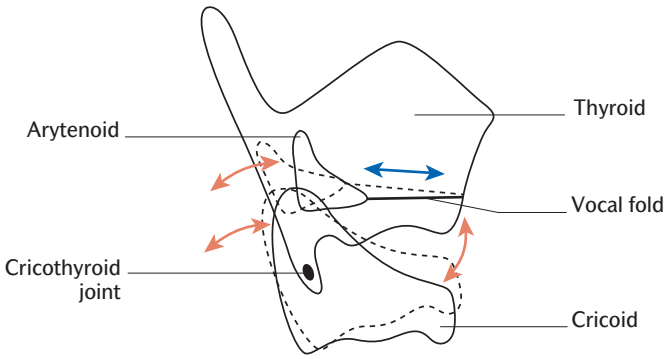


Fig. 28.9 Relative movements of the cricoid and thyroid cartilages at the cricothyroid joint produced by the cricothyroid muscle. Solid lines indicate the resting position and the dotted lines the final position.

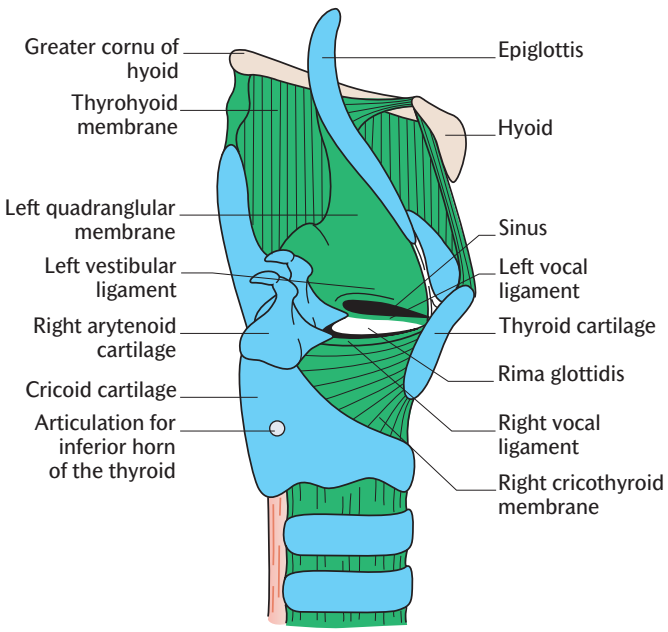


Fig. 28.10 Lateral view of right side of larynx to show the quadrangular and cricothyroid membranes. The right lamina of the thyroid cartilage and the right quadrangular membrane have been removed so the left quadrangular but right cricothyroid membranes are shown.

cuneiform cartilages are two small nodules above the corniculate cartilages in the aryepiglottic fold.

Ligaments and membranes

The gaps between the cartilages are filled by fibrous connective tissue membranes; thickened membranes are the ligaments. The membranes and ligaments also play a significant role in shaping the internal structure of the larynx as described in the next section .

The thyrohyoid membrane

The **thyrohyoid membrane** is a sheet of fibroelastic tissue. Identify it in Figures 28.8 and 28.10 and trace its attachments above to the body and greater cornua of the hyoid bone and below to the superior

borders of the laminae of the thyroid cartilage. The thyrohyoid membrane is pierced on each side by the superior laryngeal vessels and internal laryngeal nerve.

The quadrangular membrane

As illustrated in Figure 28.10, the **quadrangular membrane** attaches on each side on the anterior border of the arytenoid cartilage between the vocal process and apex and fans out to the lateral border of the epiglottis. Its upper oblique free border forms the **aryepiglottic fold**, the lateral wall of the laryngeal entrance. Its lower border is also free and is thickened to form the **vestibular ligament**. This ligament is one of the tissues that raise a ridge, the **vestibular fold** (or false vocal fold), in the mucosa lining the larynx; there are small aggregates of mucous tissue between the ligament and mucosa that bulk out the fold.

The cricothyroid membrane

The **cricothyroid membrane** can be seen on the interior view of the larynx in Figure 28.10 and from the exterior in Figure 28.13. Interiorly, it is roughly triangular in shape and is attached below to the upper border of the cricoid arch and behind to the vocal process of the arytenoid cartilage. The collagen bundles forming the membrane pass forwards to the lower border of the thyroid cartilage close to the midline and extend up on the interior aspect of the thyroid to a point more or less level with the vocal process of the arytenoids cartilage. Its upper free border, crossing from the vocal process of the arytenoid to the internal surface of the thyroid cartilage, is thickened to form the **vocal ligament;** the **vocal fold** comprises the vocalis muscle lateral to the ligament, the ligament, and mucosal covering as shown in Figure 28.11. The gap between the right and left vocal folds forms the anterior part of the **rima glottidis,** the narrowest part of the lumen of the larynx**;** the posterior part is between the vocal processes of the arytenoid cartilages. The vocal folds are mobile; their position can be altered to open and close the larynx and their length and tension adjusted to change voice pitch.

In Figure 28.11 which is a coronal section through the length of the larynx, you can see that the two cricothyroid membranes run inwards from their lower attachment to the cricoid cartilage to the vocal ligaments, forming an incomplete cone which funnels air on to the vocal folds.

This is the reason for the alternative name of the cricothyroid membrane, the **conus elasticus;** it does describe a cone, but it is not elastic tissue so the second part of the name is inappropriate.

28.4.3 The cavity and mucosa of the larynx

Examine Figure 28.11 beginning at the lower tracheal end of the larynx. Follow up the cricothyroid membrane forming the conus elasticus as just described. Its upper free border forms the outline of the vocal folds as shown. Observe how the mucosa lining the larynx forms a pouch between the vocal folds and vestibular folds called the **laryngeal sinus.** The pouch is extended upwards for a variable distance between the vestibular fold and the lamina of the thyroid cartilage as the **laryngeal saccule**. In Figure 28.11, you can see that the vestibular folds with their mucous glands form the medial wall of the laryngeal saccule. The sinus and saccule have mucous glands within their lining mucosa and these probably serves the same function as mucous glands in the paranasal

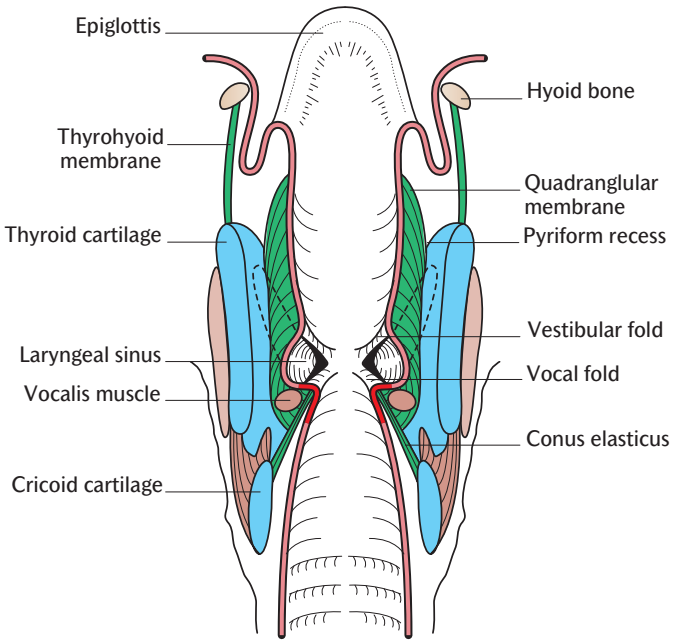


Fig. 28.11 The larynx in coronal section to show the vocal and vestibular folds and their relationship to the membranes of the larynx. Respiratory mucosa is coloured pink and stratified squamous epithelium is shown in red.

air sinuses (see Section 27.4) by providing a source of mucus which is out of the direct airstream. Due to this arrangement, the vocal folds can, in theory, be lubricated in extremes of climatic conditions when mucus from goblet cells in the laryngeal mucosa may be dried out. The **laryngeal vestibule** is the wide upper part of the larynx between the vestibular folds and the aryepiglottic folds forming the lateral walls of the laryngeal entrance. You can see in Figure 28.11, the **pyriform recesses,** two depressions in the laryngopharynx on either side of the laryngeal entrance between the aryepiglottic folds and the laminae of the thyroid. These are potential food traps and the consequences are outlined in Box 28.5.

Box 28.5 I've got a fish bone stuck in my throat!

When the epiglottis is pushed back over the laryngeal entrance during swallowing (see Section 28.1), food is deflected sideways from its convex oral surface through the pyriform recesses. The recesses are one of the places where inadvertently swallowed items like fish or chicken bones can lodge where they may cause discomfort and pain; the other is the valleculae. Coughing often does not displace the offending article so it must be removed by long forceps with visualization through a fibre optic laryngoscope, usually under sedation. The superior laryngeal nerve (see Section 28.4.5) crosses the floor of the pyriform recess just deep to the mucosa so care must be taken to avoid damaging the mucosa and underlying nerve as the object is removed. The consequences of superior laryngeal nerve damage are described in Box 28.7.

Box 28.6 Laryngeal oedema

Laryngeal oedema may arise as one of the signs and symptoms of an allergic response. Fluid leaks from capillaries into connective tissues and then tracks along fascial planes and accumulates where flow is blocked. Fluid in the laryngeal mucosa will gravitate down until stopped where the mucosa is tightly bound to the vocal folds. Fluid will then pool above the vocal folds, leading to distension of the vestibular folds and laryngeal vestibule to the point that the laryngeal lumen may be occluded. This is obvious potentially serious. It can usually be treated by injection of high doses of antihistamine drugs.

Considering the larynx is part of the respiratory system, you should anticipate that the interior of the larynx is lined with respiratory mucosa. As you can see in Figure 28.11, most of the larynx is lined by respiratory mucosa shown in pink. However, the red area in Figure 28.11 tells you the mucosa over the vocal folds is different; it is identical to lining oral mucosa so its epithelial layer is stratified squamous epithelium (see Section 25.2.1). The vocal folds make strong contact during phonation and if they were covered by respiratory mucosa, the protective epithelial layer would be worn away extremely rapidly. The mucosa over the vocal folds is tightly bound down to the underlying tissues which can have important clinical consequences as outlined in Box 28.6.

28.4.4 The laryngeal muscles

Some authors divided the muscles of the larynx into extrinsic and intrinsic groups. There is no specific extrinsic group; they are muscles arising outside the larynx and inserting into the laryngeal cartilages and include the longitudinal muscles of the pharynx and infrahyoid muscles. They elevate or depress the larynx bodily during swallowing and also stabilize the thyroid cartilage. The functions of these muscles will also be considered in the context of swallowing in Section 29.1.2.

The true laryngeal muscles have both attachments on the laryngeal cartilages and are named according to the cartilages to which they attach. The laryngeal muscles are paired, except for the transverse interarytenoids.

It is easier to understand the muscles of the larynx if you think in terms of the functions of the larynx. As shown in Figure 28.14A, the default position of the rima glottis between the vocal folds is *open* so that we can breathe in and out without impeding the airstream. We, therefore, need muscles to hold the vocal folds open. The vocal folds are closed during swallowing to prevent food entering the lower respiratory tract and during phonation so muscles are needed to close the larynx. The vocal folds are gently closed in conversational speech whereas they are held tightly together during swallowing or shouting; one set of muscles produce the basic closing movement and others act to close the vocal folds tightly. **Phonation** is the production of sound within the larynx. This requires the vocal folds to be adducted so that pressure builds up below them; they must also be tensed. When air is released and passes the tensed vocal fold, they will vibrate and produce sound. The vocal folds are lengthened, shortened, thickened and thinned, and put under varying amounts of tension to alter the pitch and intensity of voice. The changes to length, tension, and thickness during phonation are mainly very subtle small gradations using muscles designed for these movements. Smaller muscles open and narrow the laryngeal entrance.

The first four muscles to be described open and close the vocal folds.

Posterior cricoarytenoid

These muscles have sometimes been described as the most important muscles in the body because they are the muscles that abduct the vocal folds and thus the rima glottidis open, enabling us to breathe. As shown in Figure 28.12, the posterior cricoarytenoids arise from the concavities on the posterolateral surface of the cricoid lamina. Their fibres pass upwards and laterally and converge on to the muscular process of the arytenoid cartilage on the same side. The muscle dilates the rima glottidis. As indicated in Figure 28.14A, contraction of the lower more vertically directed fibres exerts a downward pull on the arytenoid which, because the base of this cartilage articulates with the sloping corner of the cricoid lamina, moves it laterally and slightly downwards; the rima glottidis opens into a triangular aperture. This is the usual configuration of the rima glottis during ventilation; the 'default position'. Contraction of the more horizontal upper fibres also rotates each arytenoid about its vertical axis; this movement results in the rima glottides opening into a wider diamond-shaped aperture used during forced ventilation.

Transverse interarytenoid

This is an unpaired muscle which adducts the vocal folds, thus closing the rima glottis. It is highly unusual because it is the only muscle in the whole body that actually crosses the midline. As you can see in Figure 28.12, it consists of transversely running fibres attached on each side to the muscular process and lateral border of the arytenoid cartilage which run in the hollow created by the concave posterior surface of the cartilages. As shown in Figure 28.14B, it draws the arytenoids together by pulling them upwards and medially along the sloping corners of the cricoid lamina, thus adducting the vocal folds.

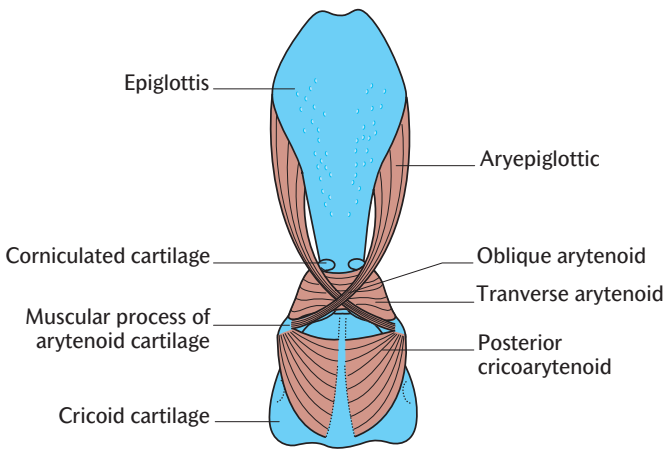


Fig. 28.12 Posterior view of the laryngeal muscles.

Lateral cricoarytenoid

As shown in Figure 28.13, these muscles arise from the superior border of the lateral aspect of the cricoid arch on each side. Each muscle passes upwards and backwards to the muscular process of the arytenoid. As shown diagrammatically in Figure 28.14C, the lateral cricoarytenoid rotates the arytenoid cartilage about its vertical axis by drawing the muscular process forwards so that the vocal process moves medially. These muscles act in concert with the thyroarytenoids to close the vocal folds tightly.

Thyroarytenoid

The thyroarytenoid is a thin sheet of muscle shown in Figure 28.13 which arises from the lower part of the inner surface of the angle of the thyroid cartilage and is inserted into the anterolateral surface of the arytenoid cartilage. The thyroarytenoid has a similar action to the lateral cricoarytenoid of twisting the arytenoid about a vertical axis and thus approximates the vocal processes and the vocal folds as illustrated in Figure 28.14C.

The next two muscles adjust the length, tension, and thickness of the vocal folds.

Vocalis

The lower fibres of thyroarytenoid running immediately laterally to the vocal ligament form a distinct band of muscle which most authorities accept has a distinct function from the upper thyroarytenoid part of the muscle. As shown in Figure 28.11, this part of thyroarytenoid is the **vocalis muscle**. As Figure 28.14D indicates, the vocalis tilts the arytenoids forwards towards the thyroid shortening, thickening, and relaxing the vocal folds which lowers the pitch of the voice.

Cricothyroid

In Figure 28.15, you can see that cricothyroid arises from a small area on the anterolateral aspect of the cricoid arch. Its fibres fan out to the anterior border of the inferior cornu and the adjacent lower border of the lamina of the thyroid cartilage. Cricothyroid produces the action shown in Figure 28.9;

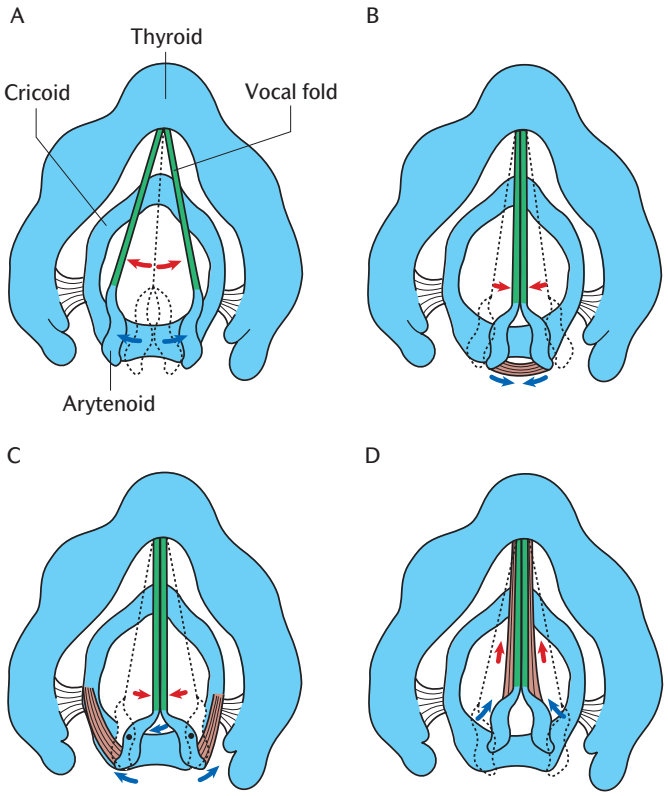


Fig. 28.14 Movements of the vocal folds produced by the laryngeal muscles. Dotted lines indicate the start position and solid lines the final position. A) 'Default' open (adducted) position during ventilation produced by the posterior cricoarytenoid muscles; B) Closure (adduction) by interarytenoid muscles; C) Rotation producing tight closure by the lateral cricoarytenoid and thyroarytenoid muscles; D) Relaxation and shortening by vocalis.

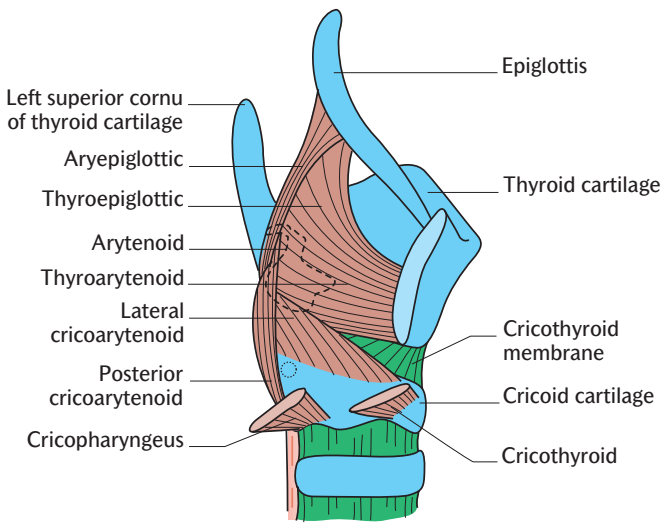


Fig. 28.13 Lateral view of the laryngeal muscles. The right thyroid lamina and cricothyroid muscle have been removed.

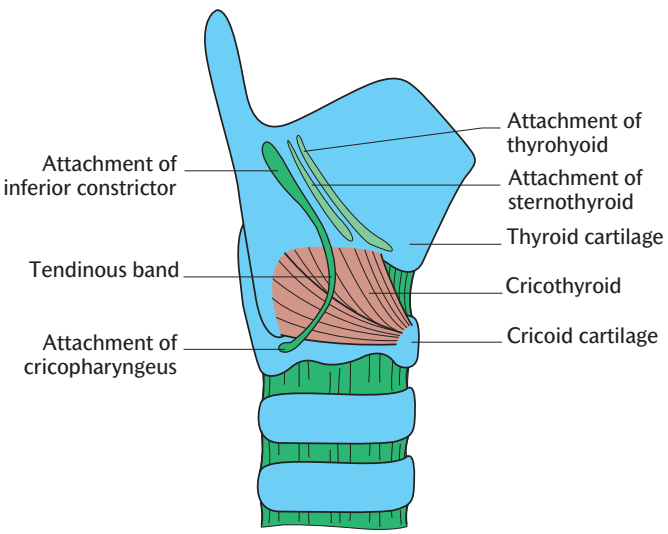


Fig. 28.15 Lateral view of the cricothyroid muscle.

Box 28.7 Laryngeal nerves in the cough reflex and laryngeal nerve injuries

The rima glottidis is the narrowest part of the upper respiratory tract so inhaled foreign bodies tend to be arrested there. In a conscious patient, the foreign body will stimulate the internal branches of the superior laryngeal nerves which are the afferent limbs of the **cough reflex**. Coughing may also be stimulated by inflammatory changes in the larynx or trachea. The **superior laryngeal nerves** carry the sensory information to the **respiratory centres** in the medulla. A short inspiration is taken followed by immediate closure of the rima glottidis. A forced expiration is initiated against the closed vocal folds, thus building up considerable pressure below the vocal folds. The rima glottidis is then suddenly opened and the powerful flow of air expels the foreign body.

A strong cough is usually sufficient to eject a foreign body into the pharynx whence it can be swallowed or spat out. In an unconscious subject or a patient under general anaesthetic, the cough reflex is suppressed and a foreign body entering the larynx may well obstruct the airway at the rima glottis. The obstruction may be dislodged by performing a **Heimlich manoeuvre**. This is a 'bear hug' given from behind which forcefully compresses the chest, thus mimicking a forced expiration which will hopefully dislodge the obstruction. If the obstruction cannot be dislodged, an emergency operation known as a **cricothyroid stab** may be performed; an incision is made into the larynx and a tube inserted for the introduction of air so that the blockage can be bypassed (see Box 28.9).

The **superior laryngeal nerve** may be damaged by removal of foreign objects from the pyriform recesses as outlined above in Box 28.5. The nerve is also susceptible to damage in people who experience gastric reflux; the stomach acid can erode the lining of the laryngeal vestibule and pyriform recesses which can damage the underlying nerve. The patient will find it quite difficult to regulate voice pitch because the internal branch of the superior laryngeal nerve supplies the cricothyroid muscle, one of the major regulators of voice pitch. They are also likely to lose the sensory supply to the larynx above the vocal folds which is the afferent limb of the cough reflex. Absence of the cough reflex results in **silent aspiration** which is described more fully in Box 29.2.

Damage to the **recurrent laryngeal nerves** will result in **laryngeal palsy**, paralysis of the majority of the laryngeal muscles ipsilateral to the nerve damage. Ninety percent of laryngeal palsies are caused by damage to the left recurrent laryngeal nerves through lung pathologies, particularly bronchial carcinoma, on their long course within the thorax adjacent to the left lung. The remaining 10% of palsies are due to inadvertent damage to the nerves during thyroid surgery.

The effect of recurrent laryngeal nerve damage is more complicated than simple flaccid paralysis of the laryngeal muscles and is a useful case study to consolidate knowledge of the actions of the laryngeal muscles. All the muscles on the affected side will be paralysed, except cricothyroid which is supplied by the superior laryngeal nerve and tenses the vocal folds. However, the vocal fold on the affected side cannot be relaxed because vocalis is paralysed. Recall that the transverse interarytenoid muscle crosses the midline. It is innervated from both recurrent laryngeal nerves and if one nerve is damaged, the muscle will still function but is weaker than normal. The vocal fold on the injured side will therefore be approximated because the posterior cricoarytenoid that abducts the vocal folds is non-functional and usually assumes a position midway between closure and the default open position. The injured vocal fold is tensed and in the airstream and will therefore vibrate whether it needs to or not; the patient's voice will be lacking power because the vocal folds cannot be closed fully and has a 'raspy' quality because of the unwanted vibration.

the arch of the cricoid is tilted upwards towards the fixed thyroid cartilage, carrying the arytenoids back away from the thyroid cartilage when the muscles contract; this increases the distance between the thyroid and arytenoid cartilage and so lengthens, thins, and tenses the vocal folds. The effect of these movements is to raise voice pitch. Cricothyroid opposes the action of the vocalis. You can verify these movements on yourself quite easily. Feel your thyroid cartilage with your index finger and your cricoid below it with your ring finger and then sing an ascending scale; you should feel the cricoid getting nearer to the thyroid as you go up the scale.

The role of the laryngeal muscles and associated structures in speech and swallowing are described in Chapter 29.

The last two laryngeal muscles adjust the size of the laryngeal entrance.

Oblique interarytenoid and aryepiglottic muscles

The **oblique arytenoid** muscles are situated superficially to the transverse arytenoid. As you can see from Figure 28.12, each consists of a band of fibres running diagonally from the muscular process of one arytenoid cartilage to the apex of the opposite cartilage, the two muscles forming an X. Fibres of each muscle continue upwards in the corresponding aryepiglottic fold to the side of the epiglottis. These fibres constitute the **aryepiglottic muscle**. The oblique arytenoids and aryepiglottic muscles are very flimsy; they are not powerful enough to overcome the elasticity of the epiglottis and therefore cannot close the laryngeal entrance. They narrow the laryngeal inlet by approximating the arytenoid cartilages and straightening each aryepiglottic fold.

Thyroepiglottic muscle

This small group of muscle fibres shown in Figure 28.13 arises from the upper part of the inner aspect of the angle of the thyroid cartilage and passes upwards to insert into the aryepiglottic fold and lateral edge of the epiglottis. It is sometimes described as an upward continuation of the thryoarytenoid. They widen the laryngeal inlet by pulling the aryepiglottic folds apart.

28.4.5 Nerve and blood supply

The superior laryngeal and recurrent laryngeal branches of the vagus innervate the larynx. The origin of these vagal branches was described in Section 28.2.3 and illustrated in Figure 28.4.

The **superior laryngeal nerve** divides into internal and external branches as it passes medial to the internal carotid artery (see Figure 28.4). The **internal laryngeal nerve** descends to the thyrohyoid membrane which it pierces above the superior laryngeal vessels and then supplies sensory processes to the mucosa of the epiglottis, aryepiglottic folds, and interior of larynx down to the level of the vocal folds. The **external laryngeal nerve** is a motor branch to the cricothyroid muscle.

The course of the **recurrent laryngeal nerves** has been described above and in Section 12.6.2. Each **recurrent laryngeal nerve** passes into the larynx from the groove between the trachea and oesophagus deep to the inferior constrictor to supply all the laryngeal muscles, except cricothyroid and the mucosa below the vocal folds.

The vocal folds are a watershed in the arterial supply and venous and lymphatic drainage of the larynx as well as being the boundary between the two areas of nerve supply. The superior laryngeal branches of the superior thyroid vessels supply the larynx above the folds and the inferior laryngeal branches of the inferior thyroid vessels below the folds. Lymph drains to the deep cervical nodes.

Box 28.7 explains the role of the laryngeal nerves in the cough reflex and the effects of nerve injuries on laryngeal function.

28.5 Lymphatic drainage of the pharynx, larynx, and deeper structures of the face

Lymph from the deeper structures of the head and neck, like that from more superficial parts, ultimately drains into the deep cervical group of nodes. Some of this drainage is direct, but in other cases, the lymph passes first through one of the groups of lymph nodes surrounding the pharynx and trachea. These include the following:

- The **retropharyngeal nodes** between the prevertebral fascia and the posterior wall of the pharynx which receive lymph from the posterior part of the nasal cavity, paranasal air sinuses, nasopharynx, soft palate, and auditory tube. They drain into the upper deep cervical nodes;
- The **paratracheal nodes** alongside the trachea and oesophagus which receive lymph from the oral and laryngeal parts of the pharynx and from the cervical part of the oesophagus. They drain into the deep cervical group;
- The **pretracheal nodes** deep to the investing layer of the deep fascia in front of the trachea. Much of the lymph from the larynx, trachea, and thyroid gland passes to the pretracheal nodes which drain into the deep cervical group. The remainder of the lymph from these structures passes directly into the deep cervical nodes.

These three groups of lymph nodes cannot be palpated so there is often no lymphadenopathy apparent to indicate the presence of malignancy from the pharynx and larynx. Cancers in these areas can be well advanced before other signs and symptoms appear.

Box 28.8 Clinical anatomy of the thyroid gland

Enlargement of the thyroid gland, or **goitre**, is a relatively common condition. It produces an unsightly lump in the neck which may exert pressure on adjacent structures such as the trachea. **Hypertrophic goitre** is most commonly a response to either reduced or excessive secretion of thyroid hormone with the corresponding physiological effects.

Iodine is a dietary trace element essential for the manufacture of thyroid hormones. Simple, non-toxic goitre is a benign compensatory enlargement of the gland in response to low levels of iodine in the diet, usually as a result of iodine being leached out of ground water by calcareous rocks. It is extremely rare in developed countries because iodine is added to table salt as a preventative measure.

Carcinoma of the thyroid gland is rare. Radioactive iodine is administered which is then concentrated by the thyroid and irradiates the malignant tissue *in situ*.

Some forms of benign goitre are treated by **thyroidectomy**. In cases of benign goitre, tissue is removed from the anterior part of the thyroid lobes so that **parathyroid tissue** is not inadvertently removed. The surgeon must be careful to avoid damage to the **recurrent laryngeal nerves** when the **inferior thyroid arteries** are isolated and ligated. The consequences of recurrent laryngeal nerve damage have been described in Box 28.7.

28.6 The thyroid gland

As illustrated in Figure 28.16, the thyroid gland consists of two lobes united across the front of the trachea at the level of the second to fourth cartilaginous rings by a narrower portion called the **isthmus**. A small **pyramidal lobe** may project upwards from the isthmus, usually to the left of the midline. The gland is within the **pretracheal fascia** and lies deep to the sternothyroid and sternohyoid muscles. The four **parathyroid glands** are situated close to the thyroid gland within the pretracheal fascia or are embedded within the gland, one at the upper pole and one at the lower pole of each lobe.

The gland receives its blood supply from the **superior** and **inferior thyroid arteries**. Each superior thyroid artery is a single vessel, but

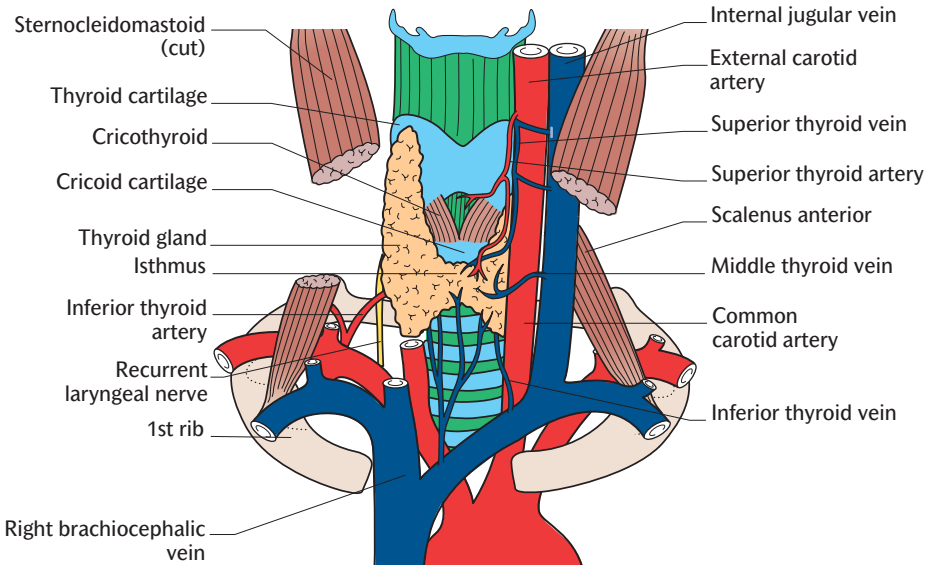


Fig. 28.16 Anterior view of the thyroid gland.

the inferior thyroid artery divides into several branches. The recurrent laryngeal nerve usually passes behind, but may pass between these branches. There are three pairs of veins—the superior and middle thyroid veins drain into the internal jugular vein and inferior thyroid veins enter the brachiocephalic veins.

The clinical anatomy of the thyroid gland is covered in Box 28.8 and Box 28.9 deals with the creation of emergency airway into the respiratory tract.

Box 28.9 Emergency access to the airway

If the larynx becomes blocked an emergency, airway into the lower respiratory tract may be created by performing a **cricothyroid stab** to bypass the blockage. The most usual indication for this procedure is oedema in the laryngeal mucosa due to an allergic response. If this should occur, the first responses are to call for expert medical help and inject the patient with adrenalin or antihistamine drugs, one of which should be available in an emergency kit in the surgery. Injection will reduce the oedema so that breathing will no longer be impaired. Emergency medical help will then be able to deal with the situation in the most appropriate way. You will gather that cricothyroid stab is no longer something that dentists would be required to attempt in the surgery in the UK.

The technique used is described here as it is a useful reminder of many aspects of the anatomy of the neck and UK guidelines do not apply universally. The objective of a cricothyroid stab is to create an opening into the airway without damaging other structures or producing excess blood loss. The patient is laid flat on their back and the neck is extended as far as possible while the head and neck straight so that the patient is looking directly forwards; the head and neck are maintained in position with cushions or other packing materials. Extension of the neck has the effect of moving the carotid sheath and its contents laterally out of danger (see Figure 23.1); the infrahyoid muscles also move laterally so that the laryngeal cartilages are covered only by skin and fascia, producing an almost bloodless pathway. The cricothyroid membrane is palpated between the thyroid and cricoids cartilages (see Section 20.3.2 and Figure 20.8) and stabbed with a sterile scalpel, keeping the scalpel vertical to avoid damage to the vocal folds just above the level of the incision. A good guide to the depth of the incision is that the diameter of the larynx and trachea is about the same as that of the *patient's* little finger; this measurement and the tracheal diameter varies according to the age of the patient. A suitably sized tracheotomy tube is then placed into the incision; the tube keeps the opening patent and oxygen may be administered through it if the patient is hypoxic. The reason for entering the larynx through the cricothyroid membrane is that it is a relatively bloodless tract and well above the very vascular thyroid gland and its blood supply. In extreme emergency, cricothyroid stab may be carried out with any sharp instrument such as a pen knife and the hole kept patent by any suitable tube like the plastic case of a biro pen.

Tracheostomy is an operation to create a more permanent airway for a variety of medical circumstances. It is carried out in the operating theatre so visualization of deep structures can be achieved. An opening is created low down in the trachea above the sternal notch where it is more cosmetically acceptable to patients; it can also be covered by a scarf or high-necked garment. The low positioning of the opening avoids the thyroid isthmus and lower poles of the thyroid lobes.

29 Swallowing and speech

Chapter contents

29.1 Swallowing

Swallowing or deglutition is a series of closely integrated actions that propel the contents of the oral cavity through the pharynx and the oesophagus to the stomach and ensuring that they do not enter the lower respiratory tract. Some of these actions are voluntary whereas others are reflex. We tend to think of swallowing merely in terms of eating and drinking. In reality, a relatively small proportion of the total number of swallows performed in a day occurs during meals; the majority of them take place to clear excess saliva from the mouth. We are generally unconscious of salivary clearance swallows, but concentrate on your swallowing as you read this chapter—you will be surprised by how often you do swallow. A single swallow usually takes 1.5 to 2 seconds from mouth to stomach. This rapid action means that it is difficult to be absolutely categorical about the precise order of events.

For convenience of description, swallowing is usually divided into three phases according to the position of the food but, in reality, the three phases are continuous with each other. The phases are:

- The **oral phase**, usually subdivided into an **oral preparatory phase** and an **oral phase**;
- The **pharyngeal phase**;
- The **oesophageal phase**.

The oral phase is voluntary whereas the second and third phases are reflex.

29.1.1 The oral phase

In the **oral preparatory phase**, food is chewed to the right consistency, mixed with saliva, and collected into a single mass, the **bolus**, on the dorsum of the tongue. This subphase requires the muscles of mastication, suprahyoids and infrahyoids, used during mastication as described in Chapter 26, together with the tongue muscles and the muscle of the lips and cheeks to push food between the teeth. The consistency of chewed food is measured by sensory receptors in the oral mucosa.

The **oral phase** is initiated when food is judged to be of the right consistency. The bolus is pushed rapidly backwards towards the oropharynx by raising the tongue against the hard palate from front to back. This action is brought about by elevating the hyoid bone by the contraction of the **suprahyoid muscles** and the musculature of the tongue itself. Towards the end of the oral phase, the tongue in front of the food is elevated by contraction of the **palatoglossus** and **intrinsic muscles** while the back of the tongue is simultaneously lowered by the **hyoglossus** muscles to form a chute. The oral phase is completed by using the **styloglossus** muscles to thrust the tongue back and tip the food down the chute into the oropharynx. The elevated part of the tongue stays in this position during the first part of the second stage and so seals the oropharyngeal isthmus to prevent the re-entry of food into the mouth.

The lips are usually closed and the teeth brought into occlusion during the oral phase to prevent food leaving the mouth. It is extremely difficult to swallow with your mouth open to any degree and the consequences are outlined in Box 29.1. In fact, if the trigger spots that elicit the reflex pharyngeal and oesophageal phases of swallowing (see Section 29.1.2) are touched with the mouth open, a gag reflex is stimulated which has the opposite effect to swallowing.

Box 29.1 Dental treatment is hard to swallow

Try to swallow with your mouth open by different amounts and you will realize how little the mouth has to be opened before it gets difficult. Now think how wide your patient's mouth is going to be open during oral examination and subsequent procedures. They are going to have real difficulty with swallowing and will soon feel uncomfortable as saliva and debris builds up even with efficient aspiration of saliva. For this reason, if you are going to be a good dental practitioner, you will stop at frequent intervals to allow your patient to swallow.

29.1.2 The pharyngeal phase

We can decide when food is the correct consistency to swallow or decide not to swallow for other reasons such as unpleasant taste. However, once food moves from the mouth into the oropharynx, a series of reflexes are triggered that execute the second and third phases of swallowing. These reflexes are designed to propel the food through the pharynx and oesophagus while preventing the food from going where it should not—the mouth, the nasopharynx and nose and, most importantly, the larynx and lower respiratory tract.

The reflex train is elicited when the bolus contacts certain sensitive **trigger areas**. These vary from person to person, but the most common areas are the mucosa covering the palatoglossal arches or the posterior wall of the oropharynx.

Contraction of the **tensor** and **levator palatini** muscles tense and elevate the soft palate which contacts the posterior pharyngeal wall at **Passavant's ridge** to seal off the nasopharynx. Respiration is *suspended* briefly to minimize the risk of inhaling food into the larynx. Simultaneously, the **stylopharyngeus** and **palatopharyngeus** muscles pull the larynx upwards behind the hyoid and under the epiglottis. The aryepiglottic folds are tensed by the **aryepiglottic** and **oblique arytenoid** muscles to narrow the laryngeal entrance. The vocal folds are strongly adducted by the **transverse interarytenoid, lateral cricoarytenoid**, and **thyroarytenoid muscles**.

The **pharyngeal constrictor muscles** now contract in sequence from above downwards, propelling the bolus of food downwards over the closed inlet of the larynx. Once the food has entered the oesophagus, it is moved onwards by peristalsis. This **oesophageal phase** is the third phase of swallowing.

As mentioned in Chapter 28, the convex oral side of the epiglottis diverts food away into the pyriform recesses on either side of the laryngeal inlet. The epiglottis keeps the inlet covered as it reopens.

The sensory neurons forming the afferent limb of the reflexes involved in the pharyngeal and oesophageal phases are carried in the **glossopharyngeal nerve**. The efferent limb comprises the motor nerves supplying the many muscles involved in the reflexes. These include

Box 29.2 Dysphagia

Considering the complex sequence of events involved, it is surprising that swallowing occurs so often without any problems. On a day-to-day basis, swallowing usually goes wrong in a way that our parents warned us about. How often have we been told 'Don't talk with your mouth full'? We need to breathe and move the soft palate downwards if we are trying to talk at the same time. The seal produced by the soft palate may be incomplete so that food or drink enters the nasopharynx and nose. Alternatively, food may be aspirated into the upper part of the larynx as we breathe. In this case, the cough reflex will be stimulated and the offending food or drink soon expelled.

More permanent difficulties in swallowing, **dysphagia**, may result from a mechanical obstruction of the pharynx or oesophagus or from disorders of the nervous system affecting the cranial nerves and central nuclei involved in the swallowing reflex. Obstruction may be caused by tumours of the pharynx or oesophagus or of structures, such as the thyroid gland or adjacent lymph nodes constricting the food passage in the neck or heart enlargement compressing the oesophagus in the thorax.

Diseases of the nervous system which affect neuromuscular coordination such as multiple sclerosis, Parkinson's disease, motor neuron disease, or stroke may be accompanied by dysphagia. In these cases, swallowing is often slow and the phases of swallowing become uncoupled instead of occurring as a smooth continuum. The patient may have difficulty transiting food from mouth to pharynx or food may pool in the pharynx instead of entering the oesophagus. If the vagal sensory innervation of the larynx is compromised in any of these diseases, the patient may be unable to detect the presence of food in the larynx and no cough reflex is elicited. This is **silent aspiration** and is potentially dangerous. The person may choke and asphyxiate or food may enter the bronchial tree, stagnate and decay, and trigger pneumonia. Recent studies of patients with silent aspiration have shown that good oral hygiene lessens the risk of subsequent lung infections considerably.

the masticatory muscles, suprahyoids, infrahyoids, facial expression, tongue, soft palate, pharyngeal constrictors, pharyngeal elevators, larynx and respiration—that is virtually all the muscles encountered so far in the chapters covering the head and neck.

The central control for the coordination to ensure all this muscular activity occurs in the correct spatial and temporal sequence is in networks of neurons in the brainstem. They integrate the activity of the cranial nerves involved in the control of the muscle groups listed so far. The brainstem centres are believed to control the regular unconscious swallows to clear the oral cavity of saliva. The control of swallowing during eating and drinking is mediated by a swallowing centre in the insular cortex which feeds into the brainstem centre.

Dysphagia is any disturbance to swallowing; some of the causes and consequences are described in Box 29.2.

29.2 Speech

Language is a cognitive process of formulating and ordering thoughts into words according to rules that are learned and mastered during childhood. We can think things in our head and they need go no further. We can then communicate our ideas to others in many ways. We can write them down or we can speak out. **Speech** is, therefore, the mechanical process of producing audible sounds to represent language. Language is processed within the language areas of the brain and defects in these areas will produce difficulties with comprehension or formulation of language known as dysphasia (see Section 15.4.2).

Speech requires the production of a controlled airstream which can then be manipulated in the larynx to produce sound (**phonation**) and within the upper vocal tract, the mouth and its contents, and nose, (**articulation**) to produce specific segments of speech called **phonemes**. Respiratory, laryngeal, and articulatory movements must be carefully coordinated to produce meaningful speech. If these mechanisms are disrupted by physical disruption or obstruction or by lack of control or coordination, speech becomes unclear, a condition known as **dysarthria**. Speech is usually accompanied by non-verbal communication to clarify meaning, including changes in facial expression, movements of the head, and hand gestures.

To make the distinction between speech and language clear, a dysphasic patient may not comprehend your request to 'open wide' or may produce speech that sounds perfectly normal, but is nonsense because words are missing or in the wrong order. A dysarthric patient would understand your request to 'open wide', but when you asked them a question while checking their medical history, their speech may sound very peculiar despite the fact that the words are all there and in the correct order; they cannot be articulated clearly.

Speech takes place on an expiratory airstream. As described in Section 11.4.1, quiet ventilation tends to be regular; each breathing cycle is divided about equally between inspiration and expiration and there are about 12–15 cycles per minute on average. During speech, this rhythm is changed drastically. Inspiration is timed to take place during a natural pause in talking, such as the end of a sentence or clause, and takes place very rapidly and is usually deeper. Expiration, on the other hand, is greatly prolonged to match the length of the utterance. To achieve this, passive elastic recoil, the normal mechanism of expiration, is counteracted by relaxing the intercostal muscles slowly rather than all at once to control breathing out.

29.2.1 Phonation

The production of sound begins in the larynx, a process called **phonation**. Phonation consists of converting an even flow of expired air passing through the larynx into an oscillating flow of different frequencies

within the range of human hearing—about 20–20 000 Hz in adults) although most speech falls within the 400–4000 Hz range. Phonation is achieved by vibrating the vocal folds. The phonemes in English and most other languages fall into two categories: **voiced** and **unvoiced** phonemes. Voiced phonemes require vibration whereas unvoiced ones do not. Sustain an 'S' sound while you palpate your thyroid cartilage, then convert the 'S' into a 'Z' sound; you should detect vibration as you make the voiced Z sound, but not on the unvoiced S; every other configuration in the vocal tract is exactly the same for both sounds.

Phonation is achieved by adducting the vocal folds and so obstructing the flow of air from the lungs. **Subglottal pressure** then builds up below the folds and eventually reaches a point where it forces the folds apart, allowing air to escape through the rima glottidis. As soon as the pressure is released, the vocal folds return to the adducted position and the cycle is repeated. In consequence, the flow of air is converted to a series of puffs. Phonation will only occur if the vocal folds are *tensed*. The frequency of vibration of the vocal folds is determined by the length, tension, and thickness of the vocal folds. These factors are not independent variables; you cannot alter one without altering the others. The best analogy for the vocal folds during phonation is a rubber band. Pluck the rubber band held between two fingers and it will simply flap about; stretch it between fingers on both hands and pluck and you will get noise. As you stretch the rubber band, it gets thinner and the tension increases so increasing the pitch of the note. Shorten the rubber band and the tension decreases and the thickness increases decreasing pitch.

The frequency with which the air oscillates determines voice **pitch**. This varies during speech to add intonation and expression to the voice. The basic voice pitch varies between individuals and depends on the resting length of the vocal folds. The longer they are, the deeper the voice; the pitch of children's voices is usually higher than that of women which is higher than that in men although the ranges overlap. This sex difference first becomes apparent during adolescence when the male voice deepens or breaks due to the greater growth of the larynx and consequent greater elongation of the vocal folds.

The loudness of the voice is controlled primarily by the pressure of the expired air which is determined by the force of expiration and the subglottal pressure which is determined by how tightly the vocal folds are adducted.

The quality or timbre of the voice is influenced by the interplay of many factors, including the extent of vertical contact between the folds, the relative amounts of time the rima glottidis is opened and closed, and variations in the articular phase.

29.2.2 Articulation

The passage of air through the upper **vocal tract** is modified by the acoustic characteristics of the tract, particularly the mouth. The highly mobile tongue and lips are capable of continuously changing the shape of the oral cavity, thus modulating the sound passing through; this is **articulation**. A sequence of speech consists of periods of relatively high sound intensity alternating with periods of low intensity. During high intensity, the vocal tract is unrestricted but its shape is modified by the action of the tongue and lips to produce the **vowel** phonemes. The shape is modified by the position of the tongue in the mouth; it may be high or low or near the front or back. In the low intensity intervals, the tract is constricted or completely occluded by the action of the tongue or lips to produce **consonants**. By making the sounds yourself, you can work out how the various vowels and consonants are produced.

To show you how changes to one articulator can alter the sound quite dramatically, make an 'ooooo' sound. Notice that your lips are pursed as if you were going to whistle or kiss. Continue making the sound, but spread your lips as you do so. You will find that the 'ooooo' has changed into an 'eeeee'. Phoneticians distinguish between high and low, front and back, and rounding and spreading vowels.

The consonant sounds are classified according to the point of maximum constriction of the vocal tract:

- Maximum constriction between the lips produces **labials**, e.g. /b/, /p/, /m/;
- Constriction between the lower lip and the upper incisors gives **labiodentals**, e.g. /f/, /v/;
- Constriction between the tip of the tongue and the anterior part of the hard palate produces **alveolars**, e.g. /d/, /t/, /r/;
- Constriction between the dorsum of the tongue and the posterior part of the hard palate gives **palatals**, e.g. /j/ and /l/);
- Constriction between the dorsum of the tongue and the soft palate, e.g. /g/ and /k/.
- The sibilants /s/, /sh/, and /z/ are produced by the passage of expired air through a narrow space between the tip of the tongue and the anterior part of the hard palate.

In all the vowels and the consonants above, the expired airstream is directed through the mouth by elevating the soft palate to close off the nasopharynx. In the consonants /m/, /n/, and /nj/, the soft palate is lowered, allowing a proportion of the air through the nose to give the sound a nasal quality.

Modification of the shape of the mouth by the loss of teeth, fitting of prostheses, or reconstructive maxillofacial surgery may profoundly affect articulation as described in Box 29.3.

Box 29.3 Dentistry and articulation

Despite repositioning of the teeth by oral surgery or orthodontic treatment, our wonderful brains are so adaptable that most patients will rapidly learn to reposition their articulators to accommodate to the change in the shape of their mouths; speech returns to normal in a relatively short time. Patients with pronounced malocclusions often develop tricks of articulation to compensate. For example, a person with a marked class II occlusion (Section 26.2) may have trouble making contact between their upper and lower lips for bilabial consonants; instead, they use the labiodental position for these phonemes.

Developmental abnormalities such as first arch syndrome (see Box 21.1) or cleft lip or palate (see Box 32.1) may interfere with voice production to the point where speech is unintelligible. Such conditions usually require elaborate maxillofacial corrective surgery that is carried out over long periods to accommodate to changes in growth and development. The patient may have to make quite large adjustments to their articulatory movements after each time their jaws and teeth are realigned. These patients are usually assisted to make their speech clearer by speech and language therapists.

30 The orbit

Chapter contents

30.1 Introduction

Some knowledge of the anatomy of the orbit is required by dental students and practitioners because it forms the upper part of the facial skeleton and some of the nerves and vessels supplying dental structures pass through it. Trauma to the middle third of the face, the upper facial skeleton, frequently involves the orbits and the structures they contain. Infections of the oral region occasionally spread to the orbit.

In the following description, the emphasis is on those aspects of orbital anatomy of dental relevance; no description of the structure of the eyeball or the mechanisms of vision is included.

30.2 The bones of the orbit

The orbital cavities contain the eyeballs (globes), their associated muscles, vessels, nerves, the lacrimal apparatus, and a large amount of fat to cushion and protect the globes. Each cavity is pyramidal in shape. The base is the orbital opening on to the face; the roof, floor, and medial and lateral walls converge to the apex at the posterior aspect of the orbit. The long axis of the orbit from apex to surface runs forwards and laterally. The bones that form the orbit are illustrated in Figure 30.1; use the figure and a dried or model skull if possible as you read the following description.

Most of the roof of the orbit is formed by the inferior surface of the **orbital part of the frontal bone** with a small posterior contribution from the **lesser wing of the sphenoid**; this is pierced by the optic canal through which the optic nerve exits the orbit. The lateral wall is formed by the **orbital surfaces of the zygomatic bone** anteriorly and the **greater wing of the sphenoid** posteriorly. It separates the orbital cavity from the infratemporal fossa anteriorly and from the middle cranial fossa posteriorly. The floor of the orbit is occupied by the thin plate of bone forming the upper surface of the **body of the maxilla**; this plate of bone is also the roof of the maxillary paranasal air sinus over most of its extent although the palatine bone forms a minute triangular area at the posteromedial corner. The bones forming the medial wall are, from front to back, the **frontal process of the maxilla**, the **lacrimal**, the **orbital plate of the ethmoidal labyrinth**, and the **body of the sphenoid**. The medial orbital wall is very thin; the ethmoid air cells can usually be seen through it in dried skulls or the air cells may be open if the orbital plate is damaged. In life, this thin bone is surprisingly strong because it is buttressed by the plates of bone separating the individual air cells.

The **superior orbital fissure** between the greater and lesser wings of the sphenoid separates the lateral wall and roof posteriorly. It transmits the third, fourth, and sixth cranial nerves to the extraocular muscles moving the eyeballs, the ophthalmic division of the trigeminal nerve, and ophthalmic veins. The lateral wall and floor are separated by the **inferior orbital fissure** between the maxilla and greater wing of sphenoid. The inferior fissure communicates anteriorly with the infratemporal fossa and the pterygopalatine fossa posteriorly. The **infraorbital groove** runs forwards across the floor of the orbit from the medial part of the inferior orbital fissure and becomes roofed over anteriorly to form a canal which eventually opens at the **infraorbital foramen**. They contain the infraorbital nerve, the continuation of the maxillary nerve.

The **lacrimal groove** is in the anterior medial wall of the orbit. It is formed by the maxilla anteriorly and the lacrimal bone behind; it houses the lacrimal sac (see Section 30.3.4). The **nasolacrimal canal** leads down from the groove through the maxilla to open into the inferior meatus of the nasal cavity. It transmits the nasolacrimal duct which drains tears from the lacrimal sac to the nose.

Fractures involving bones that contribute to the orbit are considered in Box 30.4.

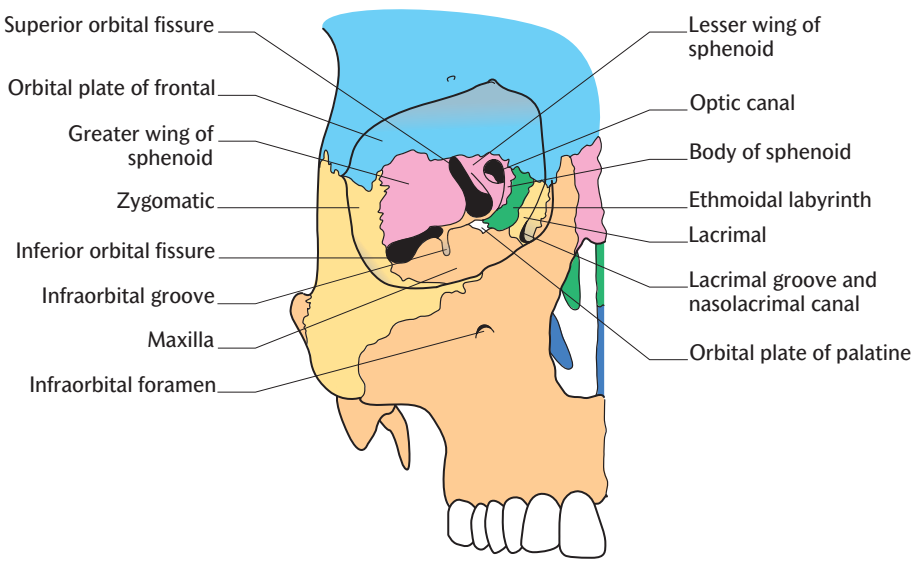


Fig. 30.1 Frontal view of the bones of the orbit.

30.3 Eyelids and lacrimal apparatus

30.3.1 Surface features

The surface features of the orbits are illustrated in Figure 30.2. Each orbit is closed by the upper and lower **eyelids** bearing **eyelashes** on their anterior edge. The **palpebral fissures** are the gaps between the lids when the eyes are open. The **lacrimal papilla** is a small prominence located near the medial end of each eyelid with a small opening on its summit called the **punctum,** this is the opening of the **lacrimal canaliculus** through which tears drain from the eye. The **lacrimal caruncle** is a pinkish fold of skin at the medial angle or **medial canthus** of the eye. In mongoloid ethnic groups, the medial canthus is covered by a small fold of skin called the **epicanthal fold**.

30.3.2 Eyelids

The eyelids are movable flaps which protect the eye. The upper is the larger and more mobile with its own elevator muscle, the **levator palpebrae superioris**, shown in Figure 30.3A.

The **orbital septum** is a connective tissue sheet attached to the margins of the bony orbit; it is deficient centrally at the palpebral fissure. It is thickened above and below the palpebral fissure into crescentic layers of strong fibrous tissue, forming the superior and inferior **tarsal plates** which are shaped to fit the curvature of the eyeball. Levator palpebrae superioris muscle attaches to the larger upper tarsal plate. The ends of the two plates are attached medially to the lacrimal bone by the **medial palpebral ligament** and laterally to the zygomatic bone by the **lateral palpebral ligament**. In Figure 30.3B, you can see that each tarsal plate is covered by thin skin anteriorly and by **conjunctiva** on its inner surface. The orbicularis oris muscle lies between the skin and tarsal plates. The **tarsal glands** are modified sebaceous glands embedded in the deep surface of the tarsal plates, opening by small ducts on to the free margin of the eyelids. They produce an oily secretion which spreads over the tear film produced by the lacrimal glands and delays its evaporation.

30.3.3 The conjunctiva

The **conjunctiva** is a transparent membrane of stratified squamous epithelium supported by a very thin layer of fibrous tissue. It covers the anterior aspect of the sclera (white) of the eyeball and becomes continuous with the corneal epithelium forming the transparent part of the eyeball

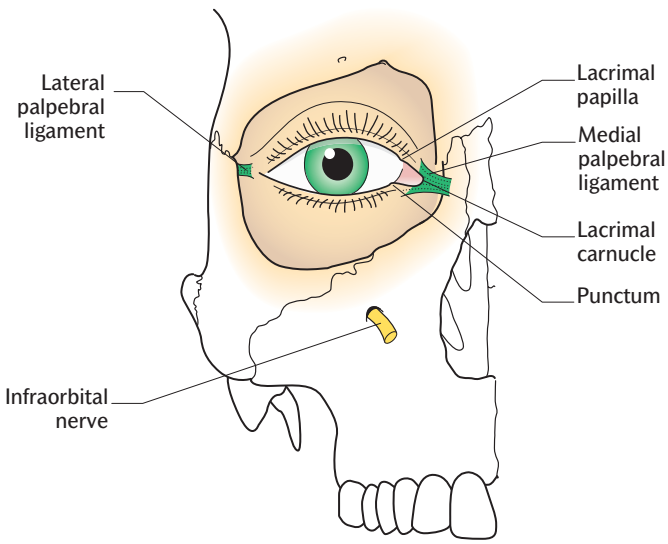
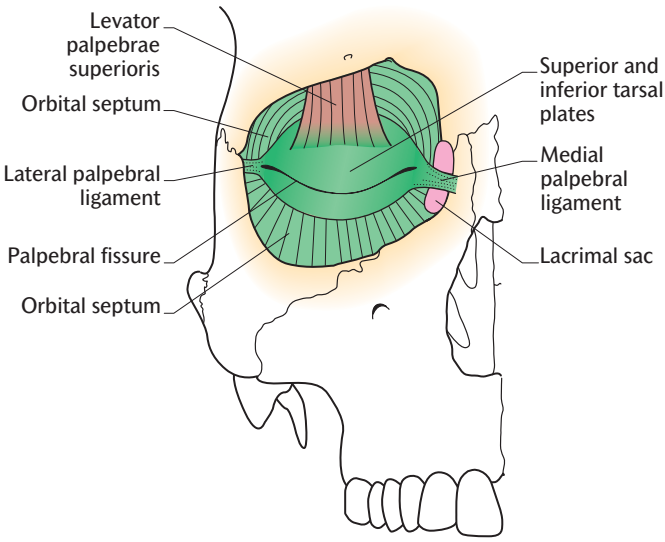


Fig. 30.2 Surface markings of the structures around the orbit.



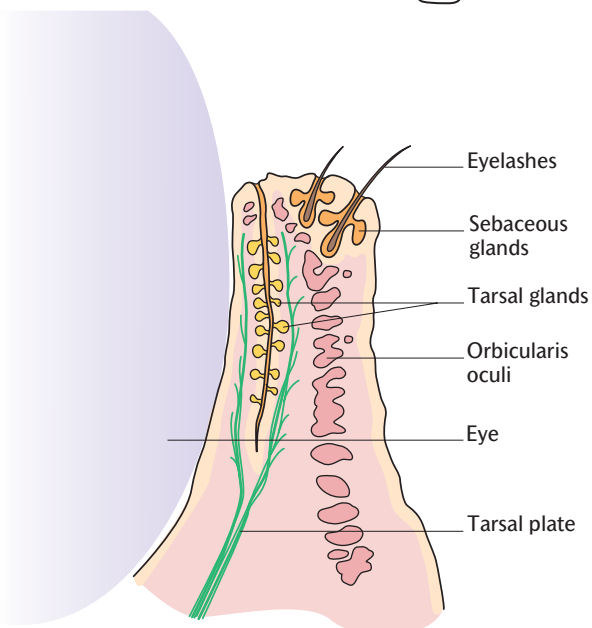


Fig. 30.3 The eyelids. A) The fibrous structures of the eyelid; B) A cross section through the lower eyelid.

Box 30.1 Clinical anatomy of the eyelids

In the upper eyelid, the connective tissue layer between the orbicularis oris muscle and tarsal plate is continuous with the subaponeurotic layer of the scalp (see Section 23.2.5); effusions of blood can easily pass down from the scalp into the upper lid, causing a 'black eye'.

Dissection through the layers of the lower eyelid (see Figure 30.3B) can be used to access the maxillae for fracture repair. Dissection is carried out between the lower tarsal plate and orbicularis oculi.

at the sclerocorneal junction. Conjunctiva also lines the inner surfaces of the eyelids and is continuous with skin at the free margins of the lids. The **conjunctival fornix**, the point where the conjunctiva reflects from the sclera on to the inner surface of the eyelid, is quite loose to allow movement of the eyelids.

The conjunctiva of the sclera and upper eyelid is innervated by branches of the **ophthalmic trigeminal nerve** and that of the lower lid by the infraorbital branch of its maxillary division.

The eyelid can be used as an access route to fractures of the maxilla as described in Box 30.1.

30.3.4 The lacrimal apparatus and circulation of tears

The structures involved in tear production and the route followed by tears from these to the nose are illustrated in Figure 30.4. The **lacrimal glands** are similar to serous salivary glandular tissue. Each gland consists of a large orbital and a smaller palpebral part. The orbital part is about the size and shape of an almond and lies in a shallow fossa in the lateral part of the roof of the orbit just within the orbital margin. It is continuous with the palpebral part of the gland which is in the lateral part of the upper eyelid deep to the orbital septum. The glands drain by about a dozen small ducts from the palpebral part which open into the upper conjunctival fornix. From here, the tears pass medially across the front of the eyeball to the medial canthus. Closing the eyelids and blinking draws tears across the eyes to prevent dryness of the cornea; blinking is reflex closure of the eyelids in response to drying of the eye, foreign bodies on the conjunctiva or cornea, or sighting foreign objects approaching the eyes. Tears pass through the lacrimal canaliculi into the **lacrimal sac** contained within the lacrimal groove in the medial wall of the orbit. Closing the eyelids helps to drain tears first by pressing the openings of the lacrimal canaliculi into the lake of tears and secondly, because the contraction of orbicularis oculi tends to dilate the lacrimal sac which sucks in the tears. The tears drain from the sac through the **nasolacrimal duct** to the nasal cavity. If the volume of tears is large, they spill out on to the face as well as travelling through the nasolacrimal duct; this is crying.

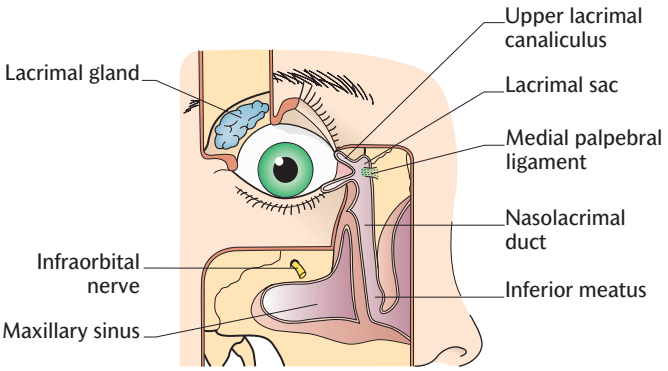


Fig. 30.4 The lacrimal apparatus.

30.4 Contents of the orbit

The principal contents of each orbit are:

- The eyeball and optic nerve;
- The extraocular muscles which move the eyeball and upper eyelid;
- The vessels and nerves that supply these structures;
- The vessels and nerves that pass through the orbit to reach structures in other regions.

30.4.1 Extraocular muscles

The anteroposterior axis of the eyeball and the long axis of the bony orbit do not coincide. The long axes of the bony orbits diverge anteriorly. In contrast, at rest, the two eyeballs face forwards with their long axes parallel to each other. If you compare the angulation of the cone of muscles passing from the apex of the orbit to the eyeball to the medial wall of the orbit in Figure 30.6, you can see that the bone runs directly forwards whereas the muscles run forwards and laterally. This point is important in understanding the functions of the extraocular muscles and the relationships of the orbital structures.

The extraocular muscles comprise six muscles in each orbit that move the eyeball and elevate the upper eyelids. They are illustrated in Figure 30.5.

Levator palpebrae superioris

This flat muscle is the elevator of the upper lid. It attaches to the bone of the roof of the orbit close to its apex posteriorly and runs forwards beneath the orbital roof and broadens out to attach to the tarsal plate, skin, and conjunctiva of the upper eyelid. Most of the fibres of the muscle are striated and innervated by the superior branch of the **oculomotor nerve**. The part of the levator that is attached to the tarsal plate also contains some smooth muscle supplied by sympathetic nerves from the **superior cervical ganglion**. Problems resulting from damage to these nerves are outlined in Box 30.2.

The rectus muscles

The four rectus muscles arise from a **common tendinous ring** attached to the bone around the opening of the optic canal and the medial end of the superior orbital fissure. The recti pass forwards from this ring as a cone of muscles surrounding the optic nerve; they attach to the fibrous

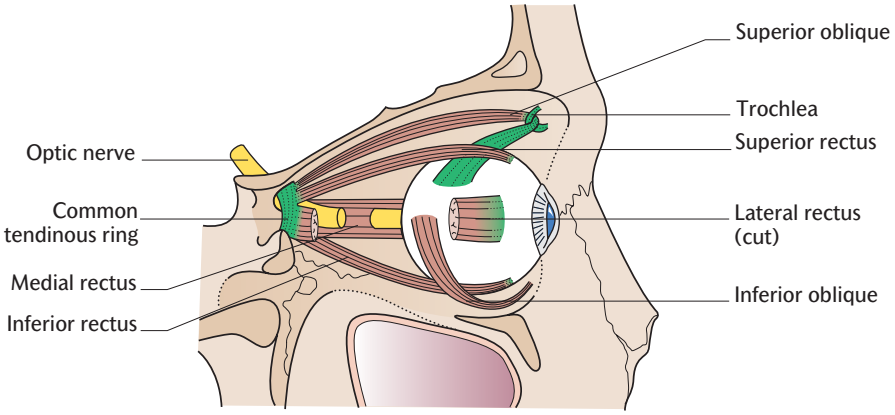


Fig. 30.5 The extraocular muscles.

Box 30.2 Ptosis

Drooping of the upper eyelid is called **ptosis**. It may arise if the sympathetic nerve supply to levator palpebrae superioris is compromised by, for example, compression of the cervical sympathetic trunk in the neck by a space-occupying lesion. In this case, ptosis may be one of the signs comprising Horner's syndrome (see Box 17.3, p. 157). Ptosis is also one of the signs and symptoms of **myasthenia gravis**, an autoimmune disease in which the body produces antibodies against its own acetyl choline receptors. Activity at neuromuscular junctions is reduced and muscles feel heavy.

sclera covering the eyeball anterior to the coronal equator which divides the eyeball into anterior and posterior halves.

As shown in Figure 30.5, the **superior rectus** lies beneath levator palpebrae superioris and attaches to the upper surface of the eyeball. It, therefore, elevates the eyeball and pulls it medially because of the lateral inclination of the muscle. The **inferior rectus** has an equivalent attachment on the inferior surface of the eyeball and acts to depress the eyeball and draw it medially. The **medial rectus** passes along the medial wall of the orbit into the medial surface of the eyeball; it turns the eye medially. The **lateral rectus** is similarly arranged on the lateral side of the eyeball and moves it laterally (abduction).

The superior rectus is innervated by the superior branch and the inferior and medial recti by the inferior branch of the oculomotor nerve. The lateral rectus is supplied by the abducens nerve.

The oblique muscles

The **superior oblique** muscle arises from the bone above and medial to the tendinous ring. Follow its course in Figure 30.5. It passes forwards above the medial rectus close to the junction of the medial wall and roof of the orbit, becoming tendinous towards the front of the orbit. The tendon passes through the **trochlear**, a loop of fibrous tissue attached to the roof of the orbit immediately behind the superomedial angle of the orbital margin. As it runs through the trochlear, the tendon turns posteriorly and laterally across the upper surface of the eyeball beneath the superior rectus, to attach to its upper surface behind the coronal equator; this is seen most clearly in Figure 30.6. The muscle pulls the posterior part of the eyeball upwards and medially, thus turning its front face downwards and laterally. It is supplied by the trochlear nerve.

The **inferior oblique muscle** is a narrow muscle which arises from the floor of the orbit lateral to the lacrimal groove as illustrated in Figure 30.5. It passes laterally and posteriorly beneath the eyeball and inferior rectus to attach to the lateral surface of the eyeball behind the coronal equator. It elevates the eyeball and draws it laterally. It is supplied by the inferior branch of the oculomotor nerve.

30.4.2 Nerves of the orbit

The nerves of the orbit comprise:

- The **optic nerve**;
- The **oculomotor, trochlear**, and **abducens nerves** to the extraocular muscles;
- The branches of the **ophthalmic division of the trigeminal nerve** which are sensory to structures within the orbit and to a wide area outside the orbit;
- The **ciliary ganglion** and associated branches which are concerned with the autonomic innervation of the eyeball;
- The **branches of the maxillary division of the trigeminal nerve** which pass through the orbit without supplying its contents.

The optic nerve

The optic nerve shown in Figure 30.5 has already been described in Section 18.4. It enters the orbit through the optic canal and passes forwards and laterally within the cone of recti muscles to enter the eyeball medial to its posterior pole.

Nerves to the extraocular muscles

The origins and functions of these nerves have already been described in Section 18.5; their intraorbital course will be described here and is illustrated in Figures 30.6 and 30.7.

The **oculomotor nerve** divides into its **superior** and **inferior branches** as it passes along the lateral wall of the cavernous sinus. These enter the orbit through the medial end of the superior orbital fissure within the common tendinous ring. The superior branch

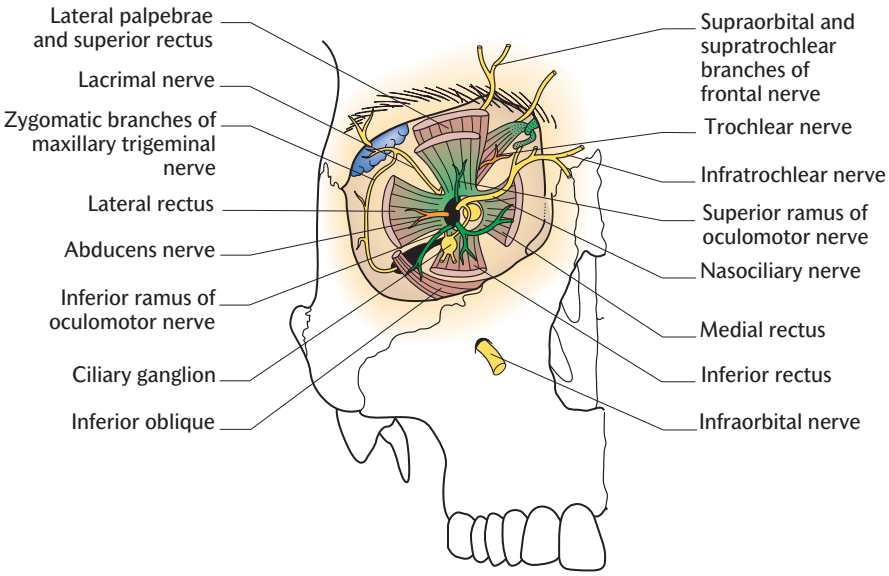


Fig. 30.6 Anterior view of the nerves of the orbit.

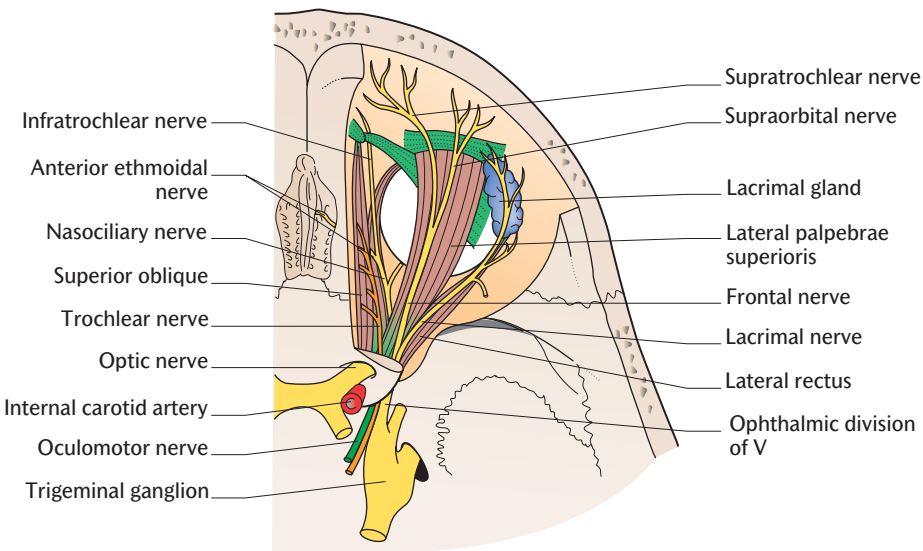


Fig. 30.7 Superior view of the ophthalmic trigeminal nerve and its branches in the right orbit after removal of the orbital part of the frontal bone.

crosses above the optic nerve to supply the superior rectus and levator palpebrae superioris muscles as you can see in Figure 30.6. The inferior branch divides immediately into branches to the medial rectus, inferior rectus, and inferior oblique muscles. The inferior branch gives off a parasympathetic branch to the ciliary ganglion.

The **trochlear nerve** enters the orbit through the superior orbital fissure outside the tendinous ring, then passes forwards to the superior oblique muscle.

The **abducens nerve** enters the orbit by passing through the superior orbital fissure within the tendinous ring and runs laterally and forwards to enter the lateral rectus muscle.

Branches of the ophthalmic division of the trigeminal nerve

The ophthalmic division of the trigeminal nerve splits into **lacrimal**, **frontal**, and **nasociliary** branches whilst still in the cavernous sinus. The course of these nerves is shown in Figures 30.6 and 30.7. All three

Box 30.3 Branches of the nasociliary nerve

The branches are, in sequence from the apex of the orbit:

- A sensory branch to **the ciliary ganglion**;
- Two **long ciliary nerves** enter the eyeball near the attachment of the optic nerve and supply sensory processes to the cornea and post-ganglionic sympathetic neurons from the superior cervical ganglion to the dilator pupillae muscle;
- The **posterior ethmoidal nerve** which exits through small foramina in the medial wall of the orbit to supply the posterior ethmoidal air cells and sphenoidal sinus;
- The two terminal branches—the anterior ethmoidal and infratrochlear branches. The **anterior ethmoidal nerve** supplies the anterior and middle ethmoidal air cells and continues as the **external nasal nerve** to innervate the skin over the nose. The **infratrochlear** nerve passes forwards below the trochlea to supply the lacrimal sac and adjacent conjunctiva and the skin of the upper eyelid and side of nose. The sensory innervation of the upper eyelids is from branches of the ophthalmic trigeminal nerve and the lower eyelids from the infraorbital branches of the maxillary trigeminal nerves.

branches enter the orbit through the superior orbital fissure, the lacrimal and frontal outside, and the nasociliary branch within the tendinous ring.

The **lacrimal nerve**, the smallest of the three branches, runs forwards along the lateral wall of the orbit above the lateral rectus muscle. It pierces the orbital septum to supply the skin of the lateral part of the upper eyelid and much of the conjunctiva. It also carries parasympathetic secretomotor nerves from the facial nerve via the pterygopalatine ganglion to the lacrimal gland.

The **frontal nerve** runs forwards between the levator palpebrae superioris and the roof of the orbit. It divides into a medial **supratrochlear** and a lateral **supraorbital** branch a short distance before the orbital margin. These supply the skin of the forehead. The supraorbital nerve also gives branches to the mucosa lining the frontal sinus.

The **nasociliary nerve** passes forwards within the cone of muscles, crosses above the optic nerve, and then divides into its two terminal branches about level with the back of the eyeball. It gives off several branches which are described in Box 30.3 for those who require this information.

The ciliary ganglion

This is a small parasympathetic ganglion lying lateral to the optic nerve some distance behind the eyeball as shown in Figure 30.7.

A branch from the inferior branch of the oculomotor carries preganglionic parasympathetic axons and synapses in the ganglion. Post-ganglionic neurons innervate **sphincter pupillae** and **ciliary muscles**. The sphincter muscles constrict the pupil and the ciliary muscles increase the lens curvature to focus the eye.

Sympathetic post-ganglionic vasoconstrictor nerves from the internal carotid sympathetic plexus pass through the ganglion to innervate blood vessels in the eyeball. As already mentioned, sympathetic nerves to dilator pupillae reach the eyeball in the long ciliary branches of the nasociliary nerve. A branch of the nasociliary nerve also passes through the ganglion without synapsing to supply the cornea and iris.

The dozen or so **short ciliary nerves** exit the ciliary ganglion and enter the back of the eyeball. They carry the parasympathetic, sympathetic, and sensory nerves from the ganglion into their various targets within the eyeball.

Orbital blood vessels

The **ophthalmic artery** branches from the internal carotid artery just as it emerges from the cavernous sinus and enters the orbit through the optic canal. The artery has branches that follow the lacrimal and nasociliary nerves to the same targets, muscular branches to the extraocular muscles, ciliary arteries to the eyeball, and the **central artery of the retina** which runs within the optic nerve to the retina.

Venous blood leaves the orbit and its contents in the **superior and inferior ophthalmic veins**. Both pass through the superior orbital fissure to drain into the cavernous sinus. The superior veins communicate with the **facial veins** and the inferior vein frequently connects with the **pterygoid plexus** through the inferior orbital fissure. These connections are, like venous connections mentioned in previous chapters, potential routes for spread of infection.

Lymph drains from the orbit to the parotid nodes and thence to the deep cervical group.

Box 30.4 Fractures of the orbit

Fractures of the facial skeleton often extend to the orbit. Nerves or extraocular muscles can be crushed or trapped by bony displacement but the consequences of a particular injury depend upon the specific structures that are injured. To reiterate an important point first raised in Chapter 18, any problem with extraocular muscles, whether through direct injury or damage to their nerve supplies, will result in the patient seeing double (**diplopia**).

A **blowout fracture** of the orbit is caused when an object smaller in diameter than the orbital rim (c 5cm), such as a squash or golf ball, impacts on the eye. The eyeball is filled with fluid and therefore cannot be compressed so the eyeball will be distorted and press on all four walls of the orbit. The medial and lateral walls and roof are robust but the floor is thin. The floor usually breaks posterior to the orbital rim but does not detach as it is held by the periosteum. The segment posterior to the fracture collapses or 'blows out' into the maxillary sinus but then swings back as the pressure is relieved; the inferior rectus and inferior oblique muscles are often trapped by the bony fragment as it hinges upwards.

Complex injuries of the facial skeleton are known as **Le Fort fractures** and are classified into three types; Figure 30.8 shows the positions of the fracture lines. A Le Fort type I fracture runs subzygomatically; Le Fort type II fractures are also known as pyramidal fractures because a pyramidal segment is broken off; Le Fort type III fractures are suprazygomatic.

In **type I injuries**, the fracture line passes through both maxillae above the apices of the teeth and the hard palate into the lower part of the lateral wall of the nose and usually involves the bones of the nasal septum. The whole tooth-bearing part and the hard palate of the upper jaw are detached from the rest of the facial skeleton as a result.

Type II fractures result in the detachment of a pyramidal-shaped segment of the facial skeleton. The fracture line passes above the apices of the roots of the posterior upper teeth and upwards through the lateral parts of the bodies of the maxillae, through the lacrimal bones or ethmoidal labyrinth and then across the frontal processes of the maxillae and the nasal bones. The lateral walls and septum of the nose are also damaged, but at a higher level than in the type I fracture.

A **type III fracture** crosses the nasal bones, close to the frontonasal suture, the frontal processes of the maxillae, and then runs posteriorly on each side along the medial wall of the orbit through the lacrimal bone and the labyrinth. The fracture line usually passes below the optic canal to the posteromedial end of the inferior orbital fissure. It continues from the anterolateral end of the fissure across the lateral wall of the orbit through the orbital part of the zygomatic bone. A further fracture line passes vertically from the inferior orbital fissure across the pterygopalatine fossa and through the pterygoid process of the sphenoid.

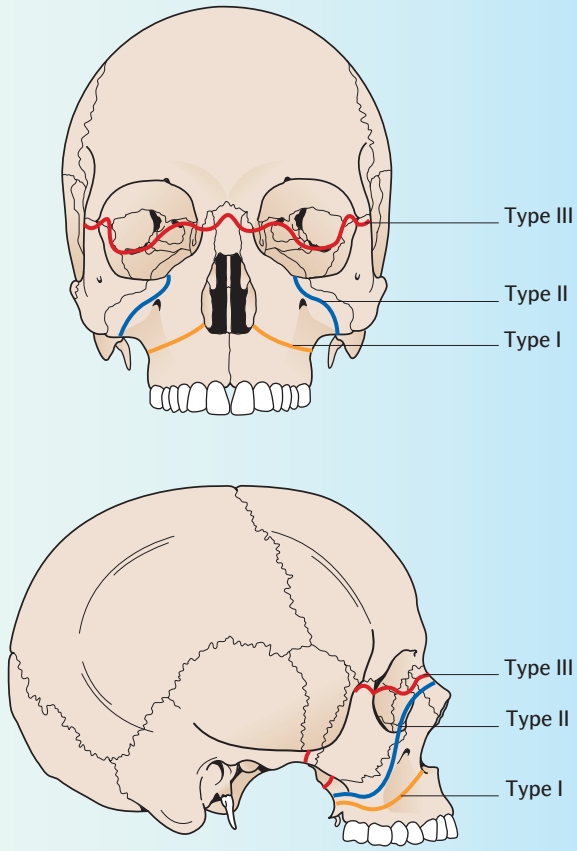


Fig. 30.8 Le Fort type I, II, and III fractures.

The zygomatic arches are also fractured. If you follow the type III fracture lines in Figure 30.8, you will realize that virtually the whole of the upper facial skeleton is detached from the remainder of the cranium.

High impact injuries are necessary to produce Le Fort type fractures. There will be considerable damage to neighbouring soft tissues with considerable haemorrhage, oedema, and pain. The involvement of the ethmoid region in the type II and III fractures may result in tearing of the meninges and consequent leakage of cerebrospinal fluid into the nose.

31

Radiological anatomy of the oral cavity

Chapter contents

31.1 Introduction

The radiographs most frequently taken in general dental practice are of the teeth and their immidiate supporting tissues for detection of dental caries or assessment of bone loss in periodontal disease. **Intraoral radiographs** are taken by placing the X-ray-sensitive film or receptor in the mouth close to the teeth being investigated. **Extraoral radiographs** use larger films or receptors positioned externally and produce a view of the entire dentition and its supporting structures on a single film; they are used to ascertain the state of development of the dentitions prior to orthodontic treatment, for example. **Dental panoramic tomographs** (**DPTs**) are the most frequent extraoral radiographs.

A radiograph is a negative photographic record. Dense structures such as bone are designated as **radio-opaque**; they absorb some X-rays and appear white on radiographs. More X-rays pass through less dense **radiolucent** structures such as air-filled cavities which show up as black areas. The contrast between different tissues of the structures which the X-ray beam passes through is determined by their radiodensity which, in turn, is largely due to their content of metallic elements. Calcium and iron are the prevalent heavy metals in the body. Calcium is combined with phosphate to form **hydroxyapatite** crystals in bones and mineralized tissues in teeth. Iron is present in haemoglobin in blood, but only large concentrations of blood, such as those found within the heart chambers, show up on X-rays. In sequence from densest to most lucent, the radiodensity of the dental and periodontal tissues are: enamel, dentine, cementum, compact bone, cancellous bone, demineralized carious enamel and dentine, dental soft tissues such as pulp and periodontal ligament, and air; gold and silver–mercury amalgam metallic restorative materials are even denser than enamel.

A radiograph is a two-dimensional representation of a three-dimensional situation. The orientation of anatomical structures relative to the X-ray beam is a major factor determining their appearance on the film. For example, a beam travelling through the long axis of a radiodense structure will produce a whiter image on the film than one passing through its shorter axis because more X-rays are absorbed; the structure will also have a different shape. Orientation also affects the degree to which structures are superimposed on each other on the radiograph; it is necessary to carefully position the part being X-rayed so that it is not obscured by another superimposed radio-opaque structure.

There are many factors that have to be considered when taking radiographs which involve the physics of X-rays and other technical aspects are outside the scope of this book.

Extraoral radiographs reveal extensive anatomy of structures adjacent to the teeth and their supporting tissues whereas smaller intraoral views only show limited areas. It is easier to appreciate the anatomy on extraoral views; the structures visible on different intraoral radiographs can then be related to the bigger picture.

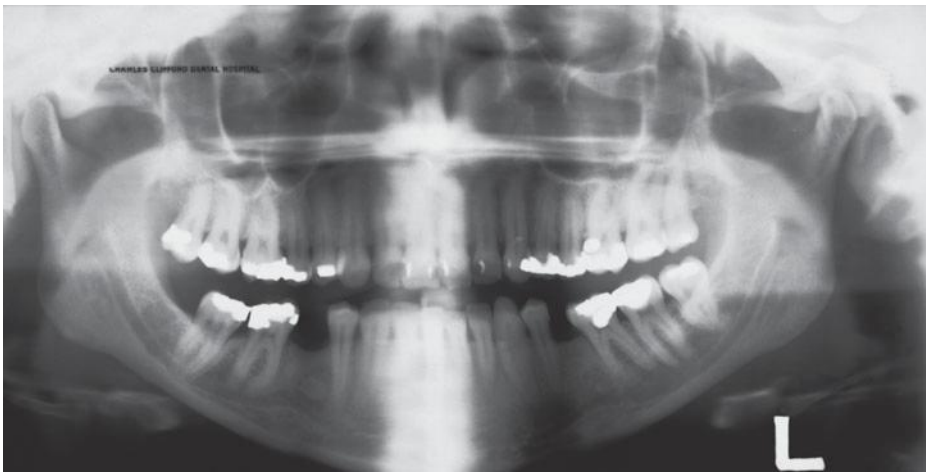
31.2 Dental panoramic tomography

Dental panoramic radiographic machines work on the principle of **tomography**. This involves simultaneously moving the film and the X-ray source in opposite directions to each other around the patient's head. The procedure is generally quicker and more comfortable for the patient because there are no intraoral films (see Section 3.4). As you can see in Figure 31.1 the resulting radiograph appears as a flat representation extending from one temporomandibular joint to the other, showing a complete picture of the entire upper and lower dentition and their supporting alveolar processes in the mandible and maxilla; these appear as a sharp image whereas structures in all other planes are blurred and effectively invisible. In DPTs, the teeth and supporting structures do not show with the sharpness and detail seen in intraoral radiographs. Panoramic radiographs are particularly useful when examining developing dentitions, large lesions, or fractures of the jaws when fine detail is often not required. For fractures, panoramic radiography has the great advantage that it can be performed without the patient needing to open their mouth.

Several anatomical structures are visible on DPTs, but many additional features can be seen. The **mandibular canal** is visible below the lower posterior mandibular teeth but becomes indistinct anteriorly; the canal is unusually broad in the DPT in Figure 31.1. The **hard palate** shows as a horizontal white line above the root apices of the maxillary dentition. The floors of the **maxillary sinuses** show clearly above the roots of the premolars and first and second molar teeth. If you examine Figure 31.1 carefully, you will see that the relationships of the upper teeth to the right and left maxillary sinuses are not completely symmetrical, which is perfectly normal. Both **mandibular condyles** can be seen with the **zygomatic arch** extending forwards anterior to them. The **zygomatic processes** of the maxilla can be seen extending upwards to the anterior part of each arch at either end of the hard palate. The **inferior orbital margin** can also be distinguished crossed by the **infraorbital canal**. The **nasal septum** is present above the midline with the **inferior conchae** visible on either side.

Many other extraoral radiographs previously used in dentistry to examine particular structures such as the temporomandibular joints or cranial base have been superseded in developed countries by CT or MRI scans (see Chapter 13) or the relatively new dental imaging technique of cone beam computerized tomography; their use is spreading to the developing world.

A



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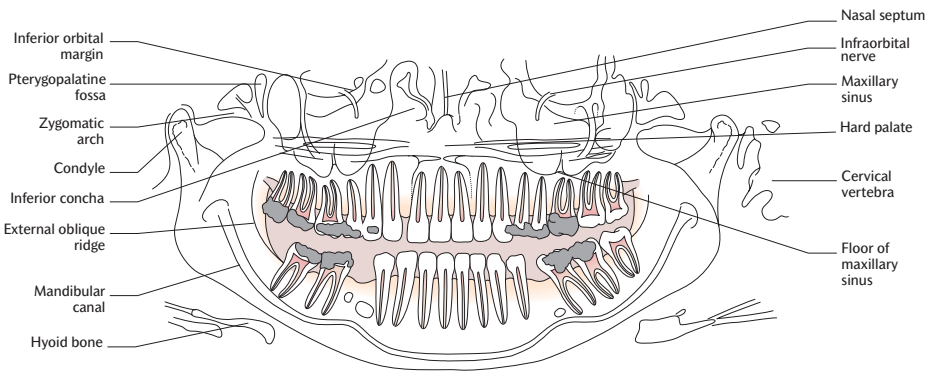


Fig. 31.1 A) A panoramic radiograph; B) Key.

31.3 Cone beam computerized tomography

A relatively recent development of targeted tomography for use in dentistry is **cone beam computerized tomography** (**cone beam CT**), also known as **digital volume tomography**. An X-ray source emitting a cone-shaped beam orbits the target area once, imaging a cylindrical volume from which computer reconstructions can be made and examined. Relatively small volumes of about 3 cm in height and 4 cm in diameter can be examined so they can be used for examination of structures usually investigated by introral radiographs.

Cone beam CT delivers a low dose of X-rays, can be carried out very quickly, and is inexpensive compared to medical CT. The technique is valuable for investigation of the temporomandibular joint, maxillary sinus, cysts and tumours of the jaw, periapical pathology, facial fractures, orthodontic assessment, and dental implants. Needless to say, there are disadvantages; metal objects produce streaked or star-shaped artefacts. In developed countries, cone beam CT is becoming available in dental schools and hospitals and is now extending into dental practice.

31.4 Intraoral radiographs

31.4.1 Bitewing radiographs

The principal purpose of these types of radiograph is to disclose caries on the inaccessible approximal surfaces of the posterior teeth. The receptors or wrapped films measure approximately 4.5 × 3 cm. The film package originally had a flange or 'wing' along the middle of the wrapping on the sensitive side which the patient bit on to hold the film in place, hence the term 'bitewing'. Modern bitewing films are placed in a suitable holder or detectors are used; these must still be retained by the patient biting on to it as shown in Figure 31.2A. The detector is adjacent to the crowns of the upper and lower teeth and the X-ray beam is directed at right angles to the plane of the film.

Examine Figure 31.3A and the corresponding key in Figure 31.4. The crowns of the upper and lower premolars and first two molars can be seen together with the coronal parts of the root and adjacent bone. You should be able to differentiate enamel, dentine, and pulp of the teeth by their differing radio-opacity. Note the clearly defined white line lining each tooth socket; this is the image of the **lamina dura**, the dense layer of alveolar bone forming the inner socket wall. The supporting trabeculae of cancellous bone of the alveolar process can be distinguished deep

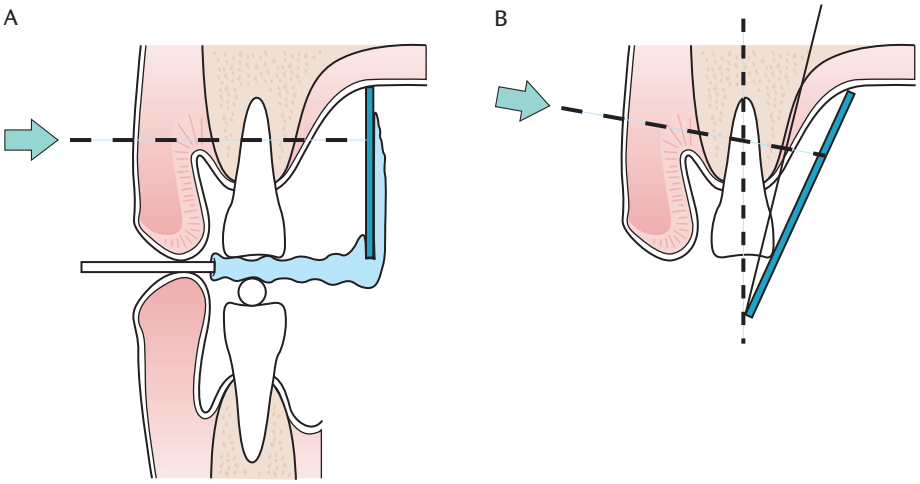


Fig. 31.2 Positioning of tube and film for: A) A bitewing radiograph for examining approximal surfaces; B) A periapical radiograph.

to the lamina dura. The **periodontal space** is the dark line between the lamina dura and the tooth root occupied by the radiolucent periodontal ligament. The cementum layer on the root surface is so thin that it cannot be distinguished from dentine.

31.4.2 Periapical radiographs

As the name suggests, these are used to examine the roots of the teeth and the periapical tissues surrounding the root apices. The crowns of the teeth are also visible. Films or receptors the same size as those used in bitewing radiography are placed in suitable holders so that the film can be placed on the internal aspect of the teeth with one of its long edges level with the occlusal surfaces or incisal edges and as nearly parallel to the long axis of the teeth as other oral structures will allow. As you can see in Figure 31.2B, the film must be placed at a considerable angle to the upper teeth because of the presence of the hard palate. To minimize distortion, the X-ray beam is directed perpendicular to the plane that bisects the angle between the long axes of the teeth and the film as indicated in Figure 31.2B.

Some of the anatomical features already described above for DPTs also appear adjacent to the teeth on periapical radiographs.

As you can see in Figures 31.3B and 31.3C and the key in Figure 31.4, the whole extent of each tooth can be seen in periapical radiographs; enamel, dentine, and pulp of the teeth, the periodontal space, lamina dura, and cancellous bone are also clear. In a healthy mouth, the lamina dura is intact around the whole of the root or roots.

In the periapical view of the upper molar region shown in Figure 31.3B, the cortical bone forming the floor of the **maxillary air sinus** appears as a rounded radiolucent area separated from the root apices of the second premolar and first and second molars. As pointed out in Section 27.4.1, the size of the sinus is extremely variable with corresponding variations in its radiographic appearance. Sometimes, the root apices of the maxillary teeth appear to be projecting into the sinus. In these cases, careful examination will show whether the lamina dura is intact around the apices, showing that the roots are not actually in the sinus but superimposed on it or that the lamina dura is perforated by the roots and is projecting into the sinus. The root of the **zygomatic process** of the maxilla usually shows as a radio-opacity overlying the shadow of the sinus and the roots of the molar teeth. The **maxillary tuberosity** is visible on periapical radiographs that include the upper third molar teeth.

The **mandibular canal** is the principal non-dental feature visible on periapical radiographs of the lower posterior teeth jaw running forwards below them. As you can see in Figure 31.3C, it shows as two dense lines, indicating the radio-opaque cortical bone forming the walls of the canal that enclose a dark band produced by the radiolucent nerves and vessels in the canal. On periapical radiographs of the lower premolars, the **mental foramen** appears as a dark area between their roots. The **genial tubercles** may be seen as small white areas on either side of the midline in periapical views of lower anterior teeth.

In a periapical radiograph of the upper incisor region, the **incisive foramen** on the bony palate appears as a dark area between the roots of the central incisors superimposed on to a linear radio-opacity produced by the **nasal septum**. These features can also be seen on maxillary standard occlusal radiographs.

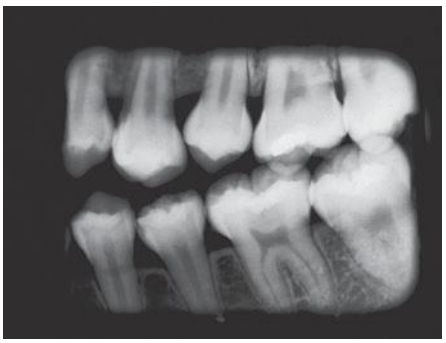
31.4.3 Occlusal radiographs

A **standard occlusal radiograph** gives a scan view of the anterior teeth and anterior part of the hard palate. The film, which is larger than a bitewing film, is held horizontally between the teeth and the X-ray beam is directed obliquely downwards through the bridge of the nose at an angle of about 65° to the film. They are frequently used to ascertain the position of unerupted teeth and the radiograph in Figure 31.3D shows an unerupted right canine tooth.

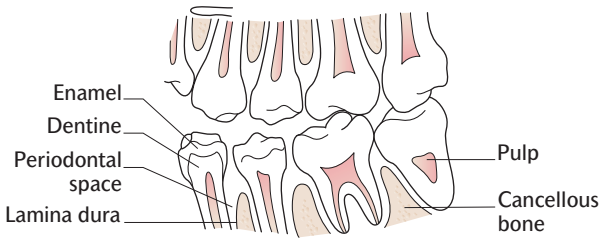
The anatomical features that show up on occlusal radiographs may also be seen on periapical radiographs of maxillary anterior teeth. In Figure 31.3D, the white radio-opaque midline **nasal septum** has a dark line superimposed upon it, produced by the **median palatine suture**; the **incisive fossa** may be visible at its anterior end. The two halves of the

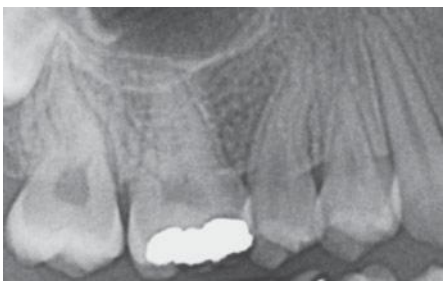
Fig. 31.3 Intraoral radiographs. A) Bitewing; B) Upper molar periapical © STEPHEN GERARD/SCIENCE PHOTO LIBRARY; C) Lower molar periapical © SOVEREIGN, ISM/SCIENCE PHOTO LIBRARY; D) Standard occlusal.

Fig. 31.4 Key to Figure 31.3.



(A)


Enamel
Dentine
Periodontal space
Lamina dura
Pulp
Cancellous bone



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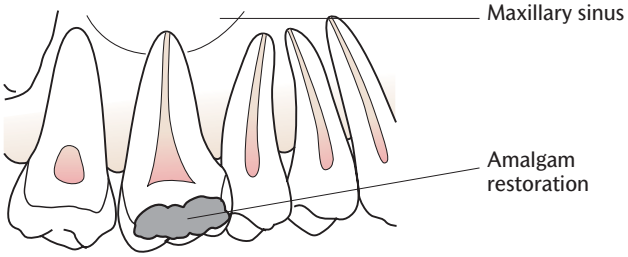
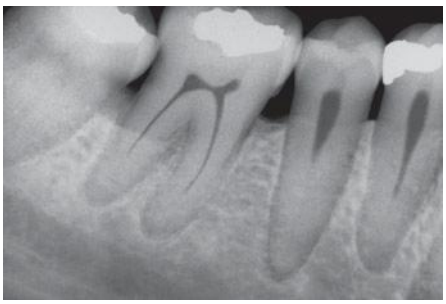
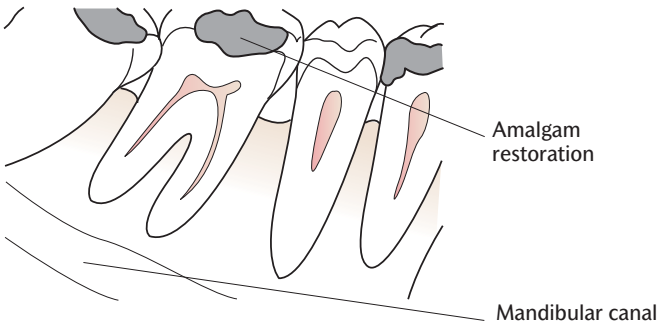
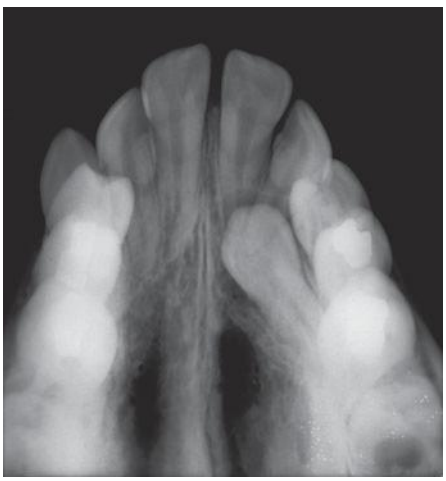

Maxillary sinus
Amalgam restoration

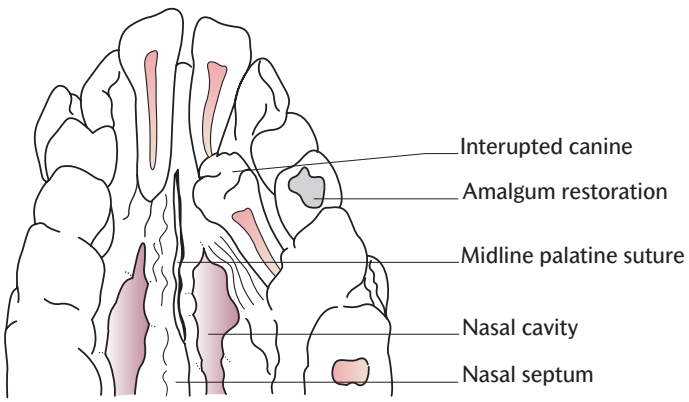


(C)


Amalgam restoration
Mandibular canal



(D)


Interupted canine
Amalgum restoration
Midline palatine suture
Nasal cavity
Nasal septum

nasal cavity are visible as radiolucencies on either side of the septum and the maxillary air sinuses show as smaller radiolucencies posterior and lateral to the nasal cavity.

Occlusal views of the mandible are taken by extending the patient's head, with the film held between the teeth (sensitive side downwards) and the X-ray beam directed at right angles to it through the floor of the mouth. They are used to determine the position of unerupted lower teeth and to show calculi in the submandibular ducts.

32

The development of the face, palate, and nose

Chapter contents

32.1 Introduction

In Chapter 21, we described the development of the pharyngeal arches and their derivatives. Craniofacial abnormalities account for about one third of all live birth defects. These arise during the development of the pharyngeal arches described in Chapter 21 or during the events described in this chapter.

The **first pharyngeal arch**, the mandibular arch, is one of the basic building blocks needed to form the face and associated structures. The other major building block is the **frontonasal process** that covers the developing forebrain (see Section 19.3).

The development of the face begins after the first pharyngeal arch forms around four weeks post-fertilization. At this stage, the head consists of a large bulge over the developing forebrain, approximating to the forehead and the mandibular arch in the position of the lower jaw. A slit between the frontonasal process and mandibular arch is continuous with the foregut tube; this slit is the primitive oral cavity or **stomodeum**. This primitive mouth cavity has no side walls where the cheeks would be and more significantly, there is no nasal cavity.

Development of the nasal and oral cavities internally and the face externally proceeds at the same time over the course of the next eight weeks of development. Essentially, the nasal cavity is formed, then divided into two and separated from the oral cavity by the palate. The sequence of events is:

- Development of the nasal cavity and first part of the palate, beginning the separation the oral and nasal cavities;
- Development of the maxillary arch from the mandibular arch to form the cheeks and important structures contributing to the palate;
- Completion of the components required to form the palate and separate the nasal cavity into right and left cavities;
- Fusion of the building blocks to complete the separation of the nasal cavity and the separation of the nasal cavity from the oral cavity.

32.2 Formation of the nasal cavity and palate

32.2.1 Formation of the primary nasal cavity and primary palate

As you can see in Figure 32.1A, the stomodeum is roofed by the frontonasal process and its floor is the fused mandibular arches. Notice how the first arch is pushed upwards by the cardiac bulge formed by the precociously developing heart. At first, the stomodeum is closed posteriorly by the **buccopharyngeal membrane**, one of the two places where ectoderm and endoderm meet without intervening mesoderm (see Chapter 8, p. 55). This membrane ruptures at about the end of the third week post-fertilization and the stomodeum becomes continuous with the foregut—the future pharynx.

Figure 32.1B shows two ectodermal thickenings called the **nasal placodes** in the ectoderm, covering the frontonasal process just above the stomodeum; they develop towards the end of the fourth week of development. The nasal placodes will eventually form the **olfactory epithelium** in the roof of the nasal cavity. During the fifth week, the placodes sink into the underlying ectomesenchyme by a combination of growth of the frontonasal process outwards around the placodes and the invagination of the placodes themselves. The placodes are now located in the floor of two shallow depressions, the **nasal pits**. As illustrated in Figure 32.1C, tissues are built up on either side of each nasal pit to form the **medial** and **lateral nasal processes**. The medial nasal processes fuse together to form the **intermaxillary segment** of the

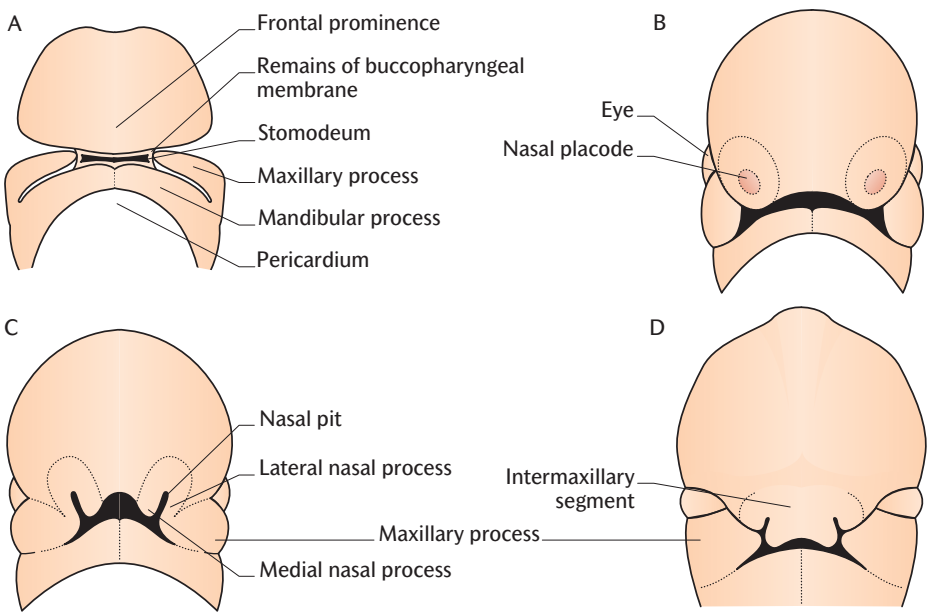


Fig. 32.1 Development of the face.

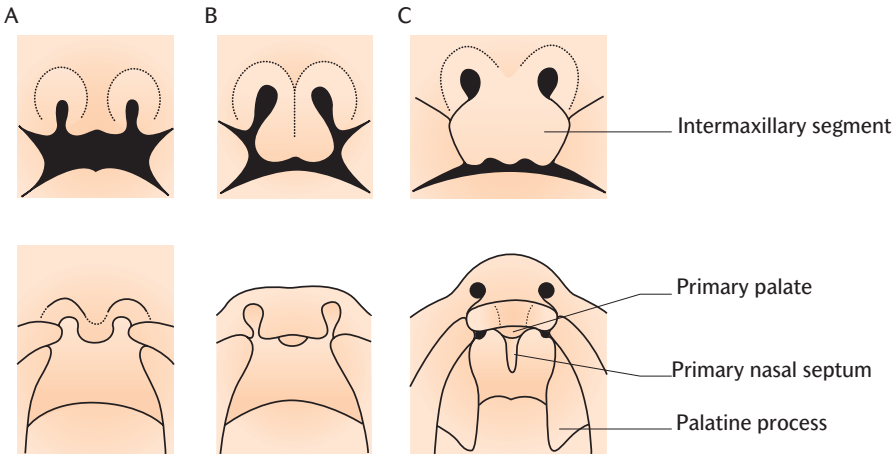


Fig. 32.2 Development of the primary palate and primary nasal septum.

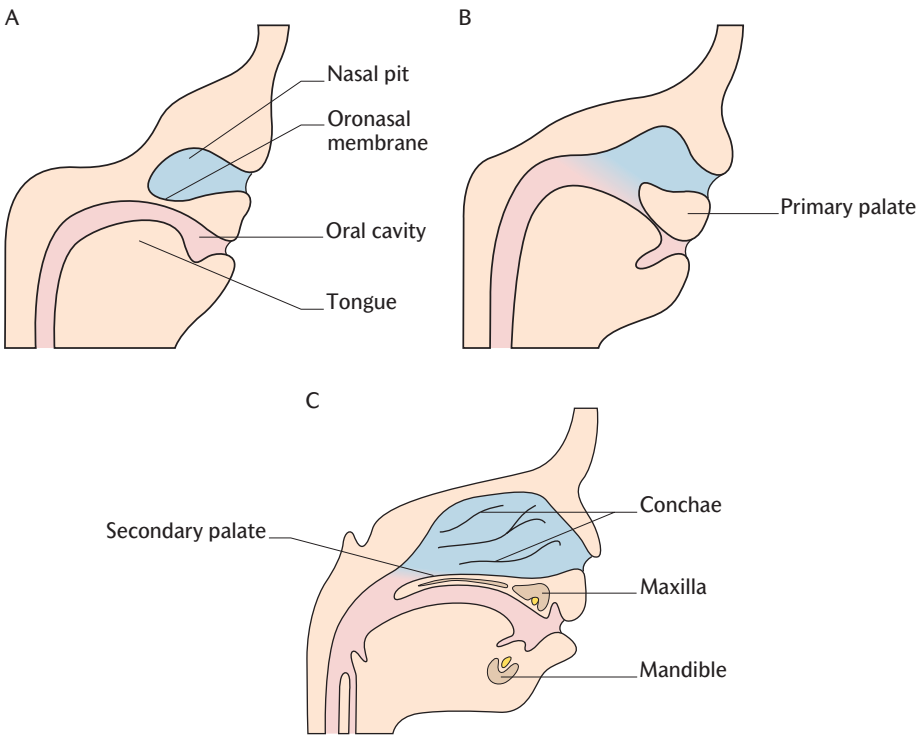


Fig. 32.3 Lateral views of the development of the nasal and oral cavities.

frontonasal process which contribute to the middle portion of the nose, the philtrum of the upper lip, and the part of the upper jaw bearing the four maxillary incisors and forming the primary palate. The lateral swellings only form small areas of the lateral parts of the external nose.

As the nasal pits push into the frontonasal process, they do not move backwards in a straight line. Instead, they converge towards the midline. The area of tissue between them is the **primary nasal septum**, forming the first component of the nasal septum. As you can see on the frontal view in Figure 32.2B, the intermaxillary segment below the nasal pits is quite bulbous and extends under the pits. The corresponding intraoral view on the second row of Figure 32.2B shows that the intermaxillary segment forms a horizontal triangular shelf with its apex directed posteriorly; this is the **primary palate** and the first component separating the nasal and oral cavities.

Figure 32.3 shows the development of the nasal pits in a lateral view; the primary palate and septum only occupy a relatively small anterior area.

32.2.2 Formation of the secondary palate

The **maxillary processes** are outgrowths from the upper posterior area of the mandibular processes. They arise at six weeks post-fertilization and grow forwards between the frontonasal process and the mandibular arches to form the cheek area of the face as you can see in Figures 32.1B and 32.1C. The secondary palate originates as two shelf-like outgrowths, the **palatine processes**; as illustrated in Figure 32.2C, they appear on the inner surface of each maxillary process. Fibroblastic growth factor 10 (FGF_{10}) is important for development and growth of

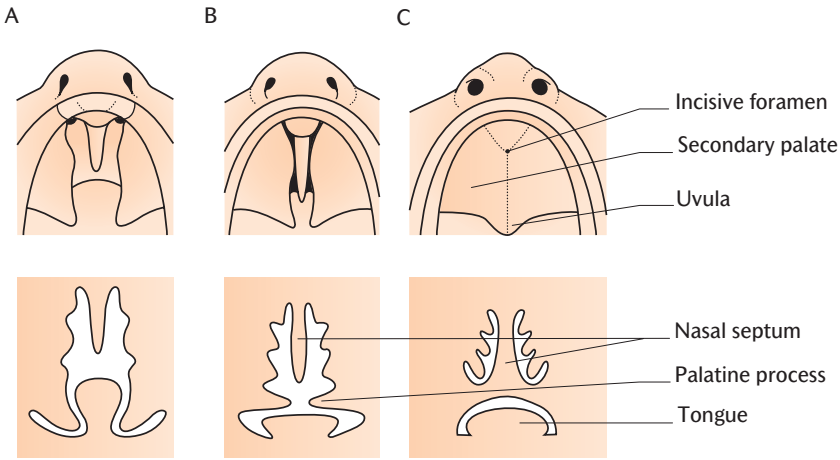


Fig. 32.4 Development of the secondary palate.

the palatine processes. If this factor is reduced or absent, the processes fail to develop or are reduced significantly.

As described in Section 21.7, the tongue develops very early quickly after the pharyngeal arches have formed. It grows and fills the oronasal cavity as shown in Figure 32.4A. The rudimentary palatine processes start to grow medially towards each other, but are then deflected downwards on either side of the developing tongue as illustrated in Figure 32.4A. Observe also in Figure 32.4A the **secondary nasal septum** growing downwards in the midline.

32.2.3 Palatal elevation

At eight weeks post-fertilization, several changes occur that alter the orientation of the palatine processes from vertical to horizontal. The developing brain acquires a cervical flexure (see Section 19.3), the result of which is that the head is raised away from the cardiac bulge. This allows space for the mandible to grow in width, which in turn allows the tongue to drop to its position in the floor of the mouth. The palatine processes are no longer impeded by the tongue and flip up in a matter of minutes into a horizontal orientation as you can see in Figure 32.4B. However, **palatal elevation** is not just a matter of removal of physical barriers. If this was the case, you would expect that the palatine processes would elevate if the tongue was depressed experimentally before the normal time it drops into the floor of the mouth. When such experiments have been performed, the palatine processes do not in fact elevate until just before their normal time. This implies that there must be something intrinsic within the palatine shelves which makes them elevate as well as the extrinsic changes just outlined. It has been found that the ectomesenchymal cells within the shelves synthesize **glycosaminoglycans** (GAGs) in large quantities. GAGs are one of the components of extracellular matrix and are strongly hydrophilic; they can bind about 100 times their own volume of water. The binding of water to the GAGs in the palatine processes provides turgor pressure to elevate the shelves; the mechanism is similar to watering a wilted plant. The elevated palatine processes, the primary palate, and secondary nasal septum grow towards each other over the next two weeks.

32.2.4 Palatal fusion

The processes meet in the tenth week and begin the process of **palatal fusion** to unite the individual components of the palate with each other to separate the oral and nasal cavities; they also fuse with the nasal septum to complete the division of the nasal cavity into right and left halves. For fusion to take place, the ectodermal **medial edge epithelium** covering the contact surfaces must be removed so that the ectomesenchyme of the contributory processes can intermix. The cells of the medial edge epithelium die off by the process of **apoptosis** or programmed cell death (see Section 8.2). When the medial edge epithelia from adjacent processes make contact, gene expression is triggered in the ectoderm. This stimulates a cascade of gene expression in the underlying ectomesenchyme that in turn stimulates production of growth factors that signal the epithelial cells to die.

First contact is made about half way along the midline between the palatal processes and fusion. Fusion continues anteriorly between the palatine processes and then between the processes and the primary palate. The fusion process continues on to the face between the intermaxillary segment of the frontonasal process forming the philtrum of the upper lip and the maxillary processes forming the lateral aspects of the upper lip. Fusion also continues posteriorly from the initial contact point to complete fusion of the palatine processes with each other and the secondary nasal septum. The lines of union between the primary palate and the two secondary palatal processes form a Y-shaped pattern which is shown by dotted lines in Figure 32.4C; the junction of the stem and two arms of the Y is marked by the **incisive foramen**. The fusion of the palate is completed in the twelfth week. Between 12 and 16 weeks, the fused palatine processes are invaded by bone anteriorly to form the bony palate and the muscles of the soft palate develop posteriorly from mesoderm invading from the first and fourth pharyngeal arches. Development abnormalities arising during formation of the palate are described and illustrated in Box 32.1.

Box 32.1 Cleft lip and palate

Craniofacial anomalies account for about one third of all birth defects. Specific anomalies clustered together constitute a **syndrome** and the collection of defects is said to be **syndromic**; an anomaly occurring in isolation is **non-syndromic**. The most common anomalies are **cleft lip, cleft palate,** or **combined cleft lip and palate**. About half of all cleft palate anomalies are syndromic and 50% non-syndromic. In the UK, this equates to an occurrence of about 1 in 1000 live births. Cleft lip (with or without cleft palate) occurs about once in every 700 live births so is slightly more prevalent. Genetic defects have been detected in some syndromic anomalies that involve cleft palate; there is no clear genetic background for non-syndromic occurrence of clefts. However, the probability of recurrence is higher in families with one affected individual than in the general population, suggesting some genetic influence.

There is some recent evidence that different molecular mechanisms may operate at different times and places during palatal and facial development. The **incisive foramen** is a useful landmark. Defects anterior to the foramen arise due to anomalies in the development and fusion of the intermaxillary segment with its neighbouring maxillary processes anteriorly and palatine processes more posteriorly. Defects posterior to the incisive foramen are due to the failure of growth or union of the palatine processes.

Anterior defects in front of the incisive foramen may be unilateral or bilateral and can extend any distance along one or both arms of the Y fusion lines in the palate anterior to the incisive foramen, the upper lip, and the nose; several examples are illustrated in Figures 32.5A–D. Severity may range from no more than a white line in the vermilion border of the lip through various degrees of lateral cleft lip to division of the maxilla. In severe bilateral defects as shown in Figure 32.5D, the central part of the lip and upper jaw are free from the structures on each side; the free segment may then swing forward in a very disfiguring manner.

Posterior defects tend to run forwards from the uvula. They vary in severity from merely having a bifid uvula to a large midline defect extending from the uvula to the incisive foramen as shown in Figure 32.5E. Combined cleft lip and cleft palate can occur together, producing a cleft which runs from the posterior border of the palate to the lip (see Figure 32.5F); combined cleft lip and palate is bilateral at its most severe.

You can probably work out the most likely causes of craniofacial defects from the events occurring in craniofacial development described in Section 32.2. They are, in temporal order of occurrence:

- Failure of formation of processes or reduced growth so that they cannot meet and fuse;
- Failure of the palatine processes to elevate;
- Failure of apoptosis at fusion sites, producing incomplete or failed fusion.

Although the likely point at which defects may occur can be surmised, the causes of palatal defects are still uncertain, except in syndromic cases with a defined genetic basis.

Pierre–Robin syndrome is an example of a syndrome that includes craniofacial anomalies. It is characterized by micrognathia (a small mandible), posterior displacement of the tongue, and upper airway restriction; cleft palate is present in the majority of affected individuals.

As well as the considerable cosmetic defect, cleft lip or palate impairs oral function; food and drink escape into the nasal cavity and speech articulation is disrupted. In developed countries, babies are assessed for cleft defects during immediate neonatal screening. A temporary repair is carried out either surgically or by placement of a denture-like device to close the palate. Repair is carried out at intervals as the child develops to produce a functional and aesthetic unit. As well as maxillofacial and reconstructive surgery, orthodontic treatment is usually required and speech therapy is necessary to develop fluent speech. Unfortunately, these extensive resources are not available in many developing countries; cleft defects remain untreated and have severe social effects on the victim.

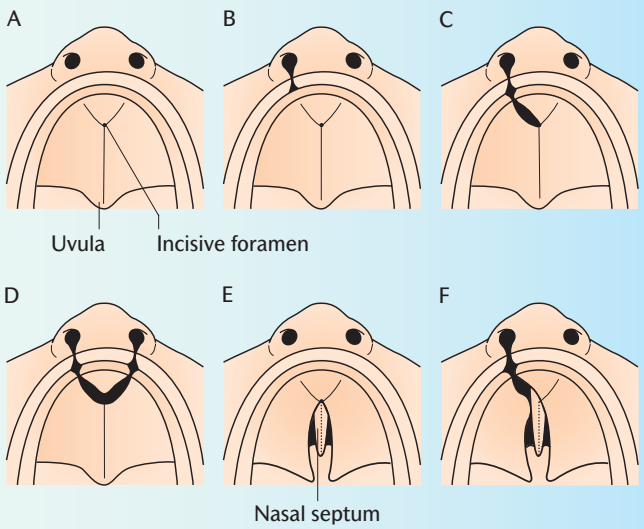


Fig. 32.5 Cleft lip and cleft palate. A = normal; B = unilateral cleft lip; C = unilateral cleft lip and anterior cleft palate; D = bilateral cleft lip and anterior cleft palate; E = posterior cleft palate; F = unilateral cleft lip and anterior and posterior cleft palate.

Box 32.2 Other craniofacial defects

There are some rarer craniofacial anomalies. Early defects in the formation of nasal placodes and intermaxillary segment produce **cyclopia** in which the two eyes fuse into a single median eye; fetuses with this anomaly usually die *in utero* and are miscarried or stillborn. **Macrostomia** (Greek = big mouth) arises when the maxillary and mandibular arches fail to fuse as far as normal; it may be unilateral or bilateral.

32.3 Formation of the external face

The development of the face externally depends to some extent on similar processes of fusion as described for the palate and also on the growth and development of the brain and special senses.

In Figure 32.1 B, you can see one of the precursors of the eye, the lens placode. Notice the eyes are positioned laterally at this very early stage of facial development. The developing forebrain pushes the eyes forwards (see Figure 32.1D) and they exert pressure on the intermaxillary segment which is pushed out as the external nose. At the same time as fusion of the palate occurs internally, fusion takes place by a similar mechanism between the inferior aspect of the maxillary arches and the mandibular arches laterally to form the entrance to the oral cavity between the lips. The lateral nasal processes and the upper aspect of the maxillary arches also fuse. Some abnormalities arising during formation of the face are outlined in Box 32.2.

A timetable of key events in the formation of the face, palate, and nose is presented in Table 32.1 to summarize this chapter.

Table 32.1 Key events in the formation of the face, palate, and nose

4 weeks post-fertilization	Formation of the pharyngeal arches
5 weeks	Formation of the nasal placodes, primary palate, and primary nasal septum
6 weeks	Outgrowth of the maxillary process from the mandibular process and formation of the palatine processes; formation of the secondary nasal septum
8 weeks	Elevation of the palatine processes
10 weeks	Fusion of the palatine processes, primary palate, and secondary nasal septum begins. Fusion of intermaxillary segment and maxillary and mandibular processes on the face begins.
12 weeks	Fusion of palatine processes, primary palate, and secondary nasal septum complete. Fusion of processes on the face complete.
12–16 weeks	Invasion of anterior palate by bone and posterior palate by muscle of first and fourth pharyngeal arches to form bony palate and soft palate.

33

Development and growth of the skull and age changes

Chapter contents

33.1 General principles of growth and development

The development of the facial bones is particularly important in the fields of paediatric dentistry and orthodontics. Dental students and dental practitioners who do not specialize in those subjects should have an appreciation of the subject to be aware of the changes to the face and jaws they are seeing in patients under continuous care as they grow, mature, and age.

Human beings increase in both size and complexity during the **growth period** which lasts from conception until maturity at about 16 to 18 years of age. As we have seen in Chapters 8, 13, 19, 21, and 32, most of the increase in complexity occurs during the **pre- embryonic** and **embryonic phases** of prenatal development although changes still occur in many organs and tissues well into post-natal life. Size increase is also rapid prenatally and continues throughout the remainder of the growth period although the growth rate changes. Changes in overall size may occur in mature individuals due to obesity or other pathological conditions but this is not growth.

33.1.2 Growth in overall proportions

Growth in overall size can be studied by examining the changes with age in easily measured parameters such as height and weight. There are two ways in which such data can be presented as shown in Figure 33.1. A **distance curve** is the simplest method illustrated in Figure 33.1A by plotting height against age on a graph. *Changes* in the rate of growth are demonstrated more clearly by plotting the increment in the measurement per unit of time such as the increase in height per year against age; this is a **velocity curve** shown in Figure 33.1B. You can see in Figure 33.1A that height increases more rapidly around the age of 14; the velocity curve in Figure 33.1B makes the rapid growth at this age much clearer.

If distance curves are plotted for different body components, the curves show specific characteristics. The overall growth of the body is accurately indicated by measures of height and weight; these measurements plotted against age produce the **somatic growth curve** shown in Figure 33.2. Growth is rapid in the prenatal and early post-natal period then begins to slow down after about 4 years of age. During the early teens starting about 11 years in girls and 13 years in boys, the gonads undergo maturation during **puberty;** this is followed almost immediately by the **adolescent (**or pubertal**) growth spurt**. During this growth spurt, height and weight increase rapidly for a few years and then slows again as maturity is approached. Bear in mind that growth may not be uniform and may proceed more rapidly in some directions than in others. The **rate, timing**, and **direction** determine the **growth pattern**.

Many other features of the body besides total height and weight follow the somatic growth pattern, including most of the internal viscera, the length of the limbs, and limb segments and their associated muscles and bones. Most significantly for dental students and practitioners, the **facial skeleton** also conforms to the somatic growth curve.

Particular organs or parts of the body have their own independent growth rate and time when growth occurs. An excellent and pertinent example is the **neural growth curve** followed by the brain and spinal cord, together with special sensory structures such as the eyeballs and organs of hearing, which is also shown in Figure 33.2. Observe how neural growth is rapid in prenatal and early post-natal life then declines smoothly and progressively; growth of the CNS and special sense organs is about 90% complete well before puberty around 8 years of age. In the context of skull growth, the **cranial vault** enclosing the brain, the **upper facial skeleton** forming the orbits around the eyeballs, and the **petrous temporal bones** enclosing the middle and inner ear follow the neural growth pattern.

The adolescent growth spurt

Growth hormone or **somatotrophin** secreted by the anterior pituitary gland is one of the principal factors governing the rate of somatic growth in the period before puberty. This hormone acts upon many target organs; in the current context, it is growth sites in the skeleton. Secretion of the sex hormones, **testosterone** and **oestrogen**, increases greatly as the gonads mature during puberty. These hormones are powerful stimulators of general body growth as well as their obvious influence on the development and growth of the primary and secondary sex organs. Their sudden increase at puberty produces the adolescent growth spurt. Sex hormones also bring about the maturation of growth sites and eventually the cessation of growth when they reach certain levels.

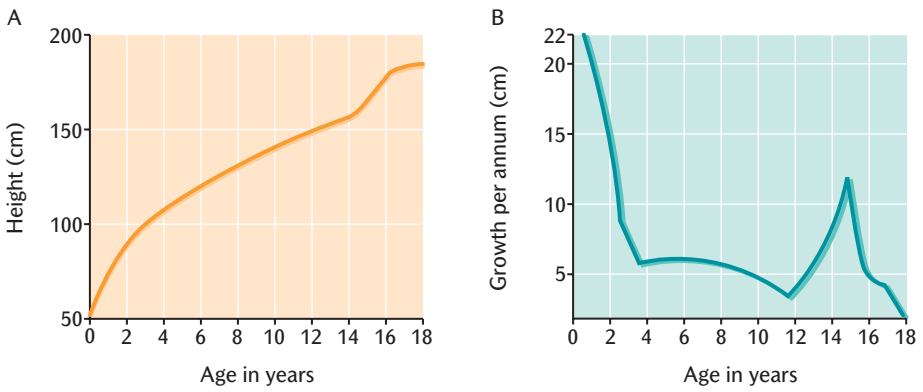


Fig. 33.1 A) Distance curve; B) Velocity curve for the growth of an individual.

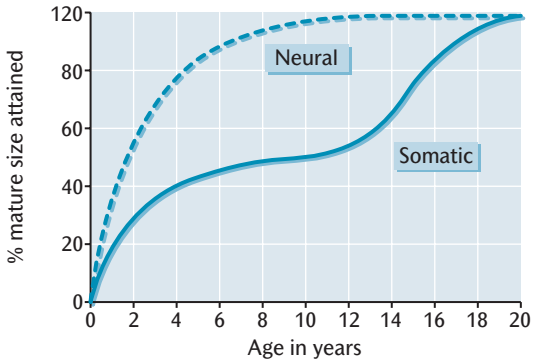


Fig. 33.2 The somatic and neural growth curves.

There is little difference in body size or shape between males and females until puberty although females are usually a little bigger than males since they are physically more mature age for age. During adolescence, this trend is reversed because the adolescent growth spurt is more pronounced and prolonged in males than females because testosterones have a greater anabolic effect than oestrogens. The differential growth effects of the two sex hormones are also responsible for the development of sexual dimorphism in body shape which becomes increasingly marked during adolescence. The sex hormones also have differential effects upon many other features, including hair growth, disposition of body fat, muscular strength, and behavioural characteristics.

Maturation

Individuals of the same chronological age may differ from each other in their rate of growth, size attained, and degree of maturity. More mature children will have progressed further along their distance growth curve and therefore, tend to be taller than those who are less mature. In the early teens, one individual may be well into the adolescent growth spurt whereas a less mature individual of the same age may not yet have entered that phase. Assessment of maturity is outlined in Box 33.1.

Box 33.1 Indices of maturity

Discrepancies between chronological age and maturity are important when assessing growth abnormalities. Retarded growth may be due to disorders of growth mechanisms or the result of delayed maturation. It is, therefore, sometimes necessary to assess maturity separately from growth using one of the **indices of maturity**. The centres of ossification in the eight wrist bones appear develop in a set sequence that can be visualized radiographically; their development is one the indices frequently used. The degree of development and eruption of the teeth is also a measure of maturity although it is not so reliable on its own as the ossification centres in the wrist. If ossification of the wrist bones is delayed, the eruption of teeth also tends to be retarded although not to the same degree.

33.2 Skull growth

33.2.1 Introduction

It is easy to think that the skeleton, including the skull, must be genetically determined to grow to a certain size. We have already seen how different tissues grow at different rates; these tissues can and do have a profound influence on growth and development of adjacent bones. Likewise, the influence of hormones on growth rates at different stages of maturity has been highlighted. It should now be coming apparent that several interrelated factors determine the growth of the skull and jaws. Some factors exert a general effect on the whole skeleton whereas others have a local influence on particular areas of the skeleton or even features of particular bones.

General effects which influence the overall shape and growth rate of bones include:

- Hormonal influences, particularly from growth and sex hormones;
- Nutritional influences; well-nourished people are usually taller and stronger than malnourished individuals;
- Genetic influences; children tend to have similar body build to one of their parents, but these characteristics are multifactorial with several genes involved;
- Socio-economic influences; children from lower socio-economic groups tend to be smaller than those from higher economic groups.

Local effects include:

- The **growth pattern** followed by a particular part of the skull; the facial skeleton follows the somatic growth pattern, the cranial vault follows the neural growth pattern, and the cranial base follows the neural pattern for a certain time, then has its own patterns;
- The effect of enclosed tissues whereby the growth of adjacent bones follows the growth of the tissues they encapsulate. Such tissues are referred to as **capsular matrices**; the eyes and the brain are good examples as you will see in Section 33.4.
- Muscle attachments, joints, adjacent soft tissues, and teeth influence the detailed architecture of bones; these are sometimes referred to as **periosteal matrices** and their specific influence on the skull will be examined when their effects on particular bones are described.

These general and specific factors all determine the growth and development of the skull in various ways.

To briefly recapitulate some aspects of development and growth of the skull outlined in Section 22.2, the skull posterior to the coronal suture is derived from **mesoderm** whereas bones anterior to the suture

are derived from **ectomesenchyme** from the neural crest. There are two types of bone formation and both start with coalescence of mesodermal or ectomesenchymal cells into dense groups, a process known as **condensation**. The condensed cells may differentiate into **chondroblasts** which form a cartilaginous template of the bone; this is **endochondral ossification (ECO)** and the cartilage is eventually replaced by bone. Some cartilages never ossify whereas other unmineralized residues of the initial cartilage template, such as the costal cartilages or xiphisternum, may partially ossify with age or pathologically. **Intramembranous ossification (IMO)** is bone formation directly in the condensation. In common with the whole of the post-cranial skeleton, the bones of the **cranial base** have cartilaginous precursors; the only exceptions are the **clavicles**. The bones of the **cranial vault** and **facial skeleton** are dermal bones which develop through IMO.

Most organs growth occurs by **interstitial growth** characterized by cell division and matrix proliferation throughout their structure. However, the mineralized matrix of bones precludes interstitial growth. Instead, bones grow by addition of new material on to pre-existing surfaces; this is **appositional growth**. Bones developing by ECO do not have the same limitations because it is the cartilage that is growing most actively during this process; cartilage can grow interstitially.

Irrespective of the mechanism of bone formation, the adult size and proportions of a bone are achieved by a combination of surface deposition and resorption, known collectively as **remodelling**. During the growth period, virtually all internal and external bone surfaces undergo deposition or resorption. **Growth sites** are surfaces or cartilages where particularly large amounts of growth take place.

33.2.2 Patterns of skull growth

There are numerous ways of measuring the pattern of skull growth. One method is to take measurements of skulls of different ages to produce **cross-sectional** growth data for a population. Measurements taken on the same child at successive intervals to follow the growth of that individual provide **longitudinal** data. Longitudinal data can be compared to cross-sectional data to determine whether the growth and development of an individual child is within normal limits for the population. Historically, skull growth was studied using lateral skull radiographs which had limitations due to distortion and superimposition of structures obscuring various features and landmarks. With the introduction of imaging methods described in Section 14.2, it has become much easier to obtain excellent three-dimensional images of growing skulls with virtually no distortion and no overlap of structures.

The development, growth, and age changes of the three major divisions of the skull will now be described in turn. The cranial base and facial skeleton are clinically the most important for dental students and practitioners, but the cranial vault illustrates some important principles of craniofacial development and growth. Briefly, the bones of the **cranial vault** are formed by **IMO** and follow the **neural growth pattern**; those of the **facial skeleton** undergo **IMO** and follow the **somatic growth curve**; the bones of the **cranial base** develop from cartilage through **ECO** and conform to the neural growth curve for several years, then adjust to the somatic growth pattern later.

The appearance of the head changes greatly with age. The braincase has completed most of its growth before puberty whereas the facial skeleton does not complete growth until maturity because of the neural and somatic growth rates influencing the cranial vault and facial skeleton, respectively. The height of the cranial vault and orbit is about eight times that of the facial skeleton at birth due to the precocious prenatal growth of the brain and orbits. The cranial vault has reached about 60% of its adult size whereas the facial skeleton is on about 40% of its final size. This ratio reduces to about 6:1 in the second year and is about 5:1 by the fifth year; the face is comparatively small and facial features are undeveloped, giving the characteristic 'childish' appearance. There is an acceleration of growth in the facial skeleton at the onset of the **adolescent growth spurt** without any change in growth of the braincase; the head rapidly attains its adult proportions of 2.5:1 and the facial features become more strongly emphasized. The sex differences in facial appearance become fully established during adolescence; the male and female sex hormones have differential effects on the growth of the facial skeleton, the development of muscle and fat, and the growth of facial hair.

Facial appearance changes less rapidly in adult life. There is usually some increase in the heaviness of the features into middle age, resulting from continued small amounts of apposition growth in the facial skeleton and the accumulation of fat. In old age, the face tends to develop a shrunken appearance, partly from loss of bone from the facial skeleton, but more so from the wasting of the muscles of the face and age changes in skin, hair, and connective tissue. Loss of some or all of the teeth can also alter the aesthetic of the face.

33.3 The cranial base

When we built a skull in Chapter 22, we started with the cranial base. This is how the skull is initiated during embryogenesis; the cartilaginous precursors of the cranial base appear before other elements of the skull. Cartilages begin to appear at about 7 weeks of development in the mesoderm or ectomesenchyme separating the brain above from the foregut below. Several small cartilages form a central stem and other cartilages outline paired lateral structures as shown in Figure 33.3A. The majority of dental courses now only require an outline of the process of development of the cranial base without naming of all the individual components; the names have been added to Figure 33.3 for those who require this information and for completeness.

In Figure 33.3A, identify the anterior end of the **notochord** in the posterior midline and the **infundibulum**, a pouch pushing down from the floor of the brain to form part of the pituitary gland. As shown in Figure 33.3A, two plates, one each side of the notochord, are the first element of the central stem to appear. They soon fuse across the midline, enclosing the front end of the notochord. The cartilage spreads backwards around the neural tube to form the foramen magnum. You should recognize in Figure 33.3C the outline of the basal part of the

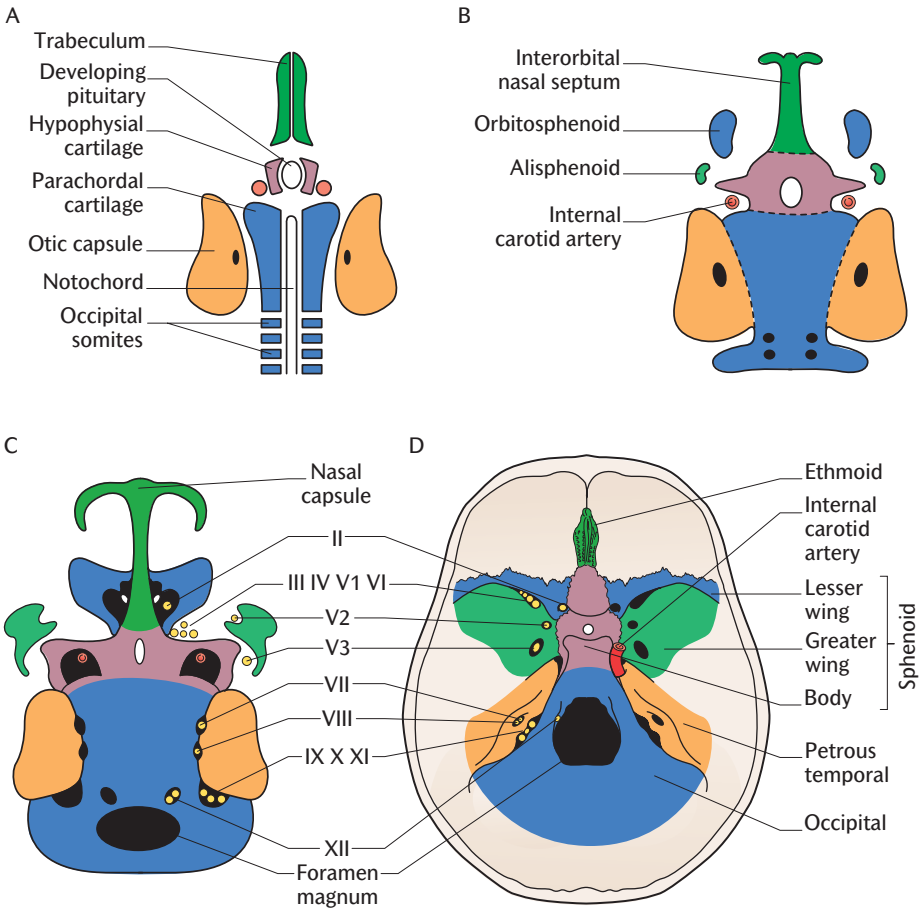


Fig. 33.3 A–C) The development of the main structural elements of the cranial base; D) Their contribution to the adult skull. Roman numerals refer to cranial nerves.

occipital bone although it does not look absolutely identical to the mature bone just yet.

Figure 33.3A also shows a pair of smaller (hypophysial) cartilages on each side of the developing pituitary gland. These soon unite in front of and beneath the developing gland, creating the **pituitary fossa** in the body of the **sphenoid bone**; it meets the **occipital bone** posteriorly at the **spheno-occipital synchondrosis** indicated by the lower horizontal dotted line in Figure 33.3B. This synchondrosis is an important growth site.

During vertebrate evolution, three cartilaginous **sense capsules** originally developed lateral to the line of cartilages, forming the central stem of the cranial base to surround the **nasal, optic**, and **otic** sense organs. In mammals, the optic capsule does not develop into cartilage; it remains as connective tissue and forms the sclera of the eye. The **otic capsule** condenses around the developing middle and inner ear to form the **petrous temporal bone**. Figure 33.3C shows the growing otic capsule meeting the occipital bone posteriorly; the glossopharyngeal, vagus, and accessory nerves occupy the medial part of this junction and the **jugular foramen** is formed at that point.

Two (trabecular) cartilages extend anterior to the developing pituitary to form the future **nasal septum** illustrated in Figure 33.3A. Two nasal capsules develop around the **nasal pits** (Section 32.2.1) to form the **nasal capsules** and their medial walls fuse with the nasal septum in the midline**;** a large central aperture remains in the midline which later becomes the **cribriform plate** of the **ethmoid bone** for passage to the olfactory nerves.

The next stage of development is shown in Figure 33.3B. Two bilaterally paired cartilages develop in the region between the otic and nasal capsules (the orbitosphenoid anteriorly and the alisphenoid posteriorly). Each anterior cartilage becomes the lesser **wing of the sphenoid**, leaving the optic canal for the passage of the optic nerve as it unites with the body of the sphenoid. The posterior cartilage becomes the **greater wing of the sphenoid**. As this grows posteriorly, it encloses the maxillary and mandibular divisions of the trigeminal nerve in the **foramen rotundum** and **foramen ovale**, respectively. The **superior orbital fissure** is formed between the lesser and greater wings of the sphenoid; the oculomotor, trochlear and abducens nerves, and the ophthalmic division of the trigeminal nerve pass through the fissure into each orbit. The **internal carotid artery** is enclosed in the carotid canal where the greater wing joins with the body of the sphenoid and the tip of the petrous temporal bone. The cranial nerves and blood vessels supplying the brain pass to and from the cranial cavity through foramina between the central stem and the lateral structures; the foramina are formed between the developing components as they grow and fuse with each other as you can see in Figure 33.3C.

The chondrocranium is fully differentiated by the end of the second month of pregnancy, only 2 weeks since the initiation of cartilage formation. Mineralization of the chondrocranium takes place at various

centres of ossification that appear in the cartilages of the cranial base and sense capsules; some form before the cranial base is completely mapped out. Figure 33.3D shows the appearance of the cranial base in the mature skull on to which the braincase and facial skeleton will be added. As we already know from earlier chapters, the cranial base comprises the ethmoid, sphenoid, temporal, and occipital bones. Note that the greater wings of the sphenoid have contributions from dermal bones forming the braincase and the pterygoid processes are dermal bones formed as part of the facial skeleton; irrespective of their origins, the seven components fuse together to form one complete sphenoid bone. The squamous parts of the temporal and occipital bones are also dermal bones.

The nasal capsules grow enormously in the early fetal period; their length doubles between 10 and 14 weeks as fusion of the facial processes occurs and grows to six times its original length by birth. The more posterior bones grow less quickly. The rapid growth of the nasal capsules is responsible for the change in the shape of the head in the late fetal period (see Section 33.3.2).

33.3.1 Synchondroses

As described in the previous section and illustrated in Figure 33.3, the growing cartilages of the cranial fuse with each other in various locations to map out the complex bones of the cranial base. The cartilaginous joints between these bones formed by remnants of this cartilage are called **synchondroses**. A typical **synchondrosis** is shown diagrammatically in Figure 33.4. A central progenitor zone containing undifferentiated cells is flanked on either side by **proliferative zones** in which the chondrocytes differentiate and divide. As the cells divide, they form into columns and deposit cartilage matrix in between. The cells enlarge as they move further along these columns in the **hypertrophic zone** until they degenerate, leaving spaces within the cartilage matrix. These spaces are invaded by osteoblasts that deposit bone which is subsequently remodelled into compact and cancellous bone. The cartilage is the growth site within synchondroses. Once these joints are established, the rate of growth is matched by the rate of bone formation so the synchondrosis maintains a constant thickness; the growth rate is not always the same on both sides of a synchondrosis.

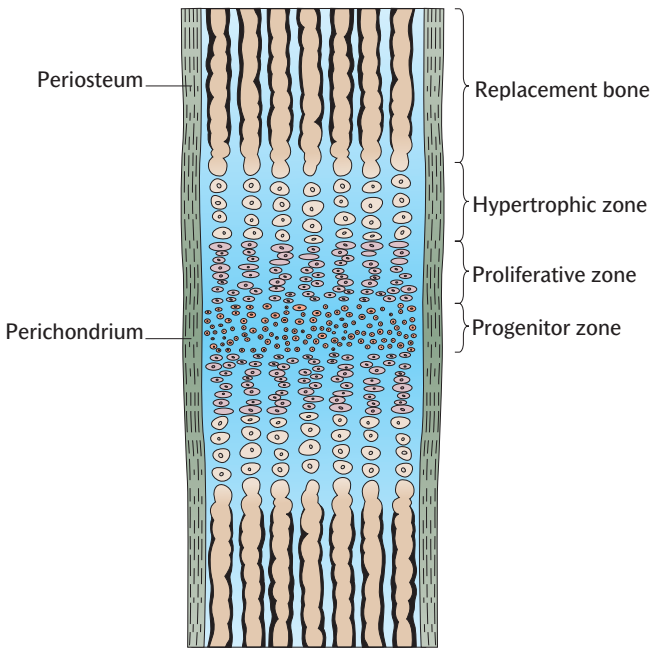


Fig. 33.4 Cross section through a synchondrosis of the cranial base.

The spheno-occipital and spheno-ethmoidal synchondroses are the two major synchondroses in the midline**;** considerable quantities of bone are formed at these active growth sites. The **spheno-ethmoidal synchondrosis** is an active growth site prenatally, but the cartilage is replaced by fibrous tissue shortly after birth where growth continues after its conversion to a suture (see Section 33.4.1). Ossification at the sphenoethmoidal synchondrosis contributes to the enlargement of the cranial base anterior to the pituitary fossa.

The **spheno-occipital synchondrosis** is the principal site of elongation of the basicranial axis and growth continues until the early teens. Elongation of the cranial base allows room for growth in length of the maxillae which in turn is vital to allow room for the second and third permanent molar teeth to erupt. The spheno-occipital synchondrosis ceases activity around the age of 12 in females and a couple of years later in males. The joint is grown over, starting on the intracranial surface and is completely obliterated at maturity; no joint is visible between the sphenoid and occipital bones after this age and is absent from virtually all dried skulls. A gap between the sphenoid and occipital bones in juvenile skulls indicates the presence of the spheno-occipital synchondrosis and can be of use in forensic dating of skeletal remains.

33.3.2 The cranial base and position of the facial skeleton

The cranial base is a key region in the growth of the skull. It forms the junction between the braincase and facial skeleton and the upper facial skeleton and mandible have separate attachments to it. In growth studies, the cranial base is divided along the midline into two zones which are illustrated in Figure 33.5. These are the **basicranial axis (BA)** stretching from the anterior border of the foramen magnum (the endobasion) to the pituitary fossa and the **anterior extension (AE)** stretching from the pituitary fossa to the **nasion,** the junction of the internasal and frontonasal sutures on the bridge of the nose. Growth changes in the cranial base are then represented by the changes that take place in the anteroposterior dimensions of the BA and AE. The **spheno-ethmoidal angle** (**SEA**) between the parts is measured on the *underside* of the cranial base as shown in Figure 33.5. Measurement of these components only delivers information about the midline and clearly does not take into account the lateral regions. Historically, growth data for the cranial base were collected from midline structures because they could be identified unambiguously in lateral head radiographs. With the advent of imaging techniques, data are now being collected that incorporate growth of the lateral structures, but BA, AE, and SEA angle measurements are still important.

From your examination of the skull in earlier chapters and Figure 33.5, you can see that the facial skeleton is attached below the AE segment of the cranial base whereas the BA segment is related to the cranial vault above and the pharynx below. The BA component tends to follow

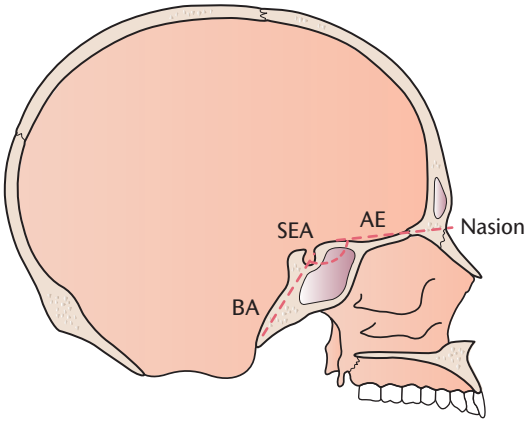


Fig. 33.5 The subdivisions of the cranial base into the basicranial axis (BA) and anterior extension (AE). The spheno-ethmoidal angle (SEA) is also shown.

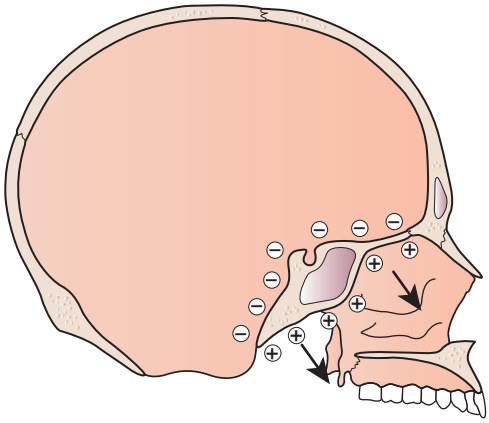


Fig. 33.6 The influence of cranial base growth on position of the facial skeleton. + = bone deposition; – = bone resorption; arrows indicate direction of growth.

the neural growth rate followed by the cranial vault. The anteroposterior dimensions of the cranial base appear to continue to elongate throughout the growth period and show a well-marked pubertal growth spurt, suggesting the somatic growth pattern. Careful measurement of subsections of the cranial base show that the BA and AE as far as the anterior edge of the **cribriform plate** closely follow the neural pattern and cease growth by about 7 years of age. In contrast, the AE from the anterior of the cribriform plate to the nasion follows the somatic pattern; there are significant changes in the thickness of the frontal bone anterior to the cribriform plate in the adolescent growth period, caused mainly by growth of the frontal air sinuses (see Section 33.4.2).

As outlined earlier, the facial skeleton lacks height at birth. The increase in height seen over the next 2 years is due to the eruption of the primary dentition and development of the alveolar processes of the maxilla and mandible (see Section 33.5). There have to be changes to maintain occlusion as the teeth erupt and many of these occur in the mandible. It has been suggested that growth changes in the angulation of the cranial base are required to maintain the relationship between the upper and lower jaws since the upper facial skeleton is attached to the anterior part of the cranial base while the mandible articulates more posteriorly. It is now accepted that there are no significant changes in the SEA angle during growth. Instead, remodelling at the periosteal surface of the bones of the cranial base is now known to play a major part in localized growth in the basicranium in the post-natal period to aid in the constant realignment of the upper and lower jaws during tooth eruption and craniofacial growth.

Bone deposition takes place over much of the extracranial surface of the base with resorption over most of the intracranial surface as illustrated in Figure 33.6. As shown by the arrows, the cranial base grows and moves anteriorly and inferiorly, carrying the facial skeleton with it. Bone deposition still takes place in localized areas of the intracranial surface, particularly the superior surfaces of the petrous temporal bones and the walls of the pituitary fossa which become increasingly prominent as growth proceeds. Deposition of bone around the anterior margin of the foramen magnum with corresponding resorption on the posterior margin also contributes to elongation of the cranial base.

The midline structures of the cranial base are relatively stable and, apart from the rapid growth of the nasal capsule, show a fairly constant growth rate until it ceases around the age of 7 or so. In comparison, the lateral parts of the cranial base forming the floors of the cranial fossae expand rapidly to accommodate the expansion of the brain until the same age. There are subsequent changes in their dimensions to adjust to growth in the cranial vault and facial skeleton.

Genetic anomalies can affect bones developing by ECO, including the cranial base; their effects are outlined in Box 33.2.

Box 33.2 Achondroplasia

Achondroplasia, a dominant genetic anomaly, has the effect its name suggests—literally, a = no, chondro = cartilage, plasia = growth—but in reality, cartilaginous growth is usually reduced rather than inhibited completely. All bones that develop by ECO are affected so stature is shorter and limb length is reduced. In the skull, the length of the cranial base is reduced significantly, but the size of cranial vault and facial skeleton are unaffected because their bones develop by IMO. However, lack of growth of the cranial base does influence the *position* of the facial skeleton; it does not move anteriorly as it should. The middle third of the face appears sunken in between a bossed forehead and a protruding mandible although these are of normal size; the resulting profile is usually referred to as 'dished face'. The backward positioning of the upper jaw in relation to the mandible means that the patient usually has a marked **Class III occlusion**. Similar 'dished face' effects may also be seen in **Down syndrome**.

33.4 The cranial vault

The vault of the skull is formed by dermal bones which develop by IMO. The growth of the cranial vault follows the neural growth pattern of the enclosed brain shown in Figure 33.2. Between birth and adulthood, the brain increases from about 400 cm^3 to about 1500 cm^3. The vault enlarges rapidly in prenatal life and growth continues at a rapid rate in the first few years of post-natal life. Head circumference is an accurate indicator of brain growth; it is about 33 cm at birth, 45 cm by the age of 1, but slows to 50 cm by the age of 3. The brain and cranial cavity attain about 90% of their adult size by the age of 8.

33.4.1 Development of the cranial vault

Condensations occur in the connective tissue surrounding the dorsal aspect of the developing brain which is derived from two sources. Bones that form anterior to the coronal suture between the frontal and parietal bones are derived from neural crest tissue and those developing posteriorly form within mesoderm. The condensed cells differentiate into osteoblasts which become **centres of ossification**. Bone formation begins during the seventh and eighth week post-fertilization and extends from specific centres to form the **frontal** and **parietal** bones and the **squamous occipital** bone. The frontal bones begin their development as two separate bones separated by the **frontal suture** which fuses and becomes obliterated by about 8 years. The **squamous temporal** bone develops initially in the maxillary process and becomes added to the lateral wall of the vault at a later stage (see Section 33.5.1).

Once IMO has been initiated at the centres of ossification, each bone expands within the fibrous membrane by radial **appositional growth**. Eventually, the bones meet each other but do not fuse; they remain separated from each other by fibrous joints or **sutures**. These are essential growth sites in the immature skull. A typical suture has five distinct layers which can be seen in Figure 33.7. A **middle zone** of fibrous tissue separates the two fibrous **capsular zones** that blend with the endosteal and periosteal layers enclosing the bones. A **cambial zone** containing osteogenic cells lines each capsular layer and covers each of the bone surfaces forming the suture. New bone is added to the sutural margins of the bones by the activity of the osteoblasts differentiating from the cambial zones, thus increasing the area of the bone.

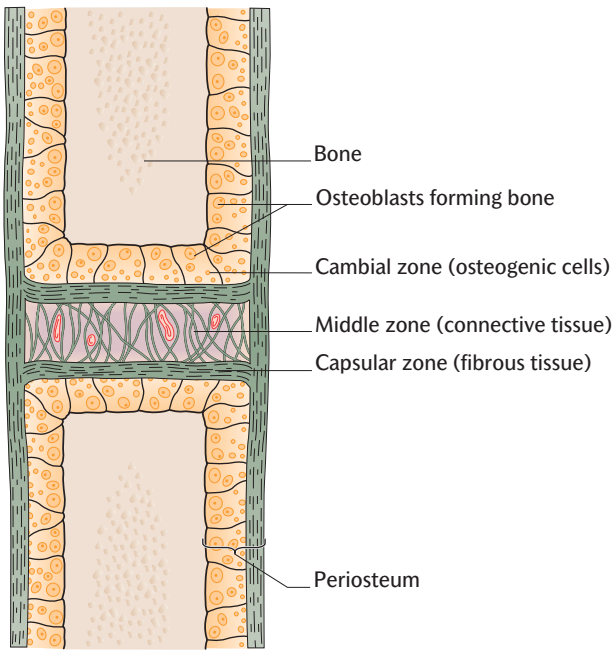


Fig. 33.7 Cross section through a suture.

In theory, growth at the margin of each bone contributing to the suture can be regulated independently in rate and timing because they are separated by the inactive tissue of the capsular and central zones. In practice, growth of adjacent bones is carefully regulated and coordinated. Tension is exerted on the sutures by the expanding brain which stimulates secretion of FGF_2 (fibroblastic growth factor 2) from mesenchymal cells. FGF_2 acts on two receptor types; osteoprogenitor cells differentiate into osteoblasts if it acts on FGF_{R1} whereas osteoprogenitor cells proliferate to enlarge the pool of cells if it acts through FGF_{R2}.

Figure 33.8 illustrates how the sutures in the cranial vault are disposed in three orientations for growth in all three dimensions:

1. In *width* by growth at the sagittally orientated **sagittal** and **frontal sutures**;
2. In *length* by addition of bone at the transversely disposed **coronal** and **lambdoid sutures**;
3. In *height* by bone deposition at horizontal sutures on the lateral aspect of the cranial vault.

Large membrane-filled deficiencies called **fontanelles** form where several sutures meet between the developing bones. Their sites are indicated in Figure 33.8. As bone growth proceeds, the fontanelles are reduced in size and eventually 'close' although the contributory sutures are still active growth sites for several years. The large **anterior fontanelle** between the sagittal, coronal, and frontal sutures closes in the second post-natal year. The **posterior fontanelle** between the coronal and lambdoid sutures closes around the second month and the **anterolateral fontanelles** between the frontal, parietal, and greater wing of the sphenoid grow over at about 3 months. The **posterolateral fontanelles** between the parietal, occipital, and squamous temporal bones close around the end of the first year. The relatively wide sutures present at birth coupled with the presence of fontanelles enables the bones of the cranial vault to override each other to some extent during childbirth, narrowing the circumference of the head as it passes through the vagina. The head of a newborn baby often looks deformed until the bones return to their normal positions within a few days.

33.4.2 Post-natal growth of the cranial vault

During the rapid post-natal enlargement of the cranial vault in response to the expansion of the underlying brain, the curvature of individual bones must decrease to accommodate it. The bones of the cranial vault are covered on their extracranial and intracranial surfaces by periosteum in common with the rest of the skeleton. Periosteum contains

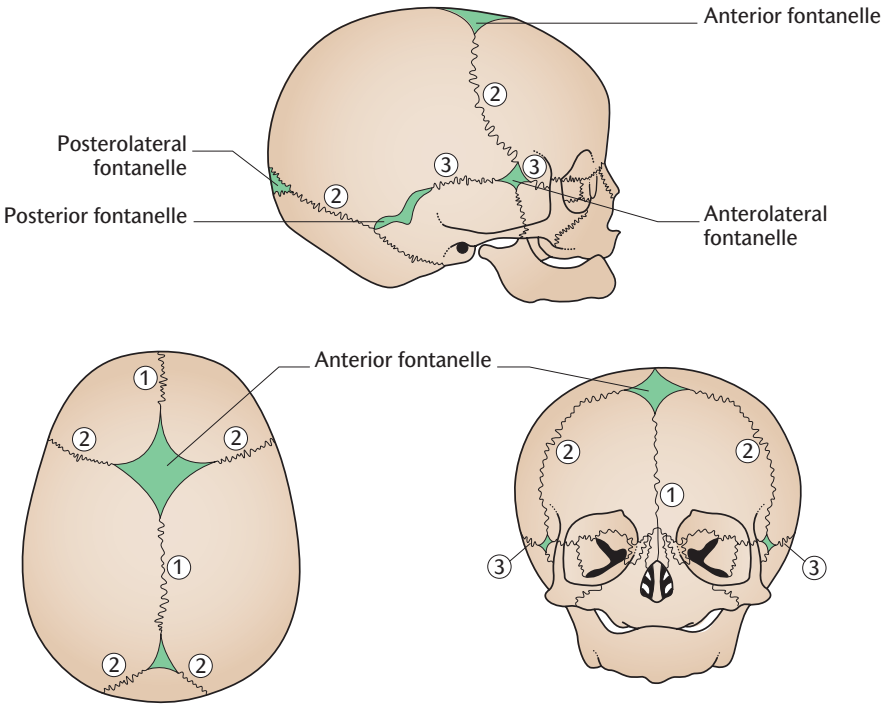


Fig. 33.8 Sutures and fontanelles in the neonatal cranial vault. 1 = sutures producing growth in width; 2 = sutures producing growth in length; 3 = sutures producing growth in height.

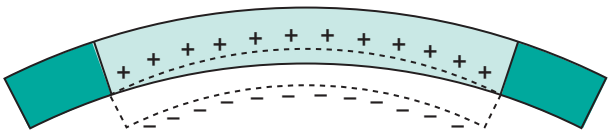


Fig. 33.9 Remodelling of the bones of the cranial vault. Sections of a vault bone at younger and older growth stages superimposed. + = bone deposition; – = bone resorption; bone added at sutures is shown in dark shading.

undifferentiated cells which can differentiate into osteoblasts for the deposition of bone; osteoclasts are recruited from blood monocytes for bone resorption. Curvature changes are achieved by remodelling on the extracranial and intracranial surfaces illustrated in Figure 33.9. The brain and cranial vault have achieved about three quarters of their growth by the age of 2, establishing the overall shape of the cranial vault in early infancy. Subsequent enlargement of the vault appears to take place in a largely concentric manner if individual skull outlines are superimposed on the anterior cranial base at progressive growth stages.

Most of the enlargement of the cranial vault is completed during the first 8 years so growth at sutures and remodelling are active until then. Growth after the age of 8 is restricted to some thickening of the cranial bones and enlargement of the superciliary arches and of the various muscle markings on the vault. As growth slows and ceases under the influence of the brain and sex hormones, the sutures subsequently develop interlocking fingers of bone which may be seen on mature skulls. The interdigitation of bones makes the cranial vault very strong. If you have access to dried skulls, you may have difficulty locating some of the sutures; the probability is that the skulls were obtained from elderly people and later in life, the sutures are obliterated as they are united by bone. Obliteration usually begins internally at the junction of the coronal and sagittal sutures and progresses to the external surfaces about 10 years later. The actual ages at which the sutures are obliterated vary; the sagittal is the first to disappear followed by the coronal and finally the lambdoid suture.

Various abnormalities of the cranial vault can occur during its growth; they are outlined in Box 33.3.

33.5 The facial skeleton

The facial skeleton is made up of dermal bones, except for part of the nasal region that develops from the nasal capsule. Cartilages of the **first pharyngeal arch** (mandibular) provide a major component of the facial skeleton for a brief period of embryonic and early fetal life. The cartilage of the second pharyngeal arch is also involved to a small degree in skull development. The contribution of the first and second arch cartilages to skull development have been described in Section 21.5.2 and are illustrated in Figure 21.5.

As illustrated in Figure 33.10, an arch of dermal bones is laid down in the ectomesenchyme of each maxillary process. These are, from front to back, the **maxilla, zygoma**, and **squamous temporal bone**. The squamous temporal bones forming part of the lateral aspect of the cranial vault fuse with the petrous temporal bones of the cranial base. The **lacrimal** and **nasal** bones are laid down on the superior aspect of each maxillary process and the **palatine bones** and **vomer** develop on the deep aspect of each maxillary process. In addition, ossification centres

Box 33.3 Abnormalities of the cranial vault

Hydrocephaly

The influence of the brain on the development and growth of the cranial vault is dramatically illustrated in circumstances where brain development is abnormal. **Hydrocephaly** is due to either excess production of cerebrospinal fluid prenatally or impairment of circulation of CSF through the ventricles, usually at the cerebral aqueduct (see Section 15.4.2). The forebrain is distorted as CSF accumulates in the lateral ventricles which, in turn, distort the cranial vault. The vault is expanded massively and overhangs the cranial base. The eyes are often downturned because the orbital roof as part of the cranial vault has grown whereas the floor, which is part of the facial skeleton, has not; this can put torsion on the optic nerves with consequent visual problems. The effects on the brain are dependent on the time at which hydrocephaly begins. If the connections between different parts of the brain have developed, intellectual function is largely unaffected despite the distortion of the brain; if it occurs before connections are established, intellectual impairment may be very severe. Hydrocephaly can be treated by placing a shunt into the lateral ventricles through the anterior fontanelle and corpus callosum and draining the excess CSF into the venous drainage. Alternatively, some of the choroid plexus may be cauterized via the same route to reduce the formation of CSF to normal levels.

Cranial synostosis

Cranial synostosis occurs when one or more of the sutures in the cranial vault fuse prematurely. Around 70% of cases of cranial synostosis are non-syndromic and may be due to prenatal malposition or mechanical restraint on the head during interuterine development. The other 30% are syndromic; cranial synostosis appears alongside other congenital malformations. Many syndromic cases are now known to be due to point mutations in the FGF_{R2} receptor which render it inactive. All activity stimulated by FGF_2 is, therefore, directed through the FGF_{R1} receptor; progenitor cells do not proliferate, but differentiate into osteoblasts which lay down bone, producing premature fusion of sutures. **Apert's syndrome** is one example and accounts for about 5% of cranial synostoses. The coronal suture fuses prematurely, thus preventing elongation of the skull and the cranial vault becomes distorted. Children with Apert's syndrome also have fused fingers and toes (syndactyly) as well as skull distortion.

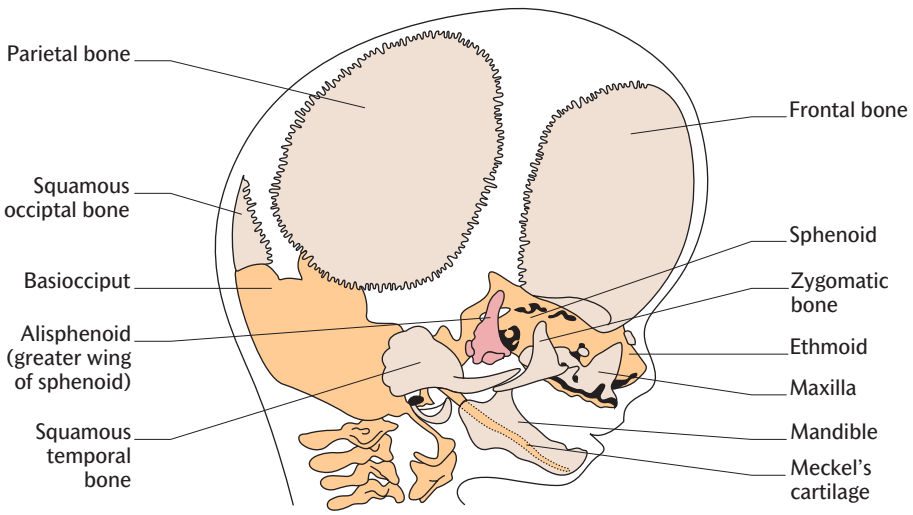


Fig. 33.10 Development of the cranial vault in a fetus aged approximately 4 months. Cream = dermal bones; dark yellow = cartilage and cartilage-replacing bone; pink = alisphenoid.

arise posteriorly for the **greater wings** and **pterygoid plates of the sphenoid**; the greater wings also have a cartilaginous component from the cranial base.

33.5.1 The maxilla and upper facial skeleton

In other mammals, two ossification centres give rise to separate maxillary and premaxillary bones; these remain as individual bones separated by a suture. It has been claimed that two independent ossification centres for the maxilla and the premaxilla arise in humans before the developing bones fuse to form the maxilla. However, it is now generally accepted that there is no separate premaxillary ossification centre.

Each maxilla ossification centre appears in the maxillary process in the sixth week post-fertilization. It is located adjacent to the lateral wall of the nasal capsule just below the point where anterior superior alveolar nerve branches off the infraorbital nerve above the tooth germ of the primary canine. Ossification spreads backwards below the orbit and forwards towards the midline to produce a curved strip of bone lateral

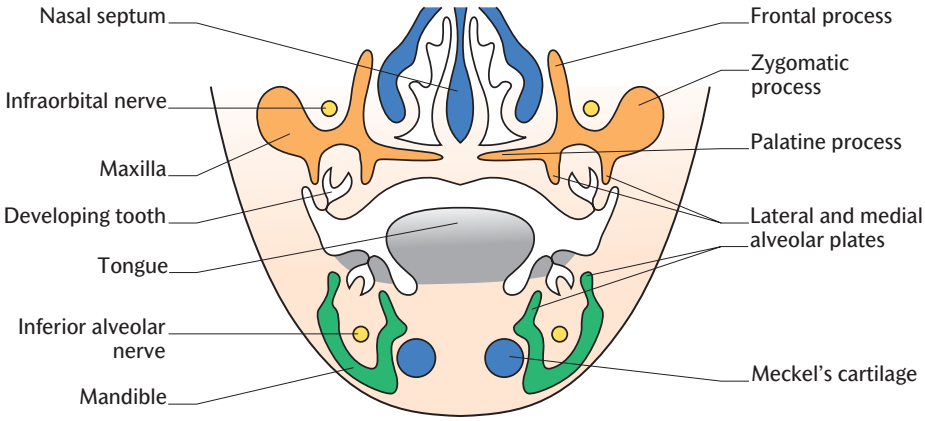


Fig. 33.11 Development of the maxillae and mandible in cross section.

to the developing tooth buds. Several extensions develop from the main mass of bone to form the various maxillary processes as shown in Figure 33.11. An upwardly directed extension forms the **frontal process** and ossification extends posteriorly to form the **zygomatic process**. After palatal fusion is completed at around 12 weeks, ossification extends medially into the palate, forming the **palatine process**. Bone extensions grow inferiorly on either side of the developing tooth germs and later fuse to enclose each tooth germ in a bony crypt.

The growth sites in the upper facial skeleton include the primary cartilage of the nasal capsule and the sutures of the upper facial skeleton and extensive remodelling of the periosteal and endosteal surfaces of the bones.

The sutures of the upper facial skeleton are identical in structure and mode of growth to those of the cranial vault. They are also orientated to promote growth in all three dimensions. On a dried skull and in Figure 33.12, you can see that sutures such as the internasal, intermaxillary, and median palatal sutures are aligned sagittally in the midline (2), promoting growth in width. All the other sutures in the facial skeleton are obliquely positioned (1) so growth at these sites contributes to increase in both the length and height of the upper face.

The **ethmoid** is the principal bone to form in the cartilaginous nasal capsules, originally components of the crania base. The ethmoid labyrinths begin to develop in the lateral walls of the nasal capsules from about the middle of prenatal life. The cartilage of the nasal septum does not begin to produce the bone of the perpendicular plate until the first post-natal year. Interstitial growth of the septal cartilage is important for facial growth in the second two trimesters of pregnancy. Despite this rapid growth, the nasal cavities are almost entirely between the orbits at birth. The **vomer** ossifies from centres which develop close to the lower edge of the septal cartilage, but for the first few years of life the perpendicular plate of the ethmoid and the vomer remain separated by septal cartilage which is a growth site. Growth at this site increases facial height, moving the hard palate downwards relative to the cranial base. Interstitial growth of the septal cartilage and later ECO at the ethmoid–vomer junction exert tension on the obliquely orientated facial sutures, promoting growth at these sites.

The major structures that influence the growth of the maxilla and other upper facial bones are:

- The orbit and eyeballs;
- The developing dentitions;
- The maxillary sinuses.

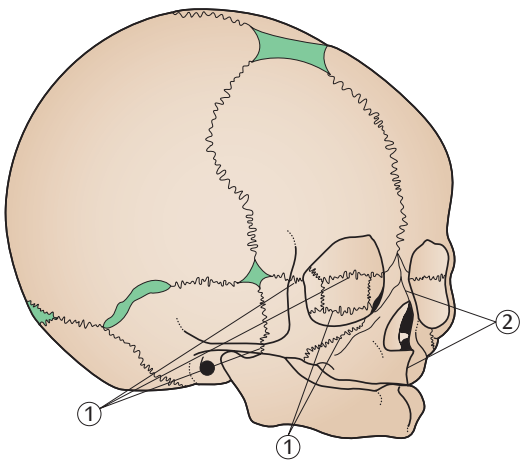


Fig. 33.12 Sutures in the facial skeleton of the newborn. 1 = some of the sutures producing growth in length and height; 2 = some of the sutures producing growth in width.

The orbital region completes its growth earlier than the lower face because it follows the neural growth pattern of the eyeballs. All babies have large appealing eyes because of their rapid prenatal growth on the neural growth curve. This continues after birth and parallels the growth of the cranial vault described in Section 33.4.2; the orbits achieve about 50% of their post-natal growth by the age of 2 and adult size around 7 years. Most of the increase in facial height taking place after infancy is in the suborbital compartment where growth in height is most obvious; there is also some growth in length and width although the face hardly protrudes beyond the forehead region of the braincase.

The **primary dentition** erupts into the oral cavity between the ages of 6 months and 2 years. Tooth eruption is the bodily displacement of the crowns of the teeth from their developmental site within the jaw bones into the mouth. As teeth erupt, their roots develop and the alveolar processes mould around them to form the tooth sockets. Just adding the height of the crowns of the primary teeth is going to increase suborbital facial height by 6 mm or so and the formation of alveolar bone increases this dimension further. The alveolar process fails to develop if teeth fail to form for any reason.

Endochondral growth in the septal cartilage allows some room for the eruption of the primary dentition together with adjustments in the growth of the mandible described in Section 33.5.3. The primary teeth are replaced by the **secondary dentition** between the ages of 6 to 12. However, there are three additional teeth in each quadrant in the permanent dentition. The first permanent molars erupt about the age of 6 years so there must be growth in the length of the maxilla to accommodate these teeth. The second permanent molars erupt at about 12 years of age which corresponds with the **adolescent growth spurt**; there is growth in all dimensions at this stage. The third molar usually erupts around maturity when the final growth increments of the somatic growth spurt hopefully allow room for this tooth.

Growth in length exhibits a gradient within the facial skeleton as you can see on Figure 33.13. It is least in the orbital region as growth in this area is completed early, more in the suborbital part of the upper face to accommodate extra teeth and maximal in the mandible for the same reasons. The lower parts of the face thus become progressively more prominent as growth proceeds.

The maxillary sinuses develop as outgrowths of the nasal mucosa of the middle meatus into the adjacent body of the maxilla about 5 months post-fertilization. By birth, the maxilla has all the processes of the adult bone, except the alveolar processes. The body of the bone lacks height due to the absence of the alveolar process because the primary teeth have not erupted and the small size of the **maxillary air sinus** which is about the size of a small pea at this stage.

The maxillary sinus enlarges post-natally by resorption of bone internally and deposition of bone on the external surfaces of the maxillary body at the same time. At puberty, the sinus is longer than it is high and is about the size of an almond in its shell. The sinus grows rapidly like everything else following the somatic growth pattern during the adolescent growth spurt. Growth is greater in height than length and the mature sinus is the size of a walnut in its shell.

Figure 33.13 illustrates the combined effect of growth in height and length; the lower part of the facial skeleton is transposed downwards and forwards relative to the braincase. Anterior points on the facial skeleton such as the anterior nasal spine and point of the chin tend to move along fairly constant anteroinferior pathways with respect to the cranial base whereas posterior points such as the posterior edge of the hard palate and angle of the mandible grow along a pathway that is more directly downwards.

As illustrated in Figure 33.14, the maxillary alveolar arch is U-shaped with the open end facing posteriorly when viewed from below. The combination of deposition on the inner aspect and tuberosities of the U and resorption from its outer aspect results in the whole region appearing to grow in a posterior direction. The tuberosities abut on to the pterygoid processes of the sphenoid which act as fixed buttresses and are sites of especially rapid bone deposition. Bone deposition on the tuberosities, therefore, results in the maxillary complex being translocated forwards. Deposition posteriorly exceeds resorption anteriorly so there is growth as well as translocation.

As shown in Figure 33.15, the maxillary alveolar processes and palate form a shallow concave arch in coronal section. The upper aspect of the bones in the floor of the nasal cavity and maxillary sinuses and the buccal surfaces of the alveolar processes are resorbed while bone is deposited on the oral surface of the palate. The growth movement

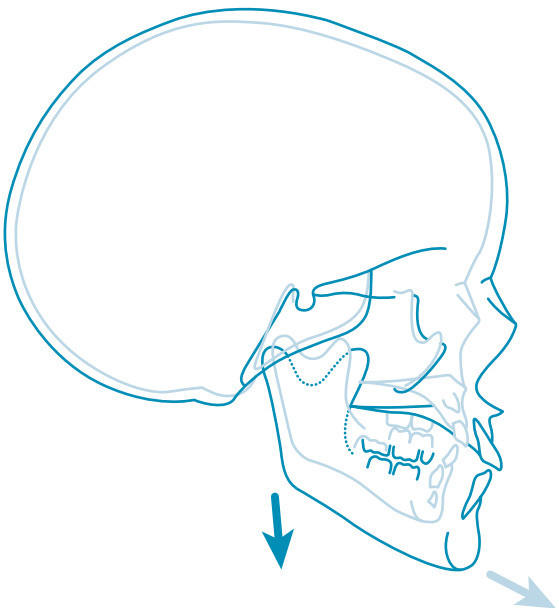


Fig. 33.13 Superimposition of radiographic outlines of the heads of a 6-year-old (thin lines) and a 16-year-old (thick lines) on to the anterior extension of cranial base to show relative growth of the facial skeleton. Note that the anterior facial skeleton moves downwards and forwards (green arrow) whereas the posterior portion moves downwards (blue arrow).

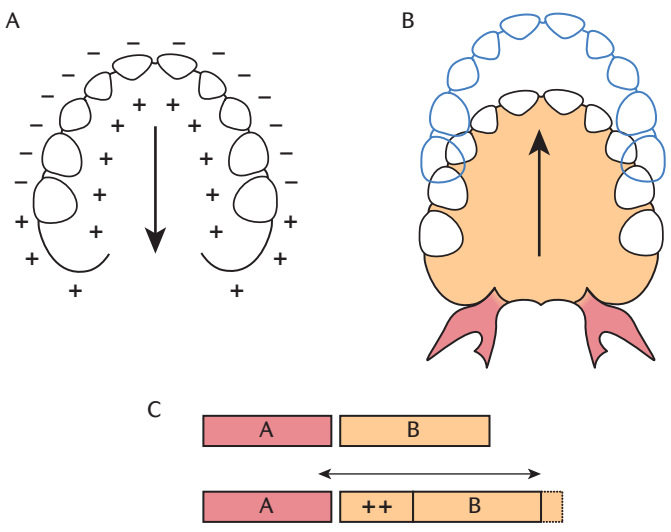


Fig. 33.14 Growth of the maxillary alveolar arch in occlusal view. + = bone deposition; – = bone resorption. A) Direction of apparent growth indicated by the arrow; B) The direction of actual growth indicated by the arrow; C) Diagrammatic representation of the growth movement—bone is added to the posterior surface of the maxilla (yellow) at its suture with the pterygoid plates (pink) and removed in lesser amount from the anterior surface of the maxilla.

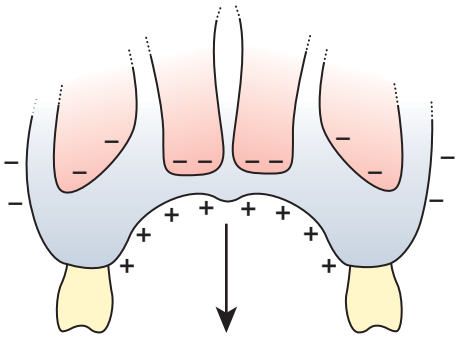


Fig. 33.15 A coronal section through the premolar region showing growth of the maxillary alveolar arch and lower nasal cavity. + = bone deposition; – = bone resorption; arrow indicates the direction of actual growth.

of the maxillary arch in the vertical plane is, therefore, downwards and adds to downward growth produced by bone deposition at the obliquely orientated facial sutures.

Development of other facial bones is described Box 33.4 for those who require this information.

33.5.2 The mandible

By 6 weeks of development, **Meckel's cartilage** forms a rod of cartilage stretching from the otic capsule to the midline in each first pharyngeal arch. The anterior ends of the two cartilages are united by a bridge of ectomesenchyme. The mandible is a dermal bone which develops in close association with, but *not* from Meckel's cartilage.

Figure 33.16 shows the ossification centre in the V of the mental and incisive nerves of each half of the mandible; they appear at 7 weeks. Ossification spreads anteriorly and posteriorly on the lateral aspect of the nerves and developing mandibular teeth then, as you can see in Figure 33.11, extends beneath these structures and spreads upwards on their medial side to form a U shape enclosing the teeth and nerves. The U is later roofed from front to back above the nerves to form the **mandibular canal**. The **body of the mandible** on each side is thus produced from a single centre of ossification and runs from the anterior midline symphysis to the area which will become the mandibular foramen. As the body of the mandible grows, it contacts Meckel's cartilage and this pressure causes resorption of the cartilage.

The **ramus** is formed by the tenth week by rapid backward spread of ossification; rudimentary backward pointing coronoid and condylar processes are visible. The part of Meckel's cartilage lying medial to the ramus is resorbed, but its perichondrium remains as the **sphenomandibular ligament**.

Surprisingly, cartilages appear in the mandible which should not happen in a dermal bone. These are called **secondary cartilages** because they are not related to **primary cartilages** formed in the pharyngeal arches. Cartilage precedes bone in primary cartilages whereas bone precedes cartilage in secondary cartilages. The secondary cartilages are not derived from undifferentiated mesenchymal or neural crest cells, but from cell lines which have already differentiated to produce dermal bone. They are growth sites producing bone by ECO.

There are three secondary cartilages in each half of the mandible. Two cartilages appear in the ramus, one in the condylar and the other in the coronoid process, and the third at the midline junction of the two halves of the mandible.

The **condylar cartilage** is the largest and most important of the secondary cartilages; it is one of the main sites of bone deposition in the mandible and is active throughout the growth period until maturity. It first appears at about 12 weeks on the superior and lateral aspects of the condylar process. It undergoes considerable enlargement and forms a conical mass in the condylar process, extending as far as the mandibular foramen within a few weeks. As the cartilage grows, it is replaced inferiorly by bone; by 5 months, much of the original cartilage has ossified, leaving only a narrow zone of growth cartilage persisting beneath the articular surface of the condylar head of the mandible. The **coronoid cartilage** also appears at about 12 weeks as a cartilaginous strip on the anterior border of the summit of the coronoid process, but ceases activity and disappears within a matter of weeks. The **symphyseal cartilages** are small nodules of secondary cartilage which develop in the midline symphysis. The mandible at birth is still separated into right and left halves by the **mental symphysis** which fuses at about the end of the first post-natal year as the cartilages ossify.

Box 33.4 Further development of other upper facial bones

The **palatine** bone develops adjacent to the cartilaginous nasal capsule in the seventh or eighth week. Ossification extends upwards to form the **perpendicular plate** and horizontally to form the **horizontal plate**, but the latter does not occur until palatal fusion is completed. The palatine bone meets the medial surface of the maxilla to form a major part of the lateral nasal wall as the nasal capsule regresses locally.

The **vomer** develops from two centres which appear during the ninth week in membrane close to the inferior margin of the septal cartilage. These unite beneath the cartilage to form a bony trough and extend backwards to meet the body of the sphenoid. A vertical plate develops on the underside of this trough and descends towards the hard palate.

The **lacrimal** bone develops in the third month from an intramembranous centre adjacent to the nasal capsule. With regression of the nasal capsule, it forms a small part of the lateral wall of the nose and of the medial wall of the orbit.

The **nasal** bone develops in the second month by IMO on the anterior roof of the nasal capsule.

The **zygomatic** bone ossifies in the second month from an intramembranous centre lateral to the eyeball. It soon makes contact with the temporal bone and maxilla and later the frontal bone.

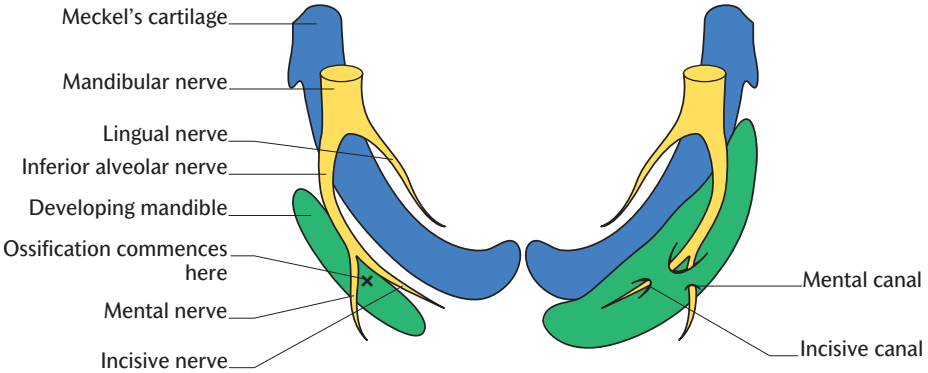


Fig. 33.16 Development of the mandible as seen from above. The beginning of ossification on the right side and the left side is about 2 weeks later. Green = dermal bone; blue = cartilage.

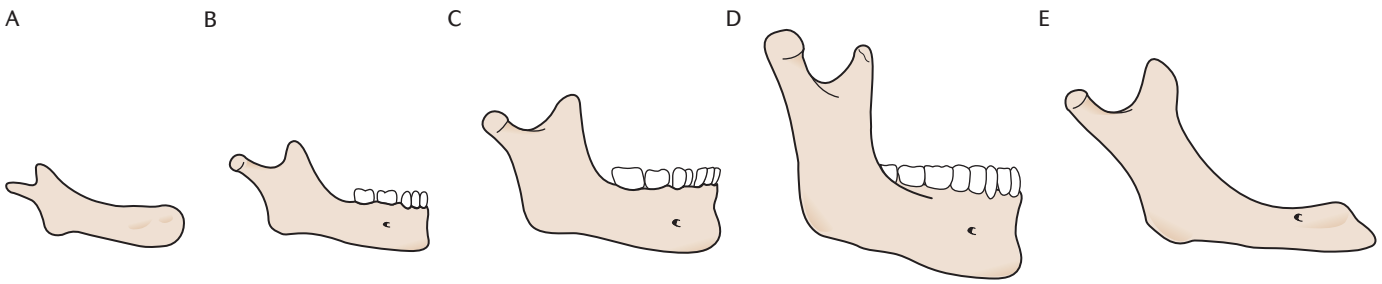


Fig. 33.17 Age changes in the mandible. A) At birth; B) After eruption of the primary dentition; C) During eruption of the permanent dentition; D) At maturity; E) An edentulous mandible.

The development of some areas of the mandible is related to the attachment and growth of muscles acting as periosteal matrices. The temporalis muscle is associated with the coronoid process, the medial pterygoid, and masseter muscles with the angle and the suprahyoid muscles with the mental region. A significant event to parents during pregnancy is when the baby starts to kick. This is literally what is happening; in the 4 months of pregnancy, the fetus begins to make reflex movements; those of the lower limbs are the ones felt as 'kicking'. The limb muscles are not the only muscles to become active at this time; the muscles of the jaw are also active and ultrasound scans at this age show the fetus opening and closing its mouth accompanied by lip and tongue movements. These movements exert a pull of the muscle on its bony attachments and these areas become more prominent in consequence.

By birth, the mandible is, like the rest of the facial skeleton, relatively small as shown in Figure 33.17A. The ramus is small relative to the body and the alveolar processes have not developed. The mandibular angle is more obtuse than in the adult because of the absence of the teeth.

As described above, the primary dentition erupts between 6 months and 2 years. As well as augmenting facial height, the presence of teeth enables the infant to chew solid food. This stimulates development of the muscles of mastication which, in turn, increase the size of their attachment sites on the mandible. A very important consequence of tooth eruption is that the angle of the mandible must be altered so that the occlusal surfaces of the upper and lower teeth remain parallel. If there was no compensation, the mouth would be forced open. The angle is adjusted by selective resorption and deposition as shown in Figure 33.17B. In addition, the position of the temporomandibular joints is altered by growth in the lateral parts of the cranial base which influence the position of the mandibular fossae. As described in Section 33.3.2, growth in width of the cranial base is most active from birth to 3 years; the widthways growth is much slower between 3 and maturity and is about the same amount as before 3. Selective resorption and deposition in the ramus adjusts the position of the head of the condyle to the position of the mandibular fossa on the cranial base.

During this time, growth at the condylar cartilages elongates the rami of the mandible. However, because the head forms one half of each temporomandibular joint against the under surface of the cranial base, this growth results in the body of the mandible being moved inferiorly so adding to the height of the facial skeleton. The length of the ramus and hence the body is also increased by periosteal bone deposition along the posterior border of the ramus while the anterior border of the ramus undergoes resorption, but at a somewhat lesser rate as shown in Figures 33.17 B and 33.17C.

As indicated in Figure 33.18, the lateral surface and anterior border of the coronoid process are resorbed and bone is deposited on the medial surface posterior to the temporal crest in the anterior part of the ramus. The reverse applies in the posterior part of the ramus, with deposition on the lateral surface and resorption on the medial surface. This combination of selective deposition and lateral resorption maintains the shape of the ramus as bone is deposited at the lingual tuberosity and also produces a small increase in the width of the mandible.

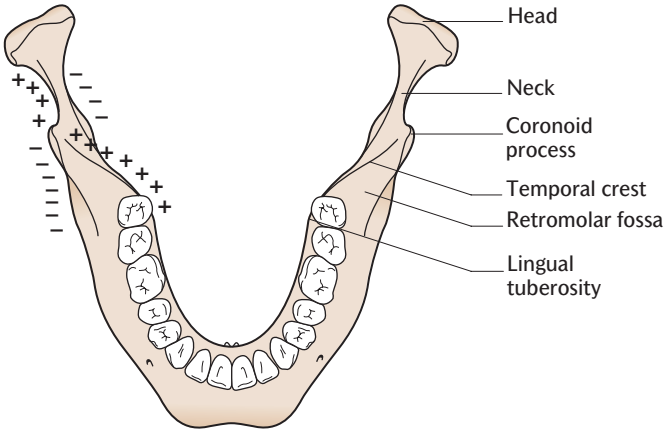


Fig. 33.18 Growth of the mandible viewed from above. + = bone deposition; – = bone resorption; arrow indicates the direction of actual growth.

The increase of the length of the mandible between the ages of 3 and 6 allows room for the eruption of the lower first permanent molars. Replacement of the primary teeth by the permanent teeth follows in the next 5 years and there is some thickening of the buccal aspects of the mandible by appositional bone deposition with a slower rate of resorption on the interior surfaces. Once the permanent dentition is established, the width of the body of the mandible barely changes between its eruption and maturity. However, length increase is still required to accommodate the second and third permanent molars. Fortuitously, the **adolescent growth spurt** produces an increase in length which coincides with eruption of the second permanent mandibular molars. The adolescent growth spurt also produces muscle hypertrophy which is more noticeable in males but does occur in females too. The extra muscular activity increases the robustness of the areas where muscles attach to the mandible, most noticeably the ramus. The mandible should grow enough between the end of the adolescent growth spurt and maturity to allow room for the eruption of the third molar tooth. An **impacted wisdom tooth** arises if there is insufficient space as outlined in Box 33.5.

As described on p. 344, the condylar cartilages are important growth sites in the mandible and are active until growth ceases at maturity. You might anticipate that the cartilage would be grown over by bone at maturity but; in fact, some cartilage remains as one or two layers of quiescent chondrocytes under the articular fibrocartilage of the mandible. Under certain circumstances described in Box 33.6, the quiescent cells can be reactivated and growth begins again.

Box 33.5 Impacted wisdom teeth

When there is insufficient space to accommodate the third permanent molar (the wisdom tooth), the short mandibular body means that the eruption pathway of the wisdom tooth is impeded by the ramus and its associated muscles. It, therefore, erupts obliquely forwards and collides with the second molar; this is an impacted wisdom tooth. They often require surgical removal as most of the tooth crown is still housed within the bone and is not accessible to extraction forceps.

Box 33.6 Acromegaly

As mentioned in the main text, one or two rows of dormant chondrocytes remain after growth has ceased in the condylar cartilage. Similar remnants of growth cartilages persist in the long bones of the hand (the phalanges and metacarpals) and foot (the phalanges and metatarsals). If there is excess of growth hormone post-maturity, usually due to a pituitary tumour, the cartilage remnants are reactivated and growth recommences in the hands, feet, and mandible. This is known as **acromegaly**. After taking the same shoe size since growth ceased, the patient suddenly finds they need progressively larger size as their feet grow; the hands and feet appear disproportionately large compared to the rest of the body. Reactivated condylar cartilages cause the mandible to grow in length and height; the length of the mandible will exceed that of the maxilla producing a **Class III occlusion**. The excess growth hormone also stimulates general **appositional growth** of bone throughout the skeleton which is most obvious in prominent areas such as the supraorbital ridges.

33.5.3 Age changes in the maxilla and mandible

Capsular and periosteal matrices that act during development still play a role in the areas they act upon by maintaining bone after growth has ceased; if the matrix is lost, then so will the bone it maintains. Half a century ago, loss of the complete dentition with age was relatively common, but in developed countries, people are retaining a substantial proportion of their teeth throughout life. This is due to a combination of factors, including improved nutrition and health status, better preventative and restorative dental treatment, and greater health and aesthetic awareness.

As we have seen earlier in the chapter, the **alveolar processes** of the jaw bones only develop as the teeth erupt and their roots form. If teeth are lost, the alveolar bone resorbs. Local resorption occurs if individual teeth are lost whereas there is extensive resorption of the alveolar process if the patient becomes **edentulous** when the entire dentition is lost through caries, periodontal disease, or other factors. There are marked changes in the mandible which you can see in Figure 33.17E. Resorption can proceed as far down as the **mylohyoid line** where the muscle attachment halts the process. The **mental foramen** is a useful marker on the external surface. In Figure 33.17, notice the changing position of the mental foramen from neonate (A) to the edentulous state (E). During growth, the mental foramen appears to move down the mandible; in reality, it is virtually static and the amount of alveolar bone above the foramen shows how much is added in successive stages of growth in Figures 33.17A to D. When you compare the profile of the edentulous mandible shown in Figure 33.17E, you will see that the mental foramen is virtually on the upper

Box 33.7 The edentulous mandible

Treatment of the edentulous mandible is a serious challenge to dental students and practitioners. Despite great effort, a satisfactory outcome is difficult because there is so little area for adhesion and retention of a conventional denture. In addition, resorption of bone down to the mylohyoid line means that muscle contraction can displace the denture. A relatively recent development to overcome these difficulties is the use of osseointegrated **dental implants**. A titanium base is surgically placed subperiosteally on the upper surface of the mandible and bone will grow into the base to integrate it with the bone; titanium is the material of choice because it is not toxic to bone cells or other tissues and can be worked and cast very accurately. The base has pegs onto which the denture is positioned so it is not reliant on adhesion to the oral mucosa for retention. Osseo-integrated implants can be used for prostheses other than full dentures. Needless to say, the use of expensive metal and the amount of skill required from dental technicians in the manufacture and involvement of the rest of the dental team in the necessary surgery and aftercare means that implants are very costly. They are only available in developed countries and even then, only to those who can afford it or who have special needs for this treatment.

surface of the bone, indicating that all the alveolar bone has been resorbed. Notice also that overall the bone looks thinner, especially in the ramus, and the angle has become more obtuse. The shape of the edentulous mandible approximates to that of the neonatal mandible shown in Figure 33.17A. Similar losses of alveolar bone occur from the maxillae, but does not appear so severe because of the vaulted shape of the palate maintaining an approximation to an alveolar ridge. The challenge of the edentulous mandible to dentists when they try to restore oral function and appearance to their patient by prosthetic dentistry is covered in Box 33.7.

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Glossary

Many anatomical terms are derived from Greek and Latin roots (G = Greek, L = Latin). Some of these are familiar because everyday words derive from them; 'canis' is Latin for 'dog' and has given us canine, meaning dog-like or the name of a specific tooth type which is very prominent in dogs. Other terms will be unfamiliar because they are more specialized and not used in everyday language. Just to confuse things, both Greek and Latin words, meaning exactly the same thing, are used in different anatomical terms. I think the explanation for the use of terms derived from both languages is a historical one. Early Greek physicians such as Hippocrates and Aristotle had started to name structures well before Latin became a universal language during the Roman Empire. It continued as the universal means of communication for religious, political, and intellectual purposes in Western Europe well into the sixteenth century and beyond. By then, terminology was becoming fixed so rather than discard terms, derivatives from both languages were used side by side. Anatomical terms also came in from other languages such as Arabic during the Renaissance, but these were usually latinized.

Many scientific words are *compound words* made up of prefixes added to the beginning and/or suffixes added to the end of words to add to or qualify their meaning. If you understand the more important prefixes and suffixes used in anatomical terminology, it can make understanding and learning so much easier and enjoyable.

Frequently used prefixes and suffixes

Prefixes

a- (G) without or negation (like the use of 'un' in English); e.g. arrhythmia = abnormal heart rhythm
ab- (L) away from; e.g. abnormal = deviating from normal
ad- (L) to; e.g. adrenal = a gland near the kidney
bi- (L) twice, double, two; e.g. bilateral = both sides
circum- (L) around; e.g. circumoral = around the mouth
contra- (L) against or other side; e.g contralateral = on the other side
di- (G) twice, double, two; e.g. digastric = a muscle with two separate parts (muscle bellies)
dis- (L) apart from or negation; e.g. dislike = do not like
dys- (G) bad, with difficulty; e.g dyslexia = reading with difficulty
ecto- (G) outer, outside; e.g. ectoderm = the outer layer of an embryo
endo- (G) inner, inside; e.g. endocrine = a gland that secretes inwards (into the bloodstream)
epi- (G) upon; e.g. epidermis = the outermost layer of skin (upon the dermis)
extra- (L) beyond, outside; e.g. extradural = outside the dural coverings of the brain
hypo- (G) under; e.g. hypoglossal (nerve) = a nerve which runs below under the tongue
infra- (L) under; e.g. infrahyoid (muscles) = muscles in the neck below the hyoid bone
[Note that in the two examples above, 'hypoglossal' uses a Greek suffix on a Greek word, but 'infrahyoid' uses a Latin suffix on a Greek word!]
ipsi- (L) the same; e.g. ipsilateral = the same side
inter- (L) between; e.g. intercostal (muscle) = muscle between the ribs
intra- (L) inside, within; e.g. intravenous (injection) = an injection into a vein
meso- (G) middle or between; e.g. mesoderm = the middle layer of the embryo
para- (G) near, beside, passing alongside; e.g. paranasal air sinuses = spaces in bones near the nose
peri- (G) around, enclosing; e.g. periondontal = tissues around the teeth
post- (L) after or behind; e.g. post-natal = after birth
pre- (L) before, in front of; e.g. prenatal = before birth
pro- (G) before, forward; e.g. prosencephalon = the most anterior part of the developing brain
retro- (L) back, behind; e.g. retromolar fossa = a space behind the molar teeth
supra- (L) above, upon; e.g. suprasternal (notch) = a notch on the upper surface of the breastbone
syn- (G) together or with; e.g. synchondrosis = a joint between two bones formed from cartilage

Suffixes

-itis inflammation; e.g. tonsillitis = inflammation of the tonsils
-culus, -culum, -cule are diminutives so tuberculum is a little lump (from tuber = lump)

Anatomical terms

The following list is composed of an anatomical term, a plural form (pl.) where this is in common use or unusual, the language of origin, and a short explanation or translation.
Alar (L) From ala = a wing, hence wing-like
Alveolus pl. alveoli (L) A cavity or socket
Amygdala; amygdaloid (G) An almond; resembling an almond
Ansa (L) Originally = jug handle, but used to describe a loop
Antrum (L) A cave

Aponeurosis (G) Originally any fibrous structure, but now used specifically for a flat tendon
Arachnoid (G) Resembling a spider's web
Arcuate (L) Arched or bow-shaped
Arytenoid (G) Shaped like a pitcher (a small jug) [*No, I can't see the resemblance either!*]
Atlas (G) The first cervical vertebra supporting the skull; named after the mythical Greek giant who carried the world on his shoulders
Atrium pl. atria (L) A chamber
Axon (G) Originally an axle or cylinder; now a nerve process
Buccal (L) From bucca = cheek, hence related to the cheek
Buccinator (L) A trumpeter; now used for the muscle of the cheek which is used during playing of wind instruments
Bulla (L) A bubble
Caecum (L) Originally meaning blind; now used for a blind-ending pouch
Calcarine (L) Spur-shaped
Canthus pl. canthi (G) Corner of the eye
Carotid (G) Originally meaning a deep sleep. The carotid artery is so called because it was believed that its compression produced drowsiness
Cauda equina (L) A horse's tail
Caudate (L) Possessing a tail
Cerebellum (L) Diminutive of cerebrum
Cerebrum (L) The brain
Cervical (L) From cervix = neck, hence relating to the neck
Chiasma (G) Derived from the Greek letter, chi (X), now meaning two lines crossing
Chondro- or -chondral (G) From chondros = cartilage or gristle, hence relating to cartilage
Chorda pl. chordae (L) A cord
Ciliary (L) From cilium = eyelash, hence related to the eye
Cingulate (L) From cingulum = a girdle
Cleido- (G) Relating to the clavicle
Colliculus pl. colliculi (L) A small mound
Commissure (L) A site of joining together; a seam
Concha pl. conchae (L) A shell
Condyle (G) A knuckle
Cornu pl. cornua (L) A horn
Coronal (L) From corona = a crown, hence related to the crown of the head or any other crown-like structure
Coronoid (G) Curved
Corpus callosum (L) (literally) a tough body. The corpus callosum appeared as a mass of fibres connecting the two cerebral hemispheres to those who chose its name; it is now known to consist of axons
Cribriform (L) Perforated like a sieve
Cricoid (G) Ring-shaped
Crista; crista galli (L) A ridge; a cock's comb
Cuneate (L) From cuneus = a wedge, hence wedge-shaped
Decussation (L) An intersection or crossing over
Dendrite (G) Tree-like—because of the branching of neuronal processes
Diencephalon (G) The 'in between' part of the brain from the suffix dia- = through or between + encephalon
Dura (L) Hard
Ectopic (G) Displaced
Encephalon (G) The brain—used with prefixes to designate parts of the brain
Epiglottis (G) Near the tongue—the highest laryngeal cartilage
Ethmoid (G) Sieve-like
Facet (L) Diminutive of facies = face, hence a smooth area on a bone
Falx (L) A sickle
Fascia pl. fasciae (L) A bandage
Fasciculus pl. fasciculi (L) Diminutive of fascis = a bundle
Foramen pl. foramina (L) An opening
Fossa pl. fossae (L) A hollow
Fovea (L) A depression
Frenum or frenulum (L) A bridle or band; a frenulum is smaller than a frenum
Funiculus pl. funiculi (L) Diminutive of funis = a cord
Ganglion pl. ganglia (G) A swelling or knot
Geniculate (L) Bent like a knee from genu = knee; genu is also used to describe structures with a knee-like bend
Genio- (G) Relating to the chin
Glosso- or -glossal (G) From glossa = tongue, hence associated with the tongue
Gracile, gracilis (L) Slender
Gyrus pl. gyri (G) A ring or circle
Hamulus (L) A little hook
Hiatus (L) A gap
Hippocampus (G) A sea horse—used to describe the area of the brain below the inferior horn of the lateral ventricle which does have some resemblance to a sea horse in coronal section
Hyoid (G) Resembling the Greek letter upsilon (υ), hence U-shaped
Incus (L) An anvil
Infundibulum (L) A funnel
Insula (L) An island
Jugular (L) From jugum = a yoke placed across the neck of beasts of burden, hence relating to the neck
Lacrimal (L) From lacrimae = tears, hence relating to tears
Lacuna (L) A pit or hole
Lambdoid (G) Shaped like the Greek letter, lambda (λ)
Lemniscus pl. lemnisci (L) A ribbon or band
Lentiform (L) Shaped like a lens or lentil
Limbic (L) From limbus = edge or border, hence something relating to an edge or border
Lingual (L) From lingua = tongue, hence relating to the tongue
Lingula (L) A little tongue
Malleus (L) A hammer
Manubrium (L) A handle
Mastoid (G) Breast-shaped
Mater (L) Literally = mother—with the connotation of one who protects, hence dura mater, arachnoid mater, and pia mater protecting the brain
Meatus pl. meati (L) A passageway
Mediastinum (L) The origin is uncertain; the word originally meant a slave or intermediary or their rooms, but is now used for the area between the lungs in the thorax

Medulla (L) Middle or inner part
Meninx pl. meninges (G) A membrane
Mental (L) From mentum = chin, hence relating to the chin
Mesencephalon (G) Midbrain
Metencephalon (G) Hindbrain
Micrometre (abbreviated to µm) One millionth (10^{-6}) of a metre or one thousandth of a millimetre
Myelencephalon (G) Midbrain
Mylohyoid (G) Mylo- derives from a Greek word meaning a corn-mill which became mola (L), also used for the grinding molar teeth; the muscle is between the molar teeth and hyoid bone
Nanometre (abbreviated to nm) one thousand millionth (10^{-9}) of a metre or one millionth of a millimetre
Neuron (G) Originally a sinew so applied to any whitish fibrous structures such as tendons, fascia, and nerves, but its use quickly became restricted to nerves; now used only for nerve cells
Nuchal (L) From nucha = the back of the neck, hence relating to the back of the neck
Occipital (L) From occiput = back of the head, hence relating to the back of the head
Ocular (L) From oculus = eye, hence relating to the eye
Ophthalmic (G) From ophthalmos = eye, hence relating to the eye
Ostium (L) An opening
Otic (G) From otos = ear, hence relating to the ear
Palpebral (L) From palpebra = eyelid, hence relating to the eyelid
Parietal (L) From paries = wall, hence relating to or resembling a wall
Parotid (G) From para = near + otos = ear
Pedicle; peduncle (L) A foot or stalk; a small foot or stalk
Petrous (G), petrosal (L) Rock-like; used to describe the solid hard part of the temporal bone
Pia (L) Tender or soft
Platysma (G) Wide or flat—the shape of the muscle of this name in the neck
Plexus pl. plexus or plexuses (L) A network
Pons (L) A bridge
Prosencephalon (G) The forebrain
Pterygoid (G) From pterion = a wing, hence wing-like.
Putamen (L) A shell
Ramus pl. rami (L) A branch
Raphe (pronounced raphé) (G) A seam
Rectus (L) Straight
Rhombencephalon (G) The hindbrain; the prefix rhomb- means kite-shaped and refers to the lozenge shape of the developing fourth ventricle
Rima (L) A chink
Sagittal (L) Shaped like an arrow; when an arrow is released, it is supposed to travel in a straight line 'as straight as an arrow', giving a secondary meaning of straight, hence the sagittal suture running in a straight line.
Scaphoid (G) Shaped like a boat
Septum (L) A ledge or fence
Skull From skalle = skull (Old Norse) possibly from skål = a drinking bowl because of the resemblance of the inverted skull cap to a bowl or more fancifully from the supposed habit of Norsemen of drinking from the skull of their victims in battle
Somatic (G) From soma = body, hence relating to the body; mainly used to contrast with visceral (related to internal organs)
Sphenoid (G) Wedge-shaped
Splanchnic (G) Related to internal organs (viscera)
Squamous (L) From squama = scale, hence resembling a scale
Stapes (L) A stirrup
Styloid (G) Resembling a pillar
Sulcus (L) A groove
Suture (L) A seam
Symphysis pl. symphyses (G) To grow together
Synapse (G) To join together
Synchondrosis pl. synchondroses (G) A cartilaginous joint, literally a joining of cartilage
Tectum (L) A roof
Telencephalon (G) The end or anterior part of the brain from telos = end + encephalon
Temporal (L) Concerning time. The temporal bone and temple overlying it are so called because the temple is the first area where hair turns to grey with aging
Tentorium (L) A tent
Thalamus (L) An inner chamber
Thyroid (G) Shield-shaped
Trigeminal (L) Triple, triplets
Trochlea, trochlear (L) A pulley, hence trochlear relating to a pulley
Tuberculum and tubercle (L) Diminutives of tuber, a protuberance
Tympanum (L) A drum
Uncus (L) A hook
Uvula (L) Little grape
Vagus (L) Wandering
Velum (L) A veil-like covering
Ventricle (L) A cavity
Viscera, visceral (L) From viscus = an internal organ, but usually used in the plural form to mean internal organs in general or the guts; used to contrast internal organs with peripheral parts of the body (somatic)
Vomer (L) A ploughshare
Xiphoid (G) Sword-like
Zygoma (G) A union, hence the adjectival form zygomatic also used as a noun for the alternative name for the same bone; possibly from the joining of two bones to form the zygomatic arch

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