

Giorgio Capogna *Editor*

Epidural Labor Analgesia

Childbirth Without Pain

 Springer

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1.1 Introduction

Labor is the process whereby the birth canal is prepared to allow the baby to pass from the uterine cavity to the outside world. In the normal course of events, it ends with a spontaneous or instrumental vaginal delivery, or Cesarean section. Conventionally, it is divided into a first stage, during which the cervix passively dilates in response to uterine contractions, a second stage where the mother pushes the baby through the vagina, and a third stage where the placenta delivers.

1.2 Onset of Labor

In the absence of interference, the normal duration of pregnancy is 40 weeks from the first day of the last menstrual period, assuming that ovulation occurred on day 14 of that cycle.

Unlike the sheep and rabbit in which a fetal-initiated fall in progesterone is the signal for labor onset, no single trigger for labor has been identified in humans. Antiprogesterone drugs induce labor [1], but there is no evidence that this is the physiological mechanism. The fetal pituitary–adrenal axis is involved, but clear

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evidence that it alters the overall balance between inhibitory hormones such as progesterone and excitatory hormones such as estrogens, prostaglandins, and oxytocin has been difficult to demonstrate. Recent research has focused on the anatomy of the closed collagenous cervix preventing local infection and the lack of connectivity of myometrial cells preventing contractions spreading throughout the uterus. Local infection and inflammatory reactions in the cervix may be crucial in the onset of labor, both at term [2] and preterm [3, 4]. Once labor has started, pituitary oxytocin is the major factor maintaining regular contractions.

1.3 Term, Preterm, and Postterm Labor

About 95 % of natural onset labor occurs between 37 + 0 and 42 + 0 weeks, so this range has traditionally been regarded as term, earlier as preterm, and later as postterm. However, recent concerns about the excess fetal risks from delivery before 38 + 6 and after 40 + 6 have led to the suggestion that 37 + 0 to 38 + 6 is named early term delivery, 39 + 0 to 40 + 6 full-term delivery, and 41 + 0 to 41 + 6 as late-term delivery [5].

1.4 The Mechanics of Labor

1.4.1 Normal

The large human brain means that the fetal head is a relatively tight fit within the ring of the maternal bony pelvis. Passage is facilitated by softening of the maternal pelvic ligaments, allowing the constituent bones to separate, and by the fetal sutures, allowing the skull bones to mould, and in severe cases to overlap. Even with the help of these factors, the fetal head has to flex and rotate correctly to achieve delivery.

Three anatomical factors need to be considered:

1. The inlet of the maternal pelvis, sometimes called the pelvic brim, formed by the sacral promontory, the ileo-pectineal line and the pubic symphysis, is functionally oval with the widest diameter in the transverse plane.
2. The outlet of the maternal pelvis, formed by the coccyx, ischial tuberosities and the inferior border of the symphysis, is functionally oval with the largest diameter in the anteroposterior plane.
3. The fetal head presents its smallest diameter when fully flexed. The flexed fetal head is also oval with the largest diameter in the suboccipitobregmatic dimension, i.e., in the fetal anteroposterior plane.

The consequence of these three factors is that in late pregnancy and early labor the fetal head enters the pelvis in a transverse position with the occiput either on the right or the left side of the maternal pelvis. Obstetricians refer to these two

transverse positions as right occipito-lateral (ROL) and left occipito-lateral (LOL), respectively.

As labor progresses and the head descends in the pelvis, it rotates until the occiput is anterior, i.e., to the occipito-anterior (OA) position. This is called internal rotation. Normal delivery occurs with extension of the head, at which point the shoulders enter the pelvic inlet in the transverse diameter. As the shoulders descend, they in turn rotate to the anteroposterior diameter and in the process rotate the delivered head back to a transverse position. This is called external rotation or restitution. Finally, the anterior and then the posterior shoulders deliver, following which the body usually slips out easily.

1.4.2 Abnormal Mechanics

If the head rotates the wrong way to an occipito-posterior (OP) position, it presents slightly larger dimensions. Labor progress tends to be slower, and the pressure of the fetal occiput on the maternal sacrum increases the pain of labor. With a modest sized baby and a roomy pelvis, spontaneous delivery of an occipito-posterior fetus is possible, the so-called “face to pubis” delivery. However, if the fit is tighter, obstetricians may need to rotate the head to occipito-anterior, either manually or with special forceps to allow a vaginal delivery.

Sometimes internal rotation fails to occur leaving the baby’s head low in the pelvis in a transverse position with the cervix fully dilated. If this fails to resolve spontaneously, or if urgent delivery is required, the obstetrician will need to rotate the head to occipito-anterior, either manually or with special forceps to facilitate vaginal delivery.

If the head fails to flex and the fetal brow presents, the dimensions are incompatible with vaginal delivery. In late labor, a brow presentation mandates Cesarean delivery.

If the head is hyperextended such that the face presents, vaginal delivery is only possible if rotation occurs to the mento-anterior position. Babies in the mento-posterior position cannot be delivered vaginally.

If labor starts with the baby lying transverse with the fetal head on the right or left side of the maternal abdomen, the shoulder will present, and as labor progresses an arm will deliver. Such a neglected transverse lie will not deliver vaginally and, if the baby is alive, cesarean delivery is mandatory.

For breech babies, the widest part is the femoral intertuberous diameter. The breech therefore enters the pelvis with this in the transverse plane (back anterior). Internal rotation occurs till the intertuberous diameter is in the anterior–posterior plane with the back to one side. After the pelvis delivers, the body rotates so the back is anterior. Unless the arms are extended above the fetal head, they usually deliver easily and as long as the head is well flexed it enters the pelvis in the same rotational sequence as for cephalic presentation, i.e., initially in the transverse position followed by rotation to occipito-anterior. For a breech baby, the delivery

of the head is by flexion instead of extension. If the arms are extended above the fetal head, special maneuvers are required to bring them into the pelvis.

1.5 The First Stage of Labor

Labor usually starts gradually with painless “Braxton Hicks” contractions increasing in frequency and strength at the end of pregnancy until they merge into labor. The onset of the first stage of labor is conventionally defined as the presence of regular painful uterine contraction in the presence of either progressive cervical effacement or dilatation or ruptured membranes. Cervical effacement is the process whereby the long tubular cervix shortens prior to dilatation. This is often difficult to define precisely and is one reason why defining the length of normal labor is also difficult.

The end of the first stage of labor is defined as full dilatation of the cervix. This is easy to define objectively so long as a vaginal examination is performed. If a vaginal examination is not done, the second stage is assumed to occur when the mother has an involuntary urge to push.

The duration of the first stage averages about 8 h for first labors and five for subsequent ones, but there is wide variation, and a duration of 18 or 15 h, respectively, would be regarded as normal.

The first stage of labor is often divided into a latent phase before cervical dilatation of 4 cm and an active phase after that. Recently, in an effort to avoid unnecessary intervention, the American College of Obstetricians and Gynaecologists has recommended that the active phase of labor be defined as starting at 6 cm [6]. Most authorities recommend that no action be taken for slow progress in the latent phase, in the absence of fetal compromise.

Once the active phase of labor has started, most experts agree that a rate of progress of about 1 cm cervical dilatation per hour is normal [7–9].

Progress in the first stage of labor in terms of cervical dilatation is often measured graphically using a partogram. Most partograms also permit the recording of fetal and maternal heart rate, maternal blood pressure, urine output, and other parameters. Although advocates argue that the use of partograms allows convenient and early diagnosis of slow progress, their format may influence practice [10] and there is little strong evidence that their use reduces adverse outcomes [11].

1.5.1 Uterine Activity in Labor

The frequency and strength of spontaneous uterine contractions increase gradually throughout the first stage of labor. Frequency is relatively easy to measure and throughout most of the first stage 3–4 contractions in every 10 min is normal. In the latter part of the first stage, the so-called “transition,” the frequency may increase up to five contractions every 10 min or even more. If such a frequency of contractions

is continued for a prolonged period, fetal compromise is almost inevitable, but this is usually pre-empted by delivery.

The force of uterine contractions can only be measured with the passage of an intrauterine pressure transducer [12]. With the possible exception of monitoring contractions when administering oxytocin to a woman with a scarred uterus, this technique is now limited to use within clinical trials. Force equals pressure multiplied by duration, and is conventionally expressed as kilo-Pascal seconds per 15 min (kPa/15 min). In first labors, normal values rise from a mean of 650 kPa/15 min at 3 cm dilatation to a mean of 1,500 kPa/15 min in the late first stage [13]. In multiparous women, the force of uterine contractions tends to be lower [14].

1.6 Second Stage of Labor

In the absence of epidural or spinal anesthesia, women usually get an uncontrollable urge to push soon after the cervix reaches full dilatation, and the duration of the second stage is typically <1 h in first labors and <30 min in subsequent ones. However, with regional anesthesia the urge to push may be abolished. If there is no fetal compromise, allowing a period of 1–2 h for the head to passively descend and rotate makes spontaneous delivery more likely. In this situation, the second stage may last for up to 3 h.

1.7 Third Stage of Labor

The third stage of labor begins after the delivery of the baby and ends with the delivery of the placenta. Immediately after the birth of the baby, the umbilical arteries within the umbilical cord go into spasm, reducing the flow of fetal blood toward the placenta. Spasm within the umbilical cord occurs first as a result of a surge of catecholamines and angiotensin caused by the stress of birth, and second as a direct effect of stretch receptors in the umbilical cord. The umbilical vein remains open and the fetus receives a net blood transfusion within the first few minutes after delivery. Some authors estimate that up to 100 ml of blood may be transfused in a term fetus, and this can take up to 10 min. If a uterotonic agent such as oxytocin or syntometrine is administered, the speed of transfusion will be increased slightly; however, if the cord is clamped soon after birth, the transfusion volume will be correspondingly reduced.

Simultaneously, uterine contraction results in the separation of the placenta from the uterine wall. Bleeding from the placental bed is controlled by the “living ligature” of the contracting uterine muscle fibres kinking and occluding the branches of the uterine arteries as they run through the myometrium. Without intervention, the third stage of labor typically lasts up to 30 min (“physiological” third stage). However, in modern obstetric practice a policy of “active management” of the third stage of labor has generally been adopted and this comprises the

administration of a uterotonic after the delivery of the anterior shoulder of the baby, early clamping of the umbilical cord, and controlled cord traction to deliver the placenta and membranes. Active management of the third stage of labor reduces the incidence of postpartum hemorrhage by approximately 60 % compared with physiological third stage [15].

1.8 Retained Placenta

Failure of the placenta to deliver is associated with an increased risk of postpartum hemorrhage. In the majority of cases (over 80 %), the placenta fails to separate and in the remaining cases, the placenta has separated but remains within the uterine cavity [16]. Weeks' findings led to the hypothesis that the administration of oxytocin via the umbilical cord might encourage separation and reduce the need for manual removal; however, this approach has since been shown to be ineffective [17].

1.9 Pain in Labor

Normal uterine contractions in labor are painful. In the first stage, pain is caused by ischemia of uterine musculature and by dilatation of the cervix. Pain signals are transmitted via spinal nerves T10-L1 and may be referred to the abdominal wall, lower back, buttocks, or thighs.

In the second stage, pain from distension of the vagina and perineum is added to uterine pain. This pain is transmitted by the pudendal nerves, via nerve roots S2–4.

1.10 Physiological Changes in Labor

1.10.1 Cardiovascular System

Cardiac output increases by 30–50 % in pregnancy, with half of this increase occurring by 8 weeks gestation. It increases further in labor and is highest in the immediate postpartum period. The maternal heart rate increases by approximately 10 beats per minute and may increase further in labor as a result of pain or dehydration.

1.10.2 Respiratory System

Tidal volume, minute ventilation, and arterial oxygen pressures increase in pregnancy, and relative hyperventilation causes a chronic respiratory alkalosis. Failed intubation is ten times more common in pregnant women than in the general

surgical population, especially in late pregnancy and labor, probably due to pharyngeal edema.

1.10.3 Gastrointestinal System

Pregnancy results in a lower esophageal sphincter pressure and delayed gastric emptying. These factors predispose to reflux and aspiration during general anesthesia.

1.10.4 Neurological System

Among women with epilepsy, 1–2 % will experience a seizure during labor and a further 1–2 % during the first 24 h after delivery. This lowered seizure threshold may be a factor in the occurrence of eclamptic convulsions in labor and makes it imperative to continue with antiepileptic medications during labor.

1.10.5 Urological System

Mild-to-moderate hydronephrosis and hydroureter occur in normal pregnancies, more commonly on the right side. Urinary retention can occur in labor, especially with epidural anesthesia, and an indwelling catheter should be considered for these women to prevent long-term complications of bladder overdistention.

1.11 Fetal Health in Labor

During uterine contractions, maternal blood flow to the placental bed is reduced. In established labor, placental perfusion ceases altogether at the height of contractions, and the fetus becomes gradually more hypoxemic as labor progresses. Normal fetuses with good glycogen reserves withstand this relative hypoxemia well, but if labor is prolonged or augmented with oxytocin, or if the fetus has poor reserves at the onset of labor due to growth restriction, fetal acidemia or even intrapartum fetal death may occur. With modern obstetric practice, the risk of fetal death during labor is approximately 1 in 2,000, making a baby's day of birth one of the most dangerous days in his/her lifetime [18].

1.11.1 Signs of Fetal Compromise

Meconium-stained liquor, visible upon the rupture of the membranes, is the only sign of fetal compromise that the mother may recognize, and is regarded as an indication for continuous electronic fetal monitoring. There are well-recognized

changes in the fetal heart rate pattern in response to fetal acidemia, which can be detected by intermittent auscultation of the fetal heart, or by continuous fetal monitoring (cardiotocography [CTG]).

Intermittent auscultation clearly prevents stillbirth. Continuous fetal monitoring probably reduces perinatal mortality further but also appears to increase the rate of Cesarean delivery. Its use is therefore generally restricted to pregnancies with recognized risk factors, for example, where oxytocin is being administered, where the fetus has passed meconium, or when the uterus is scarred by a previous cesarean birth.

1.11.2 Slow Progress in Labor

Delay in cervical dilatation may be a sign of cephalo-pelvic disproportion—the mother’s pelvis is not of an adequate size relative to the fetal head for it to pass through and allow vaginal delivery—or delay may be due to malposition of the baby such as transverse lie or a brow presentation. Typically, cervical dilatation ceases during the late first stage of labor (7-cm dilatation and above), the so-called “secondary arrest,” and in these cases Cesarean section is required. True cephalo-pelvic disproportion is relatively rare.

More typically, slow progress in labor is a result of a relative disproportion and poor uterine contractility. This is more common in first labors and typically presents with slow progress from the onset of labor—the so-called primary dysfunctional labor. In the absence of fetal compromise, management is conservative and includes administering adequate analgesia and intravenous fluids to the mother and waiting patiently for labor to progress. Artificial augmentation of labor with oxytocin may be required.

A policy of “active management of labor” became popular in the 1970s. This consisted of three main components—routine amniotomy, early use of oxytocin, and continuous one-to-one support for the laboring woman. These three components have since been evaluated in randomized controlled trials.

1.11.3 Amniotomy

In normal labor, amniotic fluid retained with intact fetal membranes tends to minimize reduction in placental blood flow and prevents cord compression. Once the membranes have ruptured, either spontaneously or artificially, the umbilical cord may prolapse through the cervix—a serious obstetric emergency because cord compression is almost inevitable.

Even in the absence of cord prolapse, the lack of amniotic fluid may allow the cord to be compressed by fetal parts. The rupture of membranes also allows infection to ascend into the uterus more easily. This is why traditionally obstetricians and midwives have tried to keep the membranes intact during labor.

Some obstetricians have advocated amniotomy as a method for the assessment of fetal well-being, by screening for meconium-stained liquor, or to augment labor. Randomized trials of this practice have shown that it does indeed shorten labor, but has little effect on fetal outcomes [19, 20].

1.11.4 Acceleration with Oxytocin

The early use of oxytocin does accelerate labor, but does not affect the mode of birth and does not affect any other major maternal or fetal outcomes [21, 23]. The use of amniotomy and oxytocin combined may have a modest effect on reduction in cesarean section [22].

1.11.5 Psychological Support in Labor

There have been several randomized trials testing the effects of continuous support in labor. Their methodological quality varies, but overall they provide good evidence that a range of adverse outcomes, including cesarean section, operative vaginal delivery, and low Apgar scores, are reduced by this intervention [24]. All well-run obstetric units now provide such support as far as midwifery staffing permits.

1.12 Summary

Labor is a challenging time for the mother and the baby. The aim of modern obstetrics is to provide optimal maternal and fetal care during labor and delivery to ensure the best possible outcomes where maternal or fetal physiology is not able to compensate for the demands of childbirth.

References

1. Hapangama D, Neilson JP (2009) Mifepristone for induction of labour. *Cochrane Database Syst Rev* 3:CD002865. doi:[10.1002/14651858.CD002865.pub2](https://doi.org/10.1002/14651858.CD002865.pub2)
2. Bollapragada S, Youssef R, Jordan F, Greer I, Norman J, Nelson S (2009) Term labor is associated with a core inflammatory response in human fetal membranes, myometrium, and cervix. *Am J Obstet Gynecol* 200:104e101–104e111
3. Muglia LJ, Katz M (2010) The enigma of spontaneous preterm birth. *N Engl J Med* 362:529–535
4. Strevens H, Allen K, Thornton JG (2010) Management of premature prelabor rupture of the membranes. *Ann NY Acad Sci* 1205:123–9. doi:[10.1111/j.1749-6632.2010.05654.x](https://doi.org/10.1111/j.1749-6632.2010.05654.x)
5. Spong C (2013) Defining “term” pregnancy recommendations from the defining “term” pregnancy workgroup. *JAMA* 309(23):2445–2446. doi:[10.1001/jama.2013.6235](https://doi.org/10.1001/jama.2013.6235)

6. Caughey AB, Cahill AG, Guise JM, Rouse DJ (2014) Safe prevention of the primary caesarean delivery. *Obstetric Care Consensus No. 1. American College of Obstetricians and Gynecologists. Obstet Gynecol* 123:693–711
7. Friedman E (1954) The graphic analysis of labor. *Am J Obstet Gynecol* 68:1568–1575
8. Philpott RH, Castle WM (1972) Cervicographs in the management of labour in primigravidae. *J Obstet Gynaecol Br Commonw* 79:592–598
9. Studd JWW (1973) Partograms and nomograms in the management of primigravida labour. *BMJ* 4:451–455
10. Cartmill RSV, Thornton JG (1992) Obstetric decision making; the effect of varying the presentation of partogram information. *Lancet* 339:1520–1522
11. Lavender T, Hart A, Smyth RMD (2013) Effect of partogram use on outcomes for women in spontaneous labour at term. *Cochrane Database Syst Rev* 7:CD005461. doi:[10.1002/14651858.CD005461.pub4](https://doi.org/10.1002/14651858.CD005461.pub4)
12. Bakker JJH, Janssen PF, van Halem K, van der Goes BY, Papatsonis DNM, van der Post JAM, Mol BWJ (2013) Internal versus external tocodynamometry during induced or augmented labour. *Cochrane Database Syst Rev* 8:CD006947. doi:[10.1002/14651858.CD006947.pub3](https://doi.org/10.1002/14651858.CD006947.pub3)
13. Gibb DM, Arulkumaran S, Lun KC, Ratnam SS (1984) Characteristics of uterine activity in nulliparous labour. *Br J Obstet Gynaecol* 91(3):220–227
14. Arulkumaran S, Gibb DM, Lun KC, Heng SH, Ratnam SS (1984) The effect of parity on uterine activity in labour. *Br J Obstet Gynaecol* 91(9):843–848
15. Begley CM, Gyte GML, Devane D, McGuire W, Weeks A (2011) Active versus expectant management for women in the third stage of labour. *Cochrane Database Syst Rev* 11:CD007412. doi: [10.1002/14651858.CD007412.pub3](https://doi.org/10.1002/14651858.CD007412.pub3)
16. Weeks AD (2008) The retained placenta. *Best Pract Res Clin Obstet Gynaecol* 22(6):1103–1117. doi:[10.1016/j.bpobgyn.2008.07.005](https://doi.org/10.1016/j.bpobgyn.2008.07.005)
17. Weeks AD, Alia G, Vernon G, Namayanja A, Gosakan R, Majeed T, Hart A, Jafri H, Nardin J, Carroli G, Fairlie F, Raashid Y, Mirembe F, Alfirevic Z (2010) Umbilical vein oxytocin for the treatment of retained placenta (release study): a double-blind, randomised controlled trial. *Lancet* 375(9709):141–147. doi:[10.1016/S0140-6736\(09\)61752-9](https://doi.org/10.1016/S0140-6736(09)61752-9)
18. Walker KF, Cohen AL, Walker SH, Allen KM, Baines DL, Thornton JG (2014) The dangers of the day of birth. *BJOG* 121(6):714–718. doi:[10.1111/1471-0528.12544](https://doi.org/10.1111/1471-0528.12544)
19. UK Amniotomy Group (1994) A multicentre randomised trial comparing routine versus delayed amniotomy in spontaneous first labour at term. *Br J Obstet Gynaecol* 101:307–309
20. Smyth RMD, Markham C, Dowswell T (2013) Amniotomy for shortening spontaneous labour. *Cochrane Database Syst Rev* 6:CD006167. doi:[10.1002/14651858.CD006167.pub4](https://doi.org/10.1002/14651858.CD006167.pub4)
21. Bugg GJ, Siddiqui F, Thornton JG (2013) Oxytocin versus no treatment or delayed treatment for slow progress in the first stage of spontaneous labour. *Cochrane Database Syst Rev* 6:CD007123. doi:[10.1002/14651858.CD007123.pub3](https://doi.org/10.1002/14651858.CD007123.pub3)
22. Wei S, Wo BL, Qi HP, Xu H, Luo ZC, Roy C, Fraser WD (2013) Early amniotomy and early oxytocin for prevention of, or therapy for, delay in first stage spontaneous labour compared with routine care. *Cochrane Database Syst Rev* 8:CD006794. doi:[10.1002/14651858.CD006794.pub4](https://doi.org/10.1002/14651858.CD006794.pub4)
23. Hinshaw K, Simpson S, Cummings S, Hildreth A, Thornton JG (2008) A randomised controlled trial of early versus delayed oxytocin augmentation to treat primary dysfunctional labour in nulliparous women. *Br J Obstet Gynaecol* 115:1289–1296
24. Hodnett ED, Gates S, Hofmeyr GJ, Sakala C (2013) Continuous support for women during childbirth. *Cochrane Database Syst Rev* 7:CD003766. doi:[10.1002/14651858.CD003766.pub5](https://doi.org/10.1002/14651858.CD003766.pub5)

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2.1 Anatomo-physiological Cardiovascular System Changes During Labor and Delivery

The major anatomo-physiological changes of the maternal cardiovascular system happen throughout gestation and include an increase of blood volume, cardiac output, maternal heart rate, decrease of arterial blood pressure, and systemic vascular resistance. These changes are almost fully reversed in the weeks and months after delivery. During labor and delivery, there is further adaptation of cardiac output which progressively rises in both stroke volume and heart rate, peaking with contractions. The cardiac output increases up to 30 % during the first stage of labor, and it can increase by as much as 50 % in the second stage because of maternal pushing efforts [1]. The rise in cardiac output with contractions (approximately 15 % on Doppler studies) [2] is complex, involving uterine contraction with squeezing of blood from the intervillous space into circulation volume (autotransfusion phenomenon), reduction of the uteroplacental shunt, sympathetic stimulation of pain, and possible relief of aortocaval compression as the contracting uterus lifts forward [3]. The volume contribution of the contraction has been estimated between 200 and 300 ml [4], but it can even achieve 500 ml. Basal cardiac output between contractions increases from a prelabor mean of 6.99–7.88 l/min at ≥ 8 cm of cervical dilatation as a result of an increase of stroke volume rather than heart rate: it actually can vary from 12 % to 31 % [5]. There are also further increases in mean blood pressure during contractions. The stress of labor affects the

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circulating levels of catecholamines. Systolic pressure rises in labor by about 35 mmHg, and diastolic pressure by about 25 mmHg [2]. Effective analgesia is known to significantly reduce the increased levels of catecholamines, mitigating the rise in blood pressure [6]. Both epinephrine and norepinephrine decrease uterine contractility. A reduction in epinephrine levels has been shown to improve uterine contractility [7]. Epinephrine but not norepinephrine levels are significantly reduced with epidural analgesia [8]. The effects of regional anesthesia on uteroplacental flow show some evidence of a small degree of autoregulation to offset small changes in maternal perfusion pressure [9]. Cardiac output peaks within 10 min of delivery [6], with the additional effect of the relief of aortocaval compression [10], and augmented venous return of parental oxytocics to offset any drop in output from blood loss. One hour after delivery, heart rate and cardiac output return to prelabor values though mean arterial pressure and stroke volume remain raised [3]. The cardiac output can remain elevated (7 l/min) for about 24 h after delivery: it can rise 80 % above prelabor values and approximately 100 % above nonpregnant measurements. The cardiac output decreases to prelabor values 24–72 h postpartum, rapidly falls over the next 2 weeks and usually returns to nonpregnant levels within 6–8 weeks after delivery; however, this adaptation can even take 6 months [11, 12]. Blood pressure falls mostly in the first 2 days [13], and then increases 3–7 days after delivery; it returns to prepregnancy levels by 6 weeks. After delivery, left atrial size remains elevated for the first 48 h due to the increased venous return of the puerperium, with a loss of the uteroplacental shunt [14]. Most cardiovascular parameters are well resolved by 2 weeks, but a mild degree of ventricular hypertrophy may persist for several months [3]. The majority of hemodynamic changes return to prepregnancy values by 3 months, but in some women full resolution may take as long as 6 months. The blood volume decreases by 10 % 3 days postdelivery. Hemoglobin levels increase steadily over the first 2 weeks postpartum, and then they stabilize. Systemic vascular resistance increases over the first 2 weeks postpartum to 30 % above delivery values. Heart rate returns to baseline over 2 weeks after delivery. Women with valvular heart disease, like aortic or mitral stenosis, or coronary arterial disease can show a severe decompensation in myocardial function during labor and especially immediately after delivery [10]. Thus, hemodynamic changes during labor are of considerable relevance in managing mothers with complicated cardiovascular function.

Hemodynamic changes can be elicited even in parturients' cerebral perfusion. In laboring women, the mean arterial pressure rises in all stages of labor with a trend toward an increase in cerebral perfusion pressure and cerebral blood flow index. However, at the peak of a contraction and during pushing, cerebral perfusion pressure significantly falls and cerebrovascular resistance significantly rises, although cerebral blood flow does not change [15]. In all stages of labor, women undergoing epidural analgesia show a clinically insignificant decrease of cerebral perfusion pressure, cerebrovascular resistance, and cerebral blood flow index.

2.2 Anatomic-physiologic Pulmonary Changes During Labor and Delivery

The pain that women experience during labor is affected by multiple physiological factors, and its intensity can vary greatly. Many women hyperventilate during labor in response to painful contractions: it might have an adverse effect on fetal oxygenation. Values of more than 3 ml/l of oxygen ventilation have been calculated, as opposed to 0.65 ml/l in healthy males; extreme additional increases in ventilation up to 300 % may occur in response to the pain of advanced labor. The additional hyperventilation decreases the PaCO₂ values and left-shifted oxyhemoglobin dissociation curve. The maternal hypocapnea might impair uterine blood flow if values are below 20 mmHg. This may be ameliorated by effective epidural analgesia. Even very apprehensive mothers might have a lower PaCO₂ (until 16 mmHg) and a greater pH (7.64), and this is not ameliorated by epidural analgesia. Acid–base state is of particular interest to the anesthesiologist. During the first stage of labor, the acid–base balance shows signs of hyperventilation and lactic acid accumulation; serum potassium levels slightly rise during labor. The creatine phosphokinase level does not change during labor, but between 2 and 4 h after delivery it is significantly elevated [16]. Profound but short periods of hypoxemia have been observed during the second stage of labor when oxygen demand is greatest as a consequence of effort [17]. The alteration of pH rather than PaCO₂ might be the controlling factor, influencing placental vascular resistance, intraplacental shunting, and the maternal and fetal oxygen dissociation curves, even if the flow volume of the uterus doesn't show any change. Hyperventilation in labor determines maternal alkalemia and, with the further left shift of the oxyhemoglobin dissociation curve, may compromise fetal oxygenation because of a reflex spasm of the umbilical vein, fetal acidosis, and fetal tachycardia [18]. In addition, the close relationship between maternal carbon dioxide levels and fetal carbon dioxide levels affects fetal cerebral oxygenation by regulating cerebral blood flow and by shifting the oxyhemoglobin dissociation curve [19]. Some investigators found maternal transcutaneous partial pressure of carbon dioxide of 28 mmHg during the first stage of labor and 20.8 mmHg in the second stage of labor: these values have a significant positive correlation with the lower umbilical venous partial pressure of oxygen, umbilical venous P(CO₂), and umbilical venous oxyhemoglobin saturation. Thus, maternal hyperventilation may interfere with optimal fetal cerebral oxygenation [20]. Most of the significant pulmonary changes will be resolved by 2 weeks postpartum, but full resolution may take 6 months.

2.3 Neurologic Changes During Labor and Delivery

The anesthesiologist should be aware that the central and peripheral nervous systems undergo significant changes during labor. An increased sensitivity of the peripheral nerve to local anesthetic has been documented in parturients because of

the progesterone or one of its active metabolites and increased endorphin concentrations [21]. There is a similar reduction of about 30 % in dose requirements for local anesthetics, for both epidural and spinal administration [22]. These changes can remain up to 36 h postpartum [23]. The minimal alveolar concentration (MAC) is reduced by one-third so that parturients show an increased sensitivity to opioids, sedatives, and general anesthetic agents [24]. Recovery usually occurs by the third day postpartum [25].

2.4 Maternal Endocrine Stress Response During Labor and Delivery

The corticotropin-releasing hormone (CRH, also known as CRF) is a 41-amino-acid neuropeptide secreted by the paraventricular nucleus of the hypothalamus in response to stress. Its major role is the regulation of the hypothalamus–pituitary–adrenal axis by the stimulation of ACTH release from the anterior pituitary gland [26]. In addition, CRF modulates behavioral, vascular, and immune responses to stress. During pregnancy, CRF is synthesized in large amounts by the placenta and released into the maternal and fetal circulations: the stimulation of fetal pituitary ACTH and fetal adrenal gland dehydroepiandrosterone sulfate release *in vitro* has been shown [27]. The placental CRF is potentially implicated in the timing of human delivery and in the physiology of parturition. The premature or accelerated activation of the placental corticotropin-releasing hormone system, as reflected by the precocious elevation of maternal CRF levels, may be associated with an earlier onset of spontaneous labor and resultant delivery, and it may be a marker of antepartum risk for preterm delivery and, therefore, it could be an indirect predictor of earlier delivery [28]. Urocortin is a 40-amino acid peptide belonging to the CRF family, expressed by human trophoblast, fetal membranes, and human placenta. CRF and urocortin share some of their biologic effects and their actions on the same specific receptors are mediated by cyclic adenosine monophosphate as a second messenger. A large-molecular-weight corticotropin-releasing factor-binding protein modulates the activity of both these peptides: they are potent local regulators of myometrial contractility and of membrane prostaglandin release. Plasma CRF and urocortin levels are higher in labor than those previously reported during pregnancy, but they do not change significantly during the different stages of labor when evaluated longitudinally [29]. Vaginal delivery is a condition associated with the highest values of maternal CRF factor levels. CRF and urocortin levels are both increased at term and preterm labor and correlate with the time of labor onset after induction. However, CRF levels are reduced and urocortin levels remain unchanged in women who are destined to experience post-term delivery. Since CRF derives from the placenta and urocortin from the fetus, the concerted expression of these neuropeptides appears to be relevant in determining the length of human gestation [30]. Increased maternal plasma CRF factor levels characterize some gestational diseases like chronic hypertension and preeclampsia; the intrauterine growth retardation is associated with an activation of the hypothalamus–pituitary–adrenal axis, reflected by increased fetal plasma concentrations of ACTH, cortisol, and CRF. In these various pathologic

states, maternal plasma corticotropin-releasing factor-binding protein levels undergo opposite changes, decreasing to very low levels. The endocrine-paracrine corticotropin-releasing factor/corticotropin-releasing factor-binding protein pathways are involved in the mechanism of human parturition [27].

Many hormones are involved during labor and delivery. The ACTH rises during all stages of labor, reaches its peak at the moment of delivery, and then rapidly decreases within 30 min. Cortisol secretion reaches its maximum during the first stage of labor and at delivery and soon after it falls by 30 min. The TSH level doesn't change during labor significantly [31]. Catecholamines and 17-alpha-hydroxyprogesterone concentrations rise during all stages of labor and decrease by 30 min after delivery. Epidural analgesia reduces maternal stress hormones at delivery, but it seems to have little or no effect on fetal endocrine stress hormones [32].

The maternal growth hormone does not change during labor. Insulin decreases during the first stage of labor, and thereafter it increases. Glucose levels do not change during the first stage, but at the moment of delivery they are significantly higher than initial levels, they remain high until 30 min after delivery, and then they rapidly fall. Free fatty acids increase as labor progresses reaching a peak at the delivery time [33]. The maternal plasma prolactin concentrations decline during labor reaching the lowest values during the first stage of labor [34]. Plasma oxytocin does not play a primary role in the initiation of labor but contributes to the formation of prostaglandins through the uterine contractions plasma oxytocin produces. Plasma oxytocin spikes have been observed to occur during labor in association with membrane rupture, vaginal examination, descent of the vertex, and especially with maximal cervical and vaginal distension. This surge represents the evidence that the Ferguson reflex exists in women. Moreover, an excess of plasma oxytocin in fetal blood is associated with hypertonic, irregular, tumultuous or prolonged labor and with mild-to-moderate fetal hypoxia and fetal distress peculiar to abnormal uterine contractions [35].

Maternal temperature during labor is affected in part by the amount of hyperventilation, perspiration, and physical activity. Calm and less active parturients have high temperatures. Neonatal rectal temperature immediately after delivery is best correlated with maternal vaginal delivery [36]. Elevated maternal temperature in labor is associated with adverse immediate and long-term neonatal outcomes. The most reliable noninvasive method of temperature monitoring in labor seems to be oral temperature having an acceptable correlation with intrauterine temperature which constitutes the fetal environment. While the temperature at the ear canal, the skin surface of the leg, and the abdomen increases as labor progresses, the temperature at the mouth is lower than the intrauterine temperature on average by 0.8 °C [37].

2.5 Hematologic Changes During Labor and Delivery

Changes in hemostasis take place around delivery time, especially at the end of labor. The coagulation and fibrinolytic systems are both further activated at delivery by placental separation with thromboplastin release, and these changes are a

protective adaptation for parturition and the risk of acute hemorrhage. At the time of delivery, there is a decrease in the levels of factors XII and XI, an increase of fibrinopeptide A, beta-thomboglobulin, and platelet factor 4, suggesting maximum platelet activation and fibrin formation. At 3 h postpartum, there is maximum fibrinolysis with an increase of D-dimer and a decrease of antiplasmin levels [38, 39]. Women remain hypercoagulable through the first 24 h after delivery, demonstrated by thromboelastography [40]. Hemostatic systems return to the normal nonpregnant state by the end of the third or fourth week postpartum [41].

Serum glutammico oxalacetic transaminase, lactic dehydrogenase, and alkaline phosphatase levels are also elevated during labor.

2.6 Anatomical-physiological Changes to Be Considered in Performing General Anesthesia in Laboring Women

General anesthesia should be avoided when it's possible in laboring women because they are at risk of gastric content aspiration as the stomach remains full before delivery. Gastric emptying time is significantly slower during labor, gastric volume is increased, and the enlarged gravid uterus divides the stomach into fundal and antral parts so that gastric pressure increases. Laboring women should always be considered to have a full stomach irrespective of the time of the last meal. In addition, narcotic analgesics decrease the tone at the lower esophageal sphincter, delay gastric emptying in labor, and make regurgitation more likely [42]. Recently, gastric content volume and its changes have been evaluated during labor using the measurement of the antral cross-sectional area. Even under epidural analgesia, authors have found a decrease in gastric volume during labor and a preserved gastric motility [43]. Gastrointestinal changes revert back to nonpregnant states within 6 weeks postpartum although the mechanical effects of the gravid uterus on the stomach are resolved in a few days.

Another reason to avoid general anesthesia is the variable activity during labor of the serum cholinesterase, a mucoprotein the importance of which lies in its ability to hydrolyze and to inactivate drugs like succinylcholine and procaine. In fact, a rise of cholinesterase activity occurs during labor (70.3 units/ml) as compared to late pregnancy (60.2 units/ml), but the increased values are significantly lower than those in the nonpregnant state (83.3 units/ml). A consistent decrease of serum cholinesterase activity has been noted 1 day (62.8 units/ml), 3 days postpartum (56.4 units/ml), and up to the sixth day, and it returns to normal by the sixth week [44, 45]. Some investigators reported reduced enzyme levels up to 25 % during labor with respect to late pregnancy and 4 h postpartum [46]. Different authors found a slight increase of serum cholinesterase during labor, a decrease up to 4 days postpartum, and an increase to normal values around the 12th postpartum day [28]. Other studies reported an increase in enzyme activity during labor as compared to late pregnancy and then a further increase in the immediate postpartum period [47]. The significance of reduced serum cholinesterase activity is that

abnormal neuromuscular response to succinylcholine has been occasionally observed during labor and particularly in the immediate postpartum period [48]. Usually, a normal dose of succinylcholine for intubation (1–1.5 mg/kg) is not associated with prolonged neuromuscular blockade [49].

There is another aspect that should be considered when the anesthesiologist is called to perform a general anesthesia during labor. Airway changes have been observed during the course of pregnancy: there is an increase in the number of the Samssoon modification of the Mallampati class 4 by 34 % at 38 weeks from that at 12 weeks of gestation due to fluid retention [50]. In laboring women, there is a further change in airways [51] owing to pharyngolaryngeal edema consequent to fluid overload in conjunction with the antidiuretic properties of oxytocin and to prolonged strenuous bear-down efforts [52–54]: it may contribute to difficult intubation. The upper airway (airway volume) has two components: the oral component, which can be assessed by Mallampati classification, and the pharyngeal component, which can be evaluated using acoustic reflectometry airway volumes. An airway volume less than 40.2 ml has been associated with a diminished ability to view glottis openings in nonpregnant subjects undergoing general anesthesia and intubation [54]. Some authors have studied the upper airways using the Samssoon modification of Mallampati, the standard airway evaluation criteria that are currently in daily practise, and the method of acoustic reflectometry, a noninvasive test, which matches computed tomography-measured airway volumes [55] and predicts the complete inability to ventilate a patient via a mask [56]. They demonstrated an increase in the Mallampati classification of one grade higher and two grades higher in 33 % and in 5 % of laboring women, respectively, and they found 50 % of parturients at the end of labor showing a class 3 or class 4 airway. The relative risk of difficult intubation in pregnant women with a class 3 airway is 7.58 times more, and increased to 11.3 times more, in women with class 4 compared with parturients with class 1 airway. This means that a change in airway from 2 to 4 in laboring women is associated with enhanced relative risk of encountering difficult intubation from 3.23 to 11.3. The women with a worsened airway class during and after labor reverted to the admission grade level within 36–48 h postpartum. The investigators also observed a significant decrease in oral airway volume, pharyngeal volume, and mean pharyngeal area after labor and delivery as compared with prelabor values. No data demonstrate the relationship between pharyngeal volume and intubation difficulties, but it is reasonable that decreasing pharyngeal volume could become an impediment to intubation. A decreased pharyngeal volume after labor assumes great importance in parturients who have Mallampati class 4 at the beginning of labor. Therefore, it is prudent to insert an epidural catheter in those parturients with a complicated labor, and it is essential to carefully reevaluate the upper airways in laboring women presenting for urgent or emergent cesarean delivery just before the commencement of anesthesia rather than obtaining this information from prelabor data, especially if there are factors increasing the risk of difficult intubation like short neck, receding mandible, protruding maxillary incisors, and morbid obesity [57, 58].

References

1. Ouzounian JG, Elkayam U (2012) Physiologic changes during normal pregnancy and delivery. *Cardiol Clin* 30:317–329
2. Robson SC, Hunter S, Boys RJ et al (1987) Cardiac output during labour. *Br Med J (Clin Res Ed)* 295:1169–1172
3. Lees MM, Scott DB, Slawson KB et al (1968) Haemodynamic changes during caesarean section. *J Obstet Gynaecol Br Commonw* 75:546–551
4. Hendricks CH (1955) The hemodynamic of a uterine contraction. *Am J Obstet Gynecol* 76:969
5. Duvetkot JJ, Peeters LL (1994) Maternal cardiovascular hemodynamic adaptation to pregnancy. *Obstet Gynecol Surv* 49(Suppl 12):S1
6. Ueland K, Hansen JM (1969) Maternal cardiovascular dynamics. Part 3. Labor and delivery under local and caudal analgesia. *Am J Obstet Gynecol* 103:8–18
7. Segal S, Csavoy AN, Datta S (1998) The tocolytic effect of catecholamines in the gravid uterus. *Anesth Analg* 87:864–869
8. Neumark J, Hammerle AF, Biegelmayr C (1985) Effects of epidural analgesia on plasma catecholamines and cortisol in parturition. *Acta Anaesthesiol Scand* 29:555–559
9. Albright GA, Jouppila R, Hollmen AI et al (1981) Epinephrine does not alter human intervillous blood flow during epidural anesthesia. *Anesthesiology* 54:131–135
10. Camann WR, Ostheimer GW (1990) Physiological adaptations during pregnancy. *Int Anesthesiol Clin* 28:2–10
11. Capeless EIL, Clapp JR (1991) When do cardiovascular parameters return to their preconception values? *Am J Obstet Gynecol* 161:883–886
12. Robson SC, Hunter S, Moore M et al (1987) Haemodynamic changes during the puerperium: a Doppler and M-mode echocardiographic study. *Br J Obstet Gynecol* 94:1028–1039
13. Robson SC, Boys RJ, Hunter S et al (1989) Maternal hemodynamics after normal delivery and delivery complicated by postpartum hemorrhage. *Obstet Gynecol* 74:234–239
14. Robson SC, Hunter S, Dunlop W (1987) Left atrial dimensions during early puerperium. *Lancet* 2:111–112
15. Williams KP, Wilson S (1999) Evaluation of cerebral perfusion pressure changes in laboring women: effects of epidural anesthesia. *Ultrasound Obstet Gynecol* 14:393–396
16. Juppila R, Hollmen A (1976) The effect of segmental epidural analgesia on maternal and foetal acid-base balance, lactate, serum potassium and creatine phosphokinase during labor. *Acta Anaesthesiol Scand* 20:259–268
17. Reed PN, Colquhoun AD, Hanning CD (1989) Maternal oxygenation during normal labour. *Br J Anaesth* 62:316
18. Müller G, Huber JC, Salzer H et al (1984) Maternal hyperventilation as a possible cause of fetal tachycardia sub partu. A clinical and experimental study. *Gynecol Obstet Invest* 17:270–275
19. Tomimatsu T, Kakigano A, Mimura K et al (2013) Maternal carbon dioxide level during labor and its possible effect on fetal cerebral oxygenation: mini review. *J Obstet Gynecol Res* 39:1–6
20. Tomimatsu T, Kakigano A, Mimura K et al (2012) Maternal hyperventilation during labor revisited: its effect on fetal oxygenation. *Reprod Sci* 19:1169–1174
21. Palahniuk RJ, Shnider SM, Eger EI II (1974) Pregnancy decreases the requirement for inhaled anesthetic agent. *Anesthesiology* 41:82
22. Datta S, Hurley RJ, Naulty JS et al (1986) Plasma and cerebrospinal fluid progesterone concentrations in pregnant and non-pregnant women. *Anesth Analg* 65:950
23. Butterworth JF IV, Walker FO, Lysak SZ (1990) Pregnancy increases median nerve susceptibility to lidocaine. *Anesthesiology* 72:962
24. Gin T, Chan MT (1994) Decreased minimum alveolar concentration of isoflurane in pregnant humans. *Anesthesiology* 81:829
25. Chan MT, Gin T (1995) Postpartum changes in the minimum alveolar concentration of isoflurane. *Anesthesiology* 82:1360

26. Wadhwa PD, Porto M, Garite TJ et al (1998) Maternal corticotropin-releasing hormone levels in the early third trimester predict length of gestation in human pregnancy. *Am J Obstet Gynecol* 179:1079–1085
27. Reis FM, Fadalti M, Florio P et al (1999) Putative role of placental corticotropin-releasing factor in the mechanisms of human parturition. *J Soc Gynecol Investig* 6:109–119
28. Florio P, Cobellis L, Woodman J et al (2002) Levels of maternal plasma corticotropin-releasing factor and urocortin during labor. *J Soc Gynecol Investig* 9:233–237
29. Torricelli M, Ignacchiti E, Giovannelli A et al (2006) Maternal plasma corticotrophin-releasing factor and urocortin levels in post-term pregnancies. *Eur J Endocrinol* 154:281–285
30. Juppila R, Hollmen A, Juppila P et al (1976) The effect of segmental epidural analgesia on maternal ACTH, cortisol and TSH during labour. *Ann Clin Res* 8:378–384
31. Westgren M, Lindahl SG, Norden NE (1986) Maternal and fetal endocrine stress response at vaginal delivery with and without an epidural block. *J Perinat Med* 14:235–241
32. Juppila R (1976) The effect of segmental epidural analgesia on maternal growth hormone, insulin, glucose and free fatty acids. *Ann Chir Gynaecol* 65:398–404
33. Juppila R, Juppila P, Moilanen K et al (1980) The effect of segmental epidural analgesia on maternal prolactin during labour. *Br J Obstet Gynaecol* 87:234–238
34. Vasicka A, Kumaresan P, Han GS et al (1978) Plasma oxytocin in initiation of labor. *Am J Obstet Gynecol* 130:263–273
35. Goodlin RC, Chapin JW (1982) Determinants of maternal temperature during labor. *Am J Obstet Gynecol* 143:97–103
36. Benerjee S, Cashman P, Yentis SM et al (2004) Maternal temperature monitoring during labor: concordance and variability among monitoring sites. *Obstet Gynecol* 103:287–293
37. Gerbasi FR, Bottoms S, Farag A et al (1990) Changes in hemostasis activity during delivery and the immediate postpartum period. *Am J Obstet Gynecol* 162:1158–1163
38. Hellgren M, Blomback M (1981) Studies on blood coagulation and fibrinolysis in pregnancy, during delivery and in the puerperium. I. Normal condition. *Gynecol Obstet Invest* 12:141–154
39. SharmaSK PJ, Wiley J (1997) Thromboelastographic changes in healthy parturients and postpartum women. *Anesth Analg* 85:94
40. Dahlman T, Hellgren M, Blomback M (1985) Changes in blood coagulation and fibrinolysis in the normal puerperium. *Gynecol Obstet Invest* 20:37–44
41. Holdsworth JD (1979) Relationship between stomach contents and analgesia in labor. *Br J Anaesth* 50:1145–1148
42. Bataille A, Rousset J, Marret E et al (2014) Ultrasonographic evaluation of gastric content during labor under epidural analgesia: prospective comfort study. *Br J Anaesth* 112:703–707
43. Pritchard JA (1961) Plasma cholinesterase activity in normal pregnancy and eclamptogenic toxemia. *Am J Obstet Gynecol* 82:132
44. Shnider Sol M (1965) Serum cholinesterase activity during pregnancy, labor and puerperium. *Anesthesiology* 26:335–339
45. Navratil E (1939) Effect of birth on serum cholinesterase. *Arch Gynak* 168:178
46. Picolli R, Longo G (1947) Serum cholinesterase in obstetrics; values during parturition and puerperium. *Boll Soc Ital Biol Sper* 23:486
47. Stefanelli S, Petronio G (1948) Relationships between serum cholinesterase activity and blood acetylcholine in labor and childbed. The influence of some substances (histamine, pituitrin, folliculin) on cholinesterase activity. *Boll Soc Ital Biol Sper* 24:644
48. Hodges RJH, Bennet JR, Turnstall ME et al (1959) Effects of oxytocin on the response to suxamethonium. *Br Med J* 1:413
49. Cohen SE (1982) Who is the pregnant patient different? *Semin Anesth* 1:3
50. Pilkington S, Carli F, Dakin MJ (1995) Increase of Mallampati score during pregnancy. *Br J Anaesth* 74:638–642
51. Farcon EL, Kim MH, Marx GF (1995) Changing Mallampati score during labor. *Can J Anaesth* 74:638–642

52. Heller PJ, Scheider EP, Marx GF (1983) Pharyngolaryngeal edema as a presenting symptom in preeclampsia. *Obstet Gynecol* 62:523–524
53. Juppila R, Juppila P, Hollmen A (1980) Laryngeal o edema as an obstetrics anaesthesia complication. *Acta Anaesthesiol Scand* 24:97–98
54. Ekmann DM, Glassemberg R, Gavriely N (1996) Acoustic reflectometry and endotracheal intubation. *Anesth Analg* 83:1084–1089
55. D’Urso AD, Rubinstein I, Lawson VG et al (1988) Comparison of glottidis areas measured by acoustic reflections versus computerized tomography. *J Appl Physiol* 64:367–370
56. Ochroch EA, Eckmann DM (2002) Clinical application of acoustic reflectometry in predicting difficult airway. *Anesth Analg* 95:645–649
57. Rocke DA, Murray WB, Rput CC et al (1992) Relative risk analysis of factors associated with difficult intubation in obstetric anesthesia. *Anesthesiology* 77:67–73
58. Hood D, Dewan D (1993) Anesthesia and obstetric outcome in morbidity obese parturients. *Anesthesiology* 79:1210–1218

York-Mui Liu, Roshan Fernando, and Wint Yu Mon

3.1 Introduction

The pain experienced in labor by women has been described by some as the worst pain ever experienced [1]. Pain perception during labor changes in intensity and nature as labor progresses, and this is associated with the behavioral changes in the laboring woman. However, these behavioral changes are not uniform, suggesting that the perception and intensity of the pain may be modulated by various emotional factors. This chapter will discuss the basics relating to the transmission of pain signals from the periphery to the central nervous system as well as discuss the changing nature of labor pain. The ways in which labor pain perception can be measured and modulated will also be discussed.

3.2 Pain Pathways

Pain has been described as an “unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage” [2]. As expected, the pathways, which transmit such complex sensations, are equally complex themselves. The major pathway which transmits pain (and temperature) from the body to the brain (Fig. 3.1) is known as the spinothalamic tract and consists of several components [3]:

1. Medium sized A δ and small unmyelinated C nerve fibers transmit signals from peripheral nociceptors, which then enter the spinal cord through the lateral division of the dorsal horns. These axons then form the Tract of Lissauer which travels up and down for one or two spinal segments on the same

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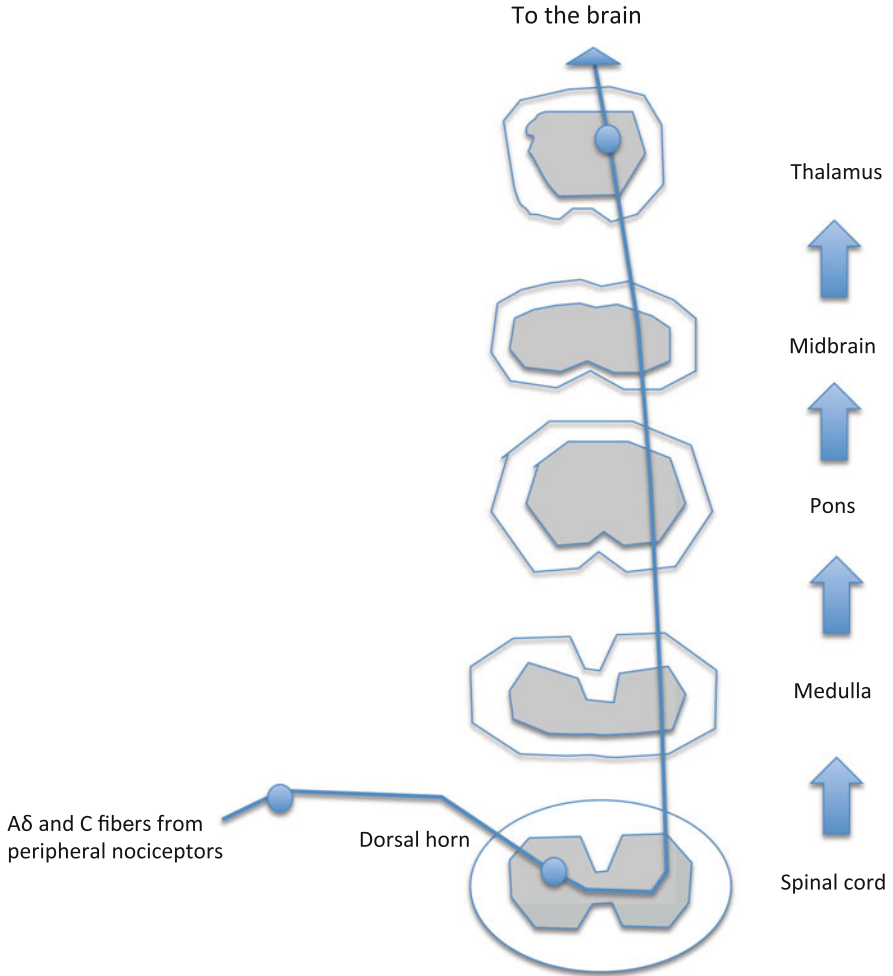


Fig. 3.1 The course of the spinothalamic tract

(ipsilateral) side of the spinal cord. These axons then enter the gray matter of the spinal cord and send projections to neurons in Rexed's laminae I (also known as the marginal zone), II (also known as the substantia gelatinosa), III, and IV (Fig. 3.2).

2. Axons in Rexed's laminae I–IV synapse with second-order neurons in Rexed's laminae V, VI, VII, and VIII, which are collectively known as the nucleus proprius. Some of the axons in Rexed's lamina I synapse with second-order neurons located within the same lamina. These second-order neurons from Rexed's laminae V–VIII along with second-order neurons from Rexed's lamina

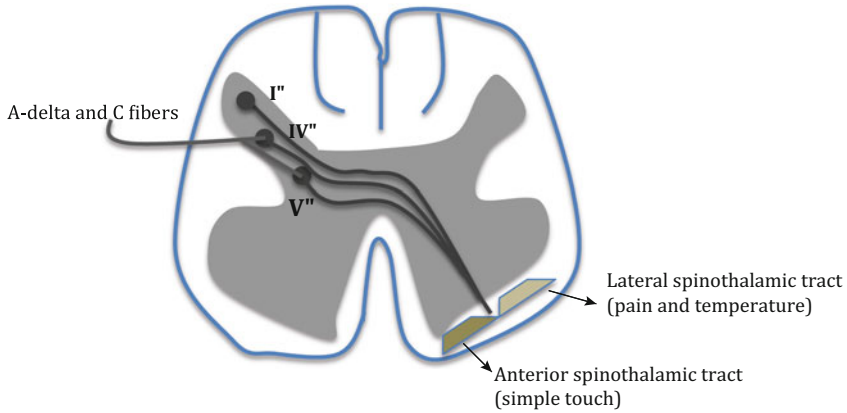


Fig. 3.2 Spinothalamic tract

I have axons, which cross the midline in the anterior white commissure and ascend to the brainstem and thalamus in the anterolateral quadrant on the contralateral half of the spinal cord as the spinothalamic tract. Pain fibers from the sacral and lower areas of the body are located laterally in the spinothalamic tract, whereas those transmitting pain from the upper half of the body are found on the medial side of the tract.

- Once in the brain, the second-order neurons synapse and terminate with neurons found in the ventro posterolateral nucleus (VPL) of the thalamus. These third-order neurons have projections to various parts of the brain such as the frontal cortex and the anterior cingulate gyrus which then modulate both the emotional and behavioral response to pain via descending pathways.

A similar pathway known as the trigeminal pain and temperature system carries pain and temperature sensations from the face to the brain.

There are two types of pain which are experienced in pregnancy:

- Visceral pain*—this is the pain transmitted by nociceptors from internal organs and may be referred to areas of the body distant to the organ. This type of pain is typically vague and difficult to localize.
- Somatic pain*—this is the pain transmitted by nociceptors in the skin and deep tissues. The pain by comparison with visceral pain is localized to the area where the nociceptors have been stimulated.

3.3 Innervation of the Uterus and Cervix During Pregnancy and Labor

The uterus is functionally formed of two components: the cervix and the body (corpus uteri). The uterus is supplied by both somatic and autonomic nerve fibers from the hypogastric plexus. The hypogastric plexus is a continuation of the aortic plexus and is found anterior to the terminal aorta, fifth lumbar vertebra, and the sacral promontory [4]. The afferent (sensory) fibers, which transmit pain from the uterus, travel in close association with sympathetic nerve fibers in the hypogastric plexus to the sympathetic chain before entering the spinal cord [5, 6]. In addition, efferent nerve fibers travel from the spinal cord via the hypogastric plexus to modulate smooth muscle activity in the cervix [7–10]. The parasympathetic supply to the uterus is from the second, third, and fourth sacral segments, collectively known as the pudendal nerve [4].

As pregnancy progresses, the nerve supply to the uterus undergoes extensive changes. The corpus uteri becomes progressively denervated as the gravid uterus increases in size, but the dense network of nerves from the hypogastric plexus to the cervix remains unchanged.

Throughout pregnancy, the cervix remains a rigid, immobile structure, which is closed and acts to protect the developing fetus from the external vaginal environment. At the onset of labor, the cervix undergoes extensive remodeling to become soft and progressively dilates to facilitate the delivery of the fetus. This process of cervical change in preparation for delivery is known as cervical ripening. What has been shown is that as cervical dilatation progresses throughout labor, the intensity of pain experienced by women increases [11]. Rat models have demonstrated increased expression of cFos, a protein, which is found in spinal cord neurons in response to painful stimuli, with progressive cervical dilatation [12]. Transection of the hypogastric nerve in rats has been associated with prevention in the increase of pain intensity as labor progresses [13] along with altered behavioral changes [14] and reduced pain perception following dilatation of the uterus [15].

The transient receptor potential vanilloid receptor subtype 1 (TRPV1) is a receptor, which exists in sensory nerve endings and plays a role in the transmission of nociceptive stimuli. The receptor responds primarily to capsaicin, an active component in chili peppers, and heat, and its presence continues to be observed in the cervix throughout pregnancy, cervical ripening, and labor [16]. Application of capsaicin to TRPV1 receptors in the cervix in mice shows a biphasic response: there is an initial burning sensation associated with nerve depolarization followed by a reduction in labor pain behavioral activity as a result of decreased nerve transmission [17].

The process of cervical ripening has been observed to be an immune-mediated inflammatory process [18–20] with the migration of macrophages [21] and inflammatory mediators [22–26] to the cervix as it undergoes extensive changes. This process appears to be mediated by nerve fibers separate to the hypogastric plexus as

transection of the plexus in pregnant mice did not stop the onset of labor and delivery of pups [27].

3.4 Neuroendocrine Aspects of Labor Pain

The pain experienced by women during labor is a complex process with both sensory and affective components, and studies have shown a variable response to pain between parturients [1, 28]. Melzack and Wall described how the perception and interpretation of pain could be modified by various behavioral, hormonal, and emotional factors by describing their Gate Control Theory [29] and introducing the concept of the Neuromatrix [30, 31]. The Neuromatrix or rather the Pain Matrix as it is known now [32] is a collection of different regions of the brain with neuronal inputs to the periaqueductal gray (PAG), which modulates the descending pain pathways to produce a response to the noxious stimulus. Imaging of the brain has shown that by distracting subjects when applying heat stimuli can actually lower their response to pain, and this is reflected in altered signals in different regions of the pain matrix [33].

During labor, there is an increase in plasma catecholamines in response to the pain and anxiety felt in labor. The uterus has both α - and β -adrenergic receptors to which both adrenaline (epinephrine) and noradrenaline (norepinephrine) may bind. Studies in pregnant rats have shown that when levels of adrenaline and noradrenaline rise to levels seen in times of stress, they have a tocolytic effect on uterine contractions [34]. Uterine contractions return when levels of catecholamines are reduced or their effects are antagonized through the use of propranolol or phentolamine [34]. This implies that high levels of stress and anxiety have a negative effect on the progress of labor, and any measure which can reduce the levels of stress such as effective labor analgesia could be beneficial.

Oxytocin is a peptide made up of nine amino acids. It is secreted by the posterior pituitary gland, and studies in rabbits [35], sheep [36], cows [37], and rhesus monkeys [38] have shown a pattern of secretion which is pulsatile and is maximal at the time of delivery of the fetus. Oxytocin binds to the oxytocin receptor, which is found in the uterine tissues and stimulates uterine contractions. The secretion of oxytocin is enhanced by Ferguson's reflex where sensory stimuli transmitted by sacral afferents travel to the midbrain to increase oxytocin release. This reflex is disrupted in spinal cord injury [39]. The use of epidural analgesia has also been shown to reduce the secretion of oxytocin [40] and therefore potentially delay the progress of labor.

At a molecular level, the transmission and propagation of pain from peripheral receptors to nerve fibers depend on the expression of various neurotransmitters at the nerve terminals. Substance P and vasoactive intestinal peptide (VIP) are examples of such neurotransmitters, which are involved in response to painful stimuli. They have been found in the nerve terminals of the hypogastric plexus

supplying the cervix [41, 42] and were originally believed to be involved in the transmission of pain experienced during labor. However, subsequent work has suggested that in the later stages of pregnancy there is a reduction in the level of plasma Substance P not associated with hemodilution [43], and that during acute labor pain, the plasma levels of Substance P appear unchanged [44].

In times of stress, the hypothalamic–pituitary axis (HPA) is activated to produce an increase in the so-called stress hormones, which prepare the body for “fight–flight”. The pain experienced in labor produces a similar response where corticotrophin-releasing factor (CRF) is produced by the hypothalamus to cause an increase in the production of the peptides β -endorphin (a neurotransmitter which modulates pain by binding to opioid receptors) and adrenocorticotrophic hormone (ACTH). β -Endorphin is also produced by the human placenta in pregnancy [45]. Studies have shown that levels of both β -endorphin and ACTH rise during pregnancy, peak at the time of delivery, and fall in the first 24 h postpartum [46, 47]. Women who had lower levels of β -endorphin toward the end of pregnancy tended to experience more pain and were more likely to request other forms of analgesia [47]. Conversely, women who exercised during pregnancy and consequently had higher levels of β -endorphin experienced less pain than those who had not exercised [48]. The analgesic effects of β -endorphin can be abolished through the administration of an opioid antagonist [49, 50]. Interestingly, the use of transcutaneous electrical nerve stimulation (TENS) therapy for labor analgesia is thought to work through a rise in β -endorphin levels [51].

Another neurohumoral change in pregnancy, which affects the perception of pain, is progesterone. Not only is the plasma concentration of progesterone raised in pregnancy, so too is the cerebrospinal fluid (CSF) concentration, and it may be this which is responsible for the reduced requirement for local anesthetic during pregnancy. Datta et al. demonstrated that the levels of progesterone in the CSF were eight times higher in pregnant women compared with nonpregnant women and that this decreased postpartum. They also demonstrated that the changes in CSF progesterone levels were inversely correlated with the dose of local anesthetic required in the neuraxial block and postulated that this was a direct effect of progesterone on the ability of the nerves to conduct painful stimuli [52]. The reduced need for a local anesthetic during pregnancy is well known [53] and may directly result from increased CSF progesterone.

3.5 Topography of Pain During Different Stages of Labor

The pain in labor is not a uniform pain experience; rather, it changes depending on the stage and progress of labor. Classically labor is divided into three stages:

1. *First stage of labor*—this stage begins with cervical ripening and lasts until the cervix is 10 cm dilated. This pain results from the physical stretching and distension in the lower uterine segment and cervix. Pain signals are conveyed

by unmyelinated slowly conducting C visceral fibers [54], which pass through both the superior and inferior hypogastric plexus to sympathetic ganglia at T10—L1. Early on in the first stage of labor, the pain is initially referred to the T11 and 12 dermatomes with progression to T10 and L1 dermatomes as cervical dilatation continues [55].

2. *Second (expulsive) stage of labor*—this stage lasts from full cervical dilatation to the delivery of the baby. The presenting part of the fetus causes distension and stretching of the pelvic floor vagina and perineum. Small myelinated A δ nerve fibers [54] transmit pain sensation via the pudendal nerve located at S2–4. The pain of the second stage of labor is localized to the vagina and the perineum.
3. *Third stage of labor*—this stage covers the delivery of the placenta.

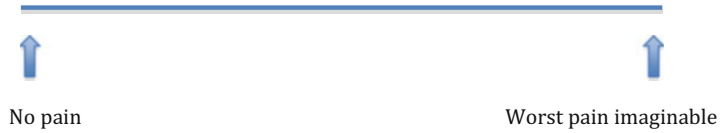
3.6 Labor Pain Evaluation

The pain experienced by women in labor not only has a sensory component but also has an affective element to it. Because this affective element is so subjective and dependent on the individual, this has made the pain in labor difficult to quantify; it makes comparisons between groups of parturients difficult [56]. There are various different methods used in the literature, which attempt to quantify the intensity of labor pain.

The visual analogue pain scale (VAPS) is a method used to assess pain other than that experienced in labor. The scale consists of a 10 cm vertical or horizontal line where at one end it is marked “no pain” and the other end is marked “severe pain”. Subjects are then asked to put a mark on the line where they believe the severity of their pain in question lies and the mark is measured from the end marked “no pain” and the distance to the closest 0.5 cm gives the severity of pain. The VAPS is a simple research tool which can be applied to the obstetric population [57], but it only gives a measure of pain intensity and does not give a measure of any of the characteristics of pain.

The verbal rating scale (VRS) is similar to the VAPS in that it measures pain intensity rather than characteristics. Instead of asking subjects to mark on a 10-cm line the severity of the pain, the subjects are asked instead to rate their pain using qualitative words such as “mild,” “moderate,” or “severe” (Fig. 3.3).

The numeric rating scale (NRS) again is similar to VAPS, but instead of marking a point on a 10-cm line, patients are asked to provide a numerical value to quantify their pain. As labor is a dynamic process and the intensity of the pain may change very quickly, the use of both VAPS and VRS may not capture the magnitude of change during each contraction as both these scales are applied at discrete times during labor, e.g., at a specific cervical dilatation [58]. Bonnel looked at the use of a Behavioral Index (BI, Fig. 3.4) which could be used during each contraction to objectively gauge the severity of the pain experienced by the parturient [59]. In this study, obstetricians or midwives were given a five-point scale on which they grade



Numerical Rating Scale (NRS)

| Score (out of 10) | Severity of pain |
|-------------------|------------------|
| 0 | No pain |
| 1,2,or 3 | Mild pain |
| 4,5, or 6 | Moderate pain |
| 7,8,9, or 10 | Severe pain |

Fig. 3.3 VAPS, VRS, and NRS

| Intensity of labor pain | Observed behavior |
|-------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| 0 | Normal respiration, no grasping, or agitated behavior seen |
| 1 | Rate and depth of respiration changes with labor contractions, all behaviors are attributed to pain, whether intentional (as a result of antenatal training) or reactional |
| 2 | As 1, signs of tension during contractions including grasping of bed, sheets, or another person’s hand, these behaviors stop when contraction has ended |
| 3 | As 2, but grasping reaction persists even after contraction has ended |
| 4 | Signs of agitation occur during and even between contractions |

Fig. 3.4 Behavioral changes seen with increasing labor pain intensity

the behavioral response exhibited by the women during the contractions. The authors found that as cervical dilatation increased and labor progressed, the observed behavior was placed in the higher two categories which correlate with severe pain and increased levels of anxiety in the parturient. However, although the BI may be considered an objective measure of pain severity in labor, its reliance on the observer who may also be the main care provider to the parturient in labor can be subject to an ethnic variation [60].

The McGill Pain Questionnaire (MPQ, Fig. 3.5) is made up from 20 descriptors which assess the characteristics of pain and a present pain intensity (PPI) index which incorporates five graded words to gauge current pain severity. This multidimensional questionnaire was first described by Melzack in 1975 and consists of 20 words which have been derived to represent different pain severities and are also considered to relate to three components of pain: sensory, affective, and evaluative [61]. The MPQ takes about 5–10 min to complete and has been used to assess pain in labor where women rate the pain they experienced highly, only superseded by digit amputation and complex regional pain syndrome (causalgia) [62]. Niven also used the MPQ to assess labor pain and noted in her study that if the parturients had previous experience of pain unrelated to labor or childbirth, their perception of pain was less when compared with women who had not experienced any pain previously [63].

However, if the MPQ is to be used to assess pain in labor, then a questionnaire, which may take up to 10 min to complete, may be considered cumbersome and may not accurately reflect the changes in pain as labor progresses. With this in mind, a shortened form of the MPQ (SF-MPQ) was developed and validated for use in pain research [64] (Fig. 3.6). The SF-MPQ comprises 15 descriptors (11 sensory and 4 affective), PPI, and a VAPS and takes 2–5 min to complete. The SF-MPQ has been used to study pain in the obstetric population by Capogna where he found that in nulliparous women in the early stages of labor, the intensity of affective and evaluative descriptors was greater than in multiparous women. In both groups, the intensity of both pain and sensory descriptors strongly correlated with the intensity of both VAPS and PPI as labor progressed [65].

3.7 Cognitive and Functional Aspects of Labor Pain

Dick-Read introduced the concept of “Childbirth without fear” [66] where it was hypothesized that increased fear led to increased muscular tension which in turn prolonged labor and increased pain. By educating the expectant women, it was postulated that tension and fear could be reduced through relaxation and breathing techniques. “Childbirth without pain” was a concept introduced by Lamaze [67]

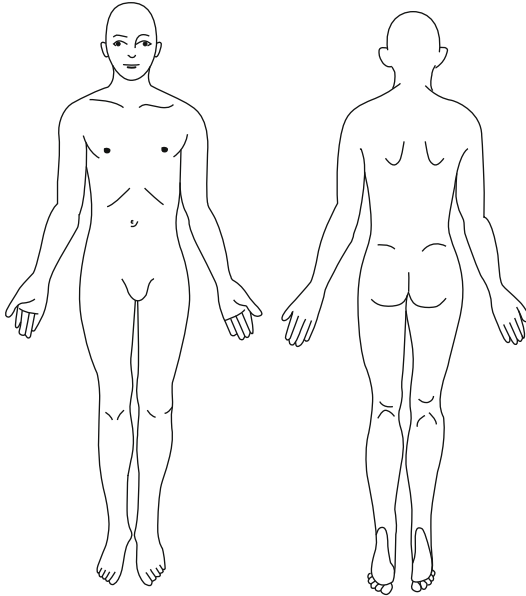
What does your pain feel like?

| | | | |
|---------------------------------------------------------------------------|----------------------------|-------------------------------------------------------------------|-----------------------|
| 1. Flickering Quivering Pulsing Throbbing Beating Pounding | — — — — — — | 11. Tiring Exhausting | — — |
| 2. Jumping Flashing Shooting | — — — | 12. Sickening Suffocating | — — |
| 3. Pricking Boring Drilling Stabbing Lancinating | — — — — — | 13. Fearful Frightful Terrifying | — — — |
| 4. Sharp Cutting Lacerating | — — — | 14. Punishing Grueling Cruel Vicious Killing | — — — — — |
| 5. Pinching Pressing Gnawing Cramping Crushing | — — — — — | 15. Wretched Blinding | — — |
| 6. Tugging Pulling Wrenching | — — — | 16. Annoying Troublesome Miserable Intense Unbearable | — — — — — |
| 7. Hot Burning Scalding Searing | — — — — | 17. Spreading Radiating Penetrating Piercing | — — — — |
| 8. Tingling Itchy Smarting Stinging | — — — — | 18. Tight Numb Drawing Squeezing Tearing | — — — — — |
| 9. Dull Sore Hurting Aching Heavy | — — — — — | 19. Cool Cold Freezing | — — — |
| 10. Tender Taut Rasping Splitting | — — — — | 20. Nagging Nauseating Agonizing Dreadful Torturing | — — — — — |
| | | How strong is your pain? | |
| | | 0 No pain | |
| | | 1 Mild | |
| | | 2 Discomforting | |
| | | 3 Distressing | |
| | | 4 Horrible | |
| | | 5 Excruciating | |

Fig. 3.5 (continued)

Where is your pain?

Please mark on the drawings where you feel pain.



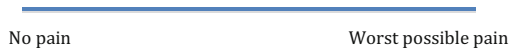
| | | | | | |
|------------------------------|-----|-----------------|-----|--------------------|-----|
| Accompanying Symptoms | | Duration | | Activity | |
| Nausea | ___ | Constant | ___ | Good | ___ |
| Headache | ___ | Periodic | ___ | Some | ___ |
| Dizziness | ___ | Brief | ___ | Little | ___ |
| Drowsiness | ___ | | | None | ___ |
| Constipation | ___ | Sleep | | | |
| Diarrhea | ___ | Good | ___ | Food intake | |
| | | Fitful | ___ | Good | ___ |
| | | Can't sleep | ___ | Some | ___ |
| | | | | Little | ___ |
| | | | | None | ___ |

Fig. 3.5 McGill Pain Questionnaire [61]. Copyright: Dr. R. Melzack, 1970, 1975. Reprinted with permission

where it was believed that by using relaxation techniques and breathing exercises, it would be possible to block or inhibit the pain signals associated with uterine contractions. Researchers have yet to provide conclusive evidence that such cognitive techniques are wholly effective [62]. However, studies looking at behavioral aspects of women in preparation for labor and childbirth have shown that the negative experience and pain of labor and childbirth could be reduced by encouraging women to believe they can cope with the pain [68–73], having the presence of

| Pain descriptors - sensory dimension | None | Mild | Moderate | Severe |
|-----------------------------------------------|-------------|-------------|-----------------|---------------|
| Throbbing | 0) ____ | 1) ____ | 2) ____ | 3) ____ |
| Soothing | 0) ____ | 1) ____ | 2) ____ | 3) ____ |
| Stabbing | 0) ____ | 1) ____ | 2) ____ | 3) ____ |
| Sharp | 0) ____ | 1) ____ | 2) ____ | 3) ____ |
| Cramping | 0) ____ | 1) ____ | 2) ____ | 3) ____ |
| Gnawing | 0) ____ | 1) ____ | 2) ____ | 3) ____ |
| Hot - burning | 0) ____ | 1) ____ | 2) ____ | 3) ____ |
| Aching | 0) ____ | 1) ____ | 2) ____ | 3) ____ |
| Heavy | 0) ____ | 1) ____ | 2) ____ | 3) ____ |
| Tender | 0) ____ | 1) ____ | 2) ____ | 3) ____ |
| Splitting | 0) ____ | 1) ____ | 2) ____ | 3) ____ |
| Pain descriptors - affective dimension | | | | |
| Tiring - exhausting | 0) ____ | 1) ____ | 2) ____ | 3) ____ |
| Sickening | 0) ____ | 1) ____ | 2) ____ | 3) ____ |
| Fearful | 0) ____ | 1) ____ | 2) ____ | 3) ____ |
| Punishing - cruel | 0) ____ | 1) ____ | 2) ____ | 3) ____ |

Visual analogue scale



Present pain intensity

- 0 No pain _____
- 1 Mild _____
- 2 Discomforting _____
- 3 Distressing _____
- 4 Horrible _____
- 5 Excruciating _____

Fig. 3.6 Short form McGill Pain Questionnaire [64]. Copyright: Dr. R. Melzack, 1984, 1987. Reprinted with permission. *Note:* Each descriptor is ranked on an intensity scale of 0 = none, 1 = mild, 2 = moderate, 3 = severe. The Present Pain Intensity (PPI) of the standard long-form McGill Pain Questionnaire (LF-MPQ) and the visual analogue (VAS) are also included to provide the overall intensity scores

a birthing partner in the delivery room [74, 75], and reducing anxiety levels [76]. Education of women to expect pain during labor may reduce the need for labor analgesia [77], and this may be in the form of antenatal classes. Capogna found that women who attended such classes tended to be more motivated and came from higher socioeconomic backgrounds [78].

3.8 Physical Factors Affecting Pain Perception

There are several physical factors which have been shown to affect pain perception:

- *Age*—older, nulliparous women experience a longer, more painful labor than their younger counterparts [79]
- *Parity*—researchers have consistently shown that nulliparous women experience a more intense sensory pain in the early stages of labor than multiparous women [80–84]
- *Obesity*—Melzack noted that women with a larger body mass index (BMI) experienced more pain in labor [85]. However, a later study did not find a correlation between a higher BMI and severity of labor pain [86]
- *History of previous pain or dysmenorrhea*—women who have had experience of severe pain, which may be non-obstetric in origin, have reduced pain scores during labor [87, 88]
- *Condition of the cervix at the time of labor*—the cervix of the nulliparous women tends to soften before the onset of labor and appears to be less sensitive to nociceptive stimuli compared with multiparous women [79]
- *Relationship of the size and position of the fetus in the birth canal*—pain scores were noted to be higher in nulliparous women when the fetal head was lower in the birth canal [65]
- *Maternal position*—while in labor, women may find walking, sitting on a birthing ball, or remaining upright helpful in labor. While there are no conclusive studies favoring one position for labor, investigators have found that those women who remained in an upright position in labor rather than a recumbent one had a short labor and were less likely to request epidural analgesia [89]
- *Immersion in water*—the use of birthing pools has been known anecdotally to ease the pain during labor, but the exact mechanism of this is presently unknown. Previous studies have observed that the use of birthing pools is associated with faster labors and a reduced requirement for labor analgesia [90] as well as a reduced rate of perineal trauma [91] and obstetric involvement with the delivery [92]. A Cochrane review found that although the use of birthing pools was associated with a significant decrease in the requests for neuraxial analgesia in labor, there were no differences in the rates of assisted or operative deliveries, perineal trauma, or infection [93]

3.9 Conclusion

The complex nature of pain in childbirth is made up from a variety of anatomical, physical, and emotional components. A variety of different strategies may be employed to lessen the intensity of pain and to improve the experience of childbirth for women. However, as yet, there is no one single strategy which is proven in the literature to be consistently successful in reducing the intensity of labor pain.

References

1. Melzack R (1984) The myth of painless childbirth (the John J Bonica lecture). *Pain* 19 (4):321–337
2. Merskey H (1979) Pain terms: a list with definitions and notes on usage recommended by the IASP subcommittee on taxonomy. *Pain* 6:249–252
3. Lin VW (ed) (2003) *Spinal cord medicine: principles and practice*. Demos Medical Publishing, New York, NY
4. Berek JS (2011) *Berek & Novak's gynecology*, 15th edn. Lippincott Williams & Wilkins, Philadelphia, PA
5. Cunningham ST, Steinman JL, Whipple B et al (1991) Differential roles of hypogastric and pelvic nerves in the analgesic and motoric effects of vaginocervical stimulation in rats. *Brain Res* 559(2):337–343
6. Sandner-Kiesling A, Pan HL, Chen SR et al (2002) Effect of kappa opioid agonists on visceral nociception induced by uterine cervical distension in rats. *Pain* 96(1–2):13–22
7. Owman C (1981) Pregnancy induces degenerative and regenerative changes in the autonomic innervation of the female reproductive tract. *Ciba Found Symp* 83:252–279
8. Stjernquist M, Owman C (1987) Interaction of noradrenaline, NPY and VIP with the neurogenic cholinergic response of the rat uterine cervix *in vitro*. *Acta Physiol Scand* 131 (4):553–562
9. Papka RE, Traurig HH (1988) Distribution of subgroups of neuropeptide Y-immunoreactive and noradrenergic nerves in the female rat uterine cervix. *Cell Tissue Res* 252(3):533–541
10. Melo RC, Machado CR (1993) Noradrenergic and acetylcholinesterase-positive nerve fibres of the uterus in sexually immature and cycling rats. *Histochem J* 25(3):213–218
11. Friedman E (1954) The graphic analysis of labor. *Am J Obstet Gynecol* 68(6):1568–1575
12. Tong C, Ma W, Shin SW et al (2003) Uterine cervical distension induces cFos expression in deep dorsal horn neurons of the rat spinal cord. *Anesthesiology* 99(1):205–211
13. Gintzler AR, Peters LC, Komisaruk BR (1983) Attenuation of pregnancy-induced analgesia by hypogastric neurectomy in rats. *Brain Res* 277(1):186–188
14. Temple JL, Bradshaw HB, Wood E et al (1999) Effects of hypogastric neurectomy on escape responses to uterine distension in the rat. *Pain Suppl* 6:S13–S20
15. Berkley KJ, Robbins A, Sato Y (1993) Functional differences between afferent fibers in the hypogastric and pelvic nerves innervating female reproductive organs in the rat. *J Neurophysiol* 69(2):533–544
16. Tingaker BK, Ekman-Ordeberg G, Facer P et al (2008) Influence of pregnancy and labor on the occurrence of nerve fibers expressing the capsaicin receptor TRPV1 in human corpus and cervix uteri. *Reprod Biol Endocrinol* 6:8. doi:10.1186/1477-7827-6-8
17. Mirza FG, Fakhoury AA, Rowley TJ et al (2013) Role of capsaicin in a murine model of labor and delivery. *Anesthesiology* 118:430–435
18. Mackler AM, Iezza G, Akin MR et al (1999) Macrophage trafficking in the uterus and cervix precedes parturition in the mouse. *Biol Reprod* 61(4):879–883

19. Richardson JD, Vasko MR (2002) Cellular mechanisms of neurogenic inflammation. *J Pharmacol Exp Ther* 302(3):839–845
20. Yellon SM, Mackler AM, Kirby MA (2003) The role of leukocyte traffic and activation in parturition. *J Soc Gynecol Investig* 10(6):323–338
21. Ekman-Ordeberg G, Stjernholm Y, Wang H et al (2003) Endocrine regulation of cervical ripening in humans—potential roles for gonadal steroids and insulin-like growth factor-1. *Steroids* 68(10–13):837–847
22. Uchiyama T, Ito A, Ikesue A et al (1992) Chemotactic factor in the pregnant rabbit uterine cervix. *Am J Obstet Gynecol* 167(5):1417–1422
23. Tanaka Y, Narahara H, Takai N et al (1998) Interleukin-1beta and interleukin-8 in cervicovaginal fluid during pregnancy. *Am J Obstet Gynecol* 179(3 Pt 1):644–649
24. Facchinetti F, Venturini P, Blasi I et al (2005) Changes in the cervical competence in preterm labour. *BJOG* 112(Suppl 1):23–27
25. Huber A, Hudelist G, Czerwenka K et al (2005) Gene expression profiling of cervical tissue during physiological cervical effacement. *Obstet Gynecol* 105(1):91–98
26. Tornblom SA, Klimaviciute A, Bystrom B et al (2005) Non-infected preterm parturition is related to increased concentrations of IL-6, IL-8 and MCP-1 in human cervix. *Reprod Biol Endocrinol* 3:39
27. Boyd JW, Lechuga TJ, Ebner CA et al (2009) Cervix remodeling and parturition in the rat: lack of a role for hypogastric innervation. *Reproduction* 137(4):739–748
28. Cardin H, Moisson Tardieu MT, Tournaire M (1986) *La péridurale*. Balland, Paris
29. Melzack R, Wall PD (1955) Pain mechanisms: a new theory. *Science* 150(3699):971–979
30. Melzack R (1999) From the gate to the neuromatrix. *Pain Suppl* 6:S121–S126
31. Melzack R (2001) Pain and the neuromatrix in the brain. *J Dent Educ* 65(12):378–382
32. Tracey I, Mantyh PW (2007) The cerebral signature for pain perception and its modulation. *Neuron* 55(3):77–91
33. Bantick SJ, Wise RG, Ploghaus A et al (2002) Imaging how attention modulates pain in humans using functional MRI. *Brain* 125:310–319
34. Segal S, Csavoy AN, Datta S (1998) The tocolytic effect of catecholamines in the gravid rat uterus. *Anesth Analg* 87:864–869
35. Fuchs AR, Dawood MY (1980) Oxytocin release and uterine activation during parturition in rabbits. *Endocrinology* 107:1117–1126
36. Glatz TH, Weitzman RE, Eliot RJ et al (1981) Ovine maternal and fetal plasma oxytocin concentrations before and during parturition. *Endocrinology* 108:1328–1332
37. Landgraf R, Schulz J, Eulenberger K et al (1983) Plasma levels of oxytocin and vasopressin before, during and after parturition in cows. *Exp Clin Endocrinol* 81:321–328
38. Hirst JJ, Haluska GJ, Cook MJ et al (1993) Plasma oxytocin and nocturnal uterine activity: maternal but not fetal concentrations increase progressively during late pregnancy and delivery in rhesus monkeys. *Am J Obstet Gynecol* 169:415–422
39. Hingson RA, Hellman LM (eds) (1956) *Anaesthesia for obstetrics*. JB Lippincott, Philadelphia, PA
40. Rahm VA, Hallgren A, Högberg H et al (2002) Plasma oxytocin levels in women during labor with or without epidural analgesia: a prospective study. *Acta Obstet Gynecol Scand* 81(11):1033–1039
41. Dalsgaard CJ, Hokfelt T, Schultzberg M et al (1983) Origin of peptide-containing fibers in the inferior mesenteric ganglion of the guinea-pig: immunohistochemical studies with antisera to substance P, enkephalin, vasoactive intestinal polypeptide, cholecystokinin and bombesin. *Neuroscience* 9(1):191–211
42. Carvalho TL, Hodson NP, Blank MA et al (1986) Occurrence, distribution and origin of peptide-containing nerves of guinea-pig and rat male genitalia and the effects of denervation on sperm characteristics. *J Anat* 149:121–141
43. Mouton S, Kamban JR, Naukura R et al (1991) Substance P levels are decreased in pregnancy. *Anesthesiology* 75(3):A842

44. Dalby PL, Ramanathan S, Rudy T et al (1997) Plasma and saliva substance P levels: The effects of acute pain in pregnant and non-pregnant women. *Pain* 69:263–267
45. Krieger DT (1982) Placenta as a source of ‘brain’ and ‘pituitary’ hormones. *Biol Reprod* 26(1):55–71
46. Fajardo MC, Florido J, Villaverde C et al (1994) Plasma levels of β -endorphin and ACTH during labor and immediate puerperium. *Eur J Obstet Gynecol Reprod Biol* 55(2):105–108
47. Dabo F, Nyberg F, Zhou Q et al (2010) Plasma levels of β -endorphin during pregnancy and use of labor analgesia. *Reprod Sci* 17(8):742–747
48. Varrassi G, Bazzano C, Edwards T (1989) Effects of physical activity on maternal plasma β -endorphin levels and perception of labor pain. *Am J Obstet Gynecol* 160:707–712
49. Ginzler A (1980) Endorphin-mediated increases in pain threshold during pregnancy. *Nature* 210:193–196
50. Iwasaki H, Collins JG, Saito Y et al (1991) Naloxone-sensitive, pregnancy induced changes in behavioural responses to colorectal distention: pregnancy induced analgesia to visceral stimulation. *Anesthesiology* 74(5):927–933
51. Lechner W, Jarosch E, Solder E et al (1991) Beta-endorphins during childbirth under transcutaneous electrical nerve stimulation. *Zentralbl Gynakol* 113:439–442
52. Datta S, Hurley RJ, Naulty JS et al (1986) Plasma and cerebrospinal fluid progesterone concentrations in pregnant and nonpregnant women. *Anesth Analg* 65:950–954
53. Bromage PR (1961) Continuous lumbar epidural analgesia for obstetrics. *Can Med Assoc J* 85:1136–1140
54. Ward ME (1997) Acute pain and the obstetric patient: recent developments in analgesia for labour and delivery. *Int Anesthesiol Clin* 35(2):83–103
55. Van Zundert AA, Crouls RJ, Korsten HH et al (1996) Spinal anaesthesia. Volume or concentration—what matters? *Reg Anesth* 21(2):112–118
56. Lowe NK (2002) The nature of labor pain. *Am J Obstet Gynecol* 186:S16–S24
57. Ludington E, Dexter F (1998) Statistical analysis of total labor pain using the visual analog scale and application to studies of analgesic effectiveness during childbirth. *Anesth Analg* 87:723–727
58. Carvalho B, Cohen SE (2013) Measuring the labor pain experience: delivery still far off. *Int J Obstet Anesth* 22(1):6–9
59. Bonnel AM, Boreau F (1985) Labor pain assessment: validity of a behavioral index. *Pain* 22:81–90
60. Sheiner EK, Sheiner E, Shoham-Vardi I et al (1999) Ethnic differences influence care giver’s estimates of pain during labour. *Pain* 81:299–305
61. Melzack R (1975) The McGill pain questionnaire: major properties and scoring methods. *Pain* 1:275–299
62. Melzack R, Taenzer P, Feldman P et al (1981) Labour is still painful after prepared childbirth training. *Can Med Assoc J* 125:357–363
63. Niven CA, Gijsbers K (1984) A study of labour pain using the McGill pain questionnaire. *Soc Sci Med* 19:1347–1351
64. Melzack R (1987) The short-form McGill pain questionnaire. *Pain* 30:191–197
65. Capogna G, Camorcia M, Stirparo S (2010) Multidimensional evaluation of pain during early and late labor: a comparison of nulliparous and multiparous women. *Int J Obstet Anesth* 19:167–170
66. Dick-Read G (1933) *Natural childbirth*. W Heinemann, London
67. Lamaze F (1984) *Painless childbirth: the Lamaze method*. Contemporary Books, Chicago, IL, Reissue of 1958 edition
68. Lowe NK (1989) Explaining the pain of active labor: the importance of maternal confidence. *Res Nurs Health* 12:237–245
69. Wuitchik M, Hesson K, Bakal D (1990) Perinatal predictors of pain and distress during labor. *Birth* 17:186–191
70. Crowe K, vom Baeyer C (1989) Predictors of a positive childbirth experience. *Birth* 16:59–63

71. Manning MM, Wright TL (1983) Self-efficacy expectancies, outcome expectancies and the persistence of pain control childbirth. *J Pers Soc Psychol* 45:421–431
72. Walker B, Erdman A (1984) Childbirth education programs: the relationship between confidence and knowledge. *Birth* 11:103–108
73. Escott D, Spiby H, Slade P et al (2004) The range of coping strategies women use to manage pain and anxiety prior to and during first experience of labor. *Midwifery* 20:144–156
74. Henneborn WJ, Cogan R (1975) The effect of husband participation on reported pain and probability of medication during labor and birth. *J Psychosom Res* 19(3):215–222
75. Kennell J, Klaus M, McGrath S et al (1991) Continuous emotional support during labor in a US hospital. A randomized controlled trial. *JAMA* 265(17):2197–2201
76. Lang AJ, Sorrell JT, Rodgers CS et al (2006) Anxiety sensitivity as a predictor of labor pain. *Eur J Pain* 10(3):263–270
77. Senden IP, van du Wetering MD (1988) Labor pain: a comparison of parturients in a Dutch and an American teaching hospital. *Obstet Gynecol* 71(4):541–544
78. Capogna G, Alahuhta S, Celleno D et al (1996) Maternal expectations and experiences of labour pain and analgesia: a multicentre study of nulliparous women. *Int J Obstet Anesth* 5(4):229–235
79. Fishman SM, Ballantyne JC, Rathmell JP (eds) (2010) *Bonica's management of pain*, 4th edn. Lippincott William & Wilkins, Philadelphia, PA
80. Brown ST, Campbell D, Kurtz A (1989) Characteristics of labor pain at two stages of cervical dilation. *Pain* 38(3):289–295
81. Sheiner E, Sheiner EK, Shoham-Vardi I (1998) The relationship between parity and labor pain. *Int J Gynecol Obstet* 63(3):287–288
82. Gaston-Johansson F, Fridh G, Turner-Norvell K (1988) Progression of labor pain in primiparas and multiparas. *Nurs Res* 37(2):86–90
83. Lowe NK (1987) Parity and pain during parturition. *J Obstet Gynecol Neonatal Nurs* 16(5):340–346
84. Ranta P, Jouppila P, Jouppila R (1996) The intensity of labor pain in grand multiparas. *Acta Obstet Gynecol Scand* 75(3):250–254
85. Melzack R, Kinch R, Dobkin P et al (1984) Severity of labour pain: influence of physical as well as psychologic variables. *Can Med Assoc J* 130:579–584
86. Ranta P, Jouppila P, Spalding M et al (1995) The effect of maternal obesity on labour and labour pain. *Anaesthesia* 50(4):322–326
87. Melzack R, Bélanger E (1988) Labour pain: correlations with menstrual pain and acute low-back pain before and during pregnancy. *Pain* 36:225–229
88. Niven C, Gijsbers K (1984) Obstetric and non-obstetric factors related to labour pain. *J Reprod Infant Psychol* 2:61–78
89. Lawrence A, Lewis L, Hofmeyr GJ et al (2009) Maternal positions and mobility during first stage labour. *Cochrane Database Syst Rev* 2009:CD003934
90. Odent M (1983) Birth under water. *Lancet* 2:1476–1477
91. Geissbuhler V, Eberhard J (2000) Waterbirths: a comparative study. A prospective study on more than 2,000 waterbirths. *Fetal Diagn Ther* 15:291–300
92. Cluett ER, Pickering RM, Getliffe K et al (2004) Randomised controlled trial of labouring in water compared with standard of augmentation for management of dystocia in first stage of labour. *BMJ* 328:314
93. Cluett ER, Burns E (2009) Immersion in water in labour and birth. *Cochrane Database Syst Rev* 2:CD000111

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4.1 The Meaning of Labor Pain in Western Culture

When we talk about the “meaning of pain,” we can do so from two main fields. First of all, the biological meaning of pain may be referred to. Second, we can talk about the function or “social meaning” of pain.

Pain has accompanied human beings throughout history, but it has not always been interpreted in the same way [1, 2]. Pain is the representation of an individual state that is unlikely to be known by somebody alien to the suffering of pain itself [3], which we can only know on the basis of the perception of the person who is suffering it [4, 5], through their personal interpretation of the painful experience [6]. Thus, its physical, anatomical, biological, and chemical component aside [7], pain is defined by the norms, values, and symbols that both the person suffering the pain and the people around him or her confer upon it [8].

Pain, as a process in which the individual tries to find meaning or significance, cannot be disassociated from its cause. Labor pain, a transitory pain, the product of a biological fact that is not an illness, the result of which is usually positive (childbirth), and which is not expected to be extended or increased in the future [9], is interpreted in a different way from any other type of pain. In labor pain, the woman is the protagonist, not only when she experiences the pain but also when she interprets it socially. However, the woman not only interprets her labor pain in

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social terms, but she also interprets other women's pain, and she is the object of interpretation of her pain for society in general.

In order to tackle labor pain in Western societies (generalizing is difficult in this area, and even more so the more different the analyzed societies) without leaving aside the meaning socially attributed to it, it is necessary to state the obvious: there are as many childbirths as women, and as many labor pains as childbirths. But this may lead to the denial of the existence of behavior and interpretation patterns in this social phenomenon, which make it more understandable. Therefore, we will refer to "ideal types" of women in the Weberian style. Thus, three "prototypes" of women have been established, which do not usually appear "purely" in society but do exemplify the main conceptions of women and their relationship with labor pain, mainly on the basis of the meaning they give to such pain in relation to their own social identity.

4.2 "Ideal" Maternity Models

Women and maternity are two concepts a priori describing objective facts, but they are as social and intersubjective as other concepts like social class or status. In this respect, women have been defined throughout history by their maternal role to a great extent, without which in certain times they lost their social role and had no identity as a social subject.

Rich was the first one to differentiate between "maternity as an institution" and "maternity as an experience," [10] and she laid the foundations for a very complex study on maternity in its public dimension, which is the main interest of social study. She states that the private and public dimensions of maternity are not only parallel but they also intertwine and influence one another. Maternity is an essential fact for society and, as such, society itself exerts a lot of pressure upon it. Although it is in principle an "individual" fact, it turns into a public debate where relatives, neighbors, acquaintances, strangers, politicians, the media, religious figures, etc., are involved.

The discourse about maternity, based on conceptions and stereotypes unifying women into a sole image due to their role as mothers [11], not only leaves aside individual differences, but also class and culture-related elements that account for the different types of maternity which exist. Furthermore, accepting that there are different types of maternity means at least calling into question the existence of an instinctive biological maternity [12].

In the social conception of women, maternity has had an almost permanent influence throughout history, although with important nuances in each period. The transformation of the "mother" figure and the way each maternity is experienced have changed as women's role has changed, as well as their increasing presence in the public sphere [11]. Today it could be said that three main types of women coincide, daughters of a certain historical development, reflecting different ways of understanding maternity. This typology, mainly based on the cyclical theories of

social change, will reveal that each archetype of woman partially emerges in contrast to the previous archetype.

4.2.1 The Traditional Woman/Mother

In the social context of the traditional woman, who holds the most traditional values of patriarchal society, the public and private spheres are two completely separate worlds, which translate into a sexual separation of productive and reproductive labor. The traditional woman is a sovereign and an executant in the private sphere, which mainly entails housekeeping and caregiving. These women build their identity mainly on the basis of their role as mothers, a maternity seen as not only a goal or objective but also as an instrument to create their identity. This is what has been called “intensive maternity” [13], where women devote all their time to taking care of their children and where the radical segregation of roles and decision spheres by sex is socially understood. These roles have been transmitted and interiorized generation after generation. From birth, women will be raised promoting the capacities and abilities typical of the private space, and their predecessors will be their perfect model. This is how stereotypes are transmitted, according to which women are emotional, sensitive, etc., characteristics that are considered appropriate for housekeeping and caregiving [12]. On the contrary, men, the source of authority, are considered the main breadwinners of the household, and that is why their role in the private sphere is secondary. When this “type” of woman has access to the public sphere, in her productive facet, she performs jobs which are reminiscent of her role as a caregiver or that were traditionally performed or occupied by marginal productive sectors, considered subsidiary jobs, men being the main supporters.

4.2.2 The Modern Woman/Mother

With the scientific development of the end at the nineteenth century, logic and reason start to dominate the different social spheres, and the traditional role of women as the owners of the most domestic sphere of life is modified.

The “idealized mother” appears in the private sphere, where women are identified as mothers, as maternity is the central goal in women’s lives [14]. In this regard, their role is similar to that of traditional women, but with a fundamental change: the loss of their decision-making ability. It is assumed that in a “natural” way (natural as a synonym of biological) only women have the ability to be connected, empathize, and give love, and that is why they are perfect for the actions of the private sphere, but, as a characteristic feature, they are protected under new basically masculine doctrines. The State imposes the values to be adopted in the domestic sphere (schooling, timetables, eating habits, sleeping habits, etc.). Women, who are responsible for everything within this sphere, but under the new

standardized criteria, lose part of their power at home, where they will be judged in their role as women [15] on the basis of new external norms.

Modern women, unlike their predecessors, enter the public sphere in order to gain financial independence from men. They do not only perform subsidiary jobs or work to assist their family economy anymore but also develop real professional careers. In this incorporation, women assume the professional world, defined in masculine terms, completely and with no evident changes. In order to be valued in the public world, women often adopt characteristics traditionally used to define men (competitiveness, toughness, etc.), becoming masculinized in many cases in order to “survive” in a world that only values these characteristics, which are new for women.

The massive incorporation of women into the public world does not result in a corresponding incorporation of men into the private world, or at least not in the same proportion. It causes the phenomenon called “double presence” [16], where women have responsibility to their families and their jobs. In some cases, this has caused a double absence, the feeling of not completely complying with either of the two fields, causing frustration and stress.

4.2.3 The Postmodern Woman

During the second half of the twentieth century, the sexual revolution of the 1960s and 1970s marked an ideal model to be followed, with more egalitarian relationships in Western countries. Traditional authority is relegated, with a less disciplinary model emerging, with greater freedom of choice and promoting personal realization [17, 18]. Transitory and superficial social relations increase exponentially, with multiplying benchmarks available in the culture, which has led to what is known as “social saturation,” one of the main characteristics of postmodernity [19], where an individual is stripped of its own identity.

The frontiers between the public and private spheres are blurred, giving rise to new ways to define parental and gender roles within the family, so that the reproductive labor starts being considered a collective task [15].

One of the characteristics of postmodern women is that maternity is not marked as the only defining condition of the new status of women, as new evaluation parameters and new ways to take part in the social game are now at stake [20]. Postmodern women assume the orientation of their own life more directly, and they are a vanguard group contributing more directly to transforming maternity’s meaning and the existence of less conventional maternity modes [21].

There are tendencies that in some cases can be considered as a return to the most traditional woman, as is the case of what has been called “New Domesticity.” Thus, there are women who decide to refuse the rhythm imposed by today’s society to embrace another way of life based on the “Do it yourself” ideas and on going back to the private sphere, among other aspects. To a large extent, this return home has been caused by the economic climate and the feeling of younger generations who do not want to make as many sacrifices for their jobs as their mothers [22].

Postmodern women decide on their bodies, their fertility, and their right to knowledge and to equal opportunities for men and women, without losing their acquired psychogenetic conditioning [23]. “Difference feminism” conceptualized these women theoretically, bestowing value on the female gender and reinforcing it, rejecting the process of acquiring masculinized behavior patterns in order to reach the spaces of power [24] partially typical of modern women.

4.3 Labor Pain Perception and Social Value of Labor Pain Relief

Like any other social fact, labor, its experience, the pain suffered or lack thereof, etc., are influenced by cultural factors, which affect the way in which a woman decides to go through labor and the perception of labor pain and its meaning.

In the past, pain was connected to evil spirits, to getting closer to God, to a rite of passage, etc., until today, when in Western societies pain is generally seen as something futile and sterile, with no function, which must be minimized. In today’s society, avoiding pain is a value, as opposed to gratuitous or avoidable suffering.

But labor pain is a much more complex phenomenon, influenced by other characteristics apart from the pain-avoiding culture. In her anthropological studies, Margaret Mead referred to the effect of culture and the education model on the intensity of labor pain [25].

But pain has not only a social meaning. The decision not to suffer it, as well as the methods and tools used, whether pharmacological or of another nature, is the object of social interpretation too. Thus, if a certain treatment or medicine is accepted to be useful and, especially “appropriate” for pain relief, it is more likely to have an effect, to be more effective to eliminate pain, than if it is socially discredited, refused, or called into question, not only because of its effectiveness but also because of its social “legitimacy.”

Pain relief methods are not alien to the social view of childbirth, maternity, and the role of women in each historical moment. Following the preceding reasoning, today three women coincide as regards pain relief methods. If we go back to the three archetypes of women previously described, we will be able to see that each of them will go through labor pain in a different way (in general descriptive terms), and they will confer a different meaning upon it.

Therefore, *traditional women* might be conferring a meaning on pain closer to a rite of passage, “experiencing childbirth” as the beginning of a stage, maternity, which defines their fundamental role as women. The discourses characterizing this painful experience would be connected to “going through” labor, and in order to do so pain needs to be experienced as an inseparable part of the experience. In general, these women refuse labor pain relief, as they confer a natural value on childbirth, which involves accepting pain as something inherent to childbirth itself. Until relatively recently, midwives played a major role in childbirth, gynecologists and obstetricians playing a secondary role [26], if there was a medical complication. It was a world of women, where childbirth is seen as something “natural,” not needing

medical monitoring, where pain is part of the process and it legitimizes a woman as a mother. As a “good mother,” a woman must renounce her own well-being and seek her child’s well-being, which brings her social recognition [27]. This position is also legitimized by historical example (“that’s how it has always been done,” “labor has always been like that”), which somehow delegitimizes those women who decide not to go through this experience in all its painful intensity.

In turn, *modern women* confer on pain a meaning much less linked to experience. This came together with the spread of the use of epidural analgesia in labor, where physicians and anesthetists play a leading role. Medical professionals redefine maternity, medicalize it, and somehow appropriate it [28]. Women accept technological and biomedical development as something “natural,” and their decision whether or not to go through pain is much more rigid, sometimes even imposed by the healthcare system, physicians or nurses, and administrative availability. The role of the woman is relegated, sometimes turned into a passive object of the childbirth process, which is monitored and managed by medical professionals, who “own” the knowledge. As compensation for stealing their limelight, women have pain relief, and pain is hence minimized and almost disappears. For these women, daughters of their social context, pain makes no sense, it is something that must be avoided if possible, and labor pain is no exception. This fact, together with the spread and popularization of medical techniques in general, and anesthetic techniques in particular, made the use of pain relief popular.

Postmodern women reclaim “what is natural.” They want their childbirth with minimum medical and biochemical intervention, a birth that is not instrumentalized and/or technified. This return to what is natural is vindicating women and their main role in this process, once again taking charge of childbirth, where they felt they had been relegated to a secondary and passive role. They also maintain a very elaborate discourse on the benefits of this kind of natural childbirth for the baby. They criticize medicine’s control of women’s bodies and reproduction, and they choose to refuse those anesthetics that prevent them from experiencing the process being “fully aware” of it. They do not see pain as something good but as something natural, which puts them in a position of not having to just accept it; hence, they demand fighting pain with other nonpharmacological but natural tools, such as different positions, natural remedies, meditation, etc. They are calling for empowerment in their own process, where they feel like protagonists. At the same time, they criminalize previous techniques, in the belief that they have a negative effect on the baby and on the labor process, as well as on subsequent breastfeeding and mother–child bonding. They opt for home births (failing that, reproducing home conditions at the hospital), surrounded by relatives and friends, with as little medical intervention as possible.

As shown above, the meaning of pain is conditioned by the characteristics of the women experiencing it and their surroundings. If pain is seen as something natural, and even “good,” it favors the labor process and their own identity, it plays an instrumental function, and so eliminating it can only be considered an error. If, on the contrary, pain is not considered to favor labor, and if it is seen as unnecessary in a society that in general benefits from the different progresses of medicine, not

turning to pain relief will be considered an error. One or other meaning is conferred by women on the labor process, but also by society as a whole.

4.4 Maternities and Epidural Anesthesia

If a woman considers that a “good labor” is essentially painless, epidural anesthesia will be the tool to achieve it. However, as we have observed, if for some women this is not the main element of labor, i.e., they confer other characteristics on their definition of what a “good labor” is, the formulas to attend to them are not so obvious. Perhaps this is why new anesthetic techniques are being developed, as well as what could be called a “new” epidural analgesia (although it is not widely used yet), also trying to minimize the complications of “traditional” epidural analgesia. The ultimate goal of these new techniques is to avoid pain without diminishing a certain control of the woman in labor over her labor process. This is the case of the combined spinal and epidural anesthetic technique (CSE), which intends to reduce some of the disadvantages of traditional epidural anesthesia [29], and especially what is known as “Walking Epidural” [30, 31], which allows the mother to have more mobility and control over her pain and her ability to push. This brings this process closer to a more natural labor in its form and possibilities.

The tendency of the most innovative healthcare systems seems to be pointing to a naturalization of the labor process, intending to avoid some protocols that were previously standardized, such as “traditional” epidural anesthesia, episiotomy, or even unnecessary Cesarean sections. In general, the simulation of a nonmedicalized labor is intended, with a greater presence of the woman in labor, but not giving up on reducing or even eliminating pain. However, from our point of view and in the light of the determinant factors defining maternity, labor and childbirth in Western contemporary societies, although satisfactory for a wide group of women, will not be enough if it is not accompanied by other measures. Portfolios of services need to include, for instance, nonpharmacological services to attend to childbirth and women in labor, which allow women to approach their labor process in accordance with the values shaping their own identities. All this set in a context of real decision-making and information on the part of future mothers which really places them at the center of the system.

4.5 Conclusions

Pain may have a physical, emotional, and psychical origin, but whatever its causes may be, it must also be seen as a cultural and socially constructed concept, involving psychological, educational, cultural, and sociological elements. Therefore, pain requires research based on subjective and contextual perceptions. In this context, studying women and their social identity, as well as the importance of their role as mothers in such identity, gains great relevance. Consequently, studying the different types of maternities in current Western societies is also essential, as they

are becoming more diverse and having different ways of gaining access to maternity, which also confer different characteristics on the maternal experience.

However, we cannot talk about maternity as a sole fact, but as maternities and different ways of experiencing maternity. Women decide whether or not to use standardized labor pain reliefs, such as epidural analgesia, in part according to the social interpretation they make of the fact of going through pain. This interpretation differs according to the profile of women taking one decision or the other, as has already been studied [1], with social characteristics and interpretations behind this decision.

However, there is a common factor in the three types of women that we have presented, which is the pressure exerted upon them for their condition as mothers, which turns them into the main providers of well-being and happiness for their children. They are increasingly idealized mothers, who must pay attention to the needs of people in their care (children or elderly people) and whose identity as women is sometimes denied to assume their identity as mothers [32]. A social sacralization of the figure of women as mothers has taken place, which requires women to absolutely devote themselves to that role, regardless of the role they play in the public sphere. This generates contradictions in women between the social ideal expected from them and what they actually do [12].

Without claiming that pain is only cultural, pregnancy and childbirth cannot only be considered biological facts, as they involve social and emotional aspects with an influence on how the entire process is experienced and developed, where women cannot be turned into mere passive subjects [33]. The social context influences the perception of pain, the way its suffering is tackled, and the decision not to experience it. However, the social context of the person who suffers is rarely analyzed, as well as the variables that may be behind its interpretation, perception or intensity. This lack of attention to the social conditioning of pain is in part due to the difficulty of analysis and empirical and scientific verification but also to the traditional biomedical conception of illness and medical research [34]. This difficulty increases when comparison among different societies and cultures is attempted. However, without this analysis the knowledge of the social meaning of pain will be incomplete when interpreting its significance.

References

1. Le Breton D (1995) *Anthropologie de la douleur*. Métailié, Paris
2. Le Bretón D (2010) Pain and the care relationship. *Soins* 749:34–35
3. Franco A (1999) El dolor en la historia. *Rev Soc Esp Dolor* 6:261–262
4. Baszanger I (1992) Deciphering chronic pain. *Sociol Health Illn* 14(2):181–215
5. Suvienen TI, Reade P, Kempainen P, Könönen M, Dworkin SF (2005) Review of aetiological concepts of temporomandibular pain disorders: towards a biopsychosocial model for integration of physical disorder factors with psychological and psychosocial illness impact factors. *Eur J Pain* 9(6):613–633
6. Leriche R (1937) *La chirurgie de la douleur*. Masson, Paris
7. Loeser JD, Melzack R (1999) Pain: an overview. *Lancet* 353(9164):1607–1609

8. Kirmayer LJ (2008) Culture and the metaphoric mediation of pain. *Transcult Psychiatry* 45 (2):318–338
9. Bayes R (1998) Psychology of suffering and death. *Anuario de Psicología* 29(4):5–17
10. Rich A (1976) *Nacida de mujer*. Noguer y Caralt Ed, Barcelona
11. Moreno M, Mira A (2005) Maternidades y Madres: un enfoque historiográfico. In: Caporale S (ed) *Discursos teóricos en torno a la(s) maternidad(es): una visión integradora*. Entinema, Madrid
12. Téllez A, Heras P (2005) Representaciones de género y maternidad: una aproximación desde la antropología sociocultural. In: Caporale S (ed) *Discursos teóricos en torno a la(s) maternidad(es): una visión integradora*. Entinema, Madrid
13. Solé C, Parella S (2004) Nuevas expresiones de la maternidad. Las madres con carreras profesionales exitosas. *Revista Española de Sociología* 4:67–92
14. Molina ME (2006) Transformaciones histórico culturales del concepto de maternidad y sus repercusiones en la identidad de la mujer. *Psyche* 15(2):93–103
15. Hays S (1996) *The cultural contradictions of motherhood*. Yale University Press, New Haven, CT
16. Balbo L (1978) *La doppia presenza*. *Inchiesta* 32:3–11
17. Ehrenberg A (2000) *La fatiga de ser uno mismo*. *Depresión y sociedad*. Nueva Visión, Buenos Aires
18. Gergen K (1991) *El yo saturado*. Paidós, Buenos Aires
19. Maffesoli M (1990) *El tiempo de las tribus, el declive del individualismo en las sociedades de masas*. Icaria, Barcelona
20. Coria C, Freixas A, Covas S (2005) *Los cambios en la vida de las mujeres. Temores, mitos y estrategias*. Paidós, Buenos Aires
21. Alberdi I, Escario P, Matas N (2000) *The young women in Spain*. Fundación La Caixa, Barcelona
22. Matchar E (2013) *Homeward bound: why women are embracing the new domesticity*. Simon & Schuster, New York, NY
23. Lipovetsky G (2002) *The third woman: the stability and shock of basis of femininity*. Anagrama, Barcelona
24. Bel-Bravo MA (2009) *Mujeres y cambio social en la Edad Moderna*. Ediciones Encuentro, Madrid
25. Macfarlane A (1977) *The psychology of childbirth*. Harvard University Press, Cambridge, MA
26. Campuzano C (2007) Guidelines for obstetric epidural analgesia within a framework of innovative management and quality and safety criteria. *Rev Soc Esp Dolor* 14(2):117–124
27. Tobío C (2002) Conciliación o contradicción: como hacen las madres trabajadoras. *RES* 97:155–186
28. Nash M (2000) Maternidad, maternología y reforma eugénica en España, 1900–1939. In: Georges D, Perrot M (eds) *Historia de las Mujeres en Occidente*. Ed. Taurusminor, Madrid
29. Fernandez-Ramos H, Cobas-Varona D, Llanos-Palmira L, Fonseca-León A (2009) Técnica combinada espinal epidural a dos espacios para anestesia obstétrica. Nuestra primera experiencia. *AMC* 13(1)
30. Kuczkowski KM (2004) Ambulation with combined spinal-epidural labor analgesia: the technique. *Acta Anaesthesiol Belg* 55(1):29–34
31. Cohen SE, Yeh JY, Riley ET, Vogel TM (2000) Walking with labor epidural analgesia: the impact of bupivacaine concentration and a lidocaine-epinephrine test dose. *Anesthesiology* 92:387–392
32. Caporale S (2005) *Discursos teóricos en torno a la(s) maternidad(es): una visión integradora*. Entinema, Madrid
33. Castro R, Bronsfman M (1993) Teoría feminista y sociológica médica: bases para una discusión. *Cad Saúde Publ Ríó de Janeiro* 9(3):375–394
34. Morris DB (1991) *The culture of pain*. University of California Press, Berkeley, CA

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5.1 Medicalization of Labor

History of labor has always fluctuated between the private and the public, the natural and the artificial. Well up to the eighteenth century, the preparation and attendance of birth was essentially considered a women's affair from which males were almost always excluded. As late as 1552, a German physician was publicly branded for having attended as a midwife in a female garb, and still in the early nineteenth century, many obstetricians lamented that during what they called "the dark ages," the care of women in childbed appertained exclusively to the female sex, especially to those of them who had acquired a certain experience in accouchement and were therefore invested of a certain authority [1]. Angélique du Coudray, the enlightened midwife who employed an anatomical model for surgical demonstrations, was one of the most famous [2]. Only with the development of modern surgery, the routine of birth saw a period of dramatic changes and lasting resistances [3]. What historians have called the "obstetric revolution" was linked to an improvement in anatomical knowledge, to the implementation of proper training, and, from a more practical point of view, to the generalized use of forceps. In this sense, obstetrics was no different from other professional activities, such as

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surgery or dentistry, which flourished under the threat of professional intrusion and the dissemination of new surgical instruments. At the same time, however, the arrival of surgeons at the birthing stool also brought about a modification in the evaluation and treatment of childbirth pains ([4, 5]; for an alternative view [6]; for a more general view [7]). The increasing professionalization of labor called into question all subjective elements involved in the process, including the way in which mothers complained about or interpreted their symptoms. The history of labor pain and labor pain relief cannot be told without this quarrel regarding the value and reliability of a mother's gestures, grimaces, and complaints. At the same time, the new emotional regimen also meant the progressive substitution of the domestic space, where traditionally mothers had delivered their children, for a new public and clinical location [8].

Historians of obstetrics and labor pain have observed remarkable continuities in both lay and professional attitudes regarding labor analgesia. This is not at all surprising. For all those involved in the history of childbirth, the main difficulty has always lied in the clarification of the natural or civilizatory character of labor pain, and therefore, in the understanding of all variations related to the possibility of a painless birth.

5.2 Labor Pain

Prior to the medicalization of labor in the mid-eighteenth century, the issue of labor analgesia was hardly raised [9]. Both in midwifery books and in the primitive obstetric treatises, the problem of a mother's suffering was a minor issue, scarcely discussed and, sometimes, even completely silenced. This oblivion remained unchanged well up to the second half of the nineteenth century, when many early handbooks on obstetrics did not even contain a single reference to labor analgesia [10]. This attitude is partly understandable, since the equation of pain and birth was less important than the possibility of a fatal result. As in the case of premenstrual pain or the symptoms that accompany menopause, childbirth pain was regarded as yet another experience that women endured as a result of their gender, and for which there scarcely existed palliative remedies ([11]; see also [12]). The discrepancies concerning the mortality rate of women during childbirth and their own expectancy of survival contrasted with the certainty of a necessary and inevitable experience. At the moment of childbirth, the death of the mother was probable, but her suffering was assured. Prior to the development of chemical anesthesia in the mid-nineteenth century, the entire history of humankind seemed to confirm this inviolable connection between pain and labor.

Before the arrival of chloroform—a substance that had already been described in the 1830s and which was initially prescribed as an antispasmodic without anyone at the time realizing its anesthetic properties—labor pain appeared not only as a consequence of a natural action but also a religious punishment imposed on women after the Fall [13]. This could explain why, in 1591, a woman named Eufame Macalayne was condemned and burned at the stake accused of asking for relief before the birth

[14]. Although this is an extreme case, the idea that maternity was an inextricably painful experience embedded the medieval mythology of procreation. Even after the Renaissance, arguments were still made against the use of any form of analgesia under the pretext that searching relief for labor pain was against the divine mandate, as laid down in the Book of Genesis: *in pain you will bring forth children*.

On many occasions, religious beliefs were combined with more secular ideas. Many surgeons and midwives understood labor suffering as a natural resource to soften the mother's body just before delivery. From their point of view, pain acted as a form of corporeal education that made delivery possible. It was a natural provision that should be accepted with patience and resignation. Since cries and screams seemed to have the effect of lessening the pain, the public expression of physical suffering invaded the delivery room. It also pervaded the use of language. The relationship between the forms of suffering and the ways of giving birth was so close that uterine contractions were often generically referred to as "the pains," while the moment of delivery was known as "the scream" [15]. For many surgeons, the most natural birth occurred when the baby was born "through the force of the pain" [16]. See Fig. 5.1. *Preceptos generales sobre las operaciones de partos*.

Though the inextricable connection between pain and labor was still widely accepted in the seventeenth century, it began to be abandoned in the eighteenth, when surgeons came to agree that the body of a mother of five was no better prepared and not less in danger than the body of a first-time mother. At the same time, the natural or supernatural understanding of pain did not prevent the proliferation of remedies. In the seventeenth century, the French surgeon Pierre Dionis had already recommended wines and liqueurs from the Canary Isles, which were shared equally between mothers and midwives [17]. To alleviate their sufferings, many



Fig. 5.1 Trelene Breathing Apparatus. Birthday National Trust Fund. Welcome Collection

women made different kinds of promises: to free a prisoner, recite a novena, or obtain the belt of Saint Margaret. Others, the most religious, asked for masses to be performed. In some cases, relics and reliquaries were arranged in the room. In others, a concoction was prepared using the head of a deer. While some women demanded holy water, the most unfortunate drank large quantities of alcohol. Traditional pharmacopeias contained recipes made from analgesic drugs, very often presented under the form of soporific sponges, which usually contained henbane, hemlock, mandrake, and ground ivy. In the Medieval world, birth came only after certain rituals had taken place. Among them, we may count baths, ventilation, the use of pessaries, or the intake of beverages prepared with herbs of different kinds, including laurel, bloodroot, or saffron. On many occasions, there were also magical or religious invocations. Especially important was the intercession of St. Margaret, the patron of pregnant women, who according to the legend managed to escape alive from a dragon's belly after having been devoured by this beast [18]. One of those remedies implied the use of special birthing chairs. In Palermo, after receiving the blessing by a local priest, a particular chair was believed to alleviate the labor pains of those who used it. It became known as the "Miraculous chair of Palermo" [19].

The arrival of surgery to the birthing chair brought about a conceptual reevaluation of pain's expressions, which turned to be interpreted in correspondence with a set of physiological signs. According to the new men-midwives, the appearance of the mother's face, the position of the baby's head, the inclination of the womb, or the phase of the moon at the moment of gestation were of no use in knowing the sex or number of the unborn. On the contrary, backache, hardening of the muscles, vomiting and nausea, difficulty in urinating, intermittent dizziness, and the presence of varicose veins were sufficient signs for identifying the beginning of gestation.¹ In 1853, Doctor Cazeaux, of the faculty of medicine in Paris, attempted to establish a correct identification of women's expressive signs and their other physiological circumstances. Within this new cartography of sensation, he classified a mother's pains as keen, frequent, dreadful, elevated, excessive, violent, or "mosquito" pains—so called due to a "comparison with the pain caused by a bite from that insect."² From a chronological point of view, he spoke about *precursor*, *preparatory*, *expelling*, or *corrupt* pains. The presence of each one of them determined a precise emotional reaction. Under the influence of *precursor* pains, for example, future mothers took on a melancholic air that grew progressively more violent. As birth progressed, the pains became more frequent and, coinciding with the dilation of the neck of the uterus, keener and closer. Each new sensation arrived with a slight shiver, which quickened and intensified the pulse. At the end of the contraction, the sufferings did not completely disappear; rather, while she was still

¹ Dionis, *Traité général des accouchemens*, Paris, Charles-Maurice d'Houry, 1718, pp. 124 and 152.

² Cazeaux, *Traité*, théorique et pratique de l'art des accouchements, Paris, F. Chamerot, 1858, p. 418.

under the power of the last pain, she began to dread the one that would come next to take its place. At the moment of the birth itself, when the abdominal muscles seemed to come to the uterus's aid, her efforts increased, and just as the baby's head emerged from the womb and the contractions became more energetic, she would cry out. The baby's transit produced a horrible pain, made up of sensations of varying intensity and transmitted through the parietal protuberances at the level of the ischium. Soon after, the head emerged. All this came to confirm that the expressive signs inscribed on the mother's body were of interest, and very much so. For the obstetrician Meigs, for example, the way in which the mother squeezed the hands of those she held onto should be enough to determine whether or not the birth had entered the expulsion phase. For him, as for others, if the duration, intensity, or frequency of the contractions were not equal to those of the pain, it was only due to differences in the age, temperament, or education of the mother. Some will protest in excess for slight sensations, whereas others will hardly complain from very strong contractions. Unfortunately, the mother frequently contaminates the natural expression of her pain, either due to her sensitivity, education, or prejudices. When the moment of birth arrives, wrote Charles Jewett, the bearing of the patient differs greatly in different women; while some of them would scream from the beginning of their contractions, destroying their throats, others remain calm for most of the process [20]. Cazeaux, for example, describes the case of a woman in labor who, following prolonged efforts and interminable suffering, suddenly changed her facial expression and began to sing the great aria from *Lucia di Lammermoor* at the top of her lungs [21]. This is not the only documented case of pain altering the nervous system to such an extent that the sufferer's behavior borders on the irrational or the ridiculous. Some doctors put forth that, with their intellectual capacities diminished, the future mothers said the most extravagant things in their delirium. According to Doctor Montgomery, this outbreak of irrationality occurred especially when the child's head emerged from the womb.³ In either case, the practice was based on an interpretive semiotics of the woman's gestures. Following Bichat, who claimed that to distinguish between true and false pain it was only necessary to take the patient's pulse, Edmund Chapman claimed that pain that caused a reddening of the face and a rapid pulse should be maintained, or even increased, to facilitate the birth [22].

The mother's ability to subvert the equation between the physiological stimulus and her expressive gestures was subject to two significant restrictions. The first, purely biological, depends entirely on anatomical conditions, for the mother's will was never thought to be able to bend the course of nature in all circumstances. The second, artificial, would take place after the introduction of anesthesia and would allow the elimination of the subjective elements from childbirth. Once put to sleep, the woman's body could be manipulated without her incidental perceptions, changes in mood, or the incomprehensible modifications of her states of consciousness. With regard to labor pains, and more in particular, to those related to the force

³ See [21], pp. 414–432.

exerted by contractions, Whitridge Williams, a North American obstetrician, considered that there had been a good deal of misconceptions and a marked tendency toward exaggeration. Like many other of his colleagues, he attempted to understand pain on the grounds of an objective measurement rather than on the always-unreliable testimony of the mother. His reasoning found his source of inspiration in the physiological reaction of the muscles involved in both contractions and expulsions. For a while some authors, like the literary novelist Laurence Sterne, estimated that the force exerted at each pain during labor amounted to 470 lb, he claimed that it should be comprehended between 4 and 37.58 lb, with an average of 16.33 lb. A greater force than this cannot possibly come into play, acknowledged Williams, as it had been shown that 1 of 120 lb was sufficient to tear the child's head from its body [23].

5.3 Chemical Analgesia

Before the development of chemical anesthesia, many of the procedures employed to lessen the pains of labor were of psychological nature. For many midwives and surgeons, the most important was to reassure the mother, or to entertain her thoughts, so she could think of something else. Fear had to be always avoided, since that emotion will have the effect of worsening the labor experience. For Doctor Chapman, for example, pregnant women should not lay their eyes on any surgical instrument that might upset them [24]. Since many pains seemed to be modulated by false expectations and emotional and educational attitudes, their lessening was frequently understood in psychological rather than physiological terms.

Ever since James Young Simpson (1811–1870), a Scottish obstetrician, administered ether to a woman in 1847, it became perfectly clear that the elimination of the mother's suffering did not interfere with the development of the birth. On the contrary, the labor could follow its course even when all painful sensations had disappeared [25]. Between January 1847 and September 1848, Simpson operated on 150 patients under the effects of ether. In November, he began to use chloroform. Administering both of these substances, he was able to prove that not only did physical suffering disappear but there was also a liberation from “*unnecessary mental anguish*,” a decrease in the fear of agony, as well as, in his own words, the disappearance of the nervous shock that often accompanied childbirth.⁴ The advantages of chemical anesthesia in the battle to legitimize obstetrics are beyond doubt. The possibility of avoiding a type of suffering that even some doctors considered agonizing, and of doing so not only in laborious births but in any birth, at will, and without interfering with nature, could only have advantages. Its employment inevitably modified the uses of pain, for even when Simpson himself understood suffering as a sign of uterine contractions, he also recognized that it was

⁴ Simpson, *On the Early History of Anaesthetic Midwifery*, Edinburg, 1848, p. 11.

not a trustworthy indicator. There could be both painless contractions, those that took place with the mother under anesthetic, and pain without contractions, namely “false” or “spurious” pains [26].

Resistance to using both substances came from many sources and acted on different fronts. Some objections were based on technical reasons, but others had ideological motivations. For Doctor Meigs, for example, annulling the pain of childbirth through the inhalation of narcotics was little more than “a questionable attempt to abolish one of the general conditions of man.”⁵ The use of ether was not only an affront to natural morals, it also spread as a consequence of excessive and exaggerated complaints which Doctor Merriman, among others, considered nonexistent in the primitive world and among savage people [27]. In other words, Western women succumbed to the sensitivity of their own education before accepting the natural provision of their suffering, an argument that will come back in the mid-twentieth century, when the practice of “natural childbirth” became a social trend. If they were not troubled by fantastical readings, poorly informed comments, or inappropriate education, the future mothers would take on the pain of birth naturally and with wise resignation. It is not barbarism, but rather the excess of civilization, that has modified the pain threshold, turning a natural event into a nervous crisis. Assuming that there were 50 contractions lasting some 30 s each during a birth of 4 h, Dr. Merriman reasoned, the woman would not suffer for more than 15 or 16 min distributed over 4 h, a proportion that, from his advantage, seemed truly insignificant.⁶

The generalized entrance of surgical practices as well as chemical anesthetics into childbirth led to a series of dilemmas. On the one hand, the elimination of subjective elements allowed the surgeon to concentrate on the objective functions of the organism, without the distractions and accompanying elements of the female condition. On the other hand, chloroform eliminated not only consciousness but also morality. Already at the end of the nineteenth century, there was little doubt that in obstetrics, as distinguished from surgical anesthesia, the purpose was to blunt, not to wholly abolish the sensibilities. From the point of view of many doctors, it was the plain duty of the obstetrician to relieve the needless sufferings of the patient. But anesthetics, however, should not be pushed beyond the stage of mere analgesia [28]. Even if we were to accept that hypersensitivity is a characteristic of civilization, the brute and inert body, subject to the arbitration of its own instincts, guarantees neither decorum nor decency. Even if anesthesia supposed an interruption and modification in the natural course of childbirth, Simpson argued, the same could be said of any other activity in the art of medicine; likewise, the progress of civilization, which permitted the use of footwear or modes of transport, should be considered equally antinatural.

⁵ Simpson, *History*, p. 43.

⁶ Meigs, *Obstetrics*, the Science and the Art, Philadelphia, Blanchard and Lea, 1849, p. 316.

5.4 The Twentieth Century

While the early twentieth century saw the hospitalization of childbirth, the main discrepancies regarding labor analgesia were not related to its use but to the practicalities involved in its employment [29]. More than being a lineal and progressive story, the history of labor pain treatment involves the superposition of different strata. During the twentieth century, the connection between physiological pain and religious guilt was still well extended. For many authors, pain was an essential part of motherhood, which implied that seeking relief equated to an explicit renunciation to develop what they understood as a “noble instinct” of women. Still in 1949, the British Minister of Health asked: “how can a woman have that motherly affection for her offspring if she bears it without pain?” [30]. At the same time, labor pain analgesia was identified by some feminists as a key element within the struggle for women’s rights.⁷ In the USA, the National Twilight Sleep Association counted among their members very active suffragists. For many other women, however, labor pain was only regarded as an extraordinary sensation that could only be labeled “pain” in the case of being pathological: “A woman giving birth, [wrote the writer Enid Bagnold], was not in torture, she was in labor.”⁸ This point of view was in accord with the ideas expressed by Grantley Dick-Read, one of the advocates of the so-called “Natural childbirth,” for whom the principal source of pain during birth was fear. If we could eliminate that dreadful emotion, he argued, most analgesics and anesthetics would be redundant. His method, which included relaxation, exercise, and diet, aimed at the reduction of the pain threshold through the gain of confidence [31]. The division between the point of view of those for whom labor pain was necessary, those for whom pain had to be avoided and those for whom the main constituent element of labor pain was fear did not have a true correspondence in political terms. While fighting against the medicalization of women’s bodies, many feminists regarded labor pain as natural. Conversely, many others considered labor analgesia as another right that women deserved to gain social visibility.

Within the medical profession, there was almost a general agreement in favor of the use of anesthetics. It was not long before the efficiency of the subarachnoidal injection of cocaine was tested upon the parturient woman. The first publication concerning the employment of this primitive epidural injection took place in 1900. It was also at the beginning of the twentieth century that pain relief for childbirth was generally described as anesthesia and not analgesia. It was only later, in the early 1930s that the medical profession tried to distinguish clearly between the two. While anesthesia prevented both sensation and consciousness, in a body under the influence of analgesia, on the other hand, the mother could answer to stimuli without feeling any pain. Among the analgesics more frequently employed in the twentieth century, the anesthesiologist R.J. Minnit designed a portable apparatus

⁷ See [30], p. 126.

⁸ See [30], p. 141.

that could administrate a fixed dose of nitrous oxide. Since the dose could not be modified, the administration of the gas could be undertaken by a midwife and could also take place at home. When oxygen was substituted by air, the addition of a much bigger tank put an end to the portability of the apparatus, which eventually led to a progressive disuse. Despite all, the *machine of Minnit*, known as the *Lucy Baldwin machine* in honor of Mrs Stanley Baldwin, one of the English pioneers in the introduction of analgesics in labor, was still among the most popular of pain-relieving methods in labor during the 1990s.⁹

In 1943, the Birthday Trust put in use a new system based on the use of trilene (trichloroethylene), a substance less toxic than chloroform, that the future Queen Mary employed in the birth of her son, Prince Charles in 1948. On some occasions, the use of hypnotism was also discussed [32]. Whether inclined to the use of chloroform, nitrous gas, ether, ethylchloride, or Nembutal, the main discrepancies within the medical professions were related to which analgesia should be employed and to the best conditions for its use. From a more practical point of view, these discussions regarding technicalities faced a clear difficulty. For one thing, the majority of the population was still anchored in all kinds of old-fashioned remedies, far away from the most modern substances. Besides, there was also a clear-cut barrier between social classes and rural and urban populations. Despite the advances in chemical anesthesia, women did not receive any substances during labor, unless there were complications. In 1929, the Central Public Health Committee of the London County Council stated that only 7 or 8 women, out of 1,747 registered births, were offered some form of pain relief.¹⁰ Both in the USA and in Europe, the majority of women delivered babies without any kind of medical relief, in part as a consequence of the connection they established between the use of those substances and the possibility of further complications. Around 1920, there were only two available methods to relieve labor pain in the United Kingdom: chloroform and the, so-called, twilight sleep. This method, which involved the administration of scopolamine and morphine, produced a state of semiconsciousness, in which women appeared to feel pain but retained no memory of it.¹¹ Despite the reluctance of the medical profession, who thought that the method was inefficient and even not suitable for private use, their advocates launched an aggressive campaign. In the USA, the National Twilight Sleep Association had close links with other feminists' organizations. For their leaders, this method imported from Germany should be regarded as a historical right that women required as a consequence of their nature, debilitated in the process of civilization. In the United Kingdom, the debate on twilight sleep took place at the end of the 1920s. The National Birthday Trust Fund, founded by women from the upper classes, fought to extend maternal services to the most humble population and to the abolition of

⁹ Williams, *Women and Childbirth in the Twentieth Century. A History of the National Birthday Trust Fund 1928–1993*, Stroud, Sutton Publishing, 1997, p. 146.

¹⁰ See [32], p. 124.

¹¹ See [32], p. 128–129.



Fig. 5.2 José Ventura Pastor, *Preceptos Genrales sobre las operaciones de partos*, Madrid, 1789-90, p. 179. Welcome Collection

childbirth pain. For them, as for many other doctors and surgeons, medicalization was a requirement of civilization, a guarantee that would allow reducing women's mortality and decreasing their pains at labor Fig. 5.2 Trelene Breathing Apparatus. For Joseph B. DeLee, an American obstetrician, childbirth could hardly be considered a natural process. On the contrary, it was difficult to believe that a process that killed thousands of women every year, that left a quarter of them more or less invalid, and that involved severe pain and tearing of tissues could be called "a natural or physiological function" [33]. This understanding of labor, as truly pathological, found a just reply by the natural birth movement of Grantly Dick-Read and the similar thesis on "painless childbirth" (psycho-prophylactic labor)

defended by the French obstetrician Fernand Lamaze. Despite their many differences, both systems argued against the medicalization of labor, the abusive use of drugs, and the paternalistic attitude of many obstetricians [34]. Their approach was not only accepted by many women, willing to free themselves from the servitudes of the rather cold and depersonalized birth clinics, but also by the Pope Pius XII, who in 1956 praised the virtues of natural childbirth.

What this cultural fusion of opinions comes to suggest is that the transition from natural to chemical childbirth or from the private home to the public hospital is far from following only one social or political direction. Labor pain analgesia is still full of resistances and accommodations. While even today many feminists claim against the medicalization of labor, defending a more intimate and humane approach to maternity, they seem to forget the feminist origins of that very medicalization, as a result of the high incidence of difficulties related to childbirth, including high maternal death rates. In a more general sense, these apparent contradictions seem the just result of a process, human childbirth, which has been historically regarded and practiced as both natural and artificial, private and public, and whose pain has been understood as either the just price of maternal instinct, the result of civilization, a divine punishment, or a physiological provision that made delivery possible. The many attempts to diminish or abolish pain will have to be also understood within the cultural milieu of all these accommodations and resistances.

References

1. Tyler Smith WM (1868) *A course of lectures of obstetrics—with an introductory lecture on the history of the art of midwifery*, by Augustus K. Gardner, 3rd edn. Robert M. De Witt, New York, p 27
2. Gelbart NR (1998) *The king's midwife. A history and mystery of Madame du Coudray*. University of California Press, Berkeley, CA
3. Wilson A (1985) Participant or patient? Seventeenth century childbirth from the mother's point of view. In: Porter R (ed) *Patients and practitioners. Lay perceptions of medicine in pre-industrial society*. Cambridge University Press, Cambridge, MA, pp 129–145, 134
4. Park K (2006) *Secrets of women. Gender, generation and the origins of human dissection*. Zone Books, New York, NY
5. Shorter W (1985) The management of the normal deliveries and the generation of William Hunter. In: Bynum WF, Porter R (eds) *William Hunter and the eighteenth century medical world*. Cambridge University Press, Cambridge, pp 371–383
6. Adrian Wilson (2002) William Hunter and the varieties of man-midwifery. In: Bynum WF, Porter R (eds) *William Hunter and the Eighteenth-Century Medical World*, Cambridge, Cambridge University Press, 1985, pp 323–343
7. Wilson A (1995) *The making of man-midwifery. Childbirth in England 1660–1770*. UCL Press, London
8. Martin E (1987) *The woman in the body. A cultural analysis of reproduction*. Beacon Press, Boston, MA
9. Gelis J (1991) *History of childbirth*. Polity Press, Cambridge, MA, pp 150
10. Maygrier JP (1841) *Midwifery illustrated*. Harper & Brothers, New York, NY
11. Stolberg M (2000) The monthly malady: a history of premenstrual suffering. *Med Hist* 44:301–322

12. Moscoso J (2012) *Pain: a cultural history*. Palgrave Macmillan, London
13. Simpson JY (1848) *On the early history and progress of Anaesthetic midwifery*. Edinburgh, 1846, p 6
14. Raper HR (1947) *Man against pain. The epic of anaesthesia*. Victor Gollancz, London, p 11
15. Wilson A (1993) The perils of early-modern procreation: childbirth with or without fear? *Br J 18th Cent Stud* 16:1–19, p 3
16. Exton B (1751) *A new and general system of midwifery*. In four parts. W. Owen, London, p 43
17. Dionis P (1724) *Traité général des accouchements, qui instruit de tout ce qu'il faut faire pour être habile accoucheur [1718]*. C.M. d'Houry, Paris, p 209
18. Laurent S (1989) *Naître au moyen age. De la conception a la naissance: la grossesse et l'accouchement*. xii-xv. Le léopard d'or, Paris, pp 186–198
19. Amanda Carson Banks (1999) *Birth chairs, midwives and medicine*. University Press of Mississippi, Jackson, MS, p 35
20. Jewett C (ed) (1899) *The practice of obstetrics*. p 201
21. Cazeaux P (1853) *Traité théorique et pratique de l'art des accouchements [1840]*. Chamerot, Paris, p 430
22. Chapman A (1753) *Treatise of the improvement of midwifery*. p xxxii
23. Williams W (1903) *Obstetrics. A text book for the use of students and practitioners*. Appleton and Company, New York, NY, p 197
24. Chapman A (1753) *Treatise of the improvement of midwifery, chiefly with regard to the operation*. John Brindely, London
25. Simpson JY (1848) *On the early history and progress of anaesthetic midwifery*. Edinburgh, 1846, p 6
26. King H (2007) *Midwifery, obstetrics and the rise of gynaecology. The uses of a sixteenth-century compendium*. Ashgate, Aldershot, p 183
27. Merriman SWJ (1848) *Arguments against the indiscriminatory employment of chloroform in midwifery*. Churchill, London
28. Ramsbotham FH (1842) *The principles and practices of obstetric medicine and surgery, in reference to the process of parturition illustrated by one hundred and forty-two figures*. Lea and Blanchard, Philadelphia, PA
29. Leavitt JW (1986) *Brought to bed. Childbearing in America, 1750–1950*. Oxford University Press, Oxford
30. Williams S (1997) *Women and childbirth in the twentieth century. A history of the National Trust Fund 1928–1993*. Sutton Publishing, Stroud, p 125
31. Cheyner JM (1981) Douleur, souffrance, violence. *Les Dossiers de l'Obstétrique*, special number, *La Douleur* 73:15–19
32. Williams (1903) *Obstetrics*. p 294
33. Flint A (1925) Responsibility of the medical profession in further reducing maternal mortality. *Am J Obstet Gynaecol* 19:864–866 (Quoted by Caton, *What a Blessing*, p 157)
34. Rosenberg C (1987) *The care of the strangers: the rise of America's hospital system*. Basic Books, New York, NY, pp 206–236

Suggested Reading

35. Kay MA (1982) *Anthropology of human birth*. F.A. Davis, Philadelphia, PA
36. Beauvalet-Boutouyrie S (1999) *Naître à l'hôpital au XIX^e siècle*. Belin, Paris
37. Carson A (1999) *Banks, birth chairs, midwives and medicine*. University Press of Mississippi, Jackson, MS
38. Caton D (1999) *What a blessing she had chloroform! The medical and social response to the pain of childbirth from 1800 to the present*. Yale University Press, New Haven, CT
39. Chatti S (2006) *Les couches royales sous l'Ancien Régime: l'étiquette obstétricale, réalisé sous la direction de monsieur le professeur Paul Vert*, Nancy

40. Gelis J (1984) *L'arbre et le fruit: la naissance dans l'Occident moderne (XVI-XIX siècles)*. Fayard, Paris
41. Gelis J (1988) *La sage femme ou le médecin*. Fayard, Paris
42. Gélis J, Morel M-F (1978) *Entrer dans la vie: naissances et enfances dans la France traditionnelle*. Gallimard-Julliard, Paris
43. Hanson C (2004) *A cultural history of pregnancy. Pregnancy, medicine and culture, 1750–2000*. Palgrave MacMillan, London
44. Knibiehler Y (2007) *Accoucher: femmes, sages-femmes et médecins depuis le milieu du XXe siècle*. Rennes, Éd. de l'École nationale de la santé publique
45. Laget M (1982) *Naissances: l'accouchement avant l'âge de la clinique*. Seuil, Paris
46. Martin E (1987) *The woman in the body. A cultural analysis of reproduction*. Beacon Press, Boston, MA
47. Moscoso J (2012) *Pain: a cultural history*. Palgrave Macmillan, London
48. Nivet V (1879) *Notice historique sur Madame Du Coudray, maîtresse sage-femme à Clermont-Ferrand en 1756*. F. Thibaud, Clermont-Ferrand
49. Sandelowski M (1984) *Pain. Pleasure and American childbirth. From the twilight sleep to the read method, 1914–1960*. Greenwood, Westport, CT
50. Tardieu N (2004) *Grossesse et sexualité à travers l'Histoire. Connaissances et savoirs*, Paris
51. Thomas M (ed) (1997) *Post-war mothers. Childbirth letters to Grantly Dick-Read, 1946–1956*. University of Rochester Press, Rochester, NY
52. Leavitt JW (1986) *Brouth to bed. Childbearing in America, 1750–1950*. Oxford University Press, Oxford
53. Williams JW (1903) *Obstetrics. A text book for the use of students and practioners*. Appleton and Company, New York, NY

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6.1 Introduction

Maternal satisfaction is a multifaceted concept, such as labor and delivery, which involves both personal attitudes and affective responses, as well as the cognitive aspect related to emotional responses.

Satisfaction, therefore, is multidimensional, meaning that the woman's expectations may be met for some aspects and not at all for others. Taking a step back, to better understand this articulated concept, there are generally speaking essentially two theories on satisfaction: the performance theory and the discrepancy theory. According to performance theory, satisfaction depends on experience; so expectations and desires are not at all taken into account. According to the discrepancy theory, satisfaction is based on the difference between expectations and desires and what you have and what you get. These two theories will be essential later to understand the concept of satisfaction.

By definition, satisfaction is “the experience which results from the subjective evaluation of the distinction between what actually occurred and what the individual thinks should have” [1], so it is defined as a cognitive evaluation, from affective responses to birth. Obviously, satisfaction with the aspects of care is strongly influenced and shaped by socio-demographic characteristics of women (level of education, age, marital status, and economic status), personal factors (values, attitudes, threshold of pain, health literacy, and personal support), as well as the sense of security and perceived control and expectations formed on the basis of previous experiences and outcomes of previous pregnancies and births [2–6]. It is, in addition, influenced not only by the perceived pain during labor and delivery or the lack of pain due to analgesia but also from obstetric care, the information

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received, and the involvement in decision-making. All these factors contribute to the childbirth's processing [7–14].

Maternal satisfaction, in fact, is influenced by four major categories: control and self-efficacy; involvement in decision-making; maternal expectations; and level of pain [15].

6.1.1 Expectations

Satisfaction with childbirth benefited most consistently from the fulfillment of expectations.

When these expectations are unrealistic, it can place incredible pressure on how women should behave in labor, and the pain relief they end up choosing to accept. Expectations of pregnant women about the level and type of pain may vary in their results. Different women can have different experiences of pain, because labor pain varies dramatically from woman to woman, and even from pregnancy to pregnancy. Labor is the result of a complex and subjective interaction of multiple physiologic and psychological factors on a woman's individual interpretation of labor stimuli. An understanding of labor pain in a multidimensional framework provides the basis for a woman-centered approach to labor pain management that includes a broad range of pharmacologic and nonpharmacologic intervention strategies [16, 17]. The multidimensional aspect and intensity of labor far beats disease conditions, since it is one of the most painful events that a woman is likely to experience [18, 19]. It is therefore not surprising that many pregnant women have concerns about the pain they will encounter and the methods of pain relief that are available during labor.

Women's lack of appropriate knowledge about the risks and benefits of the various methods of pain relief can heighten anxiety [20, 21]. There is an expectation among modern women that childbirth is the worst pain they could ever feel. Because of this, women approach childbirth with overwhelming fear of pain and attempting to remove it all in search of a good birth experience. In contrast to a negative perception, women's positive expectations are associated with the perception of a positive outcome and found that although women found pain hard to describe and often did so in opposing terms, the transition for women as they became mothers gave pain a positive meaning [22–25].

It is important to recognize the potential impact that these differences in expectations might have because choices that are made throughout labor are made on the basis of how women anticipate labor pain [23].

Expectations regarding the level of anticipated pain influenced a woman's perception or satisfaction with the birth experience, either negatively by feeling a failure as they were in greater pain than expected or positively by being pleasantly surprised as excruciating pain never came [26].

The actual experience of labor pain is found to be worse than anticipated most of all because for many women labor is the first experience with any real physical pain [26–30]. It is clear that the experience of pain for many women is different from anticipated. Following on from this, it is stated that if women expect the worst pain

imaginable then they will end up having a painful, negative experience in contrast to women whose view was more optimistic, implying that expectations shape experiences [31]. So, there is a gap between expectation and reality focusing particularly on the underestimation of pain [26–33]. This conclusion is extremely significant since it is well identified that the underestimation of the “intensity” is the primary reason for the gap in reality [34].

There are certainly differences between multiparous and primiparous women, with more primiparous women rating pain as worse than expected [31]. Pain does reduce the quality of the birth experience, but even so, pain control is not the most important factor to have a pleasurable experience [35]. Pain and pain relief, in fact, do not play a major role in childbirth satisfaction, unless expectations regarding either are unmet [5].

Expectations are related to several aspects of labor and delivery, not only pain and emotions [32, 33, 36] but also referring to a role system. The role of a laboring woman involves a set of expectations concerning her own behavior and of people in other roles such as the midwife, the partner, or the physician. By demanding the expected of one’s self and each person present, a workable order is created. Violation of expectations disturbs this order and threatens both self-evaluations and relationships with others. In other words, the deviation from what is normal or expected creates distress [37]. However, mediating factors can play a role between the discrepancy and the reaction to it [38].

Personal control, in fact, has been shown to be a stronger predictor of satisfaction with childbirth; [39] it is crucial to feeling fulfilled and empowered, even if expectations are not completely met. Women determine their satisfaction with their childbirth according to how well they perceived they could manage their own performance [2, 32, 33, 35, 40, 41]. Personal control is different from self-efficacy, which reflects a personality characteristic of confidence in the ability to cope with any stressful situation [42], which predicts a positive childbirth experience [43]. In turn, self-efficacy is also related to lower levels of pain [44, 45] and the method of delivery [15, 46].

In general, the more confident women are to be able to cope with childbirth, the less perceived pain they will feel and better they will cope [47]. If women participate actively, they are empowered by the experience of control [48].

Moreover, this empowering experience has a cumulative effect, increasing self-efficacy for the next birth [32].

Involvement in the decision-making process is also important. By taking an active role in decision-making and receiving appropriate support, in fact, women are more likely to be able to transcend their pain and experience a sense of mastery, control, and well-being, factors associated with their ability to cope with labor [49].

What was found is that women are as concerned about being involved generally [50, 51], that is, being in control [52] and being able to cope [33].

Again, even in this case, multiparous women place emphasis on being fully informed rather than primiparous women.

Determinants such as childbirth expectations [53] and personal control [54] have been shown to be strongly related to the birth environment.

6.2 Information

Regarding education, preparation helped women cope physically and psychologically with their labor; also their knowledge of pain relief helped them make informed choices [33, 55] or it is useful to socialize women about the “appropriate” ways of giving birth rather than educating them [55]. A form of antenatal education needs to be delivered which gives expectant mothers a more realistic expectation of what is likely to happen in labor [30] because if women are not able to have more accurate or realistic expectations about pain in labor they will not be able to prepare themselves appropriately for labor.

There is a gap, in fact, between expectations and actual experiences, concerning women’s expectations and experiences of pain and pain relief during labor and involvement in the decision-making process [56]. Women are inadequately prepared for the reality of labor pain and are unable to make informed choices about pain relief.

In order to close this gap, women need information prenatally about the risks and benefits of methods of pain management and opportunities to practice pain relief methods.

Woman-centered antenatal education and care are needed, as differences exist between women with regard to expectations of comfort and involvement in labor [57, 58].

Antenatal education should improve women’s experience of childbirth, as well as enhance women’s autonomy and ability to access analgesia in labor and empower women with realistic expectations and to enable them to make informed decisions. If women are well prepared during pregnancy, then they are more likely to have realistic expectations of the levels of pain, are less likely to feel a failure, and have increased confidence, which in turn can lead to more a positive experience. Women may have ideal hopes of what they would like to happen, but they need to be educated or informed to ensure that they are prepared for what might actually happen and are given the tools to deal with this.

Therefore, the mismatch between women’s expectations and their actual experiences is a mismatch between how painful women expect labor to be, how long it will last, what pain relief they will need, how in control they will be, and what the actual experience is like. Accordingly, to improve women’s experience of labor, it is necessary to look at how the expectations of these women can be brought more in line with their actual experience.

It could be useful to give women more information to support their choices about pain relief and to help them to make a decision, since women using the decision aid are more likely to consider they had enough information to make decisions about labor analgesia and are more likely to report considering health professionals’ opinions rather than making the decision alone. So, being knowledgeable and informed has been associated with being satisfied with the birth experience and with decision-making [13], and previous research has shown that women want information that includes all the risks and benefits of analgesic options they are considering [13, 59, 60].

Almost all women want to be involved in their labor analgesia decisions, and antenatally most expected to make these decisions themselves. Satisfaction with decision-making could also be affected by the practitioner's relationship rather than with the decision aid, and people find it psychologically comforting to say that they are satisfied rather than retrospectively doubt their own decisions [61]. Women want labor analgesia information and want to participate in their labor analgesia decision-making.

Without some form of education from health professionals, or childbirth educators, women have to rely on media, family, and friends for information, which may not help in forming realistic expectations.

Although not all women attend antenatal classes, it is a key vehicle for education and one which we can endeavor to change to provide a balanced approach to childbirth. It was identified that childbirth training and information on pharmacological pain relief should be regarded as compatible and complementary to other coping mechanisms.

Women need to be prepared for the possibility of pain relief, otherwise feelings of disappointment may arise [62].

6.2.1 Satisfaction

In spite of a considerable amount of study and research, maternal satisfaction has not been clearly defined, mainly because it is necessary to carry out an assessment of women belonging to different structures, with different types of assistance, and also because the studies are too dissimilar and evaluation questionnaires inaccurate [56].

The method of defining satisfaction seems to imply that a "satisfying" experience is synonymous with a "positive" experience and vice versa. Furthermore, it is unclear whether the outcome variables are meant to convey an emotional response to birth, an evaluation of the birth, or both. Specific satisfaction assessments varied widely across studies.

Lack of construct specificity limits the information that can be gained from these measures. It appears that some researchers have conceptualized satisfaction as an emotion or affective response while others regard satisfaction as a cognitive evaluation of whether the birth experience conformed to a patient's standards [63]. Based on literature, no <80 % of the women are satisfied during labor. This is essentially due to the fact that women were asked if it had been a satisfying experience, evaluating it using Visual Analogue Scale (VAS) which is equivalent to asking "How was it." "Home made" or unidimensional questionnaires tend to overestimate the sense of satisfaction, as well as those in which satisfaction is assessed in general terms. Moreover, it often happens that the interviewer is the same person who assisted in the delivery: a positive response is therefore inevitable [64]. It is also important to remember that even the timing for submitting the questionnaire is crucial. Satisfaction may have been influenced by assessment in the first 12–24 h postpartum, in which feelings of dependency and benevolence and

halo effect are common. This effect describes a lack of criticism due to social ability and fear of reprisals or due to a sensation of relief at having gone through a safe experience and having a healthy baby.

In addition, satisfaction could be saddled with denial effects and “What is, must be best” effects. Denying is a way of coping with emotional conflict, stress, painful thoughts, about anything that threatens the sense of control, refusing to acknowledge that something is wrong. Satisfaction, furthermore, may be influenced by “What is, must be best” effects, if there is no reality other than that which was experienced [5, 65].

It is therefore essential to assess maternal satisfaction through questionnaires that assessed it, taking into account all the variables that characterize and influence it. In other words, since satisfaction is difficult to define, assess, and measure, it is mandatory to use validated questionnaires to evaluate maternal satisfaction properly [66].

The birth of a child is often described as one of the most significant and memorable experiences in a woman’s life.

For many mothers, the birth experience has lasting effects despite its relative transience. Positive experiences are an important beginning of the bonding process between mothers and infants, enhancing the new family’s adjustment during the postpartum period [67]. On the other hand, extremely negative birth experiences can be viewed as traumatic and, in some instances, place women at a greater risk of developing clinically significant symptoms of postpartum post-traumatic stress disorder and depression [68]. Assessing satisfaction is therefore crucial to improving maternity care.

In medical ethics, the “goodness” is considered the standard to assess the quality of the doctors’ job (the action is good as it brings the benefit of healing that soothes the painful symptoms).

6.3 Conclusions

The new era of medicine and bioethics must go beyond the pertinence of the action with regard to the purposes to be achieved, and must also encourage medical staff to reach both a more acute sensibility and an attitude that matches the interests of society to the patient’s satisfaction.

Now satisfaction is the substantial parameter to provide an accurate assessment of the medical profession. The good relationship between a doctor and a patient has therefore become the stewardship of relationship, which implies an attitude no longer centered on the professional capability, but on the quality standards of service.

In conclusion, satisfaction is important and maternal satisfaction is even more important because childbirth is the most common and most beautiful reason for hospitalization.

Therefore, it is considerably important to establish maternal satisfaction, to know how to assess and understand its variables, for all healthcare, and for all those working in the obstetrics and gynecology field.

References

1. Janzen JA, Silvius J, Jacobs S et al (2006) What is a health expectation? Developing a pragmatic conceptual model from psychological theory. *Health Expect* 9:37–48
2. Bramadat IJ, Driedger M (1993) Satisfaction with childbirth: theories and methods of measurement. *Birth* 20(1):22–29
3. Linder-Pelz S (1982) Toward a theory of patient satisfaction. *Soc Sci Med* 16:577–582
4. Williams B (1994) Patient satisfaction—a valid concept. *Soc Sci Med* 38:509–516
5. Hodnett ED (2002) Pain and women’s satisfaction with the experience of childbirth: a systematic review. *Am J Obstet Gynecol* 186:S60–S72
6. Dencker A, Taft C, Bergqvist L, Lilja H, Berg M (2010) Childbirth experience questionnaire (CEQ): development and evaluation of a multidimensional instrument. *BMC Pregnancy Childbirth* 10:81–89
7. Hodnett ED, Gates S, Hofmeyr GJ, Sakala C (2007) Continuous support for women during childbirth. *Cochrane Database Syst Rev* 8(3):CD003766
8. Ford E, Ayers S, Wright DB (2009) Measurement of maternal perceptions of support and control in birth (SCIB). *J Womens Health* 18:245–252
9. National Institute for Health and Clinical Excellence (2007) Intrapartum care of healthy women and their babies during childbirth. RCOG, London
10. Green JM, Coupland VA, Kitzinger JV (1990) Expectations, experiences, and psychological outcomes of childbirth: a prospective study of 825 women. *Birth* 17:15–24
11. Green JM, Baston HA (2003) Feeling in control during labor: concepts, correlates, and consequences. *Birth* 30:235–247
12. Lavender T, Walkinshaw SA, Walton I (1999) A prospective study of women’s views of factors contributing to a positive birth experience. *Midwifery* 15:40–46
13. Brown S, Lumley J (1994) Satisfaction with care in labor and birth: a survey of 790 Australian women. *Birth* 21:4–13
14. Waldenstrom U (1999) Experience of labor and birth in 1111 women. *J Psychosom Res* 47:471–482
15. Christiaens W, Bracke P (2007) Assessment of social psychological determinants of satisfaction with childbirth in a cross-national perspective. *BMC Pregnancy Childbirth* 7:26–38
16. Lowe NK (1996) The pain and discomfort of labor and birth. *J Obstet Gynecol Neonatal Nurs* 25:82–92
17. Capogna G, Camorcia M, Stirparo S et al (2010) Multidimensional evaluation of pain during early and late labor: a comparison of nulliparous and multiparous women. *Int J Obstet Anesth* 19:167–170
18. Niven C, Gijsbers K (1984) Obstetric and non-obstetric factors related to labour pain. *J Reprod Infant Psychol* 2:61–78
19. Niven C (1992) *Psychological care for families: before, during and after birth*. Butterworth Heinemann, Oxford
20. Abdullah NR (2002) Pain relief in labour: parent education in the community. In: *Medicine*. Medical School, University of Newcastle, Newcastle upon Tyne
21. Raynes-Greenow CH, Roberts CL, McCaffery K, Clarke J (2007) Knowledge and decision-making for labour analgesia of Australian primiparous women. *Midwifery* 23:139–145
22. Lundgren I, Dahlberg K (1998) Women’s experience of pain during childbirth. *Midwifery* 14:105–110

23. Fenwick J, Hauck Y, Downie J, Butt J (2005) The childbirth expectations of a self-selected cohort of Western Australian women. *Midwifery* 21:23–35
24. Oweis A, Abushaikha L (2004) Jordanian pregnant women's expectations of their first childbirth experience. *Int J Nurs Pract* 10:264–271
25. Green JM, Kitzinger JV, Coupland VA (1990) Stereotypes of childbearing women: a look at some evidence. *Midwifery* 6:125–132
26. Halldorsdottir S, Karlsdottir SI (1996) Journeying through labour and delivery: perceptions of women who have given birth. *Midwifery* 12:48–61
27. Shapiro A, Fredman B, Zohar E, Olsfanger D, Jedeikin R (1998) Delivery room analgesia: an analysis of maternal satisfaction. *Int J Obstet Anesth* 7:226–230
28. Green JM (1993) Expectations and experiences of pain in labour: findings from a large prospective study. *Birth* 20:65–72
29. Peach MJ (1991) The King Edward Memorial Hospital 1000 mother survey of methods of pain relief in labour. *Anaesth Intensive Care* 19:393–399
30. Fridh G, Gaston-Johansson F (1990) Do primiparas and multiparas have realistic expectations of labour. *Acta Obstet Gynecol Scand* 69:103–109
31. Waldenstrom U, Hildingsson I, Rubertsson C, Radestad I (2004) A negative birth experience: prevalence and risk factors in a national sample. *Birth* 31:17–27
32. Slade P, MacPherson SA, Hume A, Maresh M (1993) Expectations, experiences and satisfaction with labour. *Br J Clin Psychol* 32:469–483
33. Gibbins J, Thomson AM (2001) Women's expectations and experiences of childbirth. *Midwifery* 17:302–313
34. Waldenstrom U, Borg IM, Olsson B, Skold M (1996) The childbirth experience: a study of 295 new mothers. *Birth* 23:144–153
35. Doering SG, Entwisle DR, Quinlan D (1980) Modeling the quality of women's birth experience. *J Health Soc Behav* 21:12–21
36. Booth CL, Meltzoff AN (1984) Expected and actual experience in labour and delivery and their relationship to maternal attachment. *J Reprod Infant Psychol* 2:79–91
37. Mirowsky J, Ross CE (1986) Social pattern of distress. *Annu Rev Sociol* 12:23–45
38. Pearlin LI (1989) The sociological-study of stress. *J Health Soc Behav* 30:241–256
39. Goodman P, Mackey MC, Tavakoli AS (2004) Factors related to childbirth satisfaction. *J Adv Nurs* 46:212–219
40. Humenick SS (1981) Mastery: the key to childbirth satisfaction? A review. *Birth Fam J* 8:79–83
41. Kabakian-Khasholian T, Campbell O, Shediak-Rizkallah M, Ghorayeb F (2000) Women's experiences of maternity care: satisfaction or passivity? *Soc Sci Med* 51:103–113
42. Thoits PA (1995) Stress, coping, and social support processes – where are we – what next. *J Health Soc Behav* 35:53–79
43. Crowe K, von Baeyer C (1989) Predictors of a positive childbirth experience. *Birth* 16(2):59–63
44. Larsen KE, O'Hara MW, Brewer KK, Wenzel A (2001) A prospective study of self-efficacy expectancies and labour pain. *J Reprod Infant Psychol* 19:203–214
45. Lowe NK (1989) Explaining the pain of active labor: the importance of maternal confidence. *Res Nurs Health* 12:237–245
46. Dilks FM, Beal JA (1997) Role of self-efficacy in birth choice. *J Perinat Neonatal Nurs* 11(1):1–9
47. Lowe NK (1993) Maternal confidence for labor: development of the Childbirth Self-Efficacy Inventory. *Res Nurs Health* 16:141–149
48. Davenport-Sleck B, Boylan CH (1974) Psychological correlates of childbirth pain. *Psychosom Med* 36:215–223
49. Lowe NK (2002) The nature of labor pain. *Am J Obstet Gynecol* 186:S16
50. McCrea B, Wright M, Holly E (1999) Satisfaction in childbirth and perceptions of personal control in pain relief during labour. *J Adv Nurs* 29:877–884

51. McCrea H, Wright M, Stringer M (2000) The development of a scale to assess control in pain management during labour. *J Reprod Infant Psychol* 18:105–115
52. Callister L (1995) Beliefs and perceptions of childbearing women choosing different primary health care providers. *Clin Nurs Res* 4:168–180
53. Longworth L, Ratcliffe J, Boulton M (2001) Investigating women's preferences for intrapartum care: home versus hospital births. *Health Soc Care Community* 9:404–413
54. Fleming AS, Ruble DN, Anderson V, Flett GL (1988) Place of childbirth influences feelings of satisfaction and control in 1st-time mothers. *J Psychosom Obstet Gynecol* 8:1–17
55. Carlton T, Callister LC, Stoneman E (2005) Decision making in laboring women: ethical issues for perinatal nurses. *J Perinat Neonatal Nurs* 19:145–154
56. Lally JE, Murtagh MJ, Macphail S, Thomson R (2008) More in hope than expectation: a systematic review of women's expectations and experience of pain relief in labour. *BMC Med* 6:7–17
57. Capogna G, Alahuhta S, Celleno D et al (1996) Maternal expectations and experiences of labour pain and analgesia: a multicentre study of nulliparous women. *Int J Obstet Anesth* 5:229–235
58. Raines DA, Morgan Z (2000) Culturally sensitive care during childbirth. *Appl Nurs Res* 13:167–172
59. Paech MJ, Gurrin LC (1999) A survey of parturients using epidural analgesia during labour. Considerations relevant to antenatal educators. *Aust N Z J Obstet Gynaecol* 39:21–25
60. Pattee C, Ballantyne M, Milne B (1997) Epidural analgesia for labour and delivery: informed consent issues. *Can J Anaesth* 44:918–923
61. O'Connor A, Bennett C, Stacey D, et al (2009) Decision aids for people facing health treatment or screening decisions. *Cochrane Database Syst Rev* (10):CD001431
62. Kangas-Saarela T, Kangas-Kärki K (1994) Pain and pain relief in labour: parturients' experiences. *Int J Obstet Anesth* 3:67–74
63. Stevens NR, Hamilton NA, Wallston KA (2011) Validation of the multidimensional health locus of control scales for labor and delivery. *Res Nurs Health* 34:282–296
64. Robinson PN, Salmon P, Yentis SM (1998) Maternal satisfaction. *Int J Obstet Anesth* 7:32–37
65. Waldenström U, Borg IM, Olsson B, Sköld M, Wall S (1996) The childbirth experience: a study of 295 new mothers. *Birth* 23:144–153
66. Stirparo S et al (2013) A multidimensional questionnaire to evaluate maternal satisfaction after labor analgesia. *Eur J Anaesthesiol* 30:169
67. Di Matteo MR, Kahn KL, Berry SH (1993) Narratives of birth and the postpartum: analysis of the focus group responses of new mothers. *Birth* 20:204–211
68. Ayers S, Joseph S, McKenzie-McHarg K, Slade P, Wijma K (2008) Post-traumatic stress disorder following childbirth: current issues and recommendations for future research. *J Psychosom Obstet Gynaecol* 29:240–250

Gary M. Stocks and Sarah K. Griffiths

7.1 Introduction

Neuraxial techniques for labor have consistently been shown to be the best method of providing analgesia when compared with all other modalities [1]. Both conventional epidural analgesia and combined spinal-epidural analgesia (CSE) can be used. This chapter outlines the indications and contraindications to the use of labor neuraxial techniques and details the technical and practical aspects of epidural and CSE initiation, including the choice of drugs and drug combinations used in contemporary practice. The advantages and disadvantages of the two techniques are also compared.

7.2 Indications

7.2.1 Maternal Request

Maternal request for regional analgesia in labor in the absence of any other indication is the commonest reason for the use of neuraxial blockade.

The decision regarding the type of pain relief that a woman might choose during her labor is governed by many factors such as the severity of the pain, the expectations of the mother, and the complexity of the labor. For many women the pain of labor is likely to be the most severe pain experienced in their lifetime [2] and some form of pain relief will be required. Attitudes about how best to provide this pain relief vary amongst mothers themselves and their healthcare professionals. Some believe that pain is part of the birth experience and that women should be supported through this, whilst others argue that with the availability of effective

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analgesic techniques, no woman should be expected to suffer pain in labor. All healthcare professionals should respect the wishes of laboring women and appreciate that in the dynamic situation of labor these views may change. Women should therefore be supported in their birth choice and should not be made to feel that their experience of childbirth is diminished if they decide to have epidural analgesia.

Providing women are properly informed about the advantages and disadvantages of epidural analgesia in labor, “In the absence of a medical contraindication, maternal request is a sufficient medical indication for pain relief during labor. Pain management should be provided whenever medically indicated” [3].

7.2.1.1 Timing

Women requesting epidural analgesia in labor will do so when they feel they need pain relief. For many this will be quite early in labor. Observational studies [4, 5] in the past have suggested that the early initiation of epidural analgesia may be associated with an increased risk of cesarean section (CS). In particular, one retrospective study reported that the greatest effect of epidural analgesia on the risk of CS was for nulliparous women in early labor receiving an epidural before they had reached 5 cm of cervical dilatation. Despite the obvious limitations of retrospective studies, a guideline was published in 2002 [6] that recommended that the administration of epidural analgesia in nulliparous women should be delayed until cervical dilatation had reached 4–5 cm.

These recommendations have subsequently been withdrawn because a number of randomized controlled trials have demonstrated, with a high level of evidence, that epidural analgesia administered during the early first stage of labor does not affect the progress of labor or mode of birth compared with administration later in labor [7, 8]. In the largest randomized prospective trial of labor epidurals ever to be carried out in a single centre, Wang et al. [9] randomized 12,793 nulliparous women to receive an epidural at either 1 or 4 cm of cervical dilatation and confirmed that epidural analgesia commenced early had little effect on the progress and outcome of labor. National guidelines now state that “women in labor who desire regional analgesia should not be denied it, including women in severe pain in the latent first stage of labor” [10].

7.2.2 Medical

Neuraxial techniques used for labor analgesia can assist in the attenuation of pain-mediated hypertensive and hyperventilatory responses and may reduce cardiac workload and myocardial stress during contractions. Therefore regional analgesia may be particularly beneficial in women with pre-eclampsia and in those with comorbid cardiac, cerebrovascular, or respiratory disease. It may also be medically advisable for parturients with a high body mass index ($\text{BMI} > 30 \text{ kg/m}^2$). Insertion of neuraxial blocks in these women may be technically challenging; therefore early insertion is recommended.

Subsequent intra-partum complications may necessitate a delivery by emergency CS. The presence of a working epidural catheter in labor avoids the risks of general anesthesia such as aspiration, failed intubation, and the negative

inotropic effects of positive pressure ventilation which may be particularly problematic in these at risk parturients.

7.2.3 Obstetric

Neuraxial analgesia in labor may be indicated in circumstances where vaginal delivery is expected to be prolonged, traumatic, or where there is a high probability of requiring an instrumental delivery or CS. Such situations may include multiple pregnancy, fetal macrosomia, and vaginal delivery of a breech presentation or preterm baby.

7.3 Denying Regional Analgesia and Assessment of Risk

The decision to deny regional analgesia should never be taken lightly. Each case should be considered on its own merits with an appropriate analysis of the risks and benefits. Absolute contraindications to the performance of labor neuraxial techniques include patient refusal, inadequate staffing levels, localized infection at the intended site of needle insertion, and raised intracranial pressure. Relative contraindications include congenital or acquired lesions of the spinal cord, systemic sepsis, and severe pulmonary hypertensive disease. Due to the increased risk of spinal hematoma, the performance of neuraxial techniques is a contentious issue in patients with hereditary or acquired causes of thrombocytopenia, and bleeding diatheses and in those who are receiving concurrent anticoagulant treatment. The absolute platelet count below which it is unsafe to perform neuraxial blockade is unknown. The direction of change in platelet count and the function of the platelets are also important considerations. Most anesthetists would consider non-neuraxial forms of analgesia in women with platelet counts of $50\text{--}75 \times 10^9/\text{L}$ or lower [11].

Denial of regional analgesia in labor increases the need for general anesthesia should an operative delivery be required. The risks of general anesthesia for CS are well documented and include a 1 in 200–300 chance of a failed or difficult intubation [12]. This must be compared with the relatively rare complications of central neuraxial blockade identified in the NAP 3 audit [13], in which the risk of permanent harm (including epidural hematoma) after obstetric regional blockade is quoted as 1:80,000.

The assessment of risk is difficult in parturients with abnormalities of coagulation as serious complications are rare. Table 7.1 shows some of the recommendations made by the European Society of Anaesthesiology (ESA) for regional anesthesia in the presence of antithrombotic agents [14]. In an attempt to compare risks, a recent joint publication [15] defines several high-risk scenarios. Two of which are:

1. A parturient with preeclampsia who has a falling platelet count between 75 and 100 and normal coagulation.
2. The administration of regional blockade 6–12 h after a thromboprophylactic dose and 12–24 h after a treatment dose of low molecular weight heparin.

Table 7.1 Recommended time intervals between anticoagulant drug administration and insertion of neuraxial block or epidural catheter removal

| Anticoagulant drugs | Time before insertion/catheter removal | Time after insertion/catheter removal |
|-----------------------------------|----------------------------------------|---------------------------------------|
| <i>Heparins</i> | | |
| LMWH (for prophylaxis) | 12 h | 4 h |
| LMWH (for treatment) | 24 h | 4 h |
| UFH (for prophylaxis) | 4–6 h | 1 h |
| UFH (for treatment) | 4–6 h (intravenous) | 1 h (intravenous) |
| | 8–12 h (subcutaneous) | 1 h (subcutaneous) |
| <i>Coumarins</i> | | |
| Warfarin | INR \leq 1.4 | After catheter removal |
| <i>Inhibitors of ADP binding</i> | | |
| Clopidogrel | 7 days | After catheter removal |
| Ticlopidine | 10 days | After catheter removal |
| <i>Factor Xa inhibitors</i> | | |
| Rivaroxaban (for prophylaxis) | 22–26 h | 4–6 h |
| Apixaban (for prophylaxis) | 26–30 h | 4–6 h |
| <i>Direct thrombin inhibitors</i> | | |
| Dabigatran (for prophylaxis) | Contraindicated as per manufacturer | 6 h |
| <i>NSAIDs</i> | None | None |

Partially based on data from [14]

LMWH low molecular weight heparin, *UFH* unfractionated heparin, *INR* international normalized ratio, *NSAIDs* nonsteroidal anti-inflammatory drugs

The guidelines equate the risk in these scenarios to the risk of having a general anesthetic with a full stomach in labor.

7.4 Initiation of Epidural Analgesia

Epidural analgesia is the most frequently used neuraxial technique to provide pain relief in labor. With local anesthetic skin infiltration, the epidural space is located using an epidural needle and a loss-of-resistance technique. A catheter is then threaded into the space, remaining there for the duration of the labor. Usually a mixture of local anesthetic and opioid is injected into the epidural space to provide a selective analgesic block from T10 to S5 with minimal effect on motor power. In this section some of the technical and pharmacological aspects of epidural analgesia are discussed.

7.4.1 Technical

7.4.1.1 Maternal Positioning

Labor epidurals may be inserted with the parturient in a sitting or lateral decubitus position. In order to reduce aortocaval compression, the parturient should be encouraged to allow the pregnant abdomen to rest between her thighs during epidural insertion in the sitting position and to avoid over flexion of the hips during insertion in the lateral position.

The main advantage of the sitting position compared to the lateral position is the ability to identify the midline of the back and iliac crests more easily, particularly in obese patients. Additionally, in the sitting position the distance to the epidural space is reduced [16, 17] and it is more comfortable, particularly for obese women (BMI > 30 kg/m²) [18]. Aortocaval compression is minimized in the lateral position and uteroplacental blood flow is optimized, which may be beneficial for women in preterm labor, approaching full cervical dilatation, or with evidence of fetal compromise. In addition, there is a lower incidence of epidural vein cannulation compared to the sitting position [19].

7.4.1.2 Use of Ultrasound

Ultrasound imaging of the back prior to epidural placement is increasingly popular. It can be used to identify lumbar vertebral level and midline spinous processes and to estimate the approximate distance to the epidural space [20, 21]. It may also reduce the number of attempts at epidural placement and epidural re-site rates [22], and be of benefit in obese parturients and those with scoliosis.

7.4.1.3 Aseptic Technique

The development of an infectious complication following an obstetric neuraxial technique is a rare but potentially devastating event. Anesthetists should adhere to meticulous aseptic technique during block insertion which includes thorough hand washing, the use of surgical caps, sterile gloves, gowns, a mask which covers the anesthetist's nose and mouth, and sterile draping of the patient's back. Chlorhexidine is routinely used as a skin preparation prior to block insertion. Povidone-iodine is an inferior bactericidal agent but may be used as an alternative. Both preparations are more efficacious when prepared in alcohol. Due to their devastating neurotoxic effects, it is vitally important to avoid contamination of the neuraxis with either chlorhexidine or povidone-iodine. Extreme care must be taken to follow the manufacturers' instructions, to prevent the inadvertent injection of skin preparation instead of saline, and to allow time for the skin to dry thoroughly prior to needle insertion [23, 24].

7.4.1.4 Epidural Needles and Loss of Resistance Techniques

Using a midline or paramedian approach with a loss of resistance to injection technique, a 16G or 18G Tuohy needle with a Huber tip is inserted into the epidural space. This technique is made possible due to the presence of a negative pressure within the epidural space which results in an end point experienced as a sudden

“give” when a syringe containing fluid [25, 26] or air is attached to the epidural needle and advanced through the ligamentum flavum. Needle advancement through the tissues may be either continuous or intermittent, with a continuous technique preferred when using saline or an intermittent technique with air. Loss of resistance to saline is used by the majority of anesthetists [27, 28] because it is associated with lower rates of accidental dural and vascular puncture and a less patchy block compared to air. Loss of resistance to local anesthetic is not recommended as a technique, since it carries the rare but potentially devastating risk of total spinal anesthesia if accidental dural puncture occurs.

7.4.1.5 Epidural Catheters and Fixation

Epidural catheters may be single or multi-orifice. The latter are closed at the distal end with a series of three lateral holes proximal to this which allow better spread of local anesthetic. They are the predominant type of catheter used and they result in fewer incidences of inadequate analgesia and less requirement for catheter manipulation in labor [29, 30].

Once the epidural space has been identified, the catheter is threaded and secured to the skin. Preadmission of the epidural space with saline prior to the threading of the epidural catheter has been advocated as one method of reducing the incidence of inadvertent intravenous cannulation [31]. It is common practice to leave 3–5 cm of catheter within the epidural space. Longer lengths of catheter are associated with a higher incidence of unilateral block and a greater likelihood of the tip entering an epidural blood vessel [32], whilst too little catheter length predisposes the catheter to falling out.

There is little consensus on the optimum method of catheter fixation to the skin, with a variety of sterile dressings and specialised catheter locking devices available for use. If a catheter is fixed with the mother in a flexed position it can be pulled out of the epidural space by up to 1 cm when the mother subsequently adopts the upright position [16]. Radiological contrast studies have also demonstrated that movement of an epidural catheter may still occur at the level of the ligamentum flavum, even in the absence of movement of the catheter at the level of the skin [33]. In these cases, epidural failure from catheter migration may be associated more strongly with the initial length of catheter left within the epidural space.

7.4.1.6 Choice of Local Anesthetic

The three main local anesthetics used for the initiation of labor analgesia are bupivacaine, levobupivacaine, and ropivacaine.

Bupivacaine is a chiral mixture of levo (s-) and dextro (r-) bupivacaine and provides epidural analgesia within 10 min, with a peak effect at approximately 20 min [34]. It binds strongly to cardiac muscle after inadvertent intravascular injection or where dosing has reached toxic levels. The resultant blockade of sodium and potassium channels in the myocardium has been associated with fatal or near fatal arrhythmias. The cardiotoxic effects of bupivacaine are believed to be mainly due to the dextro (r-) racemate of bupivacaine present in the chiral mixture.

Levobupivacaine is the pure *s*-enantiomer of bupivacaine, whilst ropivacaine is structurally similar to bupivacaine and differs only in the replacement of the butyl group with a propyl group resulting in a less lipid-soluble drug. These newer amide local anesthetics are considered to have a greater safety margin and a reduced potential for cardiotoxicity because they are prepared as single enantiomers [35, 36]. Since the introduction of levobupivacaine and ropivacaine into obstetric anesthesia, many studies have been performed to determine comparative analgesic potencies, in order to meaningfully compare side effect profiles with that of bupivacaine. The minimum local analgesic concentration (MLAC) of a local anesthetic is defined as the median effective local analgesic concentration (EC₅₀) in a 20 mL volume to provide epidural analgesia in the first stage of labor [37, 38].

Bupivacaine has been shown to be significantly more potent than ropivacaine by a factor of approximately 0.4 (MLAC for ropivacaine 0.100–0.176 %; MLAC for bupivacaine 0.052–0.110 %) [39, 40], with levobupivacaine and ropivacaine almost equipotent in terms of analgesic potency [41].

The choice of local anesthetic does not appear to have a significant effect on the outcome of labor. A large meta-analysis demonstrated no difference in the incidence of spontaneous vaginal delivery after the use of bupivacaine or ropivacaine for labor epidural analgesia [39].

7.4.1.7 Addition of Opioid

Fentanyl and sufentanil are highly lipid-soluble opioids which are routinely added to the local anesthetic solution used for neuraxial analgesia in labor. They have a rapid onset of action and duration of 1–2 h when given as a bolus. Fentanyl is usually used at a concentration of 2 µg/mL and sufentanil at a concentration of 1 µg/mL. They act synergistically to reduce the MLAC of local anesthetic required to provide satisfactory pain relief [42, 43]. The addition of opioid also provides greater maternal satisfaction with analgesia [44] and may reduce the degree of motor block experienced by the parturient [42]. Common maternal side effects experienced with the use of epidural opioids include nausea and vomiting, pruritus, respiratory depression, and hypotension. Fetal bradycardia may also be seen with epidural opioids, although this side effect is more commonly associated with the use of intrathecal opioid for CSE in labor.

7.4.1.8 Low-Dose Epidural Mixtures

The use of bupivacaine in concentrations ≥ 0.25 % results in a high degree of motor block in parturients [45, 46]. The combination of lower dose local anesthetic with opioid provides good analgesia with a lower total dose of local anesthetic. This not only reduces the risk of local anesthetic toxicity but results in less motor block and improved maternal satisfaction [47, 48]. The 2001 Comparative Obstetric Mobile Epidural Trial (COMET) randomized women to receive either a low-dose epidural infusion, CSE with intermittent low-dose local anesthetic and opioid top-ups, or 0.25 % bupivacaine epidural top-ups for labor. Women in the first two groups had a reduced incidence of instrumental vaginal delivery compared with those women in the latter group [49].

Table 7.2 Commonly used concentrations and doses of local anesthetic and opioid for the initiation of labor epidural analgesia

| | Concentration/dose |
|--------------------------------------|--------------------|
| <i>Local anesthetics^a</i> | |
| Bupivacaine | 0.0625–0.125 % |
| Levobupivacaine | 0.0625–0.125 % |
| Ropivacaine | 0.06–0.2 % |
| <i>Opioids</i> | |
| Fentanyl | 1–5 µg/mL |
| Sufentanil | 0.1–0.3 µg/mL |

^a10–20 mL volume routinely used for initiation of labor epidural analgesia

Most centres now favour the use of low-dose mixtures of levobupivacaine or ropivacaine with the addition of opioid to provide epidural neuraxial analgesia for women in labor. Commonly used concentrations and doses of local anesthetic and opioid for the initiation of labor analgesia are shown in Table 7.2.

7.4.1.9 Epidural Test Doses

The administration of a “test dose” down an epidural catheter is intended to identify inadvertent intrathecal or intravascular placement. In contemporary practice, maintenance of labor analgesia is via the use of dilute “low-dose” mixtures of local anesthetic and opioid. The administration of the first dose of this “low-dose” mixture as a test dose is now routine in most centres. This will reliably identify intrathecal catheter placement as evidenced by the development of a motor block at 5 min, without the risk of developing a high block or severe hypotension. A test dose of this dilute local anesthetic solution may also help to detect intravascular placement if there is a subsequent lack of analgesia together with an equivocal aspiration test for blood [50]. Since there is always the potential for migration of a previously well-positioned epidural catheter, the administration of subsequent doses should always be performed with care.

7.5 Initiation with CSE

Combined spinal-epidural analgesia is an increasingly popular technique for initiating pain relief in labor, with some units using it as their standard technique and others using it according to clinical circumstances [51, 52]. The technique involves an intrathecal injection, usually of a small dose of local anesthetic and lipophilic opioid, followed by the placement of a catheter into the epidural space to maintain analgesia during labor. As with low-dose epidural analgesia high-quality pain relief is achieved, allowing mobilization in labor and the ability to titrate and extend analgesia to anesthesia for CS. However, the main advantage of CSE compared to low-dose epidural analgesia is the ability of the spinal component to

Table 7.3 Common indications for CSE versus epidural alone

| |
|--------------------------------------------------------------------------------------------|
| Severe maternal distress regardless of cervical dilatation |
| Analgesia in the late first stage of labor (>8 cm) and the second stage of labor |
| Anesthesia for delivery (second stage) |
| Anesthesia for artificial rupture of membranes (ARM) |
| Multiparae in established labor |
| Patient has had unsatisfactory epidural in the past when spinal component was not included |
| The difficult back |

provide rapid onset of pain relief and reliable sacral analgesia. This makes CSE a particularly attractive option for the provision of analgesia in late labor or for a parturient with rapid progress in labor [53].

7.5.1 Indications for CSE in Labor

Common indications for CSE as compared to epidural alone are listed in Table 7.3. For artificial rupture of membranes (ARM), CSE provides immediate, more profound analgesia and may be allowed to wear off if labor is slow to be established; however, an uncomplicated ARM does not necessarily require analgesia. For patients with a difficult back the spinal component of a CSE confirms Tuohy needle placement.

7.5.2 Techniques for Initiation of CSE Analgesia

Several CSE techniques have been described and the advantages and disadvantages have been reviewed elsewhere [54]. Separate needle techniques can be used where the intrathecal injection is performed with one needle and the epidural catheter placed with another, in either order, using either a single intervertebral space or separate interspaces.

7.5.2.1 Needle-Through-Needle

The most commonly used technique in clinical practice is the needle-through-needle technique. Using this technique the epidural space is located in the normal way with an epidural needle. An extra-long, 25G or smaller, spinal needle is then passed through the epidural needle to puncture the dura. The epidural needle thus effectively acts as an introducer. Usually a characteristic dural “click” is felt and CSF seen in the hub of the spinal needle. An intrathecal injection is then performed, after which the spinal needle is withdrawn and an epidural catheter is threaded down the epidural needle into the epidural space.

7.5.3 Considerations Relating to the CSE Technique

7.5.3.1 Failure of the Spinal Component

When using a needle-through-needle technique, failure of the spinal component can occur for a number of reasons. The spinal needle may fail to reach or only tent the dura and not puncture it. Alternatively, the spinal needle may pass to the side of the dural sac indicating that the epidural needle is not perfectly in the midline. Finally, the spinal needle may move during the intrathecal injection because it has not been immobilized properly. This complication becomes less frequent with practice but can also be reduced by the use of needle locking devices, several of which are commercially available and have been shown to have low failure rates [55].

7.5.3.2 Inadvertent Insertion of the Catheter into the Subarachnoid Space

Initial concerns that the deliberate puncture of the dura by a spinal needle would increase the chance of threading an epidural catheter through the same hole appear to be unfounded. Epiduroscopic studies [56] suggest that it is extremely unlikely that an epidural catheter can be passed intrathecally through a small hole created with a 27G pencil point spinal needle. However, some epidural needles have been designed with “back eyes” which divert the tip of the spinal needle away from the Huber point to prevent this complication.

7.5.3.3 Testing the Epidural Catheter

One disadvantage of the CSE technique is the inability to test the location of the epidural catheter until after the spinal component has worn off. This may be problematic if an emergency CS is required at this time, but, as with conventional epidural analgesia, top-ups of high concentration local anesthetic should be given in fractionated doses. Epidural test doses in labor are discussed in Sect. 7.4.1.9.

7.5.4 Intrathecal Drug Recipes

The ideal intrathecal drug recipe has yet to be determined. The most commonly used drugs are the lipophilic opioids fentanyl or sufentanil, usually in combination with a local anesthetic, most frequently bupivacaine, levobupivacaine, or ropivacaine. Intrathecal opioid alone can be used to initiate analgesia in early labor which can do so without the motor and sympathetic block associated with local anesthetics. The optimum intrathecal dose of fentanyl alone is 25 μg [57] and the estimated ED95 of intrathecal sufentanil is 8.9 μg [58].

It is much more common to use an opioid in combination with an amide local anesthetic. The addition of fentanyl markedly reduces the ED50 dose of bupivacaine required to produce analgesia in the first stage of labor [59] and combinations of intrathecal bupivacaine and fentanyl have a supra-additive/synergistic interaction [60]. Thus the combination allows lower doses of both drugs to be used and so reduces unwanted side effects.

The most common local anesthetic used in combination with an opioid is bupivacaine, but levobupivacaine and ropivacaine are also effective. In studies without the use of opioid, Camorcia et al. reported a potency hierarchy of bupivacaine > levobupivacaine > ropivacaine [61], whilst Sia et al. demonstrated a similar potency ratio to that of Camorcia for levobupivacaine and ropivacaine but much lower ED50 values [62]. When combined with sufentanil, bupivacaine is more potent than levobupivacaine and ropivacaine, both of which were shown to be as potent as each other [63].

For the clinician, however, it is the ED95 dose that is more relevant. For bupivacaine, a dose of 2.5 mg with or without opioid has been shown to be enough to provide adequate analgesia [64]. For levobupivacaine and ropivacaine, 2.5–3.0 mg is sufficient to provide good analgesia [62]. The addition of opioid improves the quality of analgesia and prolongs its effect in a dose-dependent manner [59] and typical doses of fentanyl range from 10 to 25 µg and sufentanil 1.5–2.5 µg [65].

7.6 Low-Dose Epidural or CSE for Initiation of Labor Analgesia?

Whether to initiate analgesia in labor with a low-dose epidural or CSE remains a decision largely based on the preference and familiarity of the anesthetist with the chosen technique and the clinical circumstances of the parturient. Opinion remains divided over whether CSE should be the technique of choice in all cases or whether it should be reserved for certain indications. When evaluating techniques the advantages and disadvantages must be considered.

7.6.1 Onset and Quality of Analgesia

The main advantage of CSE is the rapid onset of reliable analgesia regardless of what stage in labor the block is sited. CSE consistently provides analgesia within 4–6 min of the intrathecal injection with the mean time of onset being 5.42 min quicker than low-dose epidural. There would appear to be no difference in the degree of maternal satisfaction between the two techniques [53].

7.6.2 Epidural Catheter Reliability

Proponents of the CSE technique argue that it is likely that the epidural catheter will subsequently function more effectively in labor because the epidural needle must be in the midline to perform the intrathecal injection. Several studies have been identified demonstrating equal or improved catheter function after CSE compared to epidural techniques [65], but a recent meta-analysis did not show any difference in the need for rescue analgesia following CSE or epidural alone [53].

7.6.3 Pruritus

Opioid-induced pruritus is a common problem and has been shown to have a dose-dependent relationship [59]. A recent Cochrane review [53] identified significantly more pruritus with CSE compared to low-dose epidural, but in practice the severity of pruritus is rarely sufficient to require treatment.

7.6.4 Infection and Neurotrauma

The major hazards of epidural and CSE analgesia are infection and neurotrauma, but fortunately these are rare [13]. In anesthetic practice there is no evidence that CSE is associated with more infectious complications than spinal or epidural [66], provided a scrupulous aseptic, non-touch technique is used. It is recognized that neurotrauma is more prevalent with intrathecal cannulation and several cases have been reported of damage to the conus medullaris when using a CSE [67]. It is very important therefore that the block be performed at or below the L3/4 interspace, as in a small number of patients the conus medullaris might extend below the L2 vertebral body.

7.6.5 Post Dural Puncture Headache

As CSE involves the deliberate puncture of the dura, there is a theoretical risk of Post Dural Puncture Headache (PDPH). However, many studies have shown no increased incidence of PDPH compared with conventional epidural analgesia including one large study with over 16,000 CSEs [53, 68].

7.6.6 Fetal Heart Rate Changes

There are a number of reports of fetal heart rate abnormalities including severe bradycardia following the administration of intrathecal opioids during the first stage of labor. A meta-analysis of 24 trials (3,513 women) found that the odds ratio for fetal bradycardia was significantly increased in patients who received intrathecal opioids [OR 1.81 (95 % CI 1.04–3.14)] compared to other forms of pain relief [69]. In a prospective randomized trial Van de Velde et al. [70] concluded that high doses of intrathecal opioid increased the incidence of significant fetal heart rate changes. The mechanism for this is not completely understood but appears to be related to several factors including the sudden drop in pain level, an imbalance of plasma catecholamines resulting in uterine hypertonus, and a possible direct central effect of intrathecal opioid. Clinically, opinion is divided as to whether these opioid-induced fetal heart rate changes are important. Case reports have described women requiring emergency CS for persistent fetal bradycardia following administration of intrathecal fentanyl [71], but trials and a meta-analysis have failed to show any

increase in emergency CS rate or neonatal morbidity [69, 70]. Nevertheless, some authors have recommended that intrathecal opioid be avoided in cases when fetal distress or uterine hypertony is present before labor analgesia is requested and to consider using local anesthetic alone when using a CSE in this circumstance [22].

7.7 Conclusion

In summary, there is no conclusive evidence to recommend one technique over another for initiation of analgesia in labor [53]. CSE provides rapid onset of pain relief and reliable sacral analgesia making it a particularly attractive option for the provision of analgesia in late labor, for those parturients with severe pain in early labor and for those who experience rapid progress in labor. However, nonreassuring fetal heart rate abnormalities occur more frequently with CSE analgesia using high-dose intrathecal opioids. The decision to initiate labor analgesia with a CSE or a low-dose epidural therefore depends on the needs of the mother, the safety of the fetus, and the expertise of the anesthetist.

References

1. Anim-Somuah M, Smyth R, Howell C (2005) Epidural versus non-epidural or no analgesia in labour. *Cochrane Database Syst Rev* (4):CD000331
2. Melzack R (1984) The myth of painless childbirth (John J Bonica lecture). *Pain* 19(4):321–337
3. American College of Obstetricians and Gynecologists Committee on Obstetric Practice (2006) Analgesia and cesarean delivery rates. ACOG Committee Opinion No. 339. *Obstet Gynecol* 107:1487–1488
4. Thorp JA, Eckert LO, Ang MS (1991) Epidural analgesia and cesarean section for dystocia: risk factors in nulliparas. *Am J Perinatol* 8(6):402–410
5. Lieberman E, Lang JM, Cohen A et al (1996) Association of epidural analgesia with cesarean delivery in nulliparas. *Obstet Gynecol* 88(6):993–1000
6. American College of Obstetricians and Gynecologists Committee on Obstetric Practice (2002) Task force on cesarean delivery rates. Evaluation of cesarean delivery. ACOG Committee Opinion No. 269
7. Wong CA, Scavone BM, Peaceman AM (2005) The risk of cesarean delivery with neuraxial analgesia given early versus late in labor. *N Engl J Med* 352(7):655–665
8. Ohel G, Gonen R, Vaida S et al (2006) Early versus late initiation of epidural analgesia in labor. Does it increase the risk of cesarean section? A randomized trial. *Am J Obstet Gynecol* 194(3):600–605
9. Wang FZ, Shen XF, Guo XR et al (2009) The Labor Analgesia Examining Group (LAEG): epidural analgesia in the latent phase of labor and the risk of cesarean delivery: a five-year randomized controlled trial. *Anesthesiology* 111:871–880
10. National Collaborating Centre for Women's and Children's Health (2007) Intrapartum care—care of healthy women and their babies during childbirth. NICE Clinical Guideline 55, Royal College of Obstetricians and Gynaecologists (RCOG), London, pp 109–137
11. Douglas MJ (2001) Platelets, the parturient and regional anesthesia. *Int J Obstet Anesth* 10:113–120
12. Rahman K, Jenkins JG (2005) Failed tracheal intubation in obstetrics: no more frequent but still managed badly. *Anaesthesia* 60:168–171

13. Cook TM, Counsell D, Wildsmith JAW (2009) Major complications of central neuraxial block: report on the Third National Audit Project of the Royal College of Anaesthetists. *Br J Anaesth* 102(2):179–190
14. Gogarten W, Vandermeulen E, Van Aken H et al (2010) Regional anaesthesia and antithrombotic agents: recommendations of the European Society of Anaesthesiology. *Eur J Anaesthesiol* 27:999–1015
15. Membership of the Working Party, Harrop-Griffiths W, Cook T, Gill H et al (2013) Regional anaesthesia and patients with abnormalities of coagulation. *Anaesthesia* 68(9):966–972
16. Hamilton CL, Riley ET, Cohen SE (1997) Changes in the position of epidural catheters associated with patient movement. *Anesthesiology* 86(4):778–784
17. Hamza J, Smida M, Benhamou D et al (1995) Parturient's posture during epidural puncture affects the distance from skin to epidural space. *J Clin Anesth* 7(1):1–4
18. Vincent RD, Chestnut DH (1991) Which position is more comfortable for the parturient during identification of the epidural space? *Int J Obstet Anesth* 1(1):9–11
19. Tsen LC (2008) Neuraxial techniques for labor analgesia should be placed in the lateral position. *Int J Obstet Anesth* 17:146–152
20. Balki M, Lee Y, Halpern S, Carvalho JCA (2009) Ultrasound imaging of the lumbar spine in the transverse plane: the correlation between estimated and actual depth to the epidural space in obese parturients. *Anesth Analg* 108(6):1876–1881
21. Arzola C, Davies S, Rofael A, Carvalho JCA (2007) Ultrasound using the transverse approach to the lumbar spine provides reliable landmarks for labor epidurals. *Anesth Analg* 104:1188–1192
22. Loubert C, Hinova A, Fernando R (2011) Update on modern neuraxial analgesia in labour: a review of the literature of the last 5 years. *Anaesthesia* 66:191–212
23. Horlocker TT, Bimbach DJ, Connis RT et al (2010) Practice advisory for the prevention, diagnosis, and management of infectious complications associated with neuraxial techniques: a report by the American Society of Anesthesiologists Task Force on Infectious Complications Associated with Neuraxial Techniques. *Anesthesiology* 112:530–545
24. Bogod D (2012) The sting in the tail: antiseptics and the neuraxis revisited. *Anaesthesia* 67:1305–1320
25. Dogliotti AM (1933) A new method of block anesthesia: segmental peridural spinal anesthesia. *Am J Surg* 20:107–118
26. Doughty A (1978) Epidural analgesia in labour: the past, the present and the future. *J R Soc Med* 71(12):879–884
27. Wantman A, Hancox N, Howell PR (2006) Techniques for identifying the epidural space: a survey of practice amongst anaesthetists in the UK. *Anaesthesia* 61:370–375
28. Cowan CM, Moore EW (2001) A survey of epidural technique and accidental dural puncture rates among obstetric anaesthetists. *Int J Obstet Anesth* 10(1):11–16
29. D'Angelo R, Foss ML, Livesay CH (1997) A comparison of multiport and uniport epidural catheters in laboring patients. *Anesth Analg* 84:1276–1279
30. Collier CB, Gatt SP (1994) Epidural catheters for obstetrics. Terminal holes or lateral eyes? *Reg Anesth* 9:378–385
31. Mhyre JM, Greenfield MVH, Tsen LC, Polley LS (2009) A systematic review of randomized controlled trials that evaluate strategies to avoid epidural vein cannulation during obstetric epidural catheter placement. *Anesth Analg* 108(4):1232–1242
32. Beilin Y, Bernstein HH, Zucker-Pinchoff B (1995) The optimal distance that a multiorifice epidural catheter should be threaded into the epidural space. *Anesth Analg* 81:301–304
33. Carrie LES, Russell R (2000) Fixation of epidural catheters (in: correspondence). *Anaesthesia* 55:1231–1233
34. Eisenach JC, Grice SC, Dewan DM (1987) Epinephrine enhances analgesia produced by epidural bupivacaine during labor. *Anesth Analg* 66:447–451

35. Knudsen K, Beckman Suurkula M, Blomberg S et al (1997) Central nervous and cardiovascular effects of i.v. infusions of ropivacaine, bupivacaine and placebo in volunteers. *Br J Anaesth* 78(5):507–514
36. Santos AC, DeArmas PI (2001) Systemic toxicity of levobupivacaine, bupivacaine, and ropivacaine during continuous intravenous infusion to non pregnant and pregnant ewes. *Anesthesiology* 95:1256–1264
37. Columb MO, Lyons G (1995) Determination of the minimum local analgesic concentrations of epidural bupivacaine and lidocaine in labor. *Anesth Analg* 81:833–837
38. Polley LS, Columb MO, Naughton NN et al (1999) Relative analgesic potencies of ropivacaine and bupivacaine for epidural analgesia in labor. *Anesthesiology* 90:944–950
39. Halpern SH, Walsh V (2003) Epidural ropivacaine versus bupivacaine for labor: a meta-analysis. *Anesth Analg* 96:1473–1479
40. Palm S, Gertzen W, Ledowski T et al (2001) Minimum local analgesic dose of plain ropivacaine vs. ropivacaine combined with sufentanil during epidural analgesia for labour. *Anaesthesia* 56(6):526–529
41. Polley LS, Columb MO, Naughton NN et al (2003) Relative analgesic potencies of levobupivacaine and ropivacaine for epidural analgesia in labor. *Anesthesiology* 99:1354–1358
42. Lyons G, Columb MO, Hawthorne L, Dresner M (1997) Extradural pain relief in labor: bupivacaine sparing by extradural fentanyl is dose dependent. *Br J Anaesth* 78:493–497
43. Robinson AP, Lyons GR, Wilson RC et al (2001) Levobupivacaine for epidural analgesia in labor: the sparing effect of epidural fentanyl. *Anesth Analg* 92:410–414
44. Vertommen JD, Vandermeulen E, Van Aken H et al (1991) The effects of the addition of sufentanil to 0.125% bupivacaine on the quality of analgesia during labor and on the incidence of instrumental deliveries. *Anesthesiology* 74:809–814
45. Brockway MS, Bannister J, McClure JH et al (1991) Comparison of extradural ropivacaine and bupivacaine. *Br J Anaesth* 66:31–37
46. Meister GC, D'Angelo R, Owen M et al (2000) A comparison of epidural analgesia with 0.125% ropivacaine with fentanyl versus 0.125% bupivacaine with fentanyl during labor. *Anesth Analg* 90:632–637
47. Collis RE, Davies DWL, Aveling W (1995) Randomised comparison of combined spinal epidural and standard epidural analgesia in labour. *Lancet* 345:1413–1416
48. Murphy JD, Henderson K, Bowden MI et al (1991) Bupivacaine versus bupivacaine plus fentanyl for epidural analgesia: effect on maternal satisfaction. *BMJ* 302:564–567
49. Comparative Obstetric Mobile Epidural Trial (COMET) Study Group UK (2001) Effect of low-dose mobile versus traditional epidural techniques on mode of delivery: a randomised controlled trial. *Lancet* 358(9275):19–23
50. Hargreaves C (2012) Test doses for epidurals—a modern and practical approach. In: Chan YK, Gatt S, Sia A (eds) *Obstetric anesthesia and analgesia*. Singapore Health Services, Singapore, pp 92–99
51. Blanshard HJ, Cook TM (2004) Use of combined spinal epidural by obstetric anaesthetists. *Anaesthesia* 59:922–923
52. Hartopp R, Hamlyn EL, Stocks G (2010) Ten years' experience with accidental dural puncture and post dural puncture headache in a tertiary obstetric anesthesia department. *Int J Obstet Anesth* 19:118
53. Simmons SW, Taghizadeh N, Dennis AT et al (2012) Combined spinal-epidural versus epidural analgesia in labour. *Cochrane Database Syst Rev* (10):CD003401. doi:[10.1002/14651858.CD003401.pub3](https://doi.org/10.1002/14651858.CD003401.pub3)
54. Cook TM (2000) Combined spinal-epidural techniques. *Anaesthesia* 55:42–64
55. Stocks GM, Hallworth SP, Fernando R (2000) Evaluation of a spinal needle locking device for use with the combined spinal epidural (CSE) technique. *Anaesthesia* 55:1185–1188
56. Holmstrom B, Rawal N, Axelsson K, Nydahl PA (1995) Risk of catheter migration during combined spinal epidural block: percutaneous epiduroscopy study. *Anesth Analg* 80:747–753

57. Palmer CM, Cork RC, Hays R et al (1998) The dose response relation of intrathecal fentanyl for labor analgesia. *Anesthesiology* 88:355–361
58. Herman NL, Calicott R, Van Decar TK et al (1997) Determination of the dose response relationship for intrathecal sufentanil in laboring patients. *Anesth Analg* 84:1256–1261
59. Stocks GM, Hallworth SP, Fernando R et al (2001) Minimum local analgesic dose of intrathecal bupivacaine in labor and the effect of intrathecal fentanyl. *Anesthesiology* 94:593–598
60. Ngan Kee WD, Khaw KS, Ng FF, Lee A (2013) Determination and quantification of the interaction of local anesthetics and lipophilic opioids administered intrathecally for labor analgesia. *Int J Obstet Anesth* 22:S6
61. Camorcica M, Capogna G, Columb MO (2005) Minimum local analgesic doses of ropivacaine, levobupivacaine and bupivacaine for intrathecal labor analgesia. *Anesthesiology* 102:646–650
62. Sia AT, Goy RW, Lim Y, Ocampo CE (2005) A comparison of median effective doses of intrathecal levobupivacaine and ropivacaine for labor analgesia. *Anesthesiology* 102:651–656
63. Van de Velde M, Dreelinck R, Dubois J et al (2007) Determination of the full dose-response relation of intrathecal bupivacaine, levobupivacaine and ropivacaine, combined with sufentanil, for labor analgesia. *Anesthesiology* 106:149–156
64. Lim Y, Ocampo CE, Sia AT (2004) A comparison of duration of analgesia of intrathecal 2.5 mg of bupivacaine, ropivacaine, and levobupivacaine in combined spinal epidural analgesia for patients in labor. *Anesth Analg* 98:235–239
65. Van de Velde M (2009) Combined spinal epidural analgesia for labor and delivery: a balanced view based on experience and literature. *Acta Anaesthesiol Belg* 60:109–122
66. Pan PH, Bogard TD, Owen MD (2004) Incidence and characteristics of failures in obstetric neuraxial analgesia and anesthesia: a retrospective analysis of 19,259 deliveries. *Int J Obstet Anesth* 13:227–233
67. Reynolds F (2001) Damage to the conus medullaris following spinal anaesthesia. *Anaesthesia* 56:238–247
68. Van de Velde M, Schepers R, Berends N et al (2008) Ten years of experience with accidental dural puncture and post-dural puncture headache in a tertiary obstetric anesthesia department. *Int J Obstet Anesth* 17:329–335
69. Mardirosoff C, Dumont L, Boulvain M, Tramer MR (2002) Fetal bradycardia due to intrathecal opioids for labour analgesia: a systematic review. *Br J Obstet Gynaecol* 109:274–281
70. Van de Velde M, Teunkens A, Hanssens M et al (2004) Intrathecal sufentanil and fetal heart rate abnormalities: a double blind, double placebo controlled trial comparing two forms of combined spinal epidural analgesia with epidural analgesia in labor. *Anesth Analg* 98:1153–1159
71. Clarke VT, Smiley RM, Finster M (1994) Uterine hyper-activity after intrathecal injection of fentanyl for analgesia during labor: a cause of fetal bradycardia? *Anesthesiology* 81:1083

Giorgio Capogna

8.1 Introduction

In the late 1970s by starting to use a low concentration of local anesthetic solution (bupivacaine 0.125 %) in a rather large volume (10 mL), most of the potential objections to the use of epidural analgesia in childbirth were overcome [1]. In fact, with this relatively low-dose, low-concentration solution, epidural analgesia was able to provide satisfactory analgesia with minimal motor block, without leading to a prolonged expulsion time, and with the maternal and neonatal plasma concentration of local anesthetic well below the toxic level. A few years later it was demonstrated that with the addition of one opioid to the above solution, the duration of analgesia was prolonged and the quality of analgesia was improved [2].

The ideal analgesic method was brought within reach, on the one hand providing effective maternal analgesia during labor and delivery and on the other hand avoiding negative effects on the mother and the fetus of interfering with the progress of labor.

Since then, the use of a low concentration of local anesthetic solution plus one opioid has become the standard routine analgesic solution worldwide. Nowadays much lower concentrations of local anesthetic solution such as bupivacaine or levobupivacaine 0.0625 % or ropivacaine 0.1 % plus a small dose of fentanyl or sufentanil are very common practice everywhere. An additional innovation was the introduction of the combined spinal epidural analgesia technique, which led to a new, combined form of administration of analgesic solutions during labor [3].

With the reduction of the dose and of the concentration of the local anesthetic solution and with the addition of a small amount of one opioid, either epidural or spinal administration (by CSE) of such analgesic mixtures may now provide a safe, reliable, and effective labor analgesia, without affecting maternal ambulation

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and without interfering with the labor outcome. Consequently, the main and most frequent question is not how to initiate labor analgesia but how to maintain it for hours throughout labor, since pain relief provided by any single-shot injection may last no more than 2–3 h with any analgesic mixture administered intrathecally or through epidural loading [4].

The ideal maintenance techniques should provide continuous, uninterrupted, and safe analgesia, allowing maternal ambulation and preserving the expulsive forces during the expulsive period. It should also reduce the incidence of breakthrough pain which requires additional pain relief and so subsequently also diminishing the physician's workload and the total local anesthetic dose [5].

Basically, analgesia can be maintained using two different kinds of methods: the first provides relief at irregular intervals when requested by the patient upon the resumption of pain and includes modalities such as manual top-ups and Patient-Controlled Epidural Analgesia (PCEA). The second prevents pain recurrence providing analgesia before its return which includes Continuous Epidural Infusion (CEI), PCEA with basal infusion, Automated Intermittent Mandatory Boluses (AMB), Programmed Intermittent Epidural Boluses (PIEB), and Computer-integrated PCEA (CIPCEA) and its associated settings.

8.2 Intermittent Epidural Bolus (Top-Up)

The intensity and the site of pain vary considerably not only between parturients but also during the course of labor in the same individual.

In theory, the intermittent administration method (top-up) permits the anesthesiologist to titrate the local analgesic solution and to vary its dose, volume, and concentration depending on the progression of labor and the severity of the pain. The question remains when will it be the time to refill the dose.

Once good analgesia has been established, we do not know in advance when the maintenance doses will be needed. According to clinical experience, just to make an example, in nulliparous women, during the first stage of labor, analgesia may last up to 3 h after a 20 mL bolus of levobupivacaine 0.0625 % with 10 µg of sufentanil. However, these statistical data are of little help to the clinician caring for the individual parturient.

The top-up technique may be given in two ways. In the first one, the top-up is given before the pain returns, for example, at fixed time intervals, and in the other, it is given upon the parturient's request. When using the first method, the return of pain can be anticipated and analgesia adapted to the progress of labor with the standard dose previously chosen. There are a number of reasons for giving a top-up to a pain-free parturient. In more prolonged labors, we can learn how long a dose works in one particular individual or in a particular type of labor (nulliparous, multiparous, dystocic, induced, augmented, and so on). In a busy maternity ward, it seems preferable to give the top-up dose in advance when the anesthesiologist is free, rather than to risk having to attend three or more parturients at the same time. In addition, with low doses it is better to inject too early rather than too late, because

it is better to prevent pain rather than to follow it. However, in practice this solution is not very often feasible and, in all cases, the physician's workload is increased.

The other most popular approach is to administer the top-up dose when the pain returns: this is the typical "peak and valley" method. There are strong arguments against this technique: a pain-free labor is preferable to some pain-free intervals; the mother may lose confidence in the anesthesiologist's ability to control her pain and it may take considerable time and effort to reestablish good analgesia once it is lost. In fact with this method of administration, the timing of the injections usually depends on the parturient's need for pain relief, but if the injections are only administered when the parturient complains of pain, the analgesia will only occur at intervals between periods of suffering and second, intermittent dosing calls for many interventions on the part of the providers. In addition, administering the top-up dose only when the pain returns may expose the mother to long periods of waiting for pain relief due to the variable period of time between the parturient's request to the midwife and the midwife's request for the anesthesiologist's intervention.

Surely one of the major advantages of using the intermittent technique is the less local anesthetic consumption and the less frequent occurrence of motor block when compared with continuous infusion [6].

8.3 Continuous Epidural Infusion

Due to the intermittent nature of pain relief provided by manual top-ups and its need for frequent interventions, many maternity units have moved to a long-standing analgesia maintenance regimen, the continuous epidural infusion of analgesics into the epidural space, which is one of the current and most frequently used techniques providing the parturient with a much smoother analgesic experience. The advantages of continuous infusion include the fact that once the effective analgesia is established, true continuous pain relief is maintained, thereby avoiding block regression and a painful maternal waiting period prior to reinjection, which commonly occurs in busy wards when the intermittent injection method is used. The initial claimed advantages of continuous infusion over the intermittent administration such as more stable cardiovascular response, less motor block, and less risk of toxic reactions are nowadays no longer an issue due to the much lower concentrations of local anesthetic solution used in clinical practice as compared to that used approximately 30 years ago, when the continuous infusion technique started to be routinely used.

However, with continuous infusion, anesthesiologists may be tempted to place the parturient on "automatic pilot," and they may be less likely to individualize the parturient's personal needs during labor. By definition, by eliminating the need for individualized top-up injections some parturients will receive more local anesthetic than they need.

Although the infusion can be modified to individualize analgesia, unilateral analgesia and breakthrough pain also frequently occur with continuous infusions

and the administration of supplementary analgesia rescue analgesic doses may still be frequently required [7].

In addition, even with analgesic solutions with a very low concentration of local anesthetic, motor block is observed in a relatively high percentage of patients [8].

For these reasons, there has been a transition to other maintenance techniques of labor analgesia which are considered to be more effective.

8.4 Patient-Controlled Epidural Analgesia

PCEA is a reliable and effective method of maintaining labor analgesia, provided that sufficient drug volumes are allowed, and a wide variety of drug combinations and settings have been used successfully [9].

This method permits the parturients to self-administer intermittent boluses of epidural solution depending on their individual level of pain which varies as the labor progresses or when labor augmentation regimens are started [10]. Every patient has thus autonomy, titrability, and a flexible analgesic regimen and drugs are delivered safely and effectively [11]. Compared to continuous infusion, there is also a lower consumption of local anesthetic with no reduction in the efficacy of the analgesia, less motor block of the lower extremities, and less frequent unscheduled clinician interventions, so reducing the workload [5, 9, 10].

Unfortunately the tendency to unilateral block during analgesia still persists significantly with PCEA [12].

Much debate has centered around the optimal settings for the PCEA lock-out interval and the bolus volume with the most recent studies deciding that there is no ideal bolus dose or lock-out interval setting for labor PCEA and no hard evidence to recommend one regimen more than another. The principle of all PCEA techniques depends on the response of the parturient when she feels the need for analgesia. This means that she must, however, experience short periods of persistent pain which may or may not be blocked depending on the PCEA settings. Maternal cultural factors, psychological characteristics, previous training, and expectations will also have a bearing on the efficacy of this technique [13].

In addition, there may be some patients who may not wish to control their analgesia because of fatigue and some may prefer to “leave it to the doctor.”

8.5 PCEA Plus Basal Infusion

In theory, PCEA plus basal infusion should result in better analgesia and greater patient satisfaction and comfort as pain is prevented rather than followed.

However, there is no clear evidence that by adding a basal infusion to PCEA, apart from a lower consumption of local anesthetic without the basal infusion, there might be some differences in outcomes such as motor block, maternal analgesia, and maternal satisfaction [9, 14].

However, decreased incidence of breakthrough pain has been described and this may determine a consequent possible reduction in the physician's workload and be some of the major benefits of using a basal infusion in a PCEA regimen, particularly in a busy labor ward where the anesthetist may not always be on hand to provide an epidural rescue bolus immediately when requested [9]. Unfortunately, what the ideal rate of this basal or background infusion should be is still ongoing [5].

8.6 PCEA Plus Automated Intermittent Bolus

A variant of the PCEA plus basal infusion is the technique of PCEA associated with an intermittent automated bolus instead of a continuous background infusion.

These automated intermittent mandatory boluses (AMB) or automated continual intermittent boluses (CIB) describe automated systems regulated to administer a bolus at programmable intervals during labor analgesia provided by a PCEA.

Many studies have reported that these systems are more effective than continuous epidural infusion in reducing labor pain. Decreasing pain scores and overall consumption of local anesthetic plus greater maternal satisfaction occur when automated intermittent mandatory boluses (AMB) are used as opposed to continual basal infusion (CBI). This is also true when AMB is linked to a PCEA regimen, showing that there is evidence of a reduction in the parturient's need for self-boluses and a longer time interval to the first PCEA demand in comparison to a PCEA and CBI regimen. The question mark still hangs over whether PCEA with AMB helps to reduce the incidence of breakthrough pain [5, 10, 15–17].

8.7 Programmed Intermittent Epidural Boluses with PCEA

An interesting alternative technique to maintain labor epidural analgesia is the programmed intermittent epidural bolus (PIEB) technique. With this technique, a fixed preprogrammed epidural bolus is given at a fixed period of time to obtain a continuous and constant analgesia. In case of breakthrough pain analgesia may be completed by using PCEA or by manual physicians' boluses. With this technique a reduction in the total amount of local anesthetic, fewer additional PCEA or manual boluses, and greater maternal satisfaction have been observed when compared to continuous epidural infusion either in multiparous or in nulliparous women [18, 19].

The optimum settings for the bolus volume and time interval in the maintenance of epidural labor analgesia were studied in nulliparous women in spontaneous labor [20] by comparing three different infusion regimens: 2.5 mL every 15 min (2.5/15), 5 mL every 30 min (5/30), and 10 mL every 60 min (10/60). The results showed that the 10 mL every 60 min regimen was the most advantageous, with a decreased local anesthetic consumption with no diminution of patient analgesia.

PIEB may also be performed by using two pumps, one set to deliver the PIB bolus, for example, 10 mL of levobupivacaine 0.0625 % with sufentanil 0.5 µg/mL, to maintain epidural analgesia, and another set to deliver a patient-controlled analgesia (PCEA) for breakthrough pain using a more concentrated solution, such as levobupivacaine 0.125 %.

With this double pump–double concentration regime, when compared to an equipotent solution given by continuous infusion, pain scores and duration of labor analgesia are the same for both PCEA and CEI, but there is less total local anesthetic consumption, less patients needing additional PCEA boluses, and a lower mean number of PCEA boluses per patient in the PIEB group. The most important difference, however, is that epidural analgesia is maintained with a lower incidence of motor block and instrumental vaginal delivery with PIEB. It is interesting to note that only instrumental vaginal delivery patients require anesthesiologist administered manual boluses [19].

There is always an increase in the level of pain as the labor progresses and multiple obstetric and obstetric management factors can alter its intensity. To confirm that, a high rate of breakthrough pain needing clinician intervention is reported in PIB studies [18, 20] where the same concentration of local anesthetic was used in both the PCEA bolus and PIEB. Hence a way to arrive at an excellent level of analgesia, while significantly reducing supplemental manual rescue boluses, might be achieved by using the double pump/double concentration method [19].

Continuous epidural infusion, PCEA with and without a background infusion, and programmed intermittent epidural bolus (PIB) regimens with or without supplemental PCEA are all sustained by the current epidural pump technology, but this technology cannot support any of the above procedures using two different concentrations of local anesthetic with the same pump. Will a different analgesic mixture for the rescue bolus diminish manual interventions and increase parturient satisfaction? Only further studies will be able to answer this question.

8.8 Mechanism of Action of Intermittent Administration

A systematic review [21] confirmed that in comparison to CEI, by using a preprogrammed intermittent bolus technique, there is a reduced local anesthetic consumption, a shorter second stage of labor, and higher maternal satisfaction.

There are many theories have been put forward to explain this finding.

It has been demonstrated that when intermittent boluses are used as opposed to continuous infusion, the spread of the infusate from a multi-orifice catheter is better, resulting in a wider and more uniform spread of contrast medium, while the continuous infusion results in a smaller spread that is exclusively through the proximal port of the epidural catheter [17, 22]. Cadaveric and experimental models [23, 24] put forward the theory that a more uniform spread of the solution in the epidural space could be achieved as a result of the higher injectate pressure generated during a bolus injection. These *in vitro* observations found that the spread of liquids

Fig. 8.1 Pressure generated by the pump set in Programmed Intermittent Bolus (PIEB) mode

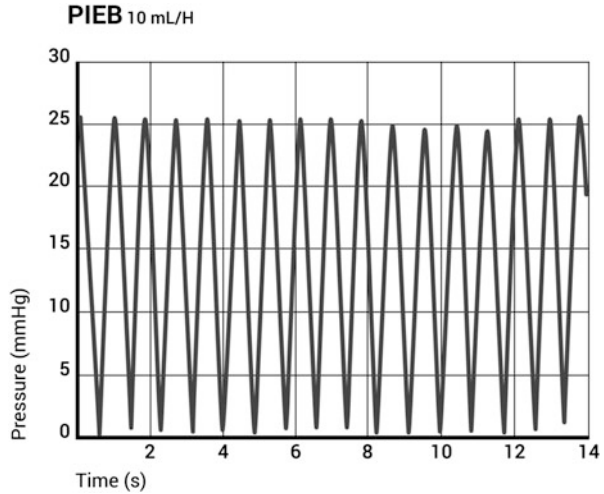
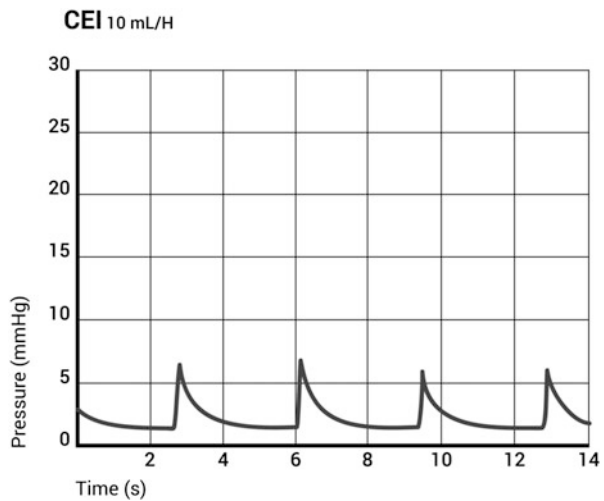


Fig. 8.2 Pressure generated by the pump set in Continuous Epidural Infusion (CEI) mode



in the epidural space is highly nonuniform in multiple small channels and suggest that the spread would be more uniform in large volumes and correspondingly high injectate pressure near the site of injection would engage the most channels.

When there is a combination of AMB and CSE, it has been hypothesized that the intrathecal space may directly receive the local anesthetic solution through the dural rent, owing to the high driving pressure created when the bolus is administered, so leading to a more efficacious analgesia [16].

In vitro (Figs. 8.1 and 8.2) there is a higher injectate pressure when boluses are administered by the pump when compared to the pressures delivered by continuous

Table 8.1 In vivo evaluation of pressure generated by Programmed Intermittent Bolus (PIEB) or Continuous Epidural Infusion (CEI)

| | Baseline (mmHg) | P_{\max} during bolus/ infusion (mmHg) | P_{\min} during bolus/ infusion (mmHg) |
|------------------|-----------------|---------------------------------------------|---------------------------------------------|
| PIEB 10 mL bolus | 18.32 (3.1) | 311.12 (22.8) | 208.66 (27.6) |
| CEI 10 mL/h | 18.45 (2.4) | 33.28 (3.7) | 22.21 (3.3) |

Values are given as mean (SD)

infusion and this in vitro evidence could explain the reason for this greater diffusion of the anesthetic solution in the epidural space [25].

This hypothesis has been confirmed by in vivo (Table 8.1) measurements of the epidural pressures in pregnant volunteers which clearly demonstrate the greater pressures generated in the epidural space by the PIEB technique when compared to the continuous infusion [24].

The way of delivering analgesia by single bolus or by continuous infusion may subsequently influence the dynamics of nerve block.

Given that there is less motor block and fewer instrumental deliveries after PIEB when compared to CEI, it might be hypothesized that creating or lessening motor block may be a direct result of the mode of delivery of the epidural solution. As stated in pharmacodynamics, the movement of local anesthetic into the nerve according to diffusion gradients can determine the production and reversal of analgesia and motor block [26].

Analgesia and motor block are produced by the movement of local anesthetic from the extraneural space into the nerve along a diffusion gradient (Fig. 8.3). After a single bolus administration, initially the concentration is greater outside of the nerve fiber, but over time, the extraneural concentration equals the intraneural one, establishing a steady state. Nerve blockade is eventually overcome when the intraneural concentration exceeds the extraneural concentration and the diffusion gradient is reversed. If a local anesthetic at very low concentration is used in intermittent boluses, the amount of local anesthetic inside the nerve fiber is sufficient to block the sensory fibers, which are small and with a short internodal distance, but blockade of motor fibers, which are greater and with long internodal distance, is unlikely, as the total amount of local anesthetic inside the nerve is insufficient to block them. In the case of continuous infusion, the extraneural concentration of local anesthetic is generally constantly higher than in the intraneural space, and the total concentration inside the nerve is therefore increased with the time and may reach the threshold for motor fiber block even if we are using a local anesthetic solution at very low concentration.

This may explain the frequent occurrence and intensification of motor block during prolonged continuous infusions like those used for labor analgesia.

This also may explain the less frequent occurrence of motor block during labor analgesia with PIEB and the preliminary observation that when PIEB is used for a more prolonged period of time, such as in post-cesarean section pain relief, it has the potential to decrease motor block, maintaining adequate analgesia [27].

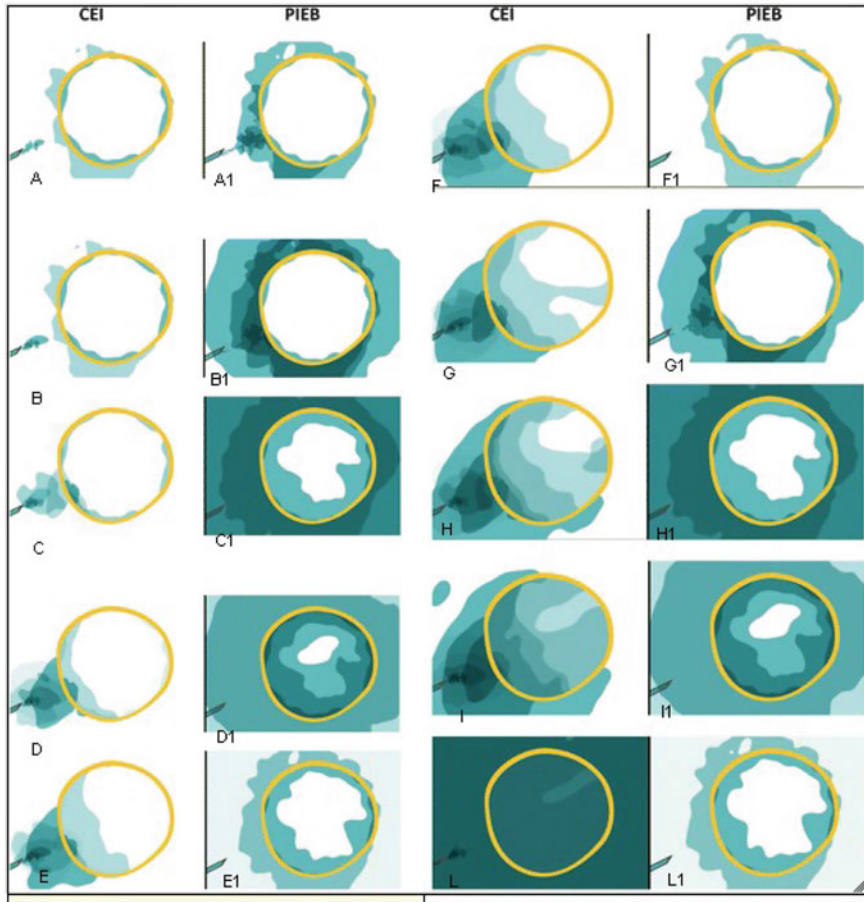


Fig. 8.3 This sequence represents the diffusion of the movement of local anesthetic into the nerve according to diffusion gradients with **CEI** and **PIEB**. After the initial loading dose, not represented in the figure, shortly after injection the local anesthetic solution reaches the nerve's mantle region first and eventually the core, blocking almost all fibers and producing anesthesia or analgesia, depending on the concentration of the local anesthetic injected. Continuous infusion (CEI) or a PIEB is usually started after an effective local anesthetic loading dose, but before the complete regression of analgesia (A). After a single bolus administration such as with PIEB, initially the concentration is greater outside of the nerve fiber (A1-D1), but over time, the extraneural concentration equals the intraneural one, establishing a steady state (E1). Nerve blockade is eventually overcome when the intraneural concentration exceeds the extraneural concentration and the diffusion gradient is reversed (F1). This cycle is repeated at regular predetermined intervals (usually every hour) (F1-L1). Therefore, if a local anesthetic at very low concentration is used in intermittent boluses, the amount of local anesthetic inside the nerve fiber is only sufficient to block the sensory fibers, but is not enough to block the motor fibers. In the case of continuous infusion, it takes a longer period of time to reach the steady state (A-G) and the diffusion of the local anesthetic solution is less uniform (D-H), but after a few hours the extraneural concentration of local anesthetic is generally constantly higher than that in the intraneural space because it is constantly refilled, and so the total concentration inside the nerve

8.9 Computer-Integrated Patient-Controlled Epidural Analgesia

A conventional PCEA pump permits the parturients, through self-administered boluses, to titrate their epidural analgesia, but it cannot provide a varying basal rate without the intervention of a clinician. Although basal infusion is not called on in early labor, it may become more important with the inevitable increase of pain as labor progresses or with the initiation of labor augmentation regimens. If a PCEA pump could allow for a variable basal infusion rate and could be responsive to the demands of the patient, the parturient may be able to experience more effective analgesia.

Devices to adjust background infusion rates according to the frequency of earlier demands have been created by converting an ordinary infusion pump into a computer-integrated (CI-PCEA) pump by devising a program based on a new clinical algorithm [28].

Depending on the number of PCEA patient requests over the last hour, this interactive pump can automatically adjust the basal infusion rate, continually recording the patient's analgesic requirements and modifying the basal infusion rate depending on whether the parturient needed one, two, or three demand boluses, respectively, in the previous hour. Should there be no demands in this hour, the pump lowers the basal infusion rate by decrements of 5 mL/h [28–30]. It has long been believed that as labor progresses there is a greater need for epidural analgesia. The CI-PCEA can meet this requirement by matching the basal infusion rate to the patient's analgesic needs. By matching the basal infusion rate to the patient's analgesic needs, the CIPCEA can meet the growing analgesic requirements of labor.

8.10 New Maintenance Techniques and Maternal Satisfaction

Maternal satisfaction is one of the frequent secondary outcomes indicated in the results of many studies which describe the new techniques of labor analgesia maintenance [5, 9, 10, 18].

On average, almost always, 80 % of women are reported to be satisfied, more with PCEA or automated bolus techniques than with CEI, but because unidimensional scales are used to measure satisfaction it is hard to arrive at particular conclusions.

Fig. 8.3 (continued) therefore increases over time and may reach the threshold for motor fiber block even if a very low concentration local anesthetic solution has been used (H-L). This difference in diffusion is even more evident as time passes. This may explain the frequent occurrence of unilateral analgesia and the frequent intensification of motor block during prolonged continuous infusions like those used for labor analgesia. Courtesy of Giorgio Capogna (2014)

However, when a more appropriate tool to investigate satisfaction is used more detailed information is obtained.

In one study the differences in maternal satisfaction between labor analgesia provided by PIEB or CEI in nulliparous women were evaluated by using a multi-dimensional questionnaire and then semistructured interviews. With CEI the women experienced more motor block, numbness, and feeling of loss of control and subsequent negative feelings because of their reduced ambulatory ability and capacity to cope with labor and delivery. They indicated less satisfaction with CEI when compared with PIEB. Actually there were consistently lower scores at the postdelivery overall satisfaction evaluation in the women who had been randomized to CEI [31].

8.11 Conclusion

Labor pain is dynamic and intensely personal and with this understanding clinicians must “tailor” the analgesic regimen for each of their parturients so that the birthing experience can be pleasant and memorable. Early labor may not necessitate basal infusion, but with the intensification of pain as the labor proceeds or labor augmentation to change the analgesic regimen may become more important.

Medical technology has now provided us with more advanced drug delivery systems [32] that have promising, but to be confirmed, potential to fulfill maternal requirements of a safe, natural, and painless childbirth.

References

1. Bleyaert A, Soetens M, Vaes L et al (1979) Bupivacaine, 0.125 per cent, in obstetric epidural analgesia: experience in three thousand cases. *Anesthesiology* 51:435–438
2. Desprats R, Mandry J, Grandjean H et al (1983) Peridural analgesia during labor: comparative study of fentanyl-marcaïne combination and marcaïne alone. *J Gynecol Obstet Biol Reprod* 12:901–905
3. Collis RE, Baxandall ML, Srikantharajah ID et al (1994) Combined spinal epidural (CSE) analgesia: technique, management, and outcome of 300 mothers. *Int J Obstet Anesth* 3:75–81
4. Heesen M, de Velde V, Klohr S et al (2014) Meta-analysis of the success of the block following combined spinal-epidural vs epidural analgesia during labour. *Anaesthesia* 69: 64–71
5. Loubert C, Hinova A, Fernando R (2011) Update on modern neuraxial analgesia in labour: a review of the literature of the last 5 years. *Anaesthesia* 66:191–212
6. Boutros A, Blary S, Bronchard R et al (1999) Comparison of intermittent epidural bolus, continuous epidural infusion and patient controlled-epidural analgesia during labor. *Int J Obstet Anesth* 8:236–241
7. Hess PE, Pratt SD, Lucas TP et al (2001) More [predictors of breakthrough pain during labor epidural analgesia](#). *Anesth Analg* 93:4141–4418
8. Beilin Y, Guin NR, Bernstein HH et al (2007) Local anesthetics and mode of delivery: bupivacaine versus ropivacaine versus levobupivacaine. *Anesth Analg* 105:756–763
9. Halpern SH, Carvahlo B (2009) Patient-controlled epidural analgesia for labor. *Anesth Analg* 108:921–928

10. Leo S, Sia ATH (2008) Maintaining labour epidural analgesia: what is the best option? *Curr Opin Anaesthesiol* 21:263–269
11. Van der Vyver M, Halpern S, Joseph G (2002) Patient-controlled epidural analgesia versus continuous infusion for labour analgesia: a meta-analysis. *Br J Anaesth* 89:459–465
12. Cappiello E, O'Rourke N, Segal S et al (2008) A randomized trial of dural puncture epidural technique compared with the standard epidural technique for labor analgesia. *Anesth Analg* 107:1646–1651
13. Chumbely GM, Hall GM, Salmon P (1988) Patient-controlled analgesia: an assessment by 200 patients. *Anaesthesia* 53:216–221
14. Okutomi T, Saito M, Mochizuki J et al (2009) A double-blind randomized controlled trial of patient controlled epidural analgesia with or without a background infusion following initial spinal analgesia for labor pain. *Int J Obstet Anesth* 18:28–32
15. Leo S, Ocampo CE, Lim Y et al (2010) A randomized comparison of automated intermittent mandatory boluses with a basal infusion in combination with patient-controlled epidural analgesia for labor and delivery. *Int J Obstet* 19:357–364
16. Chua SM, Sia AT (2004) Automated intermittent epidural boluses improve analgesia induced by intrathecal fentanyl during labour. *Can J Anaesth* 51:581–585
17. Fettes PDW, Moore CS, Whiteside JB et al (2006) Intermittent vs continuous administration of epidural ropivacaine with fentanyl for analgesia during labour. *Br J Anaesth* 97:359–364
18. Wong C, Ratliff JT, Sullivan JT et al (2006) A randomized comparison of programmed intermittent epidural bolus with continuous epidural infusion for labor analgesia. *Anesth Analg* 102:904–909
19. Capogna G, Camorcia M, Stirparo S et al (2011) Programmed intermittent epidural bolus versus continuous epidural infusion for labor analgesia: the effects on maternal motor function and labor outcome. A randomized double-blind study in nulliparous women. *Anesth Analg* 113:826–831
20. Wong CA, McCarthy RJ, Hewlett B (2011) The effect of manipulation of the programmed intermittent bolus time interval and injection volume on total drug use for labor epidural analgesia: a randomized controlled trial. *Anesth Analg* 112:904–911
21. George RB, Allen TK, Habib AS (2013) Intermittent epidural bolus compared with continuous epidural infusions for labor analgesia: a systematic review and meta-analysis. *Anesth Analg* 116:133–144
22. Kaynar AM, Shankar KB (1999) Epidural infusion: continuous or bolus? *Anesth Analg* 89:534
23. Hogan Q (2002) Distribution of solution in the epidural space: examination by cryomicrotome section. *Reg Anesth Pain Med* 27:150–166
24. Gibiino G, Distefano R, Camorcia M et al (2014) Maternal epidural pressure changes after programmed intermittent epidural bolus (PIEB) versus continuous epidural infusion (CEI). *Eur J Anaesth* 31:11AP35
25. Stirparo S, Fortini S, Espa S et al (2013) An in vitro evaluation of pressure generated by programmed intermittent epidural bolus (PIEB) or continuous epidural infusion (CEI). *Open J Anesthesiol* 3:214–217
26. De Jong RH (1994) Dynamics of nerve block. In: De Jong RH (ed) *Local anesthetics*. Mosby, St Louis, pp 230–245
27. Stirparo S, Laudani A, Haiberger R et al (2011) Postoperative analgesia after cesarean section: a comparison between programmed intermittent epidural bolus (PIEB) versus continuous epidural infusion (CEI). *Reg Anesth Pain* 36:E1–E27
28. Sia AT, Lim Y, Cecilia O (2006) Computer-integrated patient-controlled epidural analgesia: a preliminary study on a novel approach of providing pain relief in labour. *Singapore Med J* 47:951–956
29. Lim Y, Sia AT, Cecilia O (2006) Comparison of computer integrated patient controlled epidural analgesia vs conventional patient controlled epidural analgesia for pain relief in labour. *Anaesthesia* 61:339–344

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30. Sng BL, Alex S, Lim Y et al (2009) Comparison of computer integrated patient controlled epidural analgesia and patient controlled epidural analgesia with basal infusion for labor and delivery. *Anaesth Intensive Care* 37:46–53
 31. Stirparo S, Camorcia M, Capogna G (2010) Maternal satisfaction with different techniques of epidural analgesia. *Eur J Anaesth* 27:164
 32. Capogna G, Stirparo S (2013) Techniques for the maintenance of labor analgesia. *Curr Opin Anaesthesiol* 26:261–267

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9.1 Second Stage of Labor

9.1.1 Definition and Duration

The second stage of labor is generally defined as the time elapsing from the full cervical dilation to the delivery of the fetus [1]. There is, therefore, a wide variability in its accuracy as it depends on the operator's decision to perform a vaginal examination to determine whether the "diagnosis" of complete dilatation is made.

The second stage of labor may be divided into a passive phase (latent phase), where the fetal head progressively descends through the maternal pelvis and internal rotation and flexion occur in the absence of involuntary expulsive contractions, and an active phase (perineal phase) that begins when the fetal head is visible and there is the urge to push with consequent involuntary and voluntary maternal expulsive efforts resulting in the delivery of the baby [2].

The correct identification of these two different phases is essential as it is not advisable to encourage the woman to start pushing until descent and internal rotation have occurred.

The mean duration of the second stage is highly variable and there is no good evidence regarding the absolute limits of this stage. In fact, the impact of the duration of the second stage of labor on the obstetric and fetal perinatal outcomes is very controversial. While a systematic review found that a prolonged second stage of labor is associated only with an increased incidence of operative delivery with no effects on adverse neonatal outcomes [3], a successive cohort study found that an increased duration in the second stage is associated with an increase of both

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maternal and fetal adverse outcomes and this is particularly evident for duration longer than 3 h in nulliparous women and longer than 2 h in multiparous women [4].

However, even though the picture is not completely defined, both the American College of Obstetricians and Gynecologists and the NICE guidelines suggest that birth is expected to occur within 2 h from the start of the active second stage in nulliparous patients without regional analgesia and 3 h with regional analgesia and within 1 h in multiparous patients without regional analgesia and 2 h with regional analgesia [5, 6].

9.1.2 Position for Labor and Birth

There are many positions that can be used by women during the second stage and the expulsive phase that are usually categorized as either neutral (supine) or upright [7].

The supine positions include the lithotomy position, the lateral (Sim's) position, the semi-recumbent position, and the Trendelenburg's position, while the upright positions include the sitting (using obstetric chair or stool), the kneeling, and the squatting positions.

Traditionally, and still in many institutions, women deliver in a supine, semi-recumbent, or lithotomy position with the woman's legs fixed in stirrups. This is merely due to the fact that these positions are easier for care providers as they enable easier access by the midwife and obstetrician to the woman's abdomen to monitor the fetal heart rate and visit the parturient.

The Nice guidelines [5], however, recommend discouraging women from lying supine or semi-supine in the second stage of labor and encourage women to adopt any other position that they find comfortable since the adoption of the supine position in the second stage is associated with longer labors, increased vaginal instrumental birth, increased pain, and a higher incidence of FHR abnormalities [8].

The supine position, in fact, causes aorto-caval compression that may cause a significant reduction in the utero-placental perfusion with a consequent detrimental effect to the fetus especially if adopted for a significant period of time and during the expulsive period [9].

The Cochrane collaboration supports the current NICE guidance of positions for vaginal birth encouraging women with epidural analgesia to use the position that they feel more comfortable with during the second stage of labor [10].

9.1.3 Pushing Modality and Timing

The maternal urge to push is generally felt when there is direct contact of the baby to the pelvic floor. Stretch receptors in the wall of the vagina, rectum, and ultimately the perineum communicate the pressure of the fetus descending in the birth canal that, along with an increased abdominal pressure, causes the overwhelming urge to push generally described by women. This usually becomes evident during the

transition from the latent phase of the second stage to the active phase, when the urge to push becomes compulsive and involuntary.

During the active phase of the second stage of labor, contractions become less frequent but stronger and of longer duration, thanks to a number of anatomical, biochemical, endocrine, and humoral factors. One important role is played by endogenous oxytocin that is crucial not only in contributing to the onset of labor, but also in the maintenance of an active labor and delivery [11].

The urge to push felt by women in the active phase of the second stage is caused by an enhanced oxytocin release secondary to the distension of the pelvic floor caused by the fetal presenting part, also called the Ferguson reflex. This reflex involves neural input from the ascending spinal tract, and especially from sacral sensory input to the midbrain, thereby resulting in enhanced oxytocin release. Although spontaneous labor and delivery may occur in women with spinal cord injury which disrupts this tract [12], an interference with the Ferguson reflex has long been recognized as a potential adverse effect of regional anesthesia [13].

In the past, in fact, it was believed that regional anesthesia might inhibit this reflex, therefore prolonging labor, especially the second stage. However, clear evidence for this does not exist. In fact, while some studies have noted a reduction in plasma oxytocin concentrations with epidural local anesthetics [14] or intrathecal opioid analgesia [15], other studies have failed to note such a reduction [16, 17].

However, these studies involved high doses and concentrations of local anesthetic solutions that are no longer used in our current clinical practice. The motor block of the pelvic floor muscles caused by these old regimens, the reduction or absence of the perception of the contractions, and the urge to push might be responsible for the difficulty in bearing down during the second stage with a resulting increase in the duration of this phase and, consequently, an increase in the incidence of instrumental deliveries.

What happens with the low-dose, low concentration local anesthetic plus opioid mixtures currently used for labor analgesia is the maintenance of all labor sensations, including the sensation of bearing down and the ability to effectively push. The only difference in women under epidural analgesia is the ability to resist to the urge to push which may be advisable in some situations, for instance, when the mother perceives the need to push before the vertex is visible.

In women without epidural analgesia, in fact, the Ferguson reflex may initially be controlled, but it becomes increasingly compulsive, overwhelming, and involuntary during each contraction.

There are two completely different approaches to pushing: the spontaneous pushing and the coached (or directed) pushing. The spontaneous pushing follows the physiological body's natural urge to bear down, allowing the mother to push only when she feels ready, and in whatever way she feels more comfortable [18–20].

In the coached pushing, also called the Valsalva technique, women are directed on when to start pushing and how to push, and it consists in instructing the woman to take a deep breath at the beginning of a contraction, hold her breath, and push with the closed glottis as hard as she can. These efforts are thought to facilitate or

hasten the process of fetal descent, therefore minimizing the length of the second stage of labor [21] and therefore improving fetal outcome.

However, the Valsalva maneuver only causes exactly what it is thought to avoid. In fact, each time the woman performs a pushing effort, there is a significant decrease in maternal cardiac output with a consequent reduction in the fetal blood supply and oxygenation. While the transient, harmless, and physiological interruption in the fetoplacental perfusion observed during a normal contraction is physiologically well tolerated by a healthy fetus, the Valsalva technique may cause a significant, although transient, decrease in fetal oxygenation due to a prolonged reduction in the fetal placental perfusion.

In addition, the Valsalva maneuver is also associated with damage to urinary, pelvic, and perineal structures and determines only a slight shortening in the duration of second-stage labor but not a reduction of prolonged second-stage labors [22]. For all these reasons, the Valsalva pushing is not routinely indicated in current clinical practice, and in the absence of specific indications, it should be avoided and women should, therefore, be supported in using spontaneous pushing and encouraged to choose their own method of pushing [23].

Coached pushing should be utilized only in instances when the benefits are judged to outweigh the risks such as situations where expeditious birth is indicated and the woman is not yet pushing spontaneously. Unfortunately, despite it now being well established and widely accepted that the Valsalva maneuver has adverse maternal and fetal consequences, it is still widespread in modern obstetric practice.

9.1.4 Initiation of Active Pushing

Maternal expulsive efforts may commence early in the second stage (early pushing), that is, before parturients feel the urge to push or before the fetal head is visible at the perineum provided that a full cervical dilation is obtained, or they can be delayed, that is, a woman who is fully dilated, but without the urge to push, is allowed to rest and await the urge to push before actively bearing down with contractions.

Several studies and a recent meta-analysis have demonstrated that, in women with epidural analgesia, delayed pushing is associated with an increase in the likelihood of spontaneous vaginal delivery along with an overall increase in the duration of the passive second stage of labor [24–27] but with no effects on the duration of the pushing phase. In addition, delayed pushing has been associated with less maternal fatigue for nulliparous women [27].

Early pushing is conversely often associated with maternal exhaustion and may result in more frequent fetal heart rate decelerations and fetal oxygen desaturation [27, 28].

It is now, therefore, widely recommended [29–31] that women should be encouraged to push only when full cervical dilation, the fetal condition, and

engagement of the presenting part have been confirmed, and the woman feels an urge to bear down and only during uterine contractions [19]. It has also been suggested that women should not be asked to push from a high fetal station or before full cervical dilation even in the presence of the urge to bear down.

However, there are, anyway, time limits to respect regarding the duration of the pushing phase.

Pushing can be delayed up to 2 h for primiparous and up to 1 h for multiparous women with epidural analgesia [27, 28] as after this time there is an increased risk of birth asphyxia and maternal infection [32].

9.1.5 Analgesia for the Second Stage of Labor: Pain Pathways

Near the end of the first stage of labor, once the cervix is becoming fully dilated, the nociceptive stimulation from it decreases while the uterine contractions and stretching of the lower uterine segment still cause pain as in the first stage. In addition, the fetal head begins to descend causing distension of the pelvic floor, vagina, and perineum activating stretch receptors located through these structures and activating an additional source of nociceptive impulses that account for the severest pain that accompanies the second stage and delivery in particular.

Pain increases as labor progresses becoming, in the second stage, more intense and widespread and refers not only to T11–T12 dermatomes, as in the first stage, but also to segments above and below so that pain is also felt in the upper thighs and mid-sacral areas (L1, L2) and umbilical region (T10).

In addition, the severe distension and traction of the pelvic structures result in an additional source of pain that is carried by the pudendal nerve through the anterior rami of S2 through S4.

Pain during the second stage of labor is primarily somatic: is usually sharp, burning, and well localized [33].

The knowledge of the afferent neural pathways involved in labor pain is crucial as it helps to better understand the changing analgesic requirement needed during the different phases of labor.

9.1.6 Analgesia for the Second Stage

The ideal analgesia during the second stage of labor should be able to provide successful and profound analgesia until delivery in response to the increased pain that characterizes this stage. This, of course, should not interfere with the important physiological processes that occur during this stage such as the descent and rotation of the fetal head. In addition, it should enable mothers to push effectively by preserving the sensation of the contractions, the increasing rectal pressure as the fetal head descends, and the urge to bear down.

Pain during labor is a dynamic process and epidural analgesia should be adequately titrated because it is transmitted mainly by somatic nerve fibers that are

much larger and more difficult to block than the thinner visceral nerve fibers. The intensity of labor pain, in fact, increases with greater cervical dilatation [34, 35], and there is almost a threefold increase in the analgesic requirement during advanced labor and delivery [36].

Unfortunately, despite the extensive understanding of the mechanisms involved in pain transmission and pain management during the second stage of labor, women often complain that their labor analgesia wore off during the second stage and delivery in particular.

Several studies show that the effective analgesia experienced by women during the first stage begins to decrease with the progression of labor into the second stage resulting in a significant percentage of parturients being in pain during most of the second stage [37–41] peaking at delivery [42].

Nowadays, epidural analgesia for labor involves using very dilute local anesthetic plus opioid solutions that is the least likely method associated with instrumental delivery, presumably as a result of the preservation of the muscle tone and the bearing down reflex [43] but in some cases, if not adequately titrated, may not permit parturients to experience a complete pain-free delivery, particularly during the most painful second stage.

Nevertheless, often, anesthesiologists are asked to decrease the epidural infusion rate or discontinue epidural analgesia during the second stage and/or the pushing phase due to the misconception that this will improve the maternal ability to bear down, increase the duration of the second stage, and increase the likelihood of a spontaneous vaginal delivery.

However, this practice does not guarantee an improvement in the obstetric and fetal outcome, nor increases the likelihood of a spontaneous vaginal delivery but only results in greater pain for the parturient [44].

In addition the custom of withholding labor epidural analgesia is to be considered unethical and breaks the “pain-free labor agreement” between the woman and the physician previously established during the informed consent procedure.

9.1.7 Maintaining Epidural Analgesia During the II Stage

The maintenance of epidural analgesia in the second stage should be individualized and should consider several factors. The need for additional analgesia, in fact, depends on the patient’s characteristics such as the individual pain threshold, type of labor (normal or dysfunctional), fetal position, and the length of time of epidural use.

When the diagnosis of the second stage is made, anesthesiologists should at that moment assess the quality of the present analgesia, the possible presence of missed segments or unilateral block, or the discomforting sensation of rectal or vaginal pressure and this should then guide his decision to administer additional analgesic.

Generally, additional epidural top-ups of highly concentrated local anesthetic solutions are needed or, if a continuous infusion is used, an increase of the infusion rate is required.

9.1.8 De Novo II Stage Analgesia

In some situations an anesthesiologist is asked to establish a neuraxial block in the second stage of labor. This situation may offer some problems to anesthesiologists as parturient cooperation with the performance of the block is significantly reduced due to the extreme pain being experienced. The avoidance of motor block is also more difficult as higher doses and/or concentrations of local anesthetics are needed to guarantee a successful analgesia.

9.1.8.1 Epidural Analgesia

The epidural technique can be offered to establish effective labor analgesia in the second stage.

This technique, however, may be associated with higher failure rates when compared to CSE analgesia due to its slower onset of effective analgesia [45] and in particular the delay in blocking the larger sacral nerve roots involved with second-stage labor pain.

9.1.8.2 Combined Spinal Epidural Analgesia

The combined spinal epidural (CSE) technique is indicated to establish analgesia in the second stage of labor, especially in its advanced phase due to its fast onset of analgesia. The mean onset of effective analgesia of the CSE technique is, in fact, approximately 2–5 min, whereas the onset after epidural analgesia is approximately 10–15 min [45].

In addition, one important feature of this technique is that it provides the rapid onset of effective sacral analgesia that makes the CSE technique very appealing for a laboring woman asking for analgesia in the second stage of labor or a multiparous woman with a rapid progression of labor.

9.1.8.3 Single-Shot Spinal Analgesia

Single-shot spinal analgesia may represent an option for pain relief in the second stage of labor when the CSE and epidural technique are not available or when the parturient asks for analgesia when delivery is imminent. The performance of a single-shot technique is in fact not indicated early in the second stage as the duration of the drug injected is limited in time and may not cover the pain of the late second stage and delivery [46].

9.1.8.4 Continuous Spinal Analgesia

Continuous spinal anesthesia provides a rapid onset and reliable analgesia and it might be an effective option for initiating neuraxial analgesia in the second stage. However, this technique is rarely employed in the obstetric setting due to the limitations of the available equipment and, above all, for the increased risk of complications such as the unacceptably high rate of postdural puncture headache [47]. In addition, it does not offer any advantages over the epidural or CSE technique; therefore it will probably continue to be an infrequently used option in

the obstetric population except for particular clinical situations such as difficult epidural catheter placement or parturients with previous spine surgery.

9.2 Analgesia for Delivery (Perineal Analgesia)

Perineal pain is felt when the fetal head distends the perineum. At this point, anesthesiologists should provide an effective block of the sacral roots to guarantee a painless delivery.

Anesthesiologists should evaluate the adequacy of perineal analgesia before delivery and might also perform a sensory block evaluation to assess any segmental deficiencies present in the caudal area obtaining a rough estimate of the amount of analgesic solution that is likely to be needed to obtain the solid sensory blockade of all segments below T10.

If no adequate sacral analgesia is present at the time of delivery, perineal anesthesia can usually be produced with either 5–10 mL of 1–2 % mepivacaine, or lidocaine, or 2-chloroprocaine (or 0.2–0.5 % bupivacaine).

Perineal infiltration is the most commonly used regional anesthetic technique in patients that deliver without preexisting epidural analgesia. It also provides anesthesia for repair of lacerations and may also be used to supplement poorly functioning epidural analgesia or in the case of epidural catheter migration during labor.

9.3 Third Stage of Labor

9.3.1 Definition and Management

The third stage of labor is defined as the time elapsing from the moment of delivery of the baby to complete expulsion of the placenta and membranes [5].

The third stage consists of a latent phase in which all the myometrium contracts except for that behind the placenta which remains relaxed, a contraction phase where the retro-placental myometrium also contracts and leads to the separation of the placenta, and the expulsion phase where the placenta is expelled through the birth canal [48]. At this point, precise protective mechanisms occur to prevent excessive bleeding such as the contraction of the muscle fibers that surround the maternal vessels [49] and also the women's coagulation system that activates temporarily [50].

Physiologically, the third stage is characterized by a limited blood loss, as the placenta separates and is expelled. That depends on how long it takes for the placenta to separate from the uterine wall and how effectively the uterine muscle contracts in the immediate postpartum period.

However, approximately in 5 % of deliveries, postpartum hemorrhage (PPH) occurs during the third stage and in particular within the first hour after delivery and is responsible for a major part of maternal morbidity and mortality [51, 52].

The main causes of PPH are uterine atony that accounts for 90 % all of cases, retained placenta and clots, vaginal trauma or uterine injury, and also the presence of preexisting or acquired coagulopathy.

There are basically two clinical approaches for the management of the third stage: the physiological management and the active one [5, 53–55].

The physiological (expectant) management consists of a “hands-off” approach where the delivery of the placenta is spontaneous or just helped by maternal expulsive efforts while uterotonics are not given prophylactically, the cord is neither clamped nor cut early, and there is no controlled cord traction.

On the other hand, the active management of the third stage of labor consists of the clinician intervention in the process of placental expulsion that involves the prophylactic use of uterotonics, the early clamping and cutting of the umbilical cord, and the controlled umbilical cord traction to deliver the placenta.

The active management of the third stage of labor has been introduced with the primary aim of reducing significant postpartum blood loss [54].

The Cochrane collaboration found that the active management of the third stage of labor as a routine preventative measure significantly reduces primary blood loss >500 mL and reduces the risk of postpartum hemorrhage (defined as a blood loss >1,000 mL) [5, 56]. In addition, this practice also reduces the need for the manual removal of the placenta [57], the duration of the third stage, the occurrence of postnatal maternal anemia, and the need for blood transfusion [5].

However, it is worth noting that the active management of the third stage may itself have some adverse effects, and this is mainly attributed to both the administration of ergot alkaloid as the uterotonic drugs and the practice of early umbilical cord clamping.

The adverse effects related to uterotonic use consist in an increased risk of maternal nausea, vomiting, and elevated blood pressure [56, 58].

There are several different types of uterotonic drugs that can be administered as a part of the active management of the third stage such as oxytocin, ergot alkaloids, and prostaglandins, but usually, a combination of ergometrine and oxytocin or ergometrine or oxytocin alone is used.

The combination of ergometrine and oxytocin is associated with a small but statistically significant reduction in the risk of PPH when compared to oxytocin alone for blood loss >500 mL. However, this drug association is also characterized by an increased incidence of maternal side effects such as nausea and vomiting and an increase in blood pressure with respect to the use of oxytocin alone [59].

The timing of the uterotonics administration varies across different countries and it is controversial as to whether they should be administered prior or after the expulsion of the placenta [60].

The second component of the active management of the third stage is the early cord clamping that consists of the clamping of the umbilical cord within 20–60 s of birth.

The practice of early cord clamping may anyway have adverse consequences for the fetus. In fact, it reduces the volume of placental blood transfusion to the fetus leading to a reduction in the infant blood volume at birth by about 20 % [61, 62]

with consequent lower hematocrit levels and hemoglobin concentration [59, 61–63]. However the clinical relevance of this feature is not well established.

Placental delivery is an essential step that occurs during the third stage as it allows the uterus to contract, therefore reducing the physiological volume of blood loss during this stage. Failure of the placenta to be delivered in this timely manner, in fact, is an important risk factor for postpartum hemorrhage [64, 65].

There are two basic interventions to help to deliver the placenta as part of the active management of the third stage of labor: the fundal pressure or the controlled traction on the umbilical cord that represents the standard third component of the active management of the third stage. Controlled cord traction consists of the gentle traction on the umbilical cord coupled with a counter-pressure upwards on the lower abdomen in correspondence to the lower segment of the uterus, performed after clear signs of placental separation become evident. Fundal pressure (Crede maneuver) consists of placing one hand on the lower abdomen in correspondence to the uterine fundus and squeezing it to facilitate placental separation and the expulsion of the placenta through the birth canal [66].

Both these interventions, especially if not correctly performed, may have adverse outcomes, such as severe pain, hemorrhage, or even uterine inversion that represents a life-threatening and unpredictable obstetric emergency which can lead to severe hemorrhage and shock [67, 68].

Since there are no randomized controlled trials to support the use of fundal pressure rather than controlled cord traction as part of the active management of the third stage of labor, the first should continue to be chosen as the method of placental delivery in the active management of the third stage of labor.

Usually the placenta is delivered within 5–15 min from the delivery of the baby; however, in approximately 0.6 and 3.3 % of normal deliveries, it is not delivered within 30 min of the birth when the third stage is actively managed or within 60 min when physiologically managed, and a significant complication defined retained placenta occurs [5, 65, 69].

The best strategy to treat retained placenta is to administer oxytocin in the umbilical vein followed by the proximal clamping of the cord, and, if after 30 min from this intervention the placenta is still retained, or if there is concern about the maternal well-being, the manual removal of the placenta should be performed [5].

9.3.2 Pain Pathways During the Third Stage

Pain during the third stage is due to the dilation of the cervix by the passage of the placenta and also to the contraction of the uterus. These impulses, like those that occur during the first stage of labor, enter the spinal cord via the 11th and 12th thoracic nerve.

9.3.3 Analgesia for the Third Stage

The third stage of labor is not particularly painful as the cervix is already fully dilated. In addition, if an effective epidural is running, the expulsion of the placenta will not cause any pain. However, the importance of this phase is not to be underestimated.

The Nice guidelines recommend, in fact, close obstetric and anesthetic observation after delivery, indicating that a care provider should observe the parturient's physical status and vital parameters and that palpation of the uterus and the amount of vaginal blood loss should be regularly evaluated.

Anesthesiologists should be promptly available to intervene should any complication of the third stage occur like postpartum hemorrhage or retained placenta. In this latter case, the anesthesiologist might use the epidural catheter previously used for providing labor analgesia to obtain effective anesthesia.

It is therefore good practice to remove the epidural catheter 2–4 h after delivery.

9.4 Episiotomy

Episiotomy consists of the incision through the perineal tissues as the fetal head distends the perineum shortly before delivery, in order to enlarge the vaginal outlet during delivery.

There are two types of episiotomy: the median and the medio-lateral. The first one consists of an incision that begins in the midline and extends posteriorly following the natural insertion of the perineal muscles, while the medio-lateral extends from the midline in a 45° angle to either side.

The disadvantages of the first one are that it is associated with a higher incidence of anal sphincter and rectal mucosa injury and may also cause severe postpartum pain while the latter is associated with a reduced incidence of damage to the anal sphincter and less postpartum pain, is easier to repair, and causes less blood loss.

Episiotomy is usually performed in the belief that it makes the baby's birth easier and reduces the incidence of vaginal tears associated with the vaginal opening as the baby's head passes through, especially if the baby descends quickly.

Vaginal tears, in fact, are one of the most common complications that can occur during vaginal birth and may cause discomfort and pain a long time after childbirth that will affect the quality of life of the parturients.

Spontaneous trauma can involve the labia anteriorly and the perineal skin or extend to the muscles and the anal sphincter and anus. Spontaneous tears are defined as *first degree* when there is only involvement of the fourchette; *second degree* when there is involvement of the fourchette plus the perineal muscles and skin; *third degree* when there is also injury to the anal sphincter; and *fourth degree* when the injury extends through the rectal mucosa to expose the lumen of the bowel [70].

The occurrence of obstetric perineal third or fourth degree tears lacerations is an important cause of maternal morbidity as they may cause important long-term

adverse effects including, in particular, the development of anal incontinence after childbirth but also dyspareunia, chronic perineal pain, and recto-vaginal fistula [71, 72]. Although significant anal incontinence only affects a minority of women, its symptoms may have devastating effects on the quality of life for the women affected [73].

Episiotomy was largely used in the past, while its use in contemporary obstetrics is now restricted [74].

The American College of Obstetricians and Gynecologists suggested that not using episiotomy routinely as the performance of a midline episiotomy is itself associated with third and fourth degree tear and is not associated with any benefits when compared to no episiotomy [74, 75].

The Cochrane collaboration also demonstrated that there is evidence to support the restrictive use of episiotomy as compared to the routine use of episiotomy because the first is associated with a lower risk of clinically relevant morbidities such as severe perineal trauma, the need for suturing, and fewer complications. The only disadvantage shown in the restrictive use of episiotomy is an increased risk of anterior perineal trauma [76].

Epidural analgesia during labor has traditionally been regarded as a risk factor for severe perineal trauma after vaginal delivery due to the presumed interference with the second stage of labor that may lead to increased obstetric intervention such as operative deliveries and consequent perineal trauma. Old studies investigating the relationship between the use of epidural analgesia and perineal trauma gave conflicted results [77–80]. More recent research postulated, on the contrary, that epidural analgesia might have a protective effect against severe third and fourth degree lacerations due to the relaxation of the muscles of the pelvic floor induced by this technique that may allow a more controlled delivery of the fetal head, therefore reducing obstetric lacerations [77, 81, 82].

Interestingly, recent evidence suggests that the etiological factors responsible for the occurrence of obstetric anal sphincter injuries are to be found in ethnicity, operative vaginal birth, persistent occipito-posterior position, and rapid uncontrolled delivery of the fetal head rather than in the epidural use [83].

The routine use of episiotomy is not even recommended in the case of instrumental vaginal delivery. The available studies on this topic [84–86] were not able to define if a policy of routine episiotomy might reduce the incidence of perineal and genital trauma. Therefore, also in this case, the restrictive use of episiotomy is supported [30].

9.4.1 Pain Pathways

The sensory and motor innervation of the perineum is provided by the pudendal nerve that arises primarily from segments S2 to S4. This nerve divides into three branches: the inferior rectal (inferior hemorrhoidal), perineal, and clitoral nerves. A third branch of the pudendal nerve, the inferior rectal nerve, provides innervation to the perirectal skin, the anal sphincter, and parts of the musculature of the posterior

pelvic floor. The mons pubis and anterior labia are supplied by the ilioinguinal and genitofemoral nerves. These nerves arise from the lumbar plexus and travel through the inguinal canal.

9.4.2 Analgesia for Episiotomy

Episiotomy represents a surgical procedure; therefore dense and profound perineal anesthesia is required.

The degree of perineal block obtained after a few hours of effective epidural analgesia might not be dense enough to guarantee a pain-free procedure.

It is therefore advisable to always perform a careful assessment of the adequacy of perineal anesthesia prior to the incision in order to correctly titrate the dose of local anesthetic possibly needed for this phase.

Lidocaine 2 % or mepivacaine 2 %, 5 or 10 mL, may be ideal for this phase due to their rapid onset or the anesthesiologist may prefer to carry on with the same local anesthetic previously used for labor analgesia but increasing its concentration in order to obtain a surgical anesthesia of the perineum. Time must be allowed for the top-up to take effect before episiotomy is performed; usually, if epidural analgesia is running, approximately 3–5 min are needed for the perineum to be adequately anesthetized.

Following delivery of the placenta and its inspection, episiotomy or minor lacerations are repaired.

Parturients who received correct anesthesia of the perineum for episiotomy usually don't need any additional top-ups. However, it is crucial at this time to remain in the labor ward and monitor the parturient's vital signs to rule out any complications relative to delivery.

References

1. Kilpatrick SJ, Laros RK (1989) Characteristics of normal labor. *Obstet Gynecol* 74:85–87
2. Gross MM, Drobnic S, Keirse MJN (2005) Influence of fixed and time- dependent factors on duration of normal first stage labor. *Birth* 32:27–33
3. Lydon-Rochelle MT (2006) Prolonged second stage of labor and risk of adverse maternal and perinatal outcomes: a systematic review. *Birth* 33:315–322
4. Allen VM, Baskett TF, O'Connell CM et al (2009) Maternal and perinatal outcomes with increasing duration of the second stage of labor. *Obstet Gynecol* 113:1248–1258
5. National Collaborating Centre for Women's and Children's Health (UK) (ed) (2007) National Institute for Health and Clinical Excellence: guidance. Intrapartum care: care of healthy women and their babies during childbirth. RCOG, London
6. Spong CY, Berghella V, Wenstrom KD et al (2012) Preventing the first cesarean delivery: summary of a joint Eunice Kennedy Shriver National Institute of Child Health and Human Development, Society for Maternal-Fetal Medicine, and American College of Obstetricians and Gynecologists Workshop. *Obstet Gynecol* 120:1181–1193
7. Atwood RJ (1976) Parturition posture and related birth behavior. *Acta Obstet Gynecol Scand* 57:3–25

8. Gupta JK, Hofmeyr GJ, Shehmar M (2012) Position in the second stage of labour for women without epidural anaesthesia. *Cochrane Database Syst Rev* (5):CD002006. doi:[10.1002/14651858.CD002006.pub3](https://doi.org/10.1002/14651858.CD002006.pub3)
9. Carbone B, Benachi A, Leveque M et al (1996) Maternal position during labor: effects on fetal oxygen saturation measured by pulse oximetry. *Obstet Gynecol* 88:797–800
10. Kemp E, Kingswood CJ, Kibuka M et al (2013) Position in the second stage of labour for women with epidural anaesthesia (Review). *Cochrane Database Syst Rev* (1):CD008070. doi:[10.1002/14651858.CD008070.pub2](https://doi.org/10.1002/14651858.CD008070.pub2)
11. Fuchs AR, Romero R, Keefe D et al (1991) Oxytocin secretion and human parturition: pulse frequency and duration increase during spontaneous labor in women. *Am J Obstet Gynecol* 165:1515–1523
12. Hellman LM (1956) Anatomic and physiologic considerations. In: Hingson RA, Hellman LM (eds) *Anesthesia for obstetrics*. JB Lippincott, Philadelphia, PA, p 74
13. Goodfellow CF, Hull MGR, Swaab DF et al (1983) Oxytocin deficiency at delivery with epidural analgesia. *Br J Obstet Gynaecol* 99:96–100
14. Rahm VA, Hallgren A, Hognerg H et al (2002) Plasma oxytocin levels in women during labor with or without epidural analgesia: a prospective study. *Acta Obstet Gynecol Scand* 81: 1033–1039
15. Stocche RM, Klamt JG, Antunes-Rodrigues J et al (2001) Effect of intrathecal sufentanil on plasma oxytocin and cortisol concentrations in women during the first stage of labor. *Reg Anesth Pain Med* 26:545–550
16. De Geest K, Thiery M, Piron-Possuyt G et al (1985) Plasma oxytocin in human pregnancy and parturition. *J Perinat Med* 13:3–13
17. Scull TJ, Hemmings GT, Carli F et al (1998) Epidural analgesia in early labour blocks the stress response but uterine contractions remain unchanged. *Can J Anaesth* 45:626–630
18. Hanson L (2009) Second-stage labor care: challenges in spontaneous bearing down. *J Perinat Neonatal Nurs* 23:31–39
19. Roberts J, Hanson L (2007) Best practices in second stage labor care: maternal bearing down and positioning. *J Midwifery Womens Health* 52:238–245
20. Cosner KR, DeJong E (1993) Physiologic second stage labor. *MCN Am J Matern Child Nurs* 18:38–43
21. Barnett MM, Sharron S, Humenick RN (1982) Infant outcome in relation to second stage labor pushing method. *Birth* 9:221–229
22. Bloom SL, Casey BM, Schaffer JI et al (2006) A randomized trial of coached versus uncoached maternal pushing during the second stage of labor. *Am J Obstet Gynecol* 194:10–13
23. Prins M, Boxem J, Lucas C et al (2011) Effect of spontaneous pushing versus Valsalva pushing in the second stage of labour on mother and fetus: a systematic review of randomized trials. *BJOG* 118:662–670
24. Fraser WD et al (2000) Multicenter, randomized, controlled trial of delayed pushing for nulliparous women in the second stage of labor with continuous epidural analgesia. *Am J Obstet Gynecol* 182:1165–1172
25. Roberts CL, Torvaldsen S, Cameron CA et al (2004) Delayed versus early pushing in women with epidural analgesia: a systematic review and meta-analysis. *BJOG* 111:1333–1340
26. Fitzpatrick M, Harkin R, Mc Quillan K et al (2002) A randomized clinical trial comparing the effects of delayed versus immediate pushing with epidural analgesia on mode of delivery and faecal incontinence. *BJOG* 109:1359–1365
27. Hansen SL, Clark SL, Foster JC (2002) Active versus passive fetal descent in the second stage of labor: a randomized controlled trial. *Obstet Gynecol* 99:29–34
28. Simpson KR, James DC (2005) Effects of immediate versus delayed pushing during second-stage labor on fetal well-being: a randomized clinical trial. *Nurs Res* 54:149–157
29. Altman MR, Lydon-Rochelle MT (2006) Prolonged second stage of labor and risk of adverse maternal and perinatal outcomes: a systematic review. *Birth* 33:315–322

30. Royal College of Obstetricians and Gynaecologists Guidelines and Audit Committee (2011) RCOG Clinical Green Top Guidelines. Instrumental Vaginal Delivery No. 26
31. FIGO Safe Motherhood and Newborn Health (SMNH) Committee (2012) Management of the second stage of labor. *Int J Gynaecol Obstet* 119:111–116
32. Le Ray C, Audibert F, Goffinet F et al (2009) When to stop pushing: effects of duration of second-stage expulsion efforts on maternal and neonatal outcomes in nulliparous women with epidural analgesia. *Am J Obstet Gynecol* 201:361.e1–361.e7
33. Mc Donald JS (2000) Pain of childbirth. In: Loeser JD (ed) *Bonica's management of Pain*, 3rd edn. Lippincott Williams & Wilkins, Philadelphia, PA, pp 1388–1410
34. Brown ST, Campbell D, Kurtz A (1989) Characteristics of labour pain at two stages of cervical dilation. *Pain* 38:289–295
35. Lowe NK (1992) Differences in first and second stage labour pain between nulliparous and multiparous women. *J Psychosom Obstet Gynaecol* 13:243–253
36. Capogna G, Celleno D, Lyons G (1998) Minimum local anaesthetic concentration of extradural bupivacaine increases with progression of labour. *Br J Anaesth* 89:11–13
37. Chua NP, Sia AT, Ocampo CE (2001) Parturient-controlled epidural analgesia during labour: bupivacaine vs ropivacaine. *Anaesthesia* 56:1169–1173
38. Gambling DR, McMorland GH, Yu P et al (1990) Comparison of patient-controlled epidural analgesia and conventional intermittent “top- up” injections during labor. *Anesth Analg* 70: 256–261
39. Gambling D, Berkowitz J, Farrell TR et al (2013) A randomized controlled comparison of epidural analgesia and combined spinal-epidural analgesia in a private practice setting: pain scores during first and second stages of labor and at delivery. *Anesth Analg* 116:636–643
40. Rathinam S, Tilakaratna P, Plaat F (2008) Pain relief in the second stage of labour: room for improvement? *Intern J Obstet Anaesth*, P25
41. Lim Y, Ocampo CE, Supandji M et al (2008) A randomized controlled trial of three patient-controlled epidural analgesia regimens for labor. *Anesth Analg* 107:1968–1972
42. Carvalho B, Cohen SE, Giarrusso M (2005) “Ultra-light” patient-controlled epidural analgesia during labor: effects of varying regimens on analgesia and physician workload. *Anesth Analg* 14:223–229
43. Thornton JG, Capogna G (2001) Reducing likelihood of instrumental delivery with epidural anaesthesia. *Lancet* 358:2
44. Torvaldsen S, Roberts CL, Bell JC et al (2010) Discontinuation of epidural analgesia late in labour for reducing the adverse delivery outcomes associated with epidural analgesia *Cochrane Database Syst Rev* (4):CD004457. doi: [10.1002/14651858.CD004457.pub2](https://doi.org/10.1002/14651858.CD004457.pub2)
45. Simmons SW, Taghizadeh N, Dennis AT et al (2012) Combined spinal-epidural versus epidural analgesia in labour. *Cochrane Database Syst Rev* (10):CD003401. doi: [10.1002/14651858.CD003401.pub3](https://doi.org/10.1002/14651858.CD003401.pub3)
46. Minty RG, Kelly L, Minty A et al (2007) Single-dose intrathecal analgesia to control labour pain: is it a useful alternative to epidural analgesia? *Can Fam Physician* 53:437–442
47. Palmer CM (2010) Continuous spinal anesthesia and analgesia in obstetrics. *Anesth Analg* 111:1476–1479
48. Herman A, Zimmerman A, Arieli S et al (2002) Down-up sequential separation of the placenta. *Ultrasound Obstet Gynecol* 19:278–281
49. Inch S (1985) Management of third stage of labour-another cascade of intervention? *Midwifery* 1:114–122
50. BonnarJ MNGP, Douglas AS (1970) Coagulation and fibrinolysis mechanisms during and after normal childbirth. *BMJ* 103:200–203
51. Reynders FC, Senten I, Tjalma W et al (2006) Postpartum hemorrhage: a practical approach to a life- threatening complication. *Clin Exp Obstet Gynecol* 33:81–84
52. Ramanathan G, Arulkumaran S (2006) Postpartum hemorrhage. *Curr Obstet Gynecol* 16:6–13

53. Prendiville WJ, Elbourne DR (1989) Care during the third stage of labour. In: Chalmers I, Enkin M, Keirse MJNC (eds) *Effective care in pregnancy and childbirth*. Oxford University Press, Oxford, pp 1145–1169
54. Prendiville WJ, Elbourne D, McDonald S (2009) Active versus expectant management in the third stage of labour. *Cochrane Database Syst Rev* (3):CD000007. doi:[10.1002/14651858.CD000007.pub2](https://doi.org/10.1002/14651858.CD000007.pub2)
55. Rogers J, Wood J, McCandlish R et al (1998) Active versus expectant management of third stage of labour: the Hinchingsbrooke randomized controlled trial. *Lancet* 351:693–699
56. Begley CM, Gyte GM, Devane D et al (2011) Active versus expectant management for women in the third stage of labour. *Cochrane Database Syst Rev* (11):CD007412. doi:[10.1002/14651858.CD007412.pub3](https://doi.org/10.1002/14651858.CD007412.pub3)
57. Brucker MC (2001) Management of the third stage of labor: an evidence-based approach. *J Midwifery Womens Health* 46:381–392
58. MaughanKL HSW, Galazka SS (2006) Preventing postpartum hemorrhage: managing the third stage of labor. *Am Fam Physician* 73:1025–1028
59. McDonald S, Abbott JM, Higgins SP (2007) Prophylactic ergometrine-oxytocin versus oxytocin for the third stage of labor. *The Cochrane database Syst Rev* (1):CD000201. doi:[10.1002/14651858.cd000201.pub2](https://doi.org/10.1002/14651858.cd000201.pub2)
60. Soltani H, Hutchon DR, Poulouse TA (2010) Timing of prophylactic uterotonics for the third stage of labour after vaginal birth. *Cochrane Database Syst Rev* (8):CD006173. doi:[10.1002/14651858.CD006173.pub2](https://doi.org/10.1002/14651858.CD006173.pub2)
61. Hutton EK, Hassan ES (2007) Late vs early clamping of the umbilical cord in full-term neonates: a systematic review and meta-analysis of controlled trials. *JAMA* 297:1241–1252
62. McDonald SJ, Middleton P (2008) Effect of timing of umbilical cord clamping of term infants on maternal and neonatal outcomes. *Cochrane Database Syst Rev* (2):CD004074. doi:[10.1002/14651858.CD004074.pub3](https://doi.org/10.1002/14651858.CD004074.pub3)
63. Van Rheenen P, De Moor L, Escbach S et al (2007) Delayed cord clamping and haemoglobin levels in infancy: a randomised controlled trial in term babies. *Trop Med Int Health* 12: 603–616
64. Dombrowski MP, Bottoms SF, Saleh AA et al (1995) Third stage of labor: analysis of duration and clinical practice. *Am J Obstet Gynecol* 172:1279–1284
65. Combs CA, Laros RK (1991) Prolonged third stage of labor: morbidity and risk factors. *Obstet Gynecol* 77:863–867
66. Brandt ML (1993) The mechanism and management of the third stage of labour. *Am J Obstet Gynecol* 25:662–667
67. LipitzS FJ (1988) Puerperal inversion of the uterus. *Eur J Obstet Gynecol Reprod Biol* 27: 271–274
68. Miras T, Collet F, Seffert P (2002) Acute puerperal uterine inversion: two cases. *J Gynecol Obstet Biol Reprod* 31:668–671
69. Tandberg A, Albrechtsen S, Iverson DE (1999) Manual removal of placenta. *Acta Obstet Gynecol Scand* 78:33–36
70. Fernando R, Sultan AH, Kettle C et al (2006) Methods of repair for obstetric anal sphincter injury. *Cochrane Database of Systematic Reviews* (3):CD002866. doi:[10.1002/14651858.CD002866.pub2](https://doi.org/10.1002/14651858.CD002866.pub2)
71. Zetterstrom J, Lopez A, Anzen B et al (1999) Anal sphincter tears at vaginal delivery: risk factors and clinical outcome of primary repair. *Obstet Gynecol* 94:21–28
72. Zetterstrom JP, Lopez A, Anzen B et al (1999) Anal incontinence after vaginal delivery: a prospective study in primiparous women. *BJOG* 106:324–330
73. Johanson JF, Lafferty J (1996) Epidemiology of fecal incontinence: the silent affliction. *Am J Gastroenterol* 91:33–36
74. American College of Obstetricians and Gynecologists (2006) Episiotomy: clinical management guidelines for obstetrician-gynecologists. *ACOG Practice Bulletin No. 71*. Washington, DC, ACOG. *Obstet Gynecol* 107:957–962

75. Hartmann K, Viswanathan M, Palmieri R et al (2005) Outcomes of routine episiotomy: a systematic review. *JAMA* 293:2141–2148
76. Carroli G, Mignini L (2009) Episiotomy for vaginal birth. *Cochrane Database Syst Rev* (1): CD000081. doi:10.1002/14651858.CD000081.pub2
77. Walker MP, Farine D, Rolbin SH et al (1991) Epidural anesthesia, episiotomy, and obstetric laceration. *Obstet Gynecol* 77:668–671
78. Bikers WM (1970) Epidural analgesia in obstetrics. *J Reprod Med* 5:41–49
79. Legino LJ, Woods MP, Rayburn WF et al (1988) Third and fourth degree tears. 50 year's experience at a university hospital. *J Reprod Med* 33:423–426
80. Combs CA, Robertson PA, Laros RK (1990) Risk factors for third-degree and fourth-degree perineal lacerations in forceps and vacuum deliveries. *Am J Obstet Gynecol* 163:100–104
81. Albers LL, Migliaccio L, Bedrick EJ et al (2007) Does epidural analgesia affect the rate of spontaneous obstetric lacerations in normal births? *J Midwifery Womens Health* 5:31–36
82. MacDougall M, Waugh J, Morland D (2011) Epidural analgesia may be protective against third and fourth degree perineal trauma. *Arch Dis Child Fetal Neonatal* 96:1. doi:10.1136/archdischild.2011.300162.2
83. Burrell M, Dilgir S, Patton V et al (2004) Risk factors for obstetric anal sphincter injuries and postpartum anal and urinary incontinence: a case-control trial. *Int Urogynecol J*. doi:10.1007/s00192-014-2478-7
84. Murphy DJ, Macleod M, Bahl R et al (2008) A randomized controlled trial of routine versus restrictive use of episiotomy at operative delivery: a multicenter pilot study. *BJOG* 115: 1695–1702
85. Robinson JN, Norwitz ER, Cohen AP et al (1999) Episiotomy, operative vaginal delivery, and significant perineal trauma in nulliparous women. *Am J Obstet Gynecol* 181:1180–1184
86. Bodner-Adler B, Bodner K, Kimberg O et al (2003) Management of the perineum during forceps delivery. Association of episiotomy with the frequency and severity of perineal trauma in women undergoing forceps delivery. *J Reprod Med* 48:239–241

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10.1 Introduction

Labor results in severe pain for most women. In the absence of medical contraindication, maternal request is a sufficient indication for pain relief, which is performed in the vast majority of developed countries through neuraxial analgesia. Regional analgesia techniques (spinal, epidural, and combined spinal epidural) are the most flexible, effective, and least debilitating to the central nervous system, enabling an alert and active woman to deliver an alert neonate [1].

Neuraxial analgesia may, however, have some effects on maternal, placental, and labor physiology that may possibly impair maternal and neonatal well-being.

This chapter will examine the possible contribution of neuraxial analgesia to determine the FHR changes observed after labor analgesia, the probable causes, and the suggested solutions, according to the major international guidelines [2–8].

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10.2 Systemic and Fetal Consequences of Neuraxial Analgesia

Neuraxial analgesia may be associated with undesirable side effects for both the parturient and fetus [9], but their incidence and causes cannot be determined easily. They have been measured and compared with other techniques providing pain relief during labor, mainly systemic administration of opioids, in either observational or randomized controlled study [3, 4]. Neuraxial analgesia has been associated with longer second-stage labor, more frequent oxytocin augmentation, and maternal hypotension. However, it is not clear whether this association is causative or not. Neuraxial analgesia does not affect fetal oxygenation, neonatal pH, or 5-min Apgar scores by contrast with systemic opioid administration [5, 6]. All parenteral opioids have indeed a significant effect on the intra-partum fetal heart rate (FHR) tracing, due to transplacental passage. A decrease in FHR variability is commonly observed in around 15 % of cases. This may usually have no impact in the course of the labor. However, parenteral analgesia is associated with a threefold increased risk of Apgar scores lower than 7 at 5 min and a fourfold increased request for neonatal naloxone administration. On the contrary, regional anesthesia is associated with the occurrence of abnormal fetal heart rate patterns in around 15 % of cases, but without any effect on the fetal or neonatal status, and even an improved well-being in some meta-analysis [6].

Neuraxial analgesia induces a sympathetic block, whose intensity depends on the nature and amount of drugs administered at initiation and during the maintenance of analgesia and on the technique of administration (more severe with spinal than with epidural). According to the extension and the density of such sympathetic block, regional analgesia may produce maternal hypotension. The incidence was very important in the 1990s (around 25 %), when neuraxial analgesia was performed with a relatively high amount and concentration of local anesthetics [3, 5]. Most recent practices [7, 8] with low doses of local anesthetics combined with lipophilic opioids may result in less frequent hypotension (10 %). Anesthesiologists, obstetricians, and midwives should be prepared for the occurrence of hypotension, which must be treated with intravenous ephedrine to correct uterine decreased perfusion. Maintaining or providing left uterine displacement should maximize uterine perfusion in the case of hypotension. Transient fetal heart rate changes may also be observed (15 %) after the initiation of neuraxial analgesia.

10.3 Uterine Blood Flow and Fetal Heart Rate During Labor

Uterine blood flow is 700 ml/min at term pregnancy and represents 10–15 % of maternal cardiac output. 80 % of uterine blood flow is devoted to placental perfusion and is not subject to self-regulation. Therefore, maternal hypotension, if not promptly and adequately treated, may have an impact of the utero-placental perfusion, which can, in turn, induce fetal hypoxia. A decreased utero-placental blood flow over 50 % leads to fetal hypoxia and acidosis. Placental blood flow is directly proportional to the uterine perfusion pressure (UPP). UPP is the difference

between the uterine arterial and venous pressures and is inversely proportional to the uterine vascular resistance [9]. These are determined by the intrinsic vasomotor tone of vessels, which may be influenced by physiological stimuli or pharmacological agents, and by the myometrial tone determined by the basal tone and uterine contraction. Uterine contraction does not alter the physiological utero-placental hemodynamic. Utero-placental flow decreases during the peak phase of the contraction, but an offset is created by an increase in the blood volume of the intervillous space, as intrauterine pressure does not exceed spiral artery pressure. During uterine relaxation, the flow returns to normal. When uterine contraction exceeds 60 mmHg, there is a deficiency of oxygen in the intervillous space. During uterine relaxation, recovery of the oxygen debt is completed. In extreme uterine hypertonia cases and/or hyperkinetic frequency, the utero-placental flow is interrupted for a longer time and can lead to fetal hypoxia in utero. Besides decreased maternal blood pressure, other factors can modify the utero-placental blood flow, such as maternal hypocapnia or hypoxia, through norepinephrine-induced increased uterine vasoconstriction [10].

A balance between the antagonistic effects of sympathetic and parasympathetic systems determines the fetal heart rate (FHR) and its variability (Fig. 10.1). The heart rate results from the cardiac cycle sequences, and its characteristics are related to the mechanisms of fetal adaptation to stimuli and aggression. These stimuli can be a change in blood flow, or in blood pressure, for which the baroreceptors are sensitive, or a decrease in PO_2 , for which the chemoreceptors are sensitive. These chemoreceptors are also sensitive to PCO_2 and/or lower pH.

During normal uterine contraction, the fetal level of oxygenation remains globally unchanged. Umbilical blood pressure and flow do not vary, as well as PO_2 and pH. Fetal hypoxia may occur in the case of maternal hypotension, maternal hypocapnia, maternal hypoxia, and uterine hypertonus. Hypoxia produces stress with an increased release of catecholamines and fetal liver and heart glycogenolysis. Glucose consumption becomes anaerobic, which causes a rapid depletion in fetal reserves, production of lactic acid, and acidosis. The physiological hypoxic response depends on the PO_2 initial value and on both the PO_2 fall intensity and its speed. Two phases are described: (1) adaptation by increasing heart rate and blood pressure and (2) adaptation capabilities are exceeded, which causes a drop in heart rate and blood and production acidosis.

Fetal hypoxia produces an increased fetal catecholamine release. This production induces subsequently a rise in blood pressure, which causes a decrease in FHR and a reduction of cardiac output. A redistribution of cardiac output is observed, with vasoconstriction in non-essential areas, which helps maintain blood flow to vital organs. The alteration of the fetal heart rate (control over early fetal aggression) may be due to stress reactions (control of fetal adaptation) or a real cellular hypoxia.

Hypercapnia, associated with increased lactate, causes a decrease in pH. Fetal hypoxia and anoxia lead to a decrease in fetal pH by hypercapnia. This type of respiratory acidosis is easily reversible, seen when there is a difficult gaseous exchange between fetus and mother. Later, there is an increase in lactate

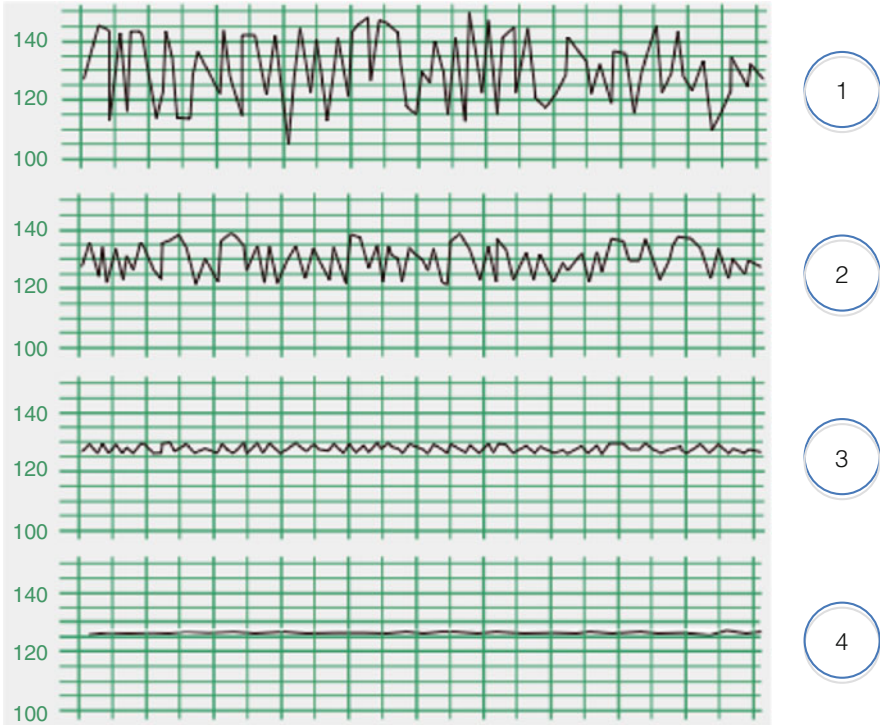


Fig. 10.1 Fetal heart rate variability

- (1) >25 bpm marked variability
- (2) Between 6 and 25 bpm normal variability
- (3) 3–5 bpm minimal variability (decreased)
- (4) No variability

production, associated with hypercapnia (mixed acidosis). At a later stage there is asphyxia associated with metabolic acidosis. Alteration of pH, at the cellular level, may cause permanent cell damage.

Late decelerations mark inadequate utero-placental exchanges, which lead to generalized hypoxia, associated with hypercapnia. Hypoxia leads firstly to the inhibition of the sympathetic and vagal stimulation and secondly to anaerobic glycolysis and metabolic acidosis. The involvement of chemoreceptors, whose response is slower than baroreceptors, is said to offset the decrease. If there is a single placental insufficiency, normal contractions can cause late decelerations. Prolonged delays are due to a sharp decrease in the placental blood flow, either by hyperactivity or by maternal hypotension. Hypoxia appears, which, like late decelerations, causes a drop in heart rate, with acidosis.

10.4 Fetal Monitoring During Labor

Fetal well-being can be appreciated by the monitoring of the fetal heart rate (FHR). FHR monitoring has, however, a good sensibility but a very poor specificity, due to the high inter- and intraindividual variability and to numerous confounding factors (maternal hyperthermia, fetal sleep, drug effects, ...) [11]. The largest prospective randomized trial on electronic fetal monitoring ever carried out [12] did not show any benefit in terms of perinatal mortality or cerebral palsy. The only significant benefit demonstrated by the use of FHR monitoring is a reduction of neonatal seizures, against an increased rate of cesarean and instrumental deliveries. Moreover, several systematic reviews concluded that the use of FHR surveillance had no benefit on neonatal mortality or on the incidence of cerebral palsy [13, 14]. Consequently, the ACOG recommends replacing the term “fetal distress” by “non-reassuring fetal status.” The presence of abnormal FHR must, however, be an alarm to possible acute fetal distress. The obstetrical team must act to avoid fetal effects, including neurological injury or even death. The normal FHR is defined by its oscillations and basic rhythm. Uterine contraction does not cause any significant change normally. The analysis involves a careful and thorough reading following an established protocol and precise terminology. An example of a commonly used classification is the classification of NICHD 2008 [15]:

- Early decelerations are defined by a deceleration of the FHR at the time of uterine contraction, and whose nadir occurs contemporaneously to the maximum peak of the uterine contraction, with a return to basic heart rate (Fig. 10.2).
- The late decelerations are defined by a deceleration of the FHR at the time of uterine contraction, which starts with a delay relative to the beginning of the contraction, with a return to basic heart rate (Fig. 10.3).
- The variable decelerations are defined by a sharp slowdown in FHR. This deceleration must be ≥ 15 bpm compared to the basic FHR, for more than or equal to 15 s but < 2 min. The onset, depth, and duration of delays vary during successive contractions (Fig. 10.4).
- The prolonged decelerations are defined by a deceleration FHR ≥ 15 bpm compared to FHR base, lasting ≥ 2 min, but < 10 min before returning to the FHR base (Fig. 10.5).
- Bradycardia corresponds to < 110 bpm FHR for a period ≥ 10 min.

Continuous fetal heart rate monitoring is recommended prior to performing any type of labor analgesia and during labor. After neuraxial analgesia, FHR abnormalities are observed with a frequency of about 15 % [2, 3].

It is commonly believed that the use of opioids systemically for labor analgesia is accompanied by a decrease in FHR variability, but a meta-analysis, which compared the frequency of abnormal FHR during analgesia produced by systemic opioids versus neuraxial analgesia, was not able to detect any differences [5].

Reports of the FHR effects of epidural analgesia suggest that decelerations are common in the first 60 min after initiation of epidural analgesia. Six reports

Fig. 10.2 Early deceleration

- Visually apparent usually symmetrical gradual decrease and return of the FHR associated with a uterine contraction
- A gradual FHR decrease is defined as from the onset to the FHR nadir of 30 s or more
- The decrease in FHR is recorded from the onset to the nadir of the deceleration
- The nadir of the deceleration occurs at the same time as the peak of the contraction
- In most cases, the onset, nadir, and recovery of the deceleration are coincident with beginning, peak, and ending of the contraction, respectively

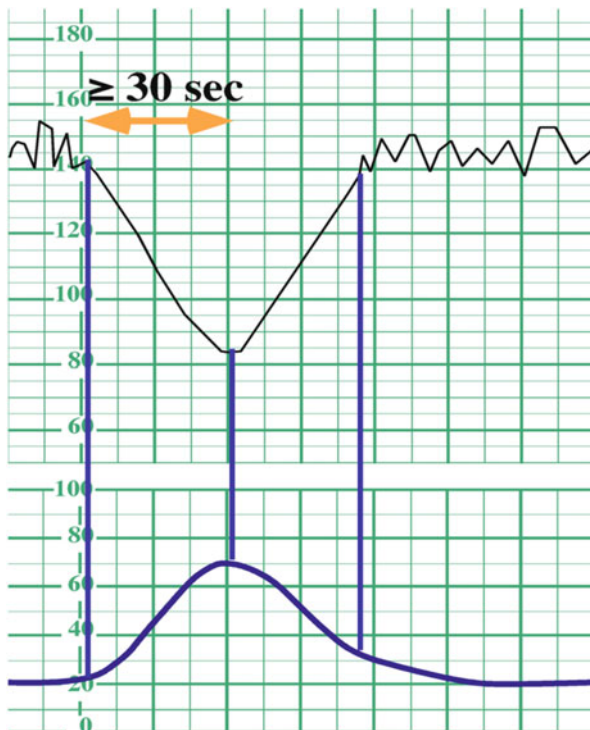


Fig. 10.3 Late deceleration

- The deceleration is delayed in timing, with the nadir of the deceleration occurring after the peak of the contraction
- In most cases, the onset, nadir, and recovery of the deceleration occur after the beginning, peak, and ending of the contraction, respectively

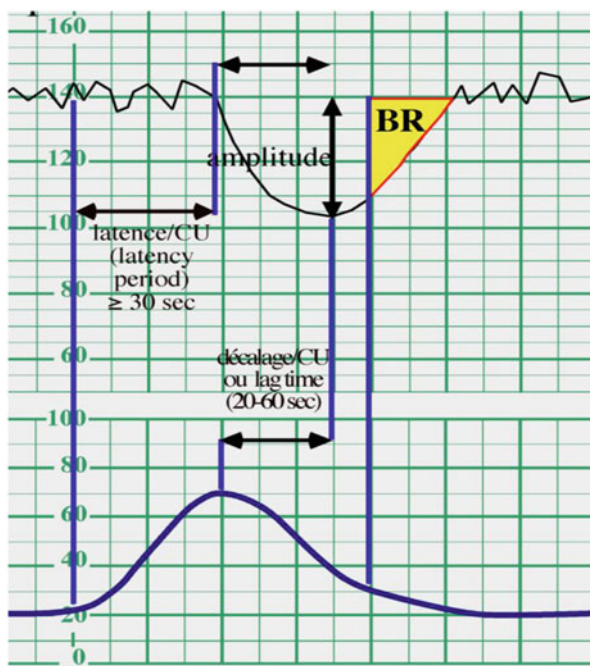
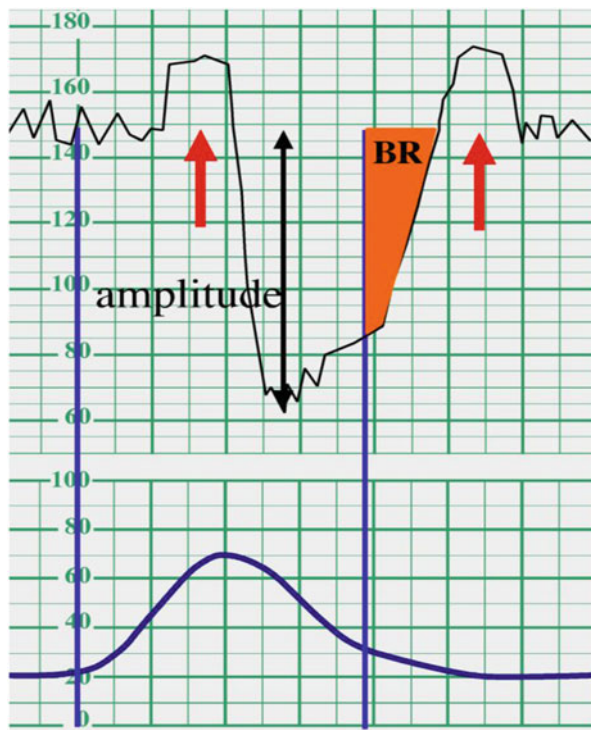


Fig. 10.4 Variable deceleration

- Visually apparent abrupt decrease in FHR
- An abrupt FHR decrease is defined as from the onset of the deceleration to the beginning of the FHR nadir of <30 s
- The decrease in FHR is recorded from the onset to the nadir of the deceleration
- The decrease in FHR is 15 bpm or greater, lasting 15 s or greater, and <2 min in duration
- When variable decelerations are associated with uterine contractions, their onset, depth, and duration commonly vary with successive uterine contractions



published between 1977 and 2003 specifically describe FHR patterns 30–60 min after injection of epidural bupivacaine. The incidence of total decelerations described in these studies varies from 8 % to 70 % within the first 60 min after epidural analgesia. Jouppila observed that 8 % of 105 women given 0.5 % bupivacaine to initiate epidural analgesia developed FHR decelerations within 30 min of anesthetic drug injection [16]. Lieberman studied 59 women given 0.375 % bupivacaine epidural analgesia and observed late decelerations in 14 % within 60 min of the injection [4]. Stavrou studied only the incidence of prolonged decelerations in the 60 min after epidural injection in 366 women and observed such decelerations in 11 % of women [17]. They reported that prolonged decelerations usually occurred within 5–10 min of epidural injection. Nielsen reported a 23.4 % incidence of late or prolonged decelerations within 60 min in 129 women who received 0.25 % bupivacaine epidural analgesia [18]. Most recently, Eberle reported that if variable decelerations were included, up to 70 % of women given epidural analgesia demonstrated FHR decelerations within 40 min of epidural injection [19]. Wolfler [20] analyzed FHR changes after epidural analgesia with ropivacaine and sufentanil and showed that epidural analgesia with ropivacaine and sufentanil was associated with transient FHR changes, without any modification in the FHR baseline. All the changes described were significantly more frequent in the first 30 min after analgesia, whereas in the following

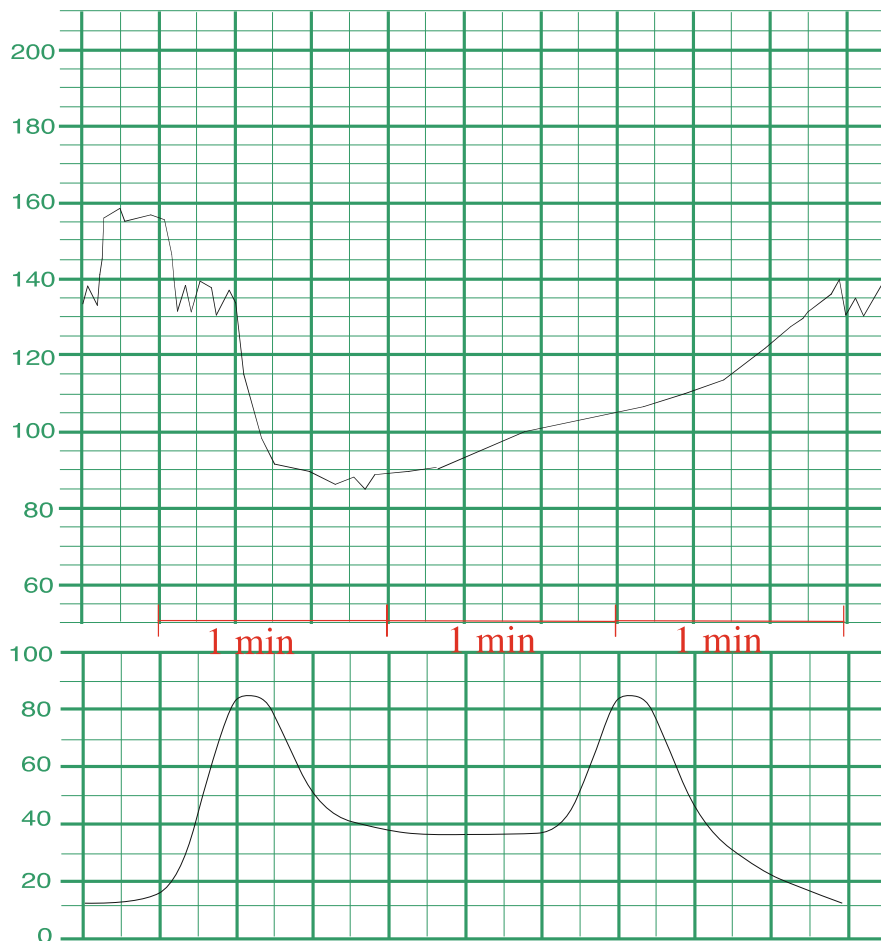


Fig. 10.5 Prolonged deceleration

- Visually apparent decrease in the FHR below the baseline
- Decrease in FHR from the baseline that is 15 bpm or more, lasting 2 min or more but <10 min in duration

60 min, even without a complete return to pre-analgesia conditions, an increase in the number of traces with accelerations and good long-term variability were observed. Decelerations were present in all trace segments after analgesia but with decreased frequency.

Late FHR decelerations attributable to epidural-induced utero-placental hypoperfusion are uncommon. In a recent French retrospective study analyzing 6,676 patients who underwent epidural analgesia, 760 (14 %) presented anomalies of FHR in the hour following the initiation of the epidural [21]. Among these anomalies, 319 (42 %) showed prolonged decelerations, 169 (22 %) variable decelerations, 122 (16 %) early decelerations, 110 (14 %) late decelerations, and

40 bradycardia (5 %). These anomalies occurred most often (73 %), while the FHR pattern prior to installation of the epidural was normal. Epidural analgesia may also deteriorate already abnormal FHR.

Some studies have suggested that there may be an increase in the frequency of non-reassuring patterns in the fetal heart rate, particularly bradycardia, with combined spinal–epidural analgesia. Intrathecal use of a large amount of lipophilic narcotics, especially sufentanil, may be associated with a small but increased incidence of profound fetal bradycardia within 60 min of administration and an increased risk of cesarean delivery for non-reassuring fetal status. This question remains open, since different studies report different results [3, 18, 22–27]. The only recommendation is probably to reduce the lipophilic opioid administered in the intrathecal part of the CSE technique to the minimal amount.

The physiological mechanisms of these abnormalities are still partly unknown, but may be mainly related to maternal hypotension due to the association between excessive sympathetic block and aorto-caval compression. Other effects related to neuraxial block-induced analgesia may also be involved. Pain relief leads to a decrease in output of the sympathetic nervous system (effective labor analgesia leads to a decrease in circulating epinephrine levels). Decreasing epinephrine, which is a tocolytic, will cause an increase in uterine tone, which may decrease placental blood flow. If placental blood flow is decreased enough, there will be a subsequent fetal bradycardia or FHR changes [28–30]. In addition, Cascio et al. showed that a faster decrease in plasma epinephrine in parturients who received spinal opioids when compared to epidural bupivacaine was observed, which offers a possible explanation for the faster onset of FHR changes in CSE compared to epidural analgesia [30]. To summarize, the rapid, reduced maternal production of epinephrine associated with a rapid onset analgesia, such as that observed with CSE, and/or a profound analgesic block, such as that obtained by the “old-fashioned” high-dose epidural analgesia, could indeed induce basal uterine hypertonus, which, combined with decreased UPP, could contribute to determine the transient FHR changes observed immediately after effective labor analgesia.

In all cases, changes in the FHR pattern after neuraxial analgesia are commonly reversible after the first 60 min and should not affect the fetal outcome or induce the obstetrician to perform an operative delivery.

10.5 International Guidelines for Maternal and Fetal Monitoring During Neuraxial Analgesia

Scientific societies and professional colleges have produced guidelines on intrapartum fetal surveillance that apply during initiation and maintenance of neuraxial analgesia [9, 16–18]. The goal of intrapartum fetal surveillance is to assure fetal well-being during labor, mainly by detecting maternal hypotension and significant fetal heart rate (FHR) abnormalities that would lead to a subsequent intervention to prevent fetal neurologic injury and death.

FHR monitoring was introduced in the 1960s to decrease the incidence of neonatal encephalopathy related to peri-partum hypoxic events. FHR monitoring is based on the ultrasonic fetal heart movement detection and coupled with external uterine contraction detectors. The quality of recording is very important for correct interpretation, and whenever there is a poor outcome for the neonates, the paper record acts as a powerful aid to any party wishing to demonstrate substandard antepartum care, i.e., at the time of neuraxial analgesia initiation.

According to the American College of Obstetricians and Gynecologists (ACOG), FHR patterns should always be interpreted according to the clinical situation and to the presence of maternal and/or fetal morbidities (preeclampsia, diabetes mellitus, prior cesarean delivery, prematurity, fetal growth restriction, . . .) [15]. Given that the available data do not show a clear benefit for the continuous use of electronic FHR monitoring, intermittent auscultation may be, however, another reasonable option in the context of uncomplicated labor for the ACOG, the Royal College of Obstetricians and Gynaecologists (RCOG), the Society of Obstetricians and Gynecologists of Canada, as well as the Royal Australian and New Zealand College of Obstetricians and Gynaecologists (RANZCOG). These four societies recommend, however, the use of continuous monitoring in women with high-risk conditions. The French Society is the only society to recommend the systematic use of FHR continuous surveillance during the active phase of labor in all parturients under neuraxial analgesia, mainly because of the French organization of care, with a low number of midwives per parturient [31].

The different national guidelines are also not in total agreement regarding FHR assessment during labor and during the initiation and maintenance of neuraxial analgesia. Fetal monitoring during neuraxial analgesia is not specified in the ACOG guidelines, but epidural analgesia and combined spinal epidural analgesia are identified as techniques affecting FHR. The 2007 Practice guidelines for Obstetric Anaesthesia state “the FHR should be monitored by a qualified individual before and after the administration of neuraxial analgesia for labor” [32]. “Continuous electronic recording of the FHR may not be necessary in every clinical setting and may not be possible during initiation of neuraxial anesthesia” is also mentioned. The French College of Obstetricians and Gynaecologists recommends that FHR should be systematically recorded before the performance of any kind of labor analgesia (professional agreement). The French Society of Anaesthesiology (SFAR) added that FHR monitoring should be maintained during the performance of neuraxial labor analgesia (professional agreement), even if its surveillance and interpretation remain fully under the responsibility of the obstetric team. In the UK, the NICE guidelines are similar, recommending “a continuous electronic fetal monitoring for at least 30 min during the establishment of regional analgesia and after the administration of each further bolus” [33].

To limit the increase of operative delivery due to abnormal FHR, including those which could be related to neuraxial analgesia, second-line techniques have been developed: fetal scalp pH or lactate measurements, fetal ECG waveform analysis (ST segment analyzer—STAN system), computerized FHR pattern analysis, and fetal pulse oximetry. Among all these methods, fetal scalp pH is the most used, as it

directly measures one criteria of fetal asphyxia. However, this is a complex procedure, uncomfortable for the woman and obstetrician alike, and so it has never been consistently employed worldwide, even though controlled trials have shown that it can limit the increase in cesarean section rates associated with not reassuring FHR tracing. However, any improvement in clinical fetal outcomes with fetal scalp pH measurement has never been demonstrated. Measuring lactate instead of pH is technically easier, but still invasive and has no other advantage over pH measurement.

In search of a less invasive replacement for fetal blood sampling, ECG waveform analysis came to the forefront. An electrode is placed on the fetal scalp to acquire fetal ECG. STAN computer analysis is based on the principle that fetal hypoxia will induce fetal ST or T wave changes secondary to catecholamine release. But appropriate interpretation of ST segment changes still requires correct FHR pattern interpretation. In addition, two recent meta-analyses conclude that the use of ST waveform analysis combined with FHR monitoring does not decrease the incidence of severe neonatal acidosis, Apgar score <7 at 5 min, neonatal encephalopathy, or the number of cesarean deliveries [34, 35]. Its use is not recommended in routine practice, neither by the Society of Obstetrics and Gynaecology of Canada nor by the French college of Obstetricians and Gynaecologists.

Pulse oximetry, using a probe placed on the fetal cheek, temple, back, or buttocks, was initially promising, but later studies have shown a poor specificity for acidosis. In 2004, a review reported that the use of fetal pulse oximetry in labor made no significant difference to any measure of outcome and several further prospective randomized controlled trials have confirmed that conclusion [36, 37]. It appears that fetal oxygen saturation is no better at predicting development of fetal acidosis than the detection of FHR late decelerations, and therefore, pulse oximetry can replace FHR monitoring but does not effectively enhance it. Because it is quite difficult to apply fetal oximetry probes to the fetus, and as the equipment is expensive, the adoption of pulse oximetry was discouraged by the ACOG and by the SOGC, and is not recommended in France.

Consequently, even if its specificity is low, FHR is still considered mandatory to detect non-reassuring or abnormal FHR patterns, in particular when associated with neuraxial analgesia initiation or maintenance.

10.6 Prevention of FHR Abnormalities Associated with Neuraxial Analgesia

Basically, the treatment of abnormal FHR associated with neuraxial labor analgesia is similar to that used for any FHR change which may occur in labor. The first step is to prevent maternal hypotension secondary to neuraxial analgesia, by slowly inducing the neuraxial block (titration and fractionation of local anesthetic administration) and by avoiding aorto-caval compression.

In a randomized trial, Preston et al. found a higher incidence of severe FHR decelerations when women were in the supine-lateral tilt position as compared to

women in the full lateral position immediately after epidural initiation [38]. Unfortunately, these results were not confirmed in two other randomized trials by Beilin and Eberle [19, 39].

Intravenous fluid loading before the initial dosing of labor neuraxial analgesia has also been proposed to prevent maternal hemodynamic alteration. But several randomized controlled trials showed that preloading as compared to no pre-hydration did not reduce the incidence of maternal hypotension and of FHR abnormalities [40, 41].

Prophylactic injection of ephedrine has been suggested, but its efficacy has never been proved. Ducros and colleagues observed that an IV bolus of ephedrine 18 mg restored uterine blood flow, even when the parturient had a normal blood pressure and without altering fetal hemodynamic parameters [42]. In a randomized controlled trial, Kreiser et al. found that a prophylactic injection of ephedrine at the time of epidural initiation significantly decreased the occurrence of FHR abnormalities without maternal adverse events [43]. But, in the context of combined spinal epidural, if the prophylactic administration of ephedrine significantly reduced the rates of maternal hypotension and of late fetal decelerations, it was also associated with fetal tachycardia. However, the rate of emergency cesarean delivery and neonatal outcomes were not significantly modified [44]. In the absence of positive fetal outcomes, the prophylactic use of ephedrine during epidural establishment is not recommended.

Pulse pressure is a hemodynamic parameter calculated by subtracting the diastolic blood pressure from the systolic blood pressure. It has been used as one of the first early predictors of intravascular fluid volume in trauma patients. A recent retrospective cohort study found a significant positive association between low maternal pulse pressure (<45 mmHg) at admission and the occurrence of FHR abnormalities in the first hour after initial dosing of a labor epidural, with an adjusted odds ratio of 29 [45]. This study suggests that maternal admission pulse pressure could be used as a predictor of new onset post-epidural FHR abnormalities. This technique is not currently used in routine practice nor recommended by international guidelines.

Concerning the effects of maternal oxygen administration on the fetus during labor, according to a systematic Cochrane Collaboration review, there is not enough evidence to support the use of prophylactic oxygen therapy for women in labor, nor to evaluate its effectiveness for fetal distress. Interestingly, in this review abnormal cord blood pH values (<7.2) were recorded significantly more frequently in the oxygenation group than the control group (RR 3.5, 95 % CI 1.3–9.1) [46].

10.7 Management of FHR Abnormalities Associated with Neuraxial Analgesia

In the case of FHR abnormal patterns, especially within the first 60 min following initiation of neuraxial analgesia, different measures of intrauterine resuscitation could be undertaken, all with the same objective: to reverse fetal hypoxia and

acidosis by increasing oxygen delivery to the fetus, by increasing maternal blood flow to the placenta, and/or increasing the oxygen content in the blood being delivered. These different measures can be used in isolation or in association; they are not specific to the context of neuraxial analgesia. Many clinicians use a stepwise method from the less invasive, moving forward until there is a resolution or plans are under way for expeditious birth. Lateral positioning is often the first intervention, followed by the discontinuation of the oxytocin IV infusion and IV fluid bolus, while medications such as oxygen, ephedrine, or terbutaline are used if the pattern does not resolve with the first-line results.

Lateral positioning prevents aorto-caval compression and supine hypotensive syndrome by increasing maternal venous return and cardiac output and ultimately uterine output. Some reports show an improvement in abnormal FHR patterns if the supine or other at-risk positions are changed to left lateral [47, 48]. This occurs even if maternal systemic blood pressure is normal, because of the relief of aortic compression. Fetal oxygen saturation has been correlated with this improvement.

In the context of abnormal FHR patterns, an IV fluid bolus is commonly administered, whether or not the mother has clinical symptoms of hypovolemia or hypotension. There is some literature favoring the use of IV crystalloid administration in the context of FHR abnormalities. Animal studies have shown that boluses of IV crystalloids increased the maternal arterial blood pressure and placental site blood flow and decreased vascular resistance [49]. A randomized controlled trial performed in healthy parturients demonstrated that a bolus of Lactated Ringer solution significantly increased fetal oxygen saturation and this increase was even higher with 1,000 ml of RL as compared to 500 ml [50].

On the other hand, vasopressors are frequently used in the case of neuraxial analgesia-induced hypotension, in particular if additional IV crystalloid and lateral maternal positioning did not result in the prompt restoration of blood pressure or in the context of severe hypotension. Traditionally, ephedrine 3–6 mg is administered to the mother.

According to the ACOG, in the context of FHR abnormalities associated with maternal hypotension, blood volume expansion or IV ephedrine or both is warranted, depending on the severity, and ephedrine is the agent traditionally administered during labor [15].

Another aspect of intrauterine resuscitation is to reduce uterine activity. Uterine contractions produce an intermittent decrease of blood flow to the intervillous space, where the oxygen exchange occurs. If the intermittent interruption of blood flow reaches an abnormal level as a result of too frequent contractions, the fetus becomes at risk of hypoxemia. Consequently, in the case of excessive contractions associated with non-reassuring FHR patterns, a reduction of uterine activity may improve fetal oxygenation. Stopping oxytocin infusion is the first immediate option and using tocolytic agents may be a second option. Three randomized controlled trials have studied the efficacy of active tocolysis versus placebo in the context of FHR abnormal patterns [51–53]. The meta-analysis of the results showed that compared to no treatment, the administration of a tocolytic agent (terbutaline, magnesium sulfate, or hexoprenaline) improves the FHR tracing,

but without any significant differences in neonatal outcomes [51]. Moreover, tocolytic agents can induce potential maternal adverse effects. Consequently, these agents should be used only in the case of non-reassuring FHR tracings associated with actual and measured uterine hyperstimulation. A randomized controlled trial from Pullen et al. compared the efficiency of terbutaline and nitroglycerin for acute intra-partum fetal resuscitation [54]. No difference was noticed between the two groups in the successful acute treatment of non-reassuring FHR tracings in labor. Due to the lack of evidence, there is no specific drug recommended as a tocolytic agent in the case of abnormal FHR patterns associated with uterine hypertonia.

Finally, the last aspect of intrauterine fetal resuscitation is maternal oxygen administration. Supplementary maternal oxygen during labor is widely accepted in the management of abnormal FHR patterns; however, its efficiency on fetal well-being is still very controversial. Indeed, both hypoxia and hyperoxia can result in the production of oxygen free radicals, which can cause oxidative stress and subsequent adverse effects such as damage to cell membranes, cell structures, cellular lipoproteins, and DNA. Prolonged hyperoxia may also induce placental vasoconstriction.

In a prospective study, Haydon et al. showed a significant increase in fetal oxygen saturation with 40 % and then 100 % FIO_2 when compared to a baseline value in a fetus with abnormal FHR patterns [55]. The highest increase in fetal oxygen saturation was observed in a fetus with the lowest initial oxygen saturation. Of note, there was no consistent change in FHR patterns after exposure to oxygen. Another randomized controlled trial compared air versus maternal oxygen administration (FiO_2 60 %) during emergency cesarean section [56]. A significant increase in indicators of fetal oxygenation was observed with oxygen administration, such as venous and arterial umbilical oxyhemoglobin saturation and oxygen content. This effect was of greater magnitude in patients with suspected fetal compromise. In contrast to the previous works with elective cesarean, there was no evidence that free radical activity was greater in patients who received oxygen. Although this study showed no difference in clinical outcome from administering oxygen, there was no evidence of harm. Further work is required to determine whether the increase in fetal oxygenation resulting from maternal administration of supplementary oxygen has a beneficial effect on the clinical outcome in the compromised fetus.

To summarize on oxygen administration as part of intrauterine resuscitation, it may be beneficial for the fetus, but it may also be potentially risky. Consequently, it should be used as a second-line treatment in intrauterine resuscitation, except in the case of sudden acute fetal deterioration, where all means are set up at the same time, and it seems to be safer to stop maternal oxygen administration as soon as possible.

To conclude on the management of non-reassuring FHR patterns, whether associated with neuraxial analgesia or not, the usual techniques of intrauterine resuscitation should be applied, even if there is a lack of evidence to support these measures. Randomized controlled trials are obviously very difficult to perform in this context, and therefore, there is no recommended standardized or

systematic approach. The more logical strategy appears to be to choose the initial intervention according to the whole clinical picture, i.e., to correct a maternal hypotension or a uterine hypertonia associated with neuraxial analgesia.

References

1. Eltzschig HK, Lieberman ES, Camann WR (2003) Regional anesthesia and analgesia for labor and delivery. *N Engl J Med* 348(4):319–332
2. Arnaout L, Ghiglione S, Figueiredo S, Mignon A (2008) Effects of maternal analgesia and anesthesia on the fetus and the newborn. *J Gynécologie Obstétrique Biol Reprod* 37(Suppl 1): S46–S55
3. Gambling DR, Sharma SK, Ramin SM, Lucas MJ, Leveno KJ, Wiley J et al (1998) A randomized study of combined spinal-epidural analgesia versus intravenous meperidine during labor: impact on cesarean delivery rate. *Anesthesiology* 89(6):1336–1344
4. Lieberman BA, Rosenblatt DB, Belsey E, Packer M, Redshaw M, Mills M et al (1979) The effects of maternally administered pethidine or epidural bupivacaine on the fetus and newborn. *Br J Obstet Gynaecol* 86(8):598–606
5. Leighton BL, Halpern SH (2002) The effects of epidural analgesia on labor, maternal, and neonatal outcomes: a systematic review. *Am J Obstet Gynecol* 186(5 Suppl Nature):S69–S77
6. Reynolds F, Sharma SK, Seed PT (2002) Analgesia in labour and fetal acid-base balance: a meta-analysis comparing epidural with systemic opioid analgesia. *BJOG* 109(12):1344–1353
7. Sharma SK, Alexander JM, Messick G, Bloom SL, McIntire DD, Wiley J et al (2002) Cesarean delivery: a randomized trial of epidural analgesia versus intravenous meperidine analgesia during labor in nulliparous women. *Anesthesiology* 96(3):546–551
8. Wong CA, Scavone BM, Peaceman AM, McCarthy RJ, Sullivan JT, Diaz NT et al (2005) The risk of cesarean delivery with neuraxial analgesia given early versus late in labor. *N Engl J Med* 352(7):655–665
9. Berman W, Goodlin RC, Heymann MA, Rudolph AM (1976) Relationships between pressure and flow in the umbilical and uterine circulations of the sheep. *Circ Res* 38(4):262–266
10. Westgren M, Lindahl SG, Nordén NE (1986) Maternal and fetal endocrine stress response at vaginal delivery with and without an epidural block. *J Perinat Med* 14(4):235–241
11. Umstad MP (1993) The predictive value of abnormal fetal heart rate patterns in early labour. *Aust N Z J Obstet Gynaecol* 33(2):145–149
12. Grant A, O'Brien N, Joy MT, Hennessy E, MacDonald D (1989) Cerebral palsy among children born during the Dublin randomised trial of intrapartum monitoring. *Lancet* 2 (8674):1233–1236
13. Thacker SB, Stroup DF, Peterson HB (1998) Intrapartum electronic fetal monitoring: data for clinical decisions. *Clin Obstet Gynecol* 41(2):362–368
14. Alfirevic Z, Devane D, Gyte GML (2013) Continuous cardiotocography (CTG) as a form of electronic fetal monitoring (EFM) for fetal assessment during labour. *Cochrane Database Syst Rev* 5:CD006066
15. American College of Obstetricians and Gynecologists (2009) ACOG Practice Bulletin No. 106: intrapartum fetal heart rate monitoring: nomenclature, interpretation, and general management principles. *Obstet Gynecol* 114(1):192–202
16. Jouppila P, Jouppila R, Käär K, Merilä M (1977) Fetal heart rate patterns and uterine activity after segmental epidural analgesia. *Br J Obstet Gynaecol* 84(7):481–486
17. Stavrou C, Hofmeyr GJ, Boezaart AP (1990) Prolonged fetal bradycardia during epidural analgesia. Incidence, timing and significance. *South Afr Med J Suid-Afr Tydskr Vir Geneesk* 77(2):66–68

18. Nielsen PE, Erickson JR, Abouleish EI, Perriatt S, Sheppard C (1996) Fetal heart rate changes after intrathecal sufentanil or epidural bupivacaine for labor analgesia: incidence and clinical significance. *Anesth Analg* 83(4):742–746
19. Eberle RL, Norris MC, Eberle AM, Naulty JS, Arkoosh VA (1998) The effect of maternal position on fetal heart rate during epidural or intrathecal labor analgesia. *Am J Obstet Gynecol* 179(1):150–155
20. Wolfer A, Salvo I, Sortino G, Bonati F, Izzo F (2010) Epidural analgesia with ropivacaine and sufentanil is associated with transient fetal heart rate changes. *Minerva Anestesiol* 76(5):340–345
21. Korb D, Bonnin M, Michel J, Oury J-F, Sibony O (2013) Analysis of fetal heart rate abnormalities occurring within one hour after laying of epidural analgesia. *J Gynécologie Obstétrique Biol Reprod* 42(6):564–569
22. Van de Velde M, Teunkens A, Hanssens M, Vandermeersch E, Verhaeghe J (2004) Intrathecal sufentanil and fetal heart rate abnormalities: a double-blind, double placebo-controlled trial comparing two forms of combined spinal epidural analgesia with epidural analgesia in labor. *Anesth Analg* 98(4):1153–1159, table of contents
23. Nicolet J, Miller A, Kaufman I, Guertin MC, Deschamps A (2008) Maternal factors implicated in fetal bradycardia after combined spinal epidural for labour pain. *Eur J Anaesthesiol* 25(9):721–725
24. Palmer CM, Maciulla JE, Cork RC, Nogami WM, Gossler K, Alves D (1999) The incidence of fetal heart rate changes after intrathecal fentanyl labor analgesia. *Anesth Analg* 88(3):577–581
25. Patel NP, Armstrong SL, Fernando R, Columb MO, Bray JK, Sodhi V et al (2012) Combined spinal epidural vs epidural labour analgesia: does initial intrathecal analgesia reduce the subsequent minimum local analgesic concentration of epidural bupivacaine? *Anaesthesia* 67(6):584–593
26. Patel NP, El-Wahab N, Fernando R, Wilson S, Robson SC, Columb MO et al (2014) Fetal effects of combined spinal-epidural vs epidural labour analgesia: a prospective, randomised double-blind study. *Anaesthesia* 69(5):458–467
27. Preston R (2007) The role of combined spinal epidural analgesia for labour: is there still a question? *Can J Anaesth* 54(1):9–14
28. Clarke VT, Smiley RM, Finster M (1994) Uterine hyperactivity after intrathecal injection of fentanyl for analgesia during labor: a cause of fetal bradycardia? *Anesthesiology* 81(4):1083
29. Segal S, Csavoy AN, Datta S (1998) The tocolytic effect of catecholamines in the gravid rat uterus. *Anesth Analg* 87(4):864–869
30. Cascio M, Pygon B, Bennett C, Ramanathan S (1997) Labour analgesia with intrathecal fentanyl decreases maternal stress. *Can J Anaesth* 44(6):605–609
31. Collège National des Gynécologues et Obstétriciens Français (2007) Recommandations pour la Pratique Clinique: Modalités de surveillance foetale pendant le travail [Internet]. http://www.cngof.asso.fr/D_PAGES/PURPC_18.HTM
32. American Society of Anesthesiologists Task Force on Obstetric Anesthesia (2007) Practice guidelines for obstetric anesthesia: an updated report by the American Society of Anesthesiologists Task Force on Obstetric Anesthesia. *Anesthesiology* 106(4):843–863
33. National Collaborating Centre for Women's and Children's Health (UK) (2007) Intrapartum care: care of healthy women and their babies during childbirth [Internet]. RCOG, London [cited 2014]. <http://www.ncbi.nlm.nih.gov/books/NBK49388/>
34. Salmelin A, Wiklund I, Bottinga R, Brorsson B, Ekman-Ordeberg G, Grimfors EE et al (2013) Fetal monitoring with computerized ST analysis during labor: a systematic review and meta-analysis. *Acta Obstet Gynecol Scand* 92(1):28–39
35. Neilson JP (2012) Fetal electrocardiogram (ECG) for fetal monitoring during labour. *Cochrane Database Syst Rev* 4:CD000116
36. Bloom SL, Spong CY, Thom E, Varner MW, Rouse DJ, Weinger S et al (2006) Fetal pulse oximetry and cesarean delivery. *N Engl J Med* 355(21):2195–2202

37. East CE, Leader LR, Sheehan P, Henshall NE, Colditz PB (2010) Intrapartum fetal scalp lactate sampling for fetal assessment in the presence of a non-reassuring fetal heart rate trace. *Cochrane Database Syst Rev* (3):CD006174
38. Preston R, Crosby ET, Kotarba D, Dudas H, Elliott RD (1993) Maternal positioning affects fetal heart rate changes after epidural analgesia for labour. *Can J Anaesth* 40(12):1136–1141
39. Beilin Y, Abramovitz SE, Zahn J, Enis S, Hossain S (2000) Improved epidural analgesia in the parturient in the 30 degree tilt position. *Can J Anaesth* 47(12):1176–1181
40. Kinsella SM, Pirllet M, Mills MS, Tuckey JP, Thomas TA (2000) Randomized study of intravenous fluid preload before epidural analgesia during labour. *Br J Anaesth* 85(2):311–313
41. Kubli M, Shennan AH, Seed PT, O'Sullivan G (2003) A randomised controlled trial of fluid pre-loading before low dose epidural analgesia for labour. *Int J Obstet Anesth* 12(4):256–260
42. Ducros L, Bonnin P, Cholley BP, Vicaut E, Benayed M, Jacob D et al (2002) Increasing maternal blood pressure with ephedrine increases uterine artery blood flow velocity during uterine contraction. *Anesthesiology* 96(3):612–616
43. Kreiser D, Katorza E, Seidman DS, Etchin A, Schiff E (2004) The effect of ephedrine on intrapartum fetal heart rate after epidural analgesia. *Obstet Gynecol* 104(6):1277–1281
44. Cleary-Goldman J, Negron M, Scott J, Downing RA, Camann W, Simpson L et al (2005) Prophylactic ephedrine and combined spinal epidural: maternal blood pressure and fetal heart rate patterns. *Obstet Gynecol* 106(3):466–472
45. Miller NR, Cypher RL, Nielsen PE, Foglia LM (2013) Maternal pulse pressure at admission is a risk factor for fetal heart rate changes after initial dosing of a labor epidural: a retrospective cohort study. *Am J Obstet Gynecol* 209(4):382.e1–382.e8
46. Fawole B, Hofmeyr GJ (2012) Maternal oxygen administration for fetal distress. *Cochrane Database Syst Rev* 12:CD000136
47. Carbonne B, Benachi A, Lévêque ML, Cabrol D, Papiernik E (1996) Maternal position during labor: effects on fetal oxygen saturation measured by pulse oximetry. *Obstet Gynecol* 88(5):797–800
48. Abitbol MM (1985) Supine position in labor and associated fetal heart rate changes. *Obstet Gynecol* 65(4):481–486
49. Crino JP, Harris AP, Parisi VM, Johnson TR (1993) Effect of rapid intravenous crystalloid infusion on uteroplacental blood flow and placental implantation-site oxygen delivery in the pregnant ewe. *Am J Obstet Gynecol* 168(5):1603–1609
50. Simpson KR, James DC (2005) Efficacy of intrauterine resuscitation techniques in improving fetal oxygen status during labor. *Obstet Gynecol* 105(6):1362–1368
51. Kulier R, Gülmezoglu AM, Hofmeyr GJ, Van Gelderen CJ (1997) Betamimetics in fetal distress: randomised controlled trial. *J Perinat Med* 25(1):97–100
52. Magann EF, Cleveland RS, Dockery JR, Chauhan SP, Martin JN, Morrison JC (1993) Acute tocolysis for fetal distress: terbutaline versus magnesium sulphate. *Aust N Z J Obstet Gynaecol* 33(4):362–364
53. Patriarco MS, Viechnicki BM, Hutchinson TA, Klasko SK, Yeh SY (1987) A study on intrauterine fetal resuscitation with terbutaline. *Am J Obstet Gynecol* 157(2):384–387
54. Pullen KM, Riley ET, Waller SA, Taylor L, Caughey AB, Druzin ML et al (2007) Randomized comparison of intravenous terbutaline vs nitroglycerin for acute intrapartum fetal resuscitation. *Am J Obstet Gynecol* 197(4):414.e1–414.e6
55. Haydon ML, Gorenberg DM, Nageotte MP, Ghamsary M, Rumney PJ, Patillo C et al (2006) The effect of maternal oxygen administration on fetal pulse oximetry during labor in fetuses with nonreassuring fetal heart rate patterns. *Am J Obstet Gynecol* 195(3):735–738
56. Khaw KS, Wang CC, Ngan Kee WD, Tam WH, Ng FF, Critchley LA et al (2009) Supplementary oxygen for emergency Caesarean section under regional anaesthesia. *Br J Anaesth* 102(1):90–96

Nutrition and Fluid Balance During Labor; Maternal Optimization and Fetal Resuscitation

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11.1 Introduction

Medicalization of the process of labor over a century has led to the imposition of many practices on women with regard to eating and drinking; many of these lack any evidence as well as logic by our current standards. Since Mendelson published his classic description of pulmonary aspiration in 1946 [1], it has become common practice to restrict oral intake during labor in order to reduce the risk of maternal mortality and morbidity from pulmonary aspiration of stomach contents should a general anesthetic be required. This accords both with the potential for unanticipated anesthesia during childbirth, as well as the physiological changes in pregnancy that are evident most clearly in the frequent occurrence of severe vomiting during labor.

11.2 Changes in Gastrointestinal Physiology During Pregnancy

As with all other body systems, pregnancy has profound effects on the gastrointestinal tract mediated through high levels of female steroid hormones (Table 11.1). Gastrointestinal symptoms including nausea and vomiting, gastro-esophageal reflux, and constipation are so frequent that they are considered a “normal” aspect of pregnancy.

Almost 70 % of women experience nausea and vomiting in pregnancy [2]. - Thirty-three percent of women have nausea without vomiting, while the life-threatening condition of hyperemesis gravidarum affects 1 %. Symptoms typically start between the 4th and 9th week of pregnancy and persist until the 12th to 16th week, although in 20 % of pregnant women symptoms persist throughout

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Table 11.1 Effects of pregnancy and labor on gastrointestinal physiology

| | Pregnancy | Labor | Postpartum |
|-------------------------|---------------------------------------------------------|-----------------------------------------|--------------------------------------------|
| Gastric motility | Reduced tone and motility | Reduced | Returns to normal by 2 days |
| Gastric emptying | Unchanged | Unchanged | Unchanged |
| Intestinal transit time | Decreased tone and motility with increased transit time | Prolonged | Returned to normal |
| Plasma osmolality | Reduced by approx 10 mOsmol | Reduced, may be exacerbated by oxytocin | Returned to prepregnancy values by 2 weeks |

pregnancy. The exact cause is unknown, but estrogen and human chorionic gonadotrophin are thought to play a major role.

Although progesterone results in reduced motility and hypotonia of the stomach, normal pregnancy does not alter gastric emptying. Reflux of alkaline duodenal contents into the stomach may occur due to incompetence of the pyloric sphincter.

Studies have shown no significant alterations in gastrointestinal transit time during the first trimester. However, there is an increase in the intestinal transit time in the third trimester which returns to normal postpartum. This slowing of intestinal transit time is thought to be hormonally mediated, notably by progesterone and estrogen acting to inhibit smooth muscle contraction.

The reduced motility and prolonged transit time of the large intestine result in increased water and sodium reabsorption and contribute to symptoms of constipation. This is compounded by dietary and exercise or activity changes plus compression of the bowel by the enlarging uterus.

Gastro-esophageal reflux disease (GORD) usually presents with heartburn and reflux; other symptoms include water brash, epigastric pain, anorexia, nausea, vomiting, and indigestion. Extra-esophageal symptoms include hoarseness, chronic laryngitis, chronic cough, and asthma. In severe cases, GORD can contribute to poor nutritional status in the mother and low birth weight in the newborn baby. The prevalence of heartburn increases with gestational age from 22 % in the first, 39 % in the second, and 72 % in the third trimester. Besides gestational age, an increased risk of heartburn is associated with prepregnancy heartburn and increased parity and a decreased risk with maternal age; body mass index before pregnancy, race, or weight gain in pregnancy do not affect the risk. Severity as well as prevalence of heartburn increases during pregnancy, especially toward the end of the second trimester. The decrease in heartburn traditionally expected during the last 3 weeks of pregnancy due to fetal head descent has not been observed in more recent studies. The pathophysiology of GORD in pregnancy is multifactorial, with causes including decreased lower esophageal sphincter pressure, changes in pressure gradients across the lower esophageal sphincter, prolonged gastrointestinal transit time, and increased intra-abdominal pressure secondary to the enlarged gravid uterus.

There are conflicting results on changes in gastric acidity during pregnancy, but the general trend appears to be toward a small reduction in acidity during the first and second trimesters, with an increase to greater than prepregnant values in the third trimester. Women with peptic ulcer disease often have an improvement in their symptoms during pregnancy. The initial decrease in acidity is thought to be hormonally (especially estrogen) mediated and also due to elevated levels of placental histaminase.

11.3 Fluid Balance and Osmolality During Pregnancy

A reduction in serum osmolality in normal pregnancy occurs from the fifth week of gestation, reaching 8–10 mOsm/kg below nonpregnant values by 10 weeks of gestation and remaining low until term. The osmolality set point for antidiuretic hormone release and thirst is also decreased. Both extracellular and intravascular volumes increase during pregnancy, with a larger expansion in the vascular component favoring placental perfusion. The exact mechanisms for water retention in pregnancy remain unclear; an increase in plasma volume occurs despite decreases in plasma osmolality and colloid osmotic pressure that would normally stimulate a feedback correction through antidiuretic hormone and aldosterone release. Estrogen and progesterone may play a role via dilation of venous capacitance vessels, which then accommodate additional volume without the stimulation of atrial baroreceptors.

In the first two trimesters, the pregnant woman manages fluid efficiently and does not display clinically obvious edema despite the lowered colloid osmotic pressure and normal blood pressure. Conversely, in the third trimester, most women have dependent edema due to a reduced diuretic response and increased venous pressure from the gravid uterus.

11.4 The Effects of Labor

The main change during labor is the slowing of stomach emptying. This may be caused by the stimulation of the sympathetic nervous system through stress, pain, and anxiety, although endogenous opioids may also have a role. The effect of administered opioids, whether systemic or epidural, is clear. Gastric emptying is markedly delayed in women given pethidine, diamorphine, or pentazocine; this effect is not reversed by metoclopramide [3]. Epidural opioids also prolong gastric emptying [4], although to a lesser degree than systemic administration [5] and possibly in a dose-dependent manner [6]. A recent study in spontaneously laboring women using gastric ultrasound has demonstrated that some stomach emptying does occur after the epidural administration of ropivacaine and sufentanil. Fifty percent of women had a gastric antral cross-sectional area of $>320 \text{ mm}^2$ (taken as the cutoff value for a risk of regurgitation and pulmonary aspiration) at epidural insertion, whereas only 13 % of women had a measurement of $>320 \text{ mm}^2$ on

reaching full dilation of the cervix. Of note, women were not allowed to drink during labor [7].

In an observational study of women who were allowed unrestricted oral intake during labor, women tended to eat during early and active labor but not as labor progressed. Nineteen percent vomited, but none experienced a poor outcome [8].

A series of randomised studies investigated the effect of food and carbohydrate drinks during labor. In the first, women who had a standardized carbohydrate meal had increased residual gastric volume compared to women who had water only. The incidence of vomiting in the eating group was 17 % compared to 19 % in the water-only group. Women who ate also had significantly larger vomit volumes than those who had water only. However, there was an increased incidence of ketosis in the water-only group [9]. A subsequent study compared women having carbohydrate drinks to those having water only. There was no difference in residual gastric volume between the two groups, but carbohydrate drinks prevented the onset of ketosis seen in the water-only group. Other outcomes were also comparable [10].

The effects of carbohydrate drinks compared to water on labor outcome are conflicting; Scheepers et al. found no beneficial effect following the consumption of carbohydrate drinks and indeed observed a higher cesarean section rate in the carbohydrate drink group [11]. In contrast, Kubli et al. demonstrated no difference between carbohydrate drink and water groups in any maternal or neonatal outcome [10].

The significance of ketosis during labor is not clear. It is commonly found when food is restricted. There is an association between ketone levels and longer labor, but causation is not proven as relevant trials are not of sufficient quality [12]. Water retention during pregnancy is worsened during labor with increased adrenocortical and antidiuretic hormone (ADH) activity. This is further enhanced by oxytocin infusion used to stimulate contractions and has led to water intoxication when infused with 5 % glucose as a carrier [13, 14].

A Cochrane review attempted to answer whether oral intake affects maternal and fetal outcomes by assessing randomized controlled trials (RCTs) and quasi-RCTs of restricting fluids and food for women in labor compared with women free to eat and drink. Five studies were identified in women in active labor and at low risk of potentially requiring a general anesthetic. One study looked at complete restriction versus giving women the freedom to eat and drink at will; two studies looked at water only versus giving women specific fluids and foods; and two studies looked at water only versus giving women carbohydrate. There were no differences in the primary outcome measures of mode of delivery, maternal satisfaction, and a 5-min Apgar score <7. This review of over 3,000 women identified no benefits or risks of restricting oral fluids and foods in labor, in women at low risk of requiring general anesthesia. Maternal satisfaction and hypoglycemia, both also primary outcomes in this review, were not reported in any of the included studies. No women included in this review suffered from regurgitation during general anesthesia or Mendelson's syndrome [15].

Few studies have assessed the impact of dietary restriction on the woman herself. A postpartum survey of women found that a quarter found food restriction and over half found fluid restriction “moderate to most stressful” [16]. Other descriptors include “unpleasant” and “harrowing” [17, 18]. Denial of food can be seen as authoritarian and intimidating and increase feelings of apprehension. The importance of this aspect of woman’s autonomy, choice, and control in labor must be considered [19].

11.5 Policies: Oral Intake During Labor

Restriction of oral intake for laboring women followed fears about the risk of pulmonary aspiration in women who were unconscious. The incidence of pulmonary aspiration has declined over half a century for several reasons, which are likely to include increased use of regional anesthesia for intrapartum surgery, improved administration of general anesthesia including training of obstetric anesthesiologists, and widespread administration of H₂ receptor antagonists and proton pump inhibitors during labor.

There are large variations in practice regarding oral intake during labor across the globe and indeed within each nation, as indicated by the results of several surveys. In the Netherlands, 79 % of clinicians allow food intake in labor [20] and in the UK 47 % of women have “access” to food and drink in labor [21], whereas in the USA a survey found that more than 70 % of units allowed fluid but no food and intake is often limited to water or ice chips only [22].

A pragmatic approach to oral intake has become widespread in the UK. Women are characterized on the basis of risk factors into “low” and “high” risk groups (Table 11.2). High-risk women have food restricted, but are usually allowed water or carbohydrate drinks freely. High-risk women are also prescribed regular gastric acid secretion blockers [23].

11.6 Maintenance Intravenous Fluid

In institutions where oral intake is restricted during labor, intravenous fluids are routinely administered, although there is no consensus on the type or volume of fluids that are required. A Cochrane review has investigated the effect of intravenous fluid on the duration of labor. Two out of nine trials demonstrated a reduction in the duration of labor with the administration of intravenous fluids compared with oral intake alone. The duration of labor in nulliparous women may be shortened by the administration of intravenous fluids at a rate of 250 ml/h rather than 125 ml/h. One trial raised concerns about the safety of 5 % glucose infusion due to an increased risk of hyponatremia. The authors concluded that there was no robust evidence to recommend the routine administration of intravenous fluids to reduce the duration of labor [24].

Table 11.2 Antenatal and intrapartum risk factors and consequences

| | Chance of operative delivery | Chance of general anesthesia/loss of consciousness | Risk of general anesthesia/stomach contents | Risk of general anesthesia/airway control |
|-------------------------------------------------------------|--------------------------------------------------------|--------------------------------------------------------------------------|---------------------------------------------|-------------------------------------------|
| <i>Antenatal</i> | | | | |
| Trial of scar | ↑ | ↑ (scar dehiscence) | | |
| Raised Body mass index | ↑ | ↑ | ↑ | ↑ |
| Pre-eclampsia | ↑ (preterm induction, intrauterine growth retardation) | ↑ (regional anesthesia contraindications, e.g., low platelets/eclampsia) | | ↑ (supraglottic/tracheal edema) |
| Breech | ↑ | ↑ (entrapment of head) | | |
| Antepartum hemorrhage before admission | ↑ | | | |
| Poor obstetric history | ↑ | | | |
| Chronic fetal condition | ↑ | | | |
| Maternal comorbidity | +/- | +/- (regional anesthesia contraindications) | +/- | +/- |
| Twins | ↑ | ↑ (breech second twin) | | |
| Insulin dependent diabetes mellitus | ↑ | | ↑ (gastroparesis) | |
| <i>Intrapartum</i> | | | | |
| Failure to progress, persistent occipito-posterior position | ↑ | | | |
| Fetal compromise/fetal blood sampling | ↑ | ↑ (fetal bradycardia) | | |
| Antepartum hemorrhage/abruption during labor | ↑ | ↑ (fetal bradycardia) | | |
| Nonengaged fetal head | | ↑ (cord prolapse) | | |
| Opioid administration | | | ↑ | |

Intravenous fluids may also be administered in cumulatively large volumes when dilute solutions of oxytocin are used to induce or augment contractions, especially in nulliparous women. Because of the reduced serum osmolality in pregnancy and the antidiuretic effect of oxytocin, the use of large volumes of intravenous fluid has the potential to cause clinical problems. This has been of special concern in pre-eclampsia where increased pulmonary capillary permeability can lead to pulmonary edema.

The effects of rapid intravenous fluid infusion (bolus) are easier to measure than slow infusion, and these are sometimes significant and unexpected. A 10 ml/kg maternal weight fluid bolus produced increases in heart rate of 11 %, stroke volume 10 %, and cardiac output 20 % and a decrease in systemic vascular resistance of 19 %, but with no change in mean arterial pressure or ejection fraction. Uterine and umbilical artery Doppler systolic–diastolic ratios measured in between contractions did not change [25].

Intravenous fluid bolus also affects the frequency of uterine contractions. Zamora et al. compared women who had either 500 ml or 1 l preload before epidural analgesia was induced. More women had an increase in uterine contraction frequency by 30 min in the 500 ml group compared to the 1 l group; more women had a decrease in contraction frequency by 30 min in the 1 l group compared to the 500 ml group. These differences were sustained at 60 min as well [26].

Fetal oxygen saturation has been shown to be increased after intravenous fluid in a dose-dependent manner, with a greater effect after 1 l than after 500 ml. The effect peaked at 5 min postbolus, and an increase was maintained at 15 min postbolus [27]. The explanation for this effect might relate to reduced uterine activity; however, it is probable that other mechanisms are also active. In women having elective cesarean section, a 1 l intravenous fluid bolus led to reduced uterine and umbilical artery Doppler resistance indices (systolic/diastolic ratio). This may be an effect of circulating volume expansion or reduced blood viscosity [28].

11.7 Effect of Regional Analgesia

Regional analgesia for labor is associated with an increased risk of cesarean section for fetal distress, but a reduced risk of neonatal acidosis and no effect on Apgar scores [29]. How can this paradox be explained? The classical paradigm is that maternal hypotension that follows regional analgesia [29] reduces uteroplacental blood flow and hence causes acute fetal compromise. An alternative view is gaining more widespread support, however, based on a physiological re-evaluation. Because external monitoring of uterine contractions with a strain gauge is the current norm, the importance of the uterine diastolic period is not readily apparent; hypertonus is defined merely as excessive contraction frequency [30]. However, uteroplacental blood flow is analogous to left ventricular blood flow; it is not only cut off completely during systole but it also dependent on diastolic time (the intercontraction period) as well as diastolic perfusion pressure (determined by the gradient between arterial pressure and uterine baseline tone).

Epidural analgesia provides effective pain relief [29], and this leads to a return of temporarily elevated blood pressure toward the prelabor baseline [31]. However, a corollary of profound analgesia is a change in uterine activity and this change may depend on the speed of the onset of analgesia. Abrao et al. evaluated uterine baseline tone using an intrauterine pressure catheter in women having regional analgesia. Combined spinal–epidural (CSE) analgesia was associated with more fetal heart rate (FHR) abnormalities than epidural; this was linked to more frequent increase in baseline tone ≥ 10 mmHg in the CSE group, with an equal incidence of systemic hypotension between both groups. When all CSE and epidural cases were combined, they noted a correlation between the degree of analgesia at 5 min and the incidence of FHR abnormalities related to increased baseline tone [32]. A recent case–control study has also shown a correlation between pain relief after CSE and fetal bradycardia [33].

Although uterine diastolic perfusion may be the most important factor in this situation, another study has also shown an increase in uterine arterial resistance during contractions 30 min after epidural analgesia [34].

There is a short-term increase in FHR abnormalities after regional analgesia, most recently shown by Patel et al. [35]. This may lead to cesarean section for fetal compromise, especially if fetal scalp pH measurements are not available to establish whether there is associated acidemia. However, once uterine activity changes are established after regional analgesia, the beneficial effects in terms of reduced maternal catecholamines then start to prevail with a reduction in fetal acidemia noted at delivery [29].

11.8 The Role of Preload Before Regional Analgesia: Maternal and Fetal Effects

The first studies on preloading before regional anesthesia for cesarean section demonstrated dramatic reductions in the frequency of hypotension. However, the subsequent decades saw a reappraisal, with a meta-analysis showing that crystalloid preload reduces the relative risk of hypotension to 0.78, which is less effective than vasopressors or leg wrapping [36]. The early enthusiasm for this maneuver was carried across to regional analgesia for labor, with similarly large benefits seen initially that were not subsequently reproduced. The first paper to study preloading during labor after high-dose (0.25 %) bupivacaine was the only one that found clear benefits; a 1 l preload reduced hypotension (relative risk 0.07) and fetal heart rate abnormalities (relative risk 0.36) [37]. Studies since then have used dilute local anesthetics for epidural or CSE analgesia, with no difference in hypotension and a nonsignificant trend to reduction in FHR abnormalities [38].

11.9 Intrauterine Resuscitation: Improving Fetal Condition

Intrauterine fetal resuscitation (IUF) consists of measures undertaken with the aim of improving oxygen delivery to the fetus when there is acute fetal compromise (see guideline below).

| |
|---------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Intrauterine resuscitation guideline [39] |
| Resuscitate |
| Oxytocin infusion off |
| Position |
| Full left lateral; continue for transfer and on operating table (if FHR remains low try right lateral/knee elbow for possible cord compression) |
| Tocolysis |
| Terbutaline 0.25 mg subcutaneous |
| Alternatively for immediate action glyceryl trinitrate sublingual spray, two puffs initially, repeat after 1 min until contractions stop, maximum three doses |
| Oxygen |
| Maximum flow (15 l/min) via tight-fitting Hudson mask with reservoir bag |
| Fluid |
| Hartmann's 1 l rapid infusion (unless fluid intake restricted, e.g., pre-eclampsia) |
| Ephedrine |
| Consider if maternal blood pressure is low |
| <i>Transfer</i> |
| <i>Reassess</i> |
| Electronic fetal monitoring should be restarted in the operating room and maintained as long as possible |

There is a good physiological basis for IUF though evidence for changes in outcome is lacking [40]. IUF is performed in the most structured way before surgical delivery for fetal compromise. Acute fetal compromise is usually caused by two factors, either increased uterine activity or poor maternal position. These can both affect either side of the placental circulation. Contractions during active labor necessarily reduce uteroplacental blood flow, but placental and fetal “reserve” ensures that this is tolerated by the healthy fetus during normal labor. The umbilical cord can be compressed during contractions if it lies between the presenting part of the fetus and the maternal pelvic wall, typically causing variable decelerations. Reduction of contractions can be seen to be a logical step in this situation as contractions will cause or exacerbate most cases of acute fetal compromise. Induction of regional analgesia may also precipitate increased uterine activity in some cases, sometimes followed by FHR abnormalities, although this usually stabilises within 60 min.

During labor, poor maternal position may produce aortocaval compression that reduces uteroplacental blood flow; it may also cause sustained umbilical cord compression, again in an unpredictable fashion depending on the exact position of cord loops. The left lateral position is associated with the highest cardiac output while recumbent [41]. Semi-recumbent (“sitting up”) is also usually acceptable

although aortic compression is detectable in some cases [39]. However when the woman takes up a maximally flexed position for the insertion of a regional anesthetic, the uterus is pressed against the blood vessels in the posterior abdomen. Cardiac output measured in flexed positions shows a decrease compared to the unflexed positions, the left lateral being worse than sitting [42, 43].

IUFR traditionally consists of maternal repositioning (usually to the left lateral position) to relieve aortocaval compression, reduction of uterine contraction frequency and baseline tone by stopping exogenous oxytocin infusion and administering a tocolytic drug, administration of high-flow maternal oxygen to increase fetal oxygenation, and administration of a rapid intravenous fluid infusion that may have beneficial effects as outlined above.

Acute deterioration in the FHR pattern may occur unexpectedly after regional analgesia is induced, with onset or exacerbation of decelerations or persisting fetal bradycardia. In these circumstances, the obstetrician may “order” a category 1 (“stat”) cesarean section. We suggest that in this situation it is the anesthesiologist’s duty to institute appropriate IUFR maneuvers to try and prevent an unnecessary operation. This will often improve fetal condition quickly. However, if fetal monitoring has already been disconnected prior to transfer to the operating room (required in all but a very few hospitals), such improvement will not be recognized unless a further assessment of fetal status is made. Assessment is ideally performed with electronic fetal monitoring, but at the least auscultation of the fetal heart should be performed.

Deterioration in fetal status may be anticipated in some cases by the obstetric anesthesiologist. Prior to inserting regional analgesia, an obstetric history may identify chronic or acute fetal stress, maternal conditions such as pre-eclampsia or diabetes mellitus, or antepartum/intrapartum sentinel events such as hemorrhage or persisting (intercontraction) pain overlying a uterine scar. FHR abnormalities are also more common late in labor, when in many units a CSE is more likely to be considered because of the more rapid onset speed, leading to a double effect of risk.

Acting on this information, the anesthesiologist may administer a generous preload such as 1 l crystalloid (modified by any prior fluid administration or requirement for fluid restriction such as in pre-eclampsia); note that this maneuver cannot be repeated without limit. The woman should initially adopt the unflexed lateral or sitting position and only curl up (flex) at the last moment and for the minimum time required till the epidural catheter is placed. Fetal heart rate abnormalities may mandate that the anesthesiologist position the woman in *either* the lateral *or* the sitting position.

If oxytocin infusion is in progress, this can be reduced or stopped. The anesthesiologist should have a low threshold for the use of a tocolytic if there is a suspicion of uterine hyperactivity; however, this is identified. Ephedrine is a useful vasopressor during labor as it has direct effects increasing fetal heart rate [44].

IUFR measures are easy to perform, do not require extensive resources, and can result in significant improvements in fetal well-being. While there is no extensive evidence-based data to support their use, IUFR techniques are commonly used and often considered as a standard of care. In most situations, when used with clinical

common sense, they are unlikely to cause harm [27]. However, there are exceptions: there are conditions that predispose women to pulmonary edema (e.g., pre-eclampsia), and in these situations, fluid restriction rather than intravenous fluid boluses may be required.

11.10 Conclusions and Recommendations

We argue for an attempt during labor to balance a reduction of the risks of anesthesia, while minimizing physical and psychological restrictions on women. It is indisputable that aspiration of food into the lungs carries a high risk of severe morbidity. It is likely that less severe risk accrues with aspiration of particulate fluid contents, acid contents, and larger volumes. Gastric contents may be aspirated either through passive regurgitation or vomiting in a woman who is unconscious or obtunded; however the precipitant for this process of transfer of stomach contents is unpredictable and nonproportional. The chances that a woman will become unconscious or obtunded are very small in uncomplicated labor, but there are certain factors that increase this risk and justify dietary restrictions.

Low-risk women should be allowed to eat a light, easily digestible diet. High-risk women should not eat food, but be allowed oral water or isotonic sports drinks as wished unless delivery is predicted within 90 min. An increased risk of using such a selective policy compared to a strict nil-by-mouth policy is unprovable, but this approach has become common in the UK with no effect on anesthetic maternal mortality.

Maintenance or replacement intravenous fluid should be administered for clearly defined indications such as vomiting, pre-existing dehydration, and pyrexia if the woman cannot drink.

An intravenous preload is not necessary before regional analgesia if there is no fetal compromise. A crystalloid preload of 1 l should be considered where there is fetal compromise, including obstetric causes also noting the aggravating effects of CSE and insertion in late labor. The presence of fetal compromise should also cause the anesthesiologist to consider prophylactic and reactive IUFM measures. However, it must be noted that intravenous fluid bolus can only be repeated a limited number of times and should not be used in women with pre-eclampsia.

References

1. Mendelson CL (1946) The aspiration of stomach contents into the lungs during obstetric anesthesia. *Am J Obstet Gynecol* 52:191–206
2. Einarson TR, Piwko C, Koren G (2013) Quantifying the global rates of nausea and vomiting of pregnancy: a meta analysis. *J Popul Ther Clin Pharmacol* 20:171–183
3. Nimmo WS, Wilson J, Prescott LF (1975) Narcotic analgesics and delayed gastric emptying during labour. *Lancet* 19:890–893
4. Ewah B, Yau K, King M, Reynolds F, Carson RJ, Morgan B (1993) Effect of epidural opioids on gastric emptying in labour. *Int J Obstet Anesth* 2:125–128

5. Holdsworth JD (1978) Relationship between stomach contents and analgesia in labour. *Br J Anaesth* 50:1145–1148
6. Porter JS, Bonello E, Reynolds F (1997) The influence of epidural administration of fentanyl infusion on gastric emptying in labour. *Anaesthesia* 52:1151–1156
7. Bataille A, Rousset J, Marret E, Bonnet F (2014) Ultrasonographic evaluation of gastric content during labour under epidural analgesia: a prospective cohort study. *Br J Anaesth* 112:703–707
8. O'Reilly SA, Perrone-Hoyer PJ, Walsh E (1993) Low risk mothers, oral intake and emesis in labour. *J Nurse Midwifery* 38:228–235
9. Scrutton M, Metcalfe G, Lowy C, Seed P, O'Sullivan G (1999) Eating in labour. A randomised controlled trial assessing the risks and benefits. *Anaesthesia* 54:329–334
10. Kubli M, Scrutton MJ, Seed PT, O'Sullivan G (2002) An evaluation of isotonic "sports drinks" during labor. *Anesth Analg* 94:404–408
11. Scheepers HCJ, Thans MCJ, de Jng PA, Essed GGM, Le Cessie S, Kanhai HHH (2002) A double-blind, randomised, placebo controlled study on the influence of carbohydrate solution intake during labour. *Br J Obstet Gynaecol* 109:178–181
12. Toohill J, Soong B, Flenady V (2008) Interventions for ketosis during labour. *Cochrane Database Syst Rev* (3):CD004230. doi: [10.1002/14651858.CD004230.pub2](https://doi.org/10.1002/14651858.CD004230.pub2)
13. Tarnow-Mordi W, Shaw J, Liu D, Gardner D, Flynn F (1981) Water intoxication and syntocinon infusion. *BMJ* 283:639–642
14. Eggers T, Fliegner J (1979) Water intoxication and syntocinon infusion. *Aust N Z J Obstet Gynaecol* 19:59–60
15. Singata M, Tranmer J, Gyte GML (2013) Restricting oral fluid and food intake during labour. *Cochrane Database Syst Rev* 8:CD003930. doi:[10.1002/14651858.CD003930.pub3](https://doi.org/10.1002/14651858.CD003930.pub3)
16. Simkin P (1986) Stress, pain, and catecholamines in labor: part 2. Stress associated with childbirth events: a pilot survey of new mothers. *Birth* 13:234–240
17. Armstrong TS, Johnston IG (2000) Which women want food during labour? Results of an audit in a Scottish DGH. *Health Bull* 58:141–144
18. Johnson C, Keirse MJNC, Enkin M, Chalmers I (1989) Nutrition and hydration in labour. In: Chalmers I, Enkin M, Keirse MJNC (eds) *Effective care in pregnancy and childbirth*. Oxford University Press, Oxford, pp 827–832
19. Pengelley L (2002) Eating and drinking in labour: the consumer's view. In: Champion P, McCormick C (eds) *Eating and drinking in labour*. Books for Midwives, Oxford, pp 111–123
20. Scheepers HC, Essed GG, Brouns F (1998) Aspects of food and fluid intake during labour. Policies of midwives and obstetricians in The Netherlands. *Eur J Obstet Gynecol Reprod Biol* 78:37–40
21. Hart D (2006) Eating and drinking during labour. In: Hall Moran V, Dykes F (eds) *Maternal and infant nutrition and nurture: controversies and challenges*. Quay Books, London, pp 102–127
22. Hawkins JL, Gibbs CP, Martin-Salvaj G, Orleans M, Beaty B (1998) Oral intake policies on labor and delivery: a national survey. *J Clin Anesth* 10:449–451
23. <http://www.oaa-anaes.ac.uk/ui/content/content.aspx?id=168>. Accessed 27 May 2014
24. Dawood F, Dowswell T, Quenby S (2013) Intravenous fluids for reducing the duration of labour in low risk nulliparous women. *Cochrane Database Syst Rev* 18(6), CD007715. doi:[10.1002/14651858.CD007715.pub2](https://doi.org/10.1002/14651858.CD007715.pub2), Review
25. Patton DE, Lee W, Miller J, Jones M (1991) Maternal, uteroplacental, and fetoplacental hemodynamic and Doppler velocimetric changes during epidural anesthesia in normal labor. *Obstet Gynecol* 77:17–19
26. Zamora JE, Rosaeg OP, Lindsay MP, Crossan ML (1996) Haemodynamic consequences and uterine contractions following 0.5 or 1.0 litre crystalloid infusion before obstetric epidural analgesia. *Can J Anaesth* 43:347–352
27. Simpson KR, James DC (2005) Efficacy of intrauterine resuscitation techniques in improving fetal oxygen status during labor. *Obstet Gynecol* 105:1362–1368

28. Giles WB, Lah FX, Trudinger BJ (1987) The effect of epidural anaesthesia for caesarean section on maternal uterine and fetal umbilical artery blood flow velocity waveforms. *Br J Obstet Gynaecol* 94:55–59
29. Anim-Somuah M, Smyth RM, Jones L (2011) Epidural versus non-epidural or no analgesia in labour. *Cochrane Database Syst Rev* 7(12), CD000331. doi:10.1002/14651858.CD000331.pub3
30. <http://guidance.nice.org.uk/CG55>. Accessed 27 May 2014
31. Kinsella SM, Black AM (1998) Reporting of ‘hypotension’ after epidural analgesia during labour. Effect of choice of arm and timing of baseline readings. *Anaesthesia* 53:131–135
32. Abrão KC, Francisco RP, Miyadahira S, Cicarelli DD, Zugaib M (2009) Elevation of uterine basal tone and fetal heart rate abnormalities after labor analgesia: a randomized controlled trial. *Obstet Gynecol* 113:41–47
33. Cheng SL, Bautista D, Leo S, Sia TH (2013) Factors affecting fetal bradycardia following combined spinal epidural for labor analgesia: a matched case-control study. *J Anesth* 27:169–174
34. Fratelli N, Prefumo F, Andrico S, Lorandi A, Recupero D, Tomasoni G, Frusca T (2011) Effects of epidural analgesia on uterine artery Doppler in labour. *Br J Anaesth* 106:221–224
35. Patel N, El-Wahab N, Fernando R, Wilson S, Robson S, Columb M, Lyons G (2014) Fetal effects of combined spinal-epidural vs epidural labour analgesia: a prospective, randomised double-blind study. *Anaesthesia* 69:458–467
36. Cyna AM, Andrew M, Emmett RS, Middleton P, Simmons SW (2006) Techniques for preventing hypotension during spinal anaesthesia for caesarean section. *Cochrane Database Syst Rev* 18:CD002251
37. Collins KM, Bevan DR, Beard DW (1978) Fluid loading to reduce abnormalities of fetal heart rate and maternal hypotension during epidural analgesia in labour. *BMJ* II:1460–1461
38. Hofmeyr G, Cyna A, Middleton P (2004) Prophylactic intravenous preloading for regional analgesia in labour. *Cochrane Database Syst Rev* (4):CD000175
39. <http://www.oaa-anaes.ac.uk/UI/Content/Content.aspx?ID=175>. Accessed 27 May 2014
40. Thurlow JA, Kinsella SM (2002) Intrauterine resuscitation: active management of fetal distress. *Int J Obstet Anesth* 11:105–116
41. Kuo CD, Chen GY, Yang MJ, Tsai YS (1997) The effect of position on autonomic nervous activity in late pregnancy. *Anaesthesia* 52:1161–1165
42. Andrews PJD, Ackerman WE, Juneja MM (1993) Aortocaval compression in the sitting and lateral decubitus positions during extradural catheter placement in the parturient. *Can J Anaesth* 40:320–324
43. Chadwick IS, Eddleston J, Candelier CK, Pollard BJ (1993) Haemodynamic effects of the position chosen for the insertion of an epidural catheter. *Int J Obstet Anesth* 2:197–201
44. Cleary-Goldman J, Negron M, Scott J et al (2005) Prophylactic ephedrine and combined spinal epidural: maternal blood pressure and fetal heart rate patterns. *Obstet Gynecol* 106:466–472

Giorgio Capogna and Michela Camorcia

12.1 Dystocia

Dystocia is the term given to laboring patients whose progress in labor stalls prior to delivery.

Dystocia can be described by a number of other terms, including failed induction of labor, active phase arrest of dilatation, and second-stage arrest of descent, but these terms relate more to the timing of the diagnosis rather than the cause. Dystocia is the leading indication listed for cesarean section in nulliparous patients and conversely is very uncommon among multiparous patients. Rates of dystocia vary markedly among practitioners, hospitals, states, regions of the country, and among countries, which is more likely a result of differences in labor management strategies rather than to differences in patient characteristics. It is commonly believed that the frequency of cesarean section for dystocia has risen dramatically nowadays and that this is a major reason for the rise in the primary cesarean delivery rate, which has also favored the rise in repeat cesareans being performed.

Dystocia is considered the result of any of the following during labor: (1) abnormalities of expulsive forces; (2) abnormalities of presentation, position, or development of the fetus; and (3) abnormalities of the maternal bony pelvis or birth canal. Frequently, combinations of these three interact to produce a dysfunctional labor.

However a strict definition of dystocia has not been established. No discreet end point exists in the latent phase to describe the length of time when vaginal delivery is no longer accomplishable, and agreement does not exist regarding the length of time for active phase arrest before intervention inappropriate.

For nulliparous patients, more than 50 years ago, Friedman [1] described the upper limit normal for the length of the latent phase as 20 h and the lower normal limit for the rate of cervical dilatation in the active phase as 1.2 cm/h. A number of practitioners

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have questioned the applicability of Friedman's findings in today's labor units. In 2002, Zhang et al. [2] found a markedly different labor curve, with labors being much slower today. They found that rates of dilatation < 1 cm/h were not uncommon among women delivering vaginally, and many patients without any dilatation noted for 2 or more hours still delivered vaginally. The recommendation that 2 h of arrest in the active phase may be sufficient for a diagnosis of dystocia has therefore been challenged.

12.2 Causes of Dystocia

The main causes of dystocia are listed in Table 12.1. Inefficient uterine action is the most common cause, comprising a number of clinical situations. Induction of labor has been associated with an increase in the rate of cesarean delivery and this risk is even higher among patients starting induction with an unfavorable cervix, and cervical ripening does not necessarily lower this risk. For patients undergoing induction of labor and those who present in spontaneous labor, some cases of dystocia could be avoided with increased or longer uterine stimulation with oxytocin [3].

In other situations, more frequent or more intense contractions cannot be attained, often due to intrauterine infection or fetal intolerance to labor as perceived by the interpretation of the fetal heart rate monitor, resulting in dystocia.

Malposition of the cephalic presentation is also a significant factor that can lead to dystocia, especially in the second stage.

Cephalopelvic disproportion (CPD) is a commonly used reason given for dystocia. Risk factors include both large fetal size and small maternal pelvic size. However, there are no established criteria for this diagnosis, and it is often made based on the lack of progress in the presence of regular uterine contractions without regard to the position of the occiput.

In the absence of a contracted pelvis, such as seen with android pelvic architecture or pelvic deformity, the diagnosis of CPD is uncertain, and dystocia could be more a function of fetal position or uterine action.

Table 12.1 Common causes of dystocia

| |
|--------------------------------------------|
| Inefficient uterine action |
| Induction |
| Inadequate stimulation of contractions |
| Failure of uterine response to stimulation |
| Malposition |
| Occiput posterior |
| Asynclitism |
| Inadequate cephalic flexion |
| Cephalopelvic disproportion |

12.3 Complications Associated with Dystocia

Complications associated with dystocia can occur and should be anticipated.

Prolonged labors have higher rates of intrauterine infection, and are associated with an increased risk of uterine atony after delivery. On rare occasions, obstructed labor can lead to a constriction ring in the uterus or rupture of the uterus.

The rising rate of cesarean delivery for a diagnosis of dystocia has led to an increased number of pregnancies occurring among patients with a prior abdominal delivery. This increase in turn has led to more complications seen with vaginal birth after cesarean, as well as major hemorrhage associated with placenta accreta.

12.4 Dystocia and Pain

It seems intuitive that the intensity of pain would be increased when labor is obstructed.

Clinical diagnosis of abnormal contractions is based on the awareness of the physician that this condition does occur and delays progressive effacement and dilation of the cervix despite apparently good uterine activity and vaginal palpation, indicating that the cervix is not under good tension at the peak of contraction. From the parturient's point of view, it is commonly observed that she reports pain and backache not only during the contraction but also between the contractions. It is also a frequent observation that women who refer to more intense pain in the latent phase of labor have longer labors and are more likely to require epidural analgesia early. It is also a common clinical observation to note a more frequent request for additional analgesic interventions due to more frequent breakthrough pain during epidural analgesia when compared to the non-dysfunctional labors. It could be hypothesized that pain arising from abnormal uterine stimulation during dysfunctional labor may be conducted via nerves that are not adequately blocked by epidural medications [4]. Laboratory evidence suggests that the transmission of abnormal pain from the uterus may involve alternative nerves, possibly including the vagus nerve [5]. Alternately, the transmission of excessive sensory stimulation may overwhelm the partial local anesthetic blockade, resulting in breakthrough pain. It is conceivable that repeated episodes of pain lead to either peripheral or central sensitization, resulting in a decreased effectiveness of epidural analgesia.

In 1989, Wuitchick et al. [6] reported that women who experienced more intense pain in latent labor had longer labors and were more likely to undergo cesarean delivery. However, the authors did not find a relationship between pain in the active phase of labor and dystocia.

Hess et al. [7] reported that women requiring supplemental epidural boluses were more likely to undergo cesarean or assisted vaginal delivery than were those who did not. Their study did not report direct measurement of patient pain but provides indirect evidence that more intense pain during labor requiring more analgesia is associated with labor dystocia.

Another paper [8] to determine if the intensity of labor pain might be associated with an increased risk of cesarean delivery for dystocia did not establish cause or effect, but strongly suggested that the need for analgesia was associated with intense pain related to dystocia. This investigation found that women who ultimately required cesarean delivery for difficult labor, self-administered larger amounts of meperidine for relief of labor pain and had more intense pain before analgesia was offered.

Observational studies cannot establish a cause and effect relationship, but merely an association; however, within this hypothesis, breakthrough pain during epidural analgesia would be a surrogate measure of the severity of labor pain possibly due to dystocia.

This hypothesis based on observational studies has been confirmed by a prospective study which demonstrated that an increased local anesthetic requirement for epidural labor analgesia is associated with more intense pain related to dystocia [9].

Women in early, clinically normal, labor but who later develop dystocia require more local anesthetic and, by inference, are experiencing more severe pain than women who deliver vaginally. In contrast to the earlier retrospective or observational trials, this study measured pain and anesthetic requirements at a fixed point in time, rather than a surrogate calculated over the course of an entire labor. In addition patients were studied well before a diagnosis of dystocia had been made clinically and patient demographics, initial pain score, stage in labor, and use of oxytocin were similar at the time of the anesthetic requirement's measurement. Finally, the analgesic used was standardized. This study strongly suggests that a woman's analgesic requirement is associated with greater pain related to labor dystocia.

The observation that more intense pain is associated with difficult labor may also alert obstetricians that such pain may also be a marker of intrinsically difficult and ultimately obstructed labor. This relationship should be considered when studying the relationship between the method of labor analgesia and potential effects on the course of labor. Women with more intense labor pain, and who therefore requested earlier epidural analgesia, may have had an increased intrinsic risk of cesarean delivery for dystocia because severe pain may be an indication of obstructed labor.

12.5 Association Between Dystocia and Epidural Analgesia

There has been considerable debate about whether or not labor analgesia can adversely affect outcome by increasing the duration of labor or by increasing the incidence of operative delivery. However, there are many important obstetric variables that may lead to an adverse outcome and some of these which lead to painful labor such as fetal malposition, macrosomia, or uterine abnormalities also lead to maternal request for analgesia.

Though women who receive epidural analgesia during labor are more likely to require instrumental or cesarean delivery, there is little evidence to suggest that the

epidural itself is to blame. The association between epidural analgesia and labor outcome is most likely not causative.

Study design is significant when assessing the evidence. Typically, in a retrospective analysis, the analgesic technique and type of delivery are reviewed following delivery. In this type of study, there is inevitable selection bias, as women with long painful labors and with increased risk of intervention are more likely to request epidural analgesia, and those women deemed at high risk are actually recommended or encouraged to have an epidural.

Impact studies involve observing labor outcome before and after the introduction of an epidural service or a marked increase in epidural rate within an individual unit. Such studies are of interest because of the large number of patients but the methodology has been criticized: confounding factors, such as changing practice over time, can influence results.

Though randomized controlled trials (RCT) are considered the gold standard for research, in labor they can be difficult to blind and therefore, there is potential for observer bias. RCTs were perceived to be difficult to accomplish in labor because of problems with consent, recruitment, and high crossover rates. However, there have recently been a number of well-designed RCTs of epidural vs non-epidural analgesia that seem to have finally addressed some of the issues surrounding epidural analgesia in labor.

Several recent large RCTs comparing epidural with non-epidural analgesia during labor have shown that epidural analgesia does not increase the cesarean section rate, whether attributable to dystocia or fetal distress. These findings are supported by meta-analysis of impact studies in which a dramatic increase in the epidural rate had no impact on operative delivery rates [10, 11].

The effect of the use of epidural analgesia on the instrumental delivery rate is more difficult to investigate due to different obstetric practice.

It is also difficult due to the presence of multiple confounding factors, such as maternal pain and the urge to bear down, the presence of neuraxial analgesia-induced motor perineal block, and the position of the fetal vertex and station. The contribution and interaction of these factors to the mode of vaginal delivery have also not usually been controlled in the studies. Although assessed as a secondary outcome in numerous trials, no randomized clinical trial has assessed the effect of neuraxial analgesia on the mode of vaginal delivery as its primary outcome.

Many impact studies have observed no difference in the instrumental vaginal delivery rate before and after the availability of neuraxial analgesia. At Tripler Army Hospital, the rate of instrumental vaginal delivery did not change (11.1 % vs. 11.9 %) despite a large increase in the rate of epidural analgesia [12].

Similarly, the rate of instrumental vaginal delivery at the National Maternity Hospital in Dublin remained unchanged despite a greater than fivefold increase in the epidural rate [13].

These findings were confirmed in a systematic review of seven impact studies involving more than 28,000 parturients, which showed no difference in instrumental vaginal delivery rates (mean change, 0.76 %; 95 % CI -1.2 to 2.8) [14].

A recent meta-analysis of nine impact studies, including over 37,000 patients, found no increase in instrumental vaginal deliveries when the epidural rate increased by more than 25 % [10].

It is widely believed that effective neuraxial analgesia may prolong the second stage of labor [15].

In a meta-analysis of RCTs of epidural vs non-epidural analgesia, epidural analgesia was found to be associated with a modest, not clinically significant (15 min) prolongation of labor and not associated with poorer neonatal outcome [16].

A prolonged second stage of labor does not result in adverse maternal or fetal outcomes provided that the fetal status is reassuring, the mother is well hydrated and has adequate analgesia, and there is progress in fetal head descent. The AGOG [17] has therefore incorporated the presence or absence of neuraxial analgesia into their definition of second-stage dystocia, which states that the need for intervention (instrumental or surgical) should not be mandated solely based on second-stage duration, especially if progress is being made.

In addition the definition of duration of labor stages may vary considerably between institutions and different obstetrical practices.

Uterine activity appears to be unaffected by the induction of regional block, but fluid preloading, although ineffective in preventing the modest reductions in blood pressure associated with low dose epidurals, may be associated with a decrease in uterine contractions [18].

The concentration of local anesthetic used for epidural analgesia may influence the spontaneous vaginal delivery rate [19], and there is no difference in the outcome of labor whether a CSE or epidural technique is used to initiate analgesia provided dilute concentrations are used [20].

Early placement of neuraxial analgesia does not influence the incidence of operative vaginal delivery [21–23]. Unfortunately, some centers discontinue epidural analgesia late in labor to improve a woman's ability to push believing in this way to reduce the rate of instrumental delivery. However, discontinuation of epidural analgesia late in labor in order to reduce its presupposed adverse delivery outcomes does not reduce the instrumental delivery rate but significantly increases inadequate pain relief [24].

12.6 Instrumental Vaginal Delivery

12.6.1 Indications and Classification

Instrumental vaginal delivery (IVD) is a relatively frequent and widely practiced obstetric intervention and refers to a delivery in which the operator uses forceps or a vacuum device to shorten the expulsive phase helping the parturient in the delivery of the baby.

An instrumental vaginal delivery should be performed only if there is a precise and well-defined indication. Basically, IVD is attempted in the case of a prolonged second stage of labor, when there is concern about fetal well-being, or when there is the need to shorten the second stage.

A prolonged second stage of labor is defined by the American College of Obstetricians and Gynecologists as “lack of progress for 3 h with regional

Table 12.2 Classification of instrumental vaginal deliveries (ACOG)

| Type of procedure | Criteria |
|-------------------|-------------------------------------------------------------------------------------------------------------------|
| Outlet | Scalp is visible at the introitus without separating the labia |
| | Fetal skull has reached the level of the pelvic floor |
| | Sagittal suture is in the anteroposterior position or in the right or left occiput anterior or posterior position |
| | If rotation of $<45^\circ$ is present in fetuses with right or left anterior or posterior position |
| Low | The rotation is 45° or less and the leading point of the fetal skull (station) is at more than +2 cm |
| Mid | The fetal head is engaged but is above +2 cm |
| | Rotation $>45^\circ$ |
| High | <i>No longer practiced</i> |

The criteria for vacuum-assisted delivery are similar to those for forceps delivery, but vacuum-assisted vaginal delivery is not recommended in cases requiring rotation of $>45^\circ$ and stations above +2 cm, since the procedure is typically unsuccessful in these situations

anesthesia or 2 h without anesthesia in nulliparous women and lack of progress for 2 h with regional anesthesia or 1 h without anesthesia in multiparous women” [25].

The presence of fetal heart rate abnormalities which suggest suspected or anticipated potential fetal compromise is the most common and widely accepted indication for operative vaginal delivery, although the interpretation of fetal heart rate tracings is subjective and highly variable [26].

Instrumental vaginal delivery can also be used to electively shorten the second stage of labor in the presence of a normal FHR tracing when there are maternal conditions that preclude effective pushing such as serious cardiovascular or neurologic disease or when maternal expulsive efforts are insufficient and also in the presence of maternal exhaustion and failure of fetal descend [25].

It is worth noting that no indication for IVD is absolute; therefore, individual clinical evaluation is essential [27].

The question of when and how to intervene should involve balancing the risks and benefits of continuing pushing versus an instrumental vaginal delivery or to consider the option to perform a cesarean section.

The type of operative vaginal delivery is classified by the American College of Obstetricians and Gynecologists [25] according to the station, position, and the degree of rotation of the fetal head within the pelvis.

If the position is unclear on clinical examination which may be seen in upwards of 25 % of cases in which operative vaginal delivery is being considered [28], an intrapartum ultrasound can be performed to confirm fetal position.

Instrumental vaginal delivery is classified as either outlet, low, mid, or high, depending on the relationship of the fetal head to the introitus and ischial spines, each of which is appropriate to specific indications (Table 12.2).

12.6.2 Prerequisites and Contraindications for IVD

There are some clear fetal and maternal prerequisites to fulfill for a safe and successful instrumental vaginal delivery: the clinical situation should be carefully assessed, in particular the cervix should be fully dilated with membranes ruptured and the fetal head should be engaged in the maternal pelvis, that is, the fetal biparietal diameter must have passed through the pelvic inlet. In addition, the exact presentation and position of the fetal head should be properly assessed and should be in the vertex position; maternal bladder should be emptied, the pelvis has to be considered adequate for the fetus [27] and a backup plan should be programmed.

A number of clinical situations exist in which operative vaginal delivery should not be attempted because of the potential risks to the fetus, in particular underlying fetal disorder, fetal malpresentation (breech, transverse lie, brow, face), and a gestational age <34 weeks or a fetal weight <2,500 g because the preterm fetus is at particularly high risk of intraventricular hemorrhage and cephaloematoma [25].

12.7 Epidemiology and Criteria of Choice

Relatively few European countries have population-based statistical data on the frequencies of cesarean section, forceps delivery, and vacuum extractions.

Although the overall rate of operative vaginal delivery has been declining, the proportion of vacuum-assisted deliveries has been increasing and now accounts for almost four times the rate of forceps-assisted vaginal births [29].

The incidence of IVD in Europe varies between 2.1 and 15.7 [30]. The incidence of IVD delivery is 10–13 % in UK and Ireland [31], around 3.5 % in the United States [32] and 10–11 % in Australia and New Zealand [33].

Vacuum extraction and forceps are both two valid options when an instrument is needed to facilitate a vaginal birth. The main function of ventouse is traction of the fetal head while forceps is able to perform both traction and rotation of the fetal head, the latter being particularly useful in the case of an occiput transverse and posterior position.

The choice of one instrument over another depends on tradition, training, practitioner experience, and the clinical circumstances. However, there are some clinical situations in particular, where one instrument may be preferred over another; for example, a forceps delivery might be preferred in the case of a delivery of an occiput posterior vertex with molding and in particular when fetal distress is observed as it is quicker than vacuum extraction, and this may be crucial [34].

In North America, forceps has generally been used more frequently than vacuum extraction, whereas the reverse is true in Europe [35].

However, recent developments may have influenced obstetricians' decisions concerning these methods. Meta-analyses of randomized trials comparing maternal

and infant outcomes between vacuum extraction and forceps deliveries have found that vacuum extraction causes less maternal trauma [36].

Vacuum extraction has also recently gained in popularity because of new designs of vacuum cups, presumably with reduced risk of injury to the infant [37].

Forceps are made of two metal handles that lock in together to provide a protective cage around the baby's head, thus preventing excessive pressure being applied, as the baby is being born. They extend and are curved in shape, to correspond with the woman's pelvic curve, and end in blades that fit over the sides of the baby's head. There have been over 700 different types of forceps developed over the years, with a variety of shapes, sizes, and lengths, generally aimed at dealing with a particular complication, or a caregiver's preference. However, there are no randomized controlled trials comparing different forceps types; therefore, the operator choice depends only on preference.

There are two types of cups for vacuum delivery: the synthetic cups (soft or rigid) and the metal cups. The synthetic cups are the handheld disposable rigid (Mityvac or Kiwi Omnicup) or the conventional soft cup ventouse (silastic). The synthetic cups have higher failure rate but cause less neonatal scalp injuries than the metal ones. The metal cups are preferred for the delivery of an occipito-posterior position [38].

The soft vacuum extractor cups are associated with a significant increase in the rate of failure but a significant reduction in puerperal scalp trauma [37].

The Kiwi Omnicup is a new vacuum device that has been reported to be both safe and effective for rotational and non-rotational IVD [39, 40], but its success rate has been reported to be inferior to the one obtained using both the metal and soft standard cups [41–43].

12.8 Complications

Both the vacuum and the forceps are associated with different benefits and potential short- and long-term maternal and fetal morbidity. Overall, maternal complications are more frequent with the use of forceps than with vacuum use while this latter is associated with increased neonatal morbidity [36].

Regarding the short-term maternal complications, forceps is associated with a high incidence of vaginal trauma, episiotomy, third or fourth degree perineal lacerations [44, 45], and damage to the anal sphincter with fecal incontinence [46].

Regarding the long-term maternal complications, anal incontinence, especially over the following year, is more frequently seen with forceps use [46, 47]. However, the advantages of one instrument over another in terms of long-term (5/10 years) maternal outcome are controversial [48, 49].

Forceps delivery is also associated with neonatal complications that may occur in addition to the causes that led to the use of IVD such as fetal anoxia and dystocia.

These complications include subgaleal hematoma, intracranial hemorrhage, and retinal hemorrhage (frequency lower than with the vacuum use) that generally have good short-term prognosis except for diffuse subcutaneous hematoma [25, 50].

In addition, other rare complications include facial lacerations, facial nerve palsy, and the depressed skull fracture that is generally asymptomatic [47].

Ventouse is associated with a lower incidence of success, significant less severe maternal genital trauma and pelvic floor injury with potential risk of anal incontinence, less postpartum perineal pain, but with an higher incidence of scalp trauma, intracranial, and subgaleal hemorrhage [38] with consequent potential subsequent neuro developmental delay and retinal hemorrhage [38] as compared to forceps. There are, however, no differences in the rate of cesarean section, long-term babies' outcomes, and women's satisfaction or psychological outcomes.

These complications of both the vacuum and forceps delivery are responsible for the increased incidence of the cesarean section rate worldwide observed in recent years [51], although it should be noticed that a cesarean section performed at full cervical dilation and with the fetal head engaged is also a potentially complicated procedure [52].

Regarding the instrument more commonly used for instrumental vaginal delivery, the vacuum represents the first choice and this is mainly due to the acknowledgment of the significant reduction in the incidence of maternal pelvic floor injuries [50, 53].

In any case, since the use of instruments is associated with maternal and fetal morbidity, it is crucial to adopt all the known strategies that reduce the need for operative vaginal delivery such as continuous support during labor [54], the use of any upright or lateral position during the expulsive period, and the practice of delayed pushing [27].

12.8.1 Failure of Vacuum Extraction

IVD with vacuum may fail due to poor patient selection, for example, an attempted vacuum extraction in the case of an undiagnosed cephalopelvic disproportion, technical errors in application, failure to apply traction in coordination with maternal pushing efforts, or traction along an incorrect plane. In all these cases, the operator should consider abandoning the procedure if it is not progressing as complications deriving from vacuum use, in particular neonatal ones, increase with the duration of the procedure.

Instrumental vaginal delivery with vacuum should be abandoned if there is no evidence of descent when applying moderate traction during contractions or when imminent delivery is not impending after three consecutive correctly applied attempts [27], while IVD with forceps should be abandoned when fetal descend fails to progress after three consecutive contractions performed applying adequate force [47].

On some occasions, after the failure of a vacuum application, clinicians sequentially perform a forceps delivery (in difficult cases, or in the case of fetal distress). In this case, the rates of both maternal and neonatal morbidity are significantly increased [55, 56].

The American College of Obstetricians and Gynecologists does not generally support multiple attempts at vaginal delivery using different instruments because of concerns about a higher rate of maternal and neonatal injury [25, 55].

12.9 Analgesia for Instrumental Delivery

Epidural analgesia and other regional blocks such as the pudendal block offer excellent conditions for instrumental vaginal delivery, providing profound analgesia and adequate perineal relaxation to facilitate the instrumental application. The intensity of analgesia needed for instrumental delivery is surgical anesthesia, and therefore it is unlikely that the commonly used low dose, low concentration local anesthetic solutions used for labor analgesia will be sufficient for instrumental delivery. Vacuum delivery generally requires anesthetic levels similar to those of outlet forceps.

There is a paucity of literature concerning analgesia for instrumental delivery. A Cochrane review about analgesia for forceps delivery [57] examined four randomized controlled studies, but unfortunately the included trials had a high or unclear risk of bias and were not of a high quality. Each of the four included trials was conducted prior to 1980 and assessed anesthetic agents no longer used in clinical practice today.

If an already tested, functioning epidural catheter is already in place, 1–2 % lidocaine or mepivacaine or a more concentrated solution of the pipicolilxyldine previously used for labor, depending on how fast the block must be provided, may be given. Small increments of 5 mL epidural bolus up to a volume of 10 mL are usually sufficient to provide good perineal analgesia; however, each patient should be evaluated individually and the dose of local anesthetic titrated to effect.

Forceps use is associated with a greater requirement for analgesia compared with vacuum use [36].

Unfortunately, a considerable number of women are delivered by vacuum extraction without pain relief [58].

The high proportion might demonstrate that clinical staff do not always consider pain relief to be of high priority in vacuum extraction deliveries or that they fear impaired pushing forces. If labor analgesia was not provided (or if the epidural catheter is misplaced or is not working as well), the anesthetic choice can be spinal anesthesia. It should be performed in the sitting position with low dose local anesthetic to attempt to limit the motor block of the perineum and to maintain the maternal expulsive force. The level of analgesia should extend to the T10 dermatome.

Greater postpartum surveillance is needed following operative vaginal delivery compared with spontaneous delivery, and the anesthesiologist should be promptly available to reintervene as soon as possible if requested. In fact, the higher doses and concentrations of local anesthetics administered to provide surgical anesthesia of the perineum and the increased blood loss associated with IVD may account for the occurrence of maternal hemodynamic impairment.

Although any attempted forceps delivery can result in prolonged fetal bradycardia requiring cesarean section, this is most likely to occur with a midforceps trial. Therefore, it is recommended that a midforceps trial be attempted in the operating room prepared for an operative delivery rather than in the labor room. Also, it should be taken into account that each failed forceps can easily become a stat cesarean section; therefore, anesthesiologists should be prepared for this event testing adequately the epidural catheter for a possible extension of the epidural block.

References

1. Friedman EA (1995) Primigravidlabor: a graphicostatistical analysis. *Obstet Gynecol* 6:567–587
2. Zhang J, Troendle JF, Yancey MK (2002) Reassessing the labor curve in nulliparous women. *Am J Obstet Gynecol* 187:824–828
3. American College of Obstetricians and Gynecologists (2009) ACOG Practice Bulletin, vol 114, 2 part 1
4. Hubscher CH, Berkley KJ (1995) Spinal and vagal influences on the responses of rat solitary nucleus neurons to stimulation of uterus, cervix and vagina. *Brain Res* 702:251–254
5. Temple JL, Bradshaw HB, Wood E, Berkley KJ (1999) Effects of hypogastricneurectomy on escape responses to uterine distention in the rat. *Pain Suppl* 6:S13–S20
6. Wuitchik M, Bakal D, Lipshitz J (1989) The clinical significance of pain and cognitive activity in latent labor. *Obstet Gynecol* 73:35–42
7. Hess PE, Pratt SD, Lucas TP et al (2001) Predictors of breakthrough pain during labor epidural analgesia. *Anesth Analg* 93:414–418
8. Alexander JM, Sharma SK, McIntire DD et al (2001) Intensity of labor pain and cesarean delivery. *Anesth Analg* 92:1524–1528
9. Panni MK, Segal S (2003) Local anesthetic requirements are greater in dystocia than in normal labor. *Anesthesiology* 98:957–963
10. Mc Grady E, Litchfield K (2004) Epidural analgesia in labour. *Contin Educ Anaesth Crit Care Pain* 4:114–117
11. Cambic CR, Wong CA (2010) Labour analgesia and obstetric outcomes. *Br J Anaesth* 105: i50–i60
12. Yancey MK, Pierce B, Schweitzer D, Daniels D (1999) Observations on labor epidural analgesia and operative delivery rates. *Am J Obstet Gynecol* 180:353–359
13. Impley L, Robson M, MacQuillan K (2000) Epidural analgesia need not increase operative delivery rates. *Am J Obstet Gynecol* 182:358–363
14. Segal S, Su M, Gilbert P (2000) Effect of a rapid change in availability of epidural analgesia on the cesarean delivery rate: a meta-analysis. *Am J Obstet Gynecol* 183:974–978
15. Sharma SK, McIntire DD, Wiley J, Leveno KJ (2004) Labor analgesia and cesarean delivery: an individual patient meta-analysis of nulliparous women. *Anesthesiology* 100:142–148
16. Halpern SH, Leighton BL, Ohlsson A, Barrett JFR, Rice A (1998) Effect of epidural versus parenteral opioid analgesia on the progress of labor. A meta-analysis. *JAMA* 280:2105–2110
17. American College of Obstetricians and Gynecologists (2003) Dystocia and augmentation of labor. ACOG Practice Bulletin No. 49, December 2003. *Obstet Gynecol* 102:1445–1454
18. Cheek TG, Samuels P, Miller F, Tobin M (1996) Normal saline i.v. fluid load decreases uterine activity in active labour. *Br J Anaesth* 77:632–635
19. Comparative Obstetric Mobile Epidural Trial (COMET) Study Group UK (2001) Effect of low-dose mobile versus traditional epidural techniques on mode of delivery: a randomized controlled trial. *Lancet* 358:19–23

20. Heesen M, Van de Velde M, Klohr S et al (2014) Meta-analysis of the success of the block following combined spinal-epidural vs epidural analgesia during labour. *Anaesthesia* 69:64–71
21. Marucci M, Cinnella G, Perchiazzi G, Brienza N, Fiore T (2007) Patient-requested neuraxial analgesia for labor: impact on rates of cesarean and instrumental vaginal delivery. *Anesthesiology* 106:1035–1045
22. Wong CA, Scavone BM, Peaceman AM, McCarthy RJ, Sullivan JT, Diaz NT, Yaghmour E, Marcus RJ, Sherwani SS, Sproviero MT, Yilmaz M, Patel R, Robles C, Grouper S (2005) The risk of cesarean delivery with neuraxial analgesia given early versus late in labor. *N Engl J Med* 352:655–665
23. Wang F, Shen X, Guo X, Peng Y, Gu X, Labor Analgesia Examining Group (2009) Epidural analgesia in the latent phase of labor and the risk of cesarean delivery: a five-year randomized controlled trial. *Anesthesiology* 4:871–880
24. Torvaldsen S, Roberts CL, Bell JC, Raynes-Greenow CH (2004) Discontinuation of epidural analgesia late in labour for reducing the adverse delivery outcomes associated with epidural analgesia. *Cochrane Database Syst Rev* 4:CD004457
25. The American College of Obstetricians and Gynecologists (2000) Operative vaginal delivery. ACOG Practice Bulletin No 17. The American College of Obstetricians and Gynecologists, Washington, DC
26. Macones GA, Hankins GD, Spong CY et al (2008) The 2008 National Institute of Child Health and Human Development workshop report on electronic fetal monitoring: update on definitions, interpretation, and research guidelines. *Obstet Gynecol* 112:661–666
27. Royal College of Obstetricians and Gynecologists (2011) Green-top guidelines n 26. Operative vaginal delivery
28. Akmal S, Kametas N, Tsoi E et al (2003) Comparison of transvaginal digital examination with intra partum sonography to determine fetal head position before instrumental delivery. *Ultrasound Obstet Gynecol* 21:437–440
29. Clark SL, Belfort MA, Hankins GD et al (2007) Variation in the rates of operative delivery in the United States. *Am J Obstet Gynecol* 196:526e.1–526e.5
30. EURO-PERISTAT (2008) EURO-PERISTAT Project, with SCPE, EUROCAT, EURONEOSTAT. European Perinatal Health Report. www.europeristat.com
31. Hehir MP, Reidy FR, Wilkinson MN et al (2013) Increasing rates of operative vaginal delivery across two decades: accompanying outcomes and instrument preferences. *Eur J Obstet Gynecol Reprod Biol* 171:40–43
32. Martin JA, Hamilton BE, Ventura SJ et al (2013) Births: final data for 2011. *Natl Vital Stat Rep* 1:69–72
33. The Royal Australian and New Zealand College of Obstetricians and Gynecologists (RANZCOG) (2012) College Statement: C-Obs 16. Instrumental vaginal delivery
34. Okunwobi-Smith Y, Cooke I, MacKenzie IZ (2000) Decision to delivery intervals for assisted vaginal vertex delivery. *Br J Obstet Gynaecol* 107:467–471
35. Lomas J, Enkin M (1991) Variations in operative delivery rates. In: Chalmers I, Enkin M, Keirse MJNC (eds) *Effective care in pregnancy and childbirth*, vol II. Oxford University Press, Oxford, pp 1182–1195
36. Johanson R, Menon V (2010) Vacuum extraction versus forceps for assisted vaginal delivery. *Cochrane Database Syst Rev*. doi:10.1002/14651858.CD000446
37. Johanson RB, Rice C, Doyle M et al (1993) A randomized prospective study comparing the new vacuum extractor policy with forceps delivery. *Br J Obstet Gynaecol* 100:524–530
38. O'Mahony F, Hofmeyr GJ, Menon V (2010) Choice of instruments for assisted vaginal delivery. *Cochrane Database Syst Rev* (11):CD005455. doi:10.1002/14651858.CD005455
39. O'Grady JP, Pope CS, Patel SS (2000) Vacuum extraction in modern obstetric practice: a review and critique. *Curr Opin Obstet Gynecol* 12:475
40. Hayman R, Gilby J, Arulkumaran S (2002) Clinical of a “hand pump” vacuum delivery device. *Obstet Gynecol* 100:1190–1195

41. Attilakos G, Sibanda T, Winter C et al (2005) A randomized controlled trial of a new handheld vacuum extraction device. *BJOG* 112:1510–1515
42. Groom KM, Jones BA, Miller N et al (2006) A prospective randomized controlled trial of the Kiwi Omnicup versus conventional ventouse cups for vacuum assisted vaginal delivery. *BJOG* 113:183–189
43. Baskett TF, Fanning CA, Young DC (2008) A prospective observational study of 1000 vacuum assisted deliveries with the Omnicup devise. *J Obstet Gynaecol Can* 30:573–580
44. Wen S, Liu S, Kramer M et al (2001) Comparison of maternal and infant outcomes between vacuum extraction and forceps deliveries. *Am J Epidemiol* 153:103–107
45. Johanson JH, Figueroa R, Garry D et al (2004) Immediate maternal and neonatal effects of forceps and vacuum assisted deliveries. *Obstet Gynecol* 103:513–518
46. Fitzpatrick M, Behan M, O'Connell PR et al (2003) Randomized clinical trial to assess anal sphincter function following forceps or vacuum assisted vaginal delivery. *Br J Obstet Gynaecol* 110:424–429
47. Vayssiere C, Beucher G, Dupuis O et al (2011) Instrumental delivery: clinical practice guidelines from the French College of Gynaecologists and Obstetricians. *Eur J Obstet Gynecol Reprod Biol* 159:43–48
48. Johanson RB, Heycock E, Carter J et al (1999) Maternal and child health after assisted vaginal delivery: five-year follow up of a randomised controlled study comparing forceps and ventouse. *BJOG* 106:544–549
49. Sultan AH, Johanson RB, Carter JE (1998) Occult anal sphincter trauma following randomized forceps and vacuum delivery. *Int J Gynaecol Obstet* 61:113–119
50. Hehir MP, Reidy FR, Wilkinson MN et al (2013) Increasing rates of operative vaginal delivery across two decades: accompanying outcomes and instrument preference. *Eur J Obstet Gynecol Repr Biol* 171:40–43
51. Brennan DJ, Murphy M, Robson MS et al (2011) The singleton, cephalic, nulliparous woman after 36 weeks of gestation: contribution to overall cesarean delivery rates. *Obstet Gynecol* 117:273–279
52. Murphy DJ, Liebling RE, Verity L et al (2001) Early maternal and neonatal morbidity associated with operative delivery in second stage of labour: a cohort study. *Lancet* 358:1203–1207
53. Chalmers JA, Chalmers I (1989) The obstetric vacuum extractor is the instrument of first choice for operative delivery. *Br J Obstet Gynaecol* 96:505–506
54. Hodnett ED, Gates S, Hofmeyr GJ et al (2007) Continuous support for women during childbirth. *Cochrane Database Syst Rev* (3):CD003766
55. Towner D, Castro MA, Eby-Wilkens E et al (1999) Effect of mode of delivery in nulliparous women on neonatal intracranial injury. *N Engl J Med* 341:1709–1714
56. Murphy DJ, Macleod M, Bahl R et al (2011) A cohort study of maternal and neonatal morbidity in relation to use of sequential instruments at operative vaginal delivery. *Eur J Obstet Gynecol Reprod Biol* 156:41–45
57. Nikpoor P, Bain E (2013) Analgesia for forceps delivery. *Cochrane Database Syst Rev* (9): CD008878
58. Ahlberg M, Saltvedt S, Ekeus C (2013) Insufficient pain relief in vacuum extraction deliveries: a population-based study. *Acta Obstet Gynecol Scand* 92:306–311

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13.1 Induction

Induction of labor is the artificial stimulation of uterine contractions for the purpose of vaginal birth. It is one of the most commonly practiced procedures in obstetrics, occurring in over 20 % of pregnancies [1].

Reasons for this increase of inductions relate to the widespread availability of better cervical ripening agents, pressure from patients, physician's desire to arrange a convenient time of delivery, and litigious constraints [2].

Labor induction is indicated when the maternal or fetal benefits from delivery outweigh the risks of prolonging the pregnancy. Indications for induction may be for medical, obstetrical, or elective reasons (Table 13.1).

Risks associated with labor induction include prolonged labors, uterine contractile abnormalities, and an increased propensity for cesarean section [3].

There are a number of different definitions for failed induction of labor.

For the most part, however, the most commonly accepted definition would involve the inability of the patient to gain entry to active labor after application of maximally accepted doses of cervical ripening agents and oxytocin infusion. The presence or absence of cervical "ripening" can influence the probability of induction success. An assessment of cervical readiness for labor induction can be established by using the modified Bishop score [4] (Table 13.2).

The original scoring methodology was first described to assess the likelihood of spontaneous labor following the cervical examination in multiparous women and thus was not intended for its contemporary use. Currently, the modified Bishop score allows a systematic assessment of cervical status that facilitates choosing an induction agent and predicting induction success. A Bishop score <6 indicates an

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Table 13.1 Selection criteria for induction of labor

| |
|---------------------------------------------------------------------------------------------------------------------|
| <i>Indications</i> |
| Gestational hypertension |
| Preeclampsia, eclampsia |
| Maternal medical problems (e.g., diabetes mellitus, renal disease, chronic hypertension, antiphospholipid syndrome) |
| Abruptio placentae |
| Chorioamnionitis |
| Post-term gestation |
| Fetal compromise (e.g., severe fetal growth restriction, isoimmunization, oligohydramnios) |
| Fetal demise |
| Logistic factors (e.g., risk of rapid labor, distance from hospital, psychosocial reasons) |
| <i>Contraindications</i> |
| Complete placenta previa or vasa previa |
| Transverse fetal lie |
| Umbilical cord prolapse |
| Prior classical uterine incision |
| Active genital herpes infection |
| Previous myomectomy with entry into the endometrial cavity |

Table 13.2 Bishop pelvic scoring system

| Component | Sub-score | | | |
|----------------|-----------|--------|----------|----------|
| | 0 | 1 | 2 | 3 |
| Dilation (cm) | 0 | 1–2 | 3–4 | 5–6 |
| Station | –3 | –2 | –1 or 0 | +1 or +2 |
| Effacement (%) | 0–30 | 40–50 | 60–70 | 80 |
| Consistency | Firm | Medium | Soft | – |
| Position | Posterior | Mild | Anterior | – |

unfavorable cervix which may require a pre-labor cervical ripening agent. The higher the Bishop score, the greater is the likelihood of induction success.

A simplified Bishop score including only dilatation, station, and effacement attains a similarly high predictive ability of successful induction as the original score [5].

13.2 Cervical Ripening Agents

Choices of induction agents include some mechanical and others pharmacological.

13.2.1 Mechanical Agents

13.2.1.1 Membrane Stripping

Induction of labor by “stripping” the amniotic membranes is a common and safe practice. It is performed by manually separating the membranes from the lower uterine segment during a cervical examination, resulting in an increase in phospholipase A2 and endogenous prostaglandin F2 release, which are known to precede the spontaneous onset of labor [6].

Women who undergo membrane stripping may experience discomfort, vaginal bleeding, and irregular contractions after the procedure is performed.

13.2.1.2 Intracervical Balloon Catheter Placement

Mechanical dilation of the cervix with a balloon catheter includes intracervical Foley balloon (14–26 F) and the Atad double balloon device. These devices work by applying local pressure on the cervix by filling the balloon (or balloons) after placement in the endocervical canal. This pressure facilitates cervical ripening most likely by stimulating the release of local prostaglandins and triggering the Ferguson reflex.

13.2.2 Pharmacological Agents

13.2.2.1 Prostaglandin E2

Vaginal PGE2 should be the preferred method of induction of labor, unless there are specific clinical reasons for not using it (in particular the risk of uterine hyperstimulation). It should be administered as a gel, tablet, or controlled-release pessary.

PGE2 when applied to the female reproductive tract alters the cervical collagen milieu, which results in separation of tightly knit collagen bundles and an increase in the intervening ground substance, resulting in softening and effacement of the cervix.

There are two prostaglandin E2 compounds for cervical ripening for medically indicated inductions of labor. One dinoprostone intracervical gel to be placed intracervically and the other is dinoprostone timed-release vaginal insert.

13.2.2.2 Prostaglandin E1

Misoprostol, a synthetic PGE1, is used off-label for a variety of obstetrical and gynecological indications, including cervical ripening and labor induction. It has numerous advantages over other prostaglandin compounds including temperature stability and low cost.

When used in higher doses, misoprostol has been reported to have a higher rate of tachysystolic uterine contractions (six or more contractions in 10 min averaged over 30 min) when compared with either placebo or PGE2. An important consideration in the use of misoprostol for labor induction is the reported increased occurrence of uterine tachysystole with or without fetal heart rate abnormalities

and the potential for disruption of the uterine scar in women with a previous cesarean delivery [7].

13.3 Labor-Inducing Procedures and Agents

13.3.1 Amniotomy

Amniotomy can safely and effectively induce or augment labor, particularly in women with favorable Bishop scores. This observation of the effect of amniotomy stems from the release of prostaglandins, which stimulate uterine contractions.

When performing amniotomy, care should be taken to ensure the fetal head is well applied to the cervix and the umbilical cord is not presenting. The fetal heart rate should be recorded immediately following amniotomy.

Labor will usually ensue thereafter, although the timing of the onset of labor may be unpredictable. If oxytocin is being used concomitantly, its dosage may need adjustment.

13.3.2 Oxytocin

Oxytocin is one of the most widely used medications in obstetrical practice. Oxytocin may be used for induction or augmentation of labor, although it has proven inferior as a cervical ripening agent when the cervical condition is found to be unfavorable, compared to other pharmacological approaches based on doses and dosing intervals [8]. There are many regimens described in the literature. Recommendations for selection of a particular regimen vary, although in the current medical climate, which focuses on safety, some authorities have suggested standardized oxytocin dosing regimens [9–11] (Table 13.3).

Unfortunately none of these dosing regimens have been tested in a scientific fashion or compared to other dosing regimens by which to demonstrate efficacy let alone safety.

Table 13.3 Examples of low- and high-dose regimens oxytocin infusion protocol for labor stimulation

| Regimen | Starting dose (mU/min) | Incremental increase (mU/min) | Maximum dose (mU/min) |
|-----------|------------------------|-------------------------------|-----------------------|
| Low dose | 0.5–2 | 1–2 | 15–40 |
| High dose | 6 | 3–6 | 15–40 |

13.4 Pain Relief and Induction of Labor

There is some evidence that reported that the prostaglandin induction of labor produces a greater analgesic requirement than does spontaneous labor, supporting the observation that women may experience induced labor as being more painful than spontaneous labor [12].

Women treated with oxytocin have more pain at the start of labor [13].

Concerning the effects of epidural analgesia on induced labor, there are no differences in the length of labor or mode of birth when epidural analgesia is given at the beginning of oxytocin induction or after labor entered the active phase, and therefore, there is no benefit in waiting until labor has started to give an epidural [14, 15].

Preliminary investigation suggests that meperidine, compared to bupivacaine, speeds cervical dilation during the latent phase of induced labor and this possible collagenolytic activity of epidural opioids and their clinical use in induced labors should be investigated in the future [16].

13.5 Conclusion

Labor inductions have become increasingly more common and the upward trend is continuing. Despite therapeutic advances and continued research into the initiation of human parturition, the clinical features which are most critical for determining induction management and predicting success are the cervical condition at the start of the induction and gestational age, among other maternal demographic characteristics such as multiparity and normal weight. Induced labor may be more painful and maternal request of labor analgesia may be made earlier.

13.6 Vaginal Birth After Cesarean Section

The current cesarean section rate is rising in Europe, and many believe that a trial of labor after previous cesarean delivery (TOLAC) provides women who desire a vaginal delivery with the possibility of achieving a vaginal birth after cesarean delivery (VBAC), promoting, at the same time, a reduction of the overall cesarean section rate.

Despite the current emphasis on evidence-based medicine, there has never been a randomized trial to prove definitively that maternal and neonatal outcomes are better with either a trial of labor after cesarean or repeat cesarean delivery.

Contemporary issues that affect VBAC rates include the right for women to have a cesarean with no medical indication (“on maternal request”), the fear and possibility of future pelvic disorders after vaginal delivery, and medical legal risks should uterine rupture occur. Consequently, deciding between trial of labor and repeat cesarean is a challenge for both physicians and patients.

13.7 Pre-labor Counseling

The decision for a trial of labor (TOLAC) after a previous cesarean involves balancing risks vs benefits (Table 13.4).

Vaginal delivery is associated with fewer complications, is less expensive, has a faster recovery, and for many women there is an important satisfaction factor. Published series indicate that about 60–80 % of trials of labor after a previous cesarean result in successful vaginal births [17].

However, these rates often represent a selected population. Patients inappropriate for trial of labor usually have been excluded, so the exact percentage of women with a previous cesarean who undergo trial of labor is not known. A woman who has delivered vaginally at least once before or after her previous cesarean is more likely to have a successful trial of labor than the woman who has not yet delivered vaginally. The chance of success for those with a previous diagnosis of dystocia is consistently lower (40–70 %) than for those with nonrecurring indications [17].

On the other hand, a repeat cesarean may be more practical and safe in certain settings. It can be scheduled, is predictable, avoids a failed trial of labor with its frustration and morbidity, and basically eliminates the risk of uterine rupture. However, an elective cesarean carries with it a likelihood of more cesareans with their future risks. The incidence of placenta previa and accreta progressively increases with each cesarean and is as high as 67 % with four or more previous cesareans, and severe bleeding associated with these conditions now accounts for over half of peripartum hysterectomies [18].

The final decision for trial of labor vs repeat elective cesarean should be made by the patient and her physician after careful consideration and discussion in the prenatal record. Once the decision for trial of labor is made, the patient deserves

Table 13.4 Criteria most predictive of a safe and successful trial of labor

| |
|-----------------------------------------------------------------------------------------------------------------------|
| One (or two) prior low-segment transverse cesarean section |
| Clinically adequate pelvis and normal fetal size |
| No other uterine scars, anomalies, or previous rupture |
| Patient consent |
| Spontaneous labor |
| Physician available capable of monitoring labor, the fetus, and performing a cesarean |
| Anesthesia, blood bank, and adequate personnel available |
| <i>Potential contraindications</i> |
| Prior classical or T-shaped incision or previous uterine surgery |
| Contracted pelvis and/or macrosomia |
| Medical or obstetric condition precluding vaginal delivery |
| Patient refusal |
| Unripe cervix, induction, and augmentation |
| Inability to perform emergency cesarean because of unavailable obstetrician, anesthesia staff, or inadequate facility |

support and encouragement. This does not mean that the plan cannot be altered if the situation changes.

13.8 Management of Labor and Delivery

A plan of management of VBAC should be outlined and documented.

Induction of labor is associated with an increased risk of uterine rupture and therefore is not usually advisable [19].

Once labor has begun, the patient should be promptly evaluated and monitored and continuous electronic monitoring is usually preferable. It is important for personnel to be familiar with the potential complications of VBAC and to watch closely for fetal heart tone abnormalities and inadequate progress of labor. These women are at high risk for labor problems in view of the 20–40 % rate of unsuccessful trial of labor. Timely diagnosis and prompt management of labor abnormalities are essential in any woman with a uterine scar to avoid the added risk of obstructed labor.

13.9 Uterine Rupture

Rupture of the uterine scar is the most serious complication of VBAC, and it can be life threatening for both mother and baby. During labor, the rupture usually involves the previous scar and lower uterine segment, but it may extend intraperitoneally or retroperitoneally.

Associated risk factors include excessive amounts of oxytocin, dysfunctional labor, more than one cesarean delivery, multiparity, and even a previous nonpregnant-uterine perforation. However, in most cases the reason for rupture is unclear, and adverse outcomes can occur even in appropriate VBAC candidates. The rate of rupture is related to the type and location of the previous incision. The risk of uterine rupture is higher with the old-fashioned, classical or T incision (4–9 %) and lower with the modern, low-transverse incision (0.5–1.5 %) [20, 21].

13.10 Diagnosis

Uterine rupture is sometimes difficult to diagnose, and close surveillance is necessary since signs and symptoms may progress rapidly but also gradually. The most common signs are fetal heart rate abnormalities. A fetal heart rate pattern with subtle variable decelerations which rapidly evolve into late decelerations, bradycardia, and undetectable fetal heart tones during a trial of labor can suggest a uterine rupture. Uterine contractions often diminish in intensity and frequency. Vaginal or intra-abdominal bleeding produces anxiety, restlessness, weakness, dizziness, gross hematuria, shoulder pain, and shock. This clinical picture has sometimes been

mistaken for abruption. Loss of station of the presenting part on vaginal examination is diagnostic.

Uterine or abdominal pain is not the most common symptom and usually occurs in the area of the previous incision but may range from mild to “tearing” in nature [22].

13.11 Treatment

Any of these findings in a patient undergoing trial of labor warrant an immediate exploratory laparotomy. The condition of the infant is dependent on the severity of the rupture and relationship to the placenta and umbilical cord. The risk of an adverse perinatal outcome at term among women with a previous cesarean delivery undergoing a trial of labor is quantitatively small (1 in 200 trials of labor; 0.46 per 1,000), but greater than that associated with elective repeated cesarean delivery [23]. When uterine rupture occurs, placental or fetal extrusion is the most important factor associated with severe metabolic acidosis and hypoxic-ischemic encephalopathy [24].

13.12 Epidural and VBAC

Epidural analgesia is not contraindicated. In fact, adequate pain relief may allow more women to choose trial of labor. In addition, effective regional analgesia should not be expected to mask signs and symptoms of uterine rupture, particularly because the most common sign of rupture is fetal heart tracing abnormalities.

Sudden or atypical maternal abdominal pain occurs more rarely than do decelerations or bradycardia: in a review of 10,967 patients undergoing a TOL, only 22 % of complete uterine ruptures presented with abdominal pain, while 76 % presented with signs of fetal distress diagnosed by continuous electronic fetal monitoring. In addition, in this review, there were no differences in presenting symptoms of uterine rupture in parturients with and without epidural analgesia [22].

Thus, abdominal pain is an unreliable and uncommon sign of uterine rupture. Initial concerns that epidural analgesia might mask the pain caused by uterine rupture have not been verified, and there have been no reports of epidural analgesia delaying the diagnosis of uterine rupture. A guideline from the ACOG suggests there is no absolute contraindication to epidural analgesia for a trial of labor because epidurals rarely mask the signs and symptoms of uterine rupture [25].

In addition, the commonly used analgesic mixtures with a very low concentration of local anesthetic and opioids produce effective analgesia but are ineffective to relieve the uterine rupture pain and therefore a sudden, unexpected breakthrough pain during labor analgesia in women undergoing a VBAC may help to diagnose a uterine rupture rather than to occult it. Moreover, frequent epidural dosing may be a marker for impending uterine rupture in parturients who attempt vaginal birth after cesarean delivery [26].

Furthermore, in the case of an emergency cesarean section, the epidural catheter is already in place and this might favor a rapid extension of the epidural block.

13.13 Conclusions

VBAC was enthusiastically supported by many groups during the past three decades. With more experience, it has become apparent that there are rare but significant risks to the mother and infant. Poor perinatal outcome associated with uterine rupture is now a common cause of litigation.

Most problems occur when the patient is not under direct observation or the diagnosis of uterine rupture is delayed. In situations where attempted VBAC is not safe or the patient does not want it, elective cesarean section is a reasonable alternative.

Epidural analgesia is not contraindicated during VBAC since it does not mask the signs and symptoms of uterine rupture. Conversely, the presence of an epidural catheter already in place can favor a rapid extension of the epidural block in the case of an emergency cesarean section.

References

1. Martin JA, Hamilton BE, Ventura SJ et al (2013) Births: final data for 2011. *Natl Vital Stat Repv* 62:4–19
2. Rayburn WF, Zhang J (2002) Rising rates of labor induction: present concerns and future strategies. *Obstet Gynecol* 100:16
3. Macones GA, Hankins GDV, Spong CY et al (2008) The 2008 National Institute of Child Health and Human Development Workshop Report on electronic fetal monitoring. *Obstet Gynecol* 11:661–666
4. Bishop EH (1964) Pelvic scoring for elective induction. *Obstet Gynecol* 24:266–268
5. Laughon SK, Zhang J, Troendle J et al (2011) Using a simplified Bishop score to predict vaginal delivery. *Obstet Gynecol* 117:805–811
6. Adair CD (2000) Nonpharmacologic approaches to cervical priming and labor induction. *Clin Obstet Gynecol* 43:447–454
7. Wing DA, Lovett K, Paul RH (1998) Disruption of uterine incision following misoprostol for labor induction in women with previous cesarean delivery. *Obstet Gynecol* 91:828–830
8. Kelly AJ, Tan B (2001) Intravenous oxytocin alone for cervical ripening and induction of labour. *Cochrane Database Syst Rev* 3:CD003246
9. Clark S, Belfort M, Saade G et al (2007) Implementation of a conservative checklist-based protocol for oxytocin administration: maternal and newborn outcomes. *Am J Obstet Gynecol* 197:480.e1–480.e5
10. Hayes EJ, Weinstein L (2008) Improving patient safety and uniformity of care by a standardized regimen for the use of oxytocin. *Am J Obstet Gynecol* 198:622.e1–622.e7
11. American College of Obstetricians and Gynecologists (2009) Induction of labor. *ACOG Practice Bulletin* 107. ACOG, Washington, DC
12. Capogna G, Pargaglioni R, Lyons G et al (2001) Minimum analgesic dose of epidural sufentanil for first-stage labor analgesia: a comparison between spontaneous and prostaglandin-induced labors in nulliparous women. *Anesthesiology* 94:740–744

13. Conell-Price J, Hong JB, Shafer S et al (2008) The development and validation of a dynamic model to account for the progress of labor in the assessment of pain. *Anesth Analg* 106:1509–1515
14. Balladur A (1989) When should epidural analgesia be started in cases of induction of labour? The results of a randomised prospective study. *J Gynecol Obstet Biol Reprod* 18:249–254
15. Wong CA, McCarthy RG, Sullivan JT et al (2009) Early compared with late neuraxial analgesia in nulliparous labor induction: a randomized controlled trial. *Obstet Gynecol* 113:1066–1074
16. Kiselev MJ, Tornatore JM, Leighton LB et al (2001) Latent phase cervical dilation is faster during epidural meperidine than during epidural bupivacaine labor analgesia in nulliparous, induced-labor patients. *Anesthesiology* 94:1A–1061A
17. Landon MB, Leindeker S, Spong CY et al (2005) The MFMU cesarean registry: factors affecting the success of trial of labor after previous caesarean delivery. *Am J Obstet Gynecol* 193:1016–1023
18. Silver RM, Landon MB, Rouse DJ et al (2006) Maternal morbidity associated with multiple repeat caesarean delivery. *Obstet Gynecol* 107:1226–1232
19. Ravasia DJ, Wood SL, Pollard JK (2000) Uterine rupture during induced trial of labor among women with previous cesarean delivery. *Am J Obstet Gynecol* 183:1176–1179
20. Rosen MG, Dickinson JC, Westhoff CL (1991) Vaginal birth after cesarean: a meta-analysis of morbidity and mortality. *Obstet Gynecol* 77:465–470
21. Flamm BL, Goings JR, Liu Y et al (1994) Elective repeat caesarean delivery versus trial of labor. A prospective multicentre study. *Obstet Gynecol* 83:927–932
22. Johnson C, Oriol N (1990) The role of epidural anesthesia in trial of labor. *Reg Anesth* 15:304–308
23. Landon MB, Hauth JC, Leveno KJ et al (2004) Maternal and perinatal outcomes associated with a trial of labor after prior cesarean delivery. *N Engl J Med* 351:2581–2586
24. Bujold E, Gauthier RJ (2002) Neonatal morbidity associated with uterine rupture: what are the risk factors? *Am J Obstet Gynecol* 186:311–314
25. American College of Obstetricians and Gynecologists (2004) Vaginal birth after previous cesarean delivery. ACOG practice bulletin no. 54. Washington, DC
26. Cahill AG, Odibo AO, Allsworth JE et al (2010) Frequent epidural dosing as a marker for impending uterine rupture in patients who attempt vaginal birth after cesarean delivery. *Am J Obstet Gynecol* 202(355):e1–e5

Vegard Dahl and Leiv Arne Rosseland

14.1 Introduction

Laboring women with epidural for pain relief may need anesthesia for cesarean delivery.

Due to substantial differences in resources and traditions, the rate of cesarean deliveries varies widely from country to country. Worldwide, the rate of cesarean section (CS) is estimated to be approximately 15 %, with South America having the highest rates (29 %) and Africa the lowest with only 3.5 %. The average CS rate in Europe in 2007 was 19 %, highest in Italy (36 %) and Portugal (30.2 %) and lowest in Serbia, Montenegro, and Moldavia (6.2–8 %) [1, 2]. In most developed countries, the rate has been rising steadily during the last decades, although the rate of planned cesarean deliveries has remained stable (Fig. 14.1).

In Norway, with 62,000 deliveries yearly, the rate of cesarean delivery was 16.8 % in 2012 [3]. Out of these, 64 % were unplanned cesarean deliveries, and 32.4 % of all cesarean deliveries were performed using an epidural top-up technique. Clearly, epidural top-up technique is widely used in our country. According to a large-scale audit in the United Kingdom (UK) from 2008, epidural top-up technique was used in 26 % of all cesarean sections [4], and similar results have been reported in other countries. Although much in use, substantial controversies remain around the different aspects of the top-up technique. The best way to convert

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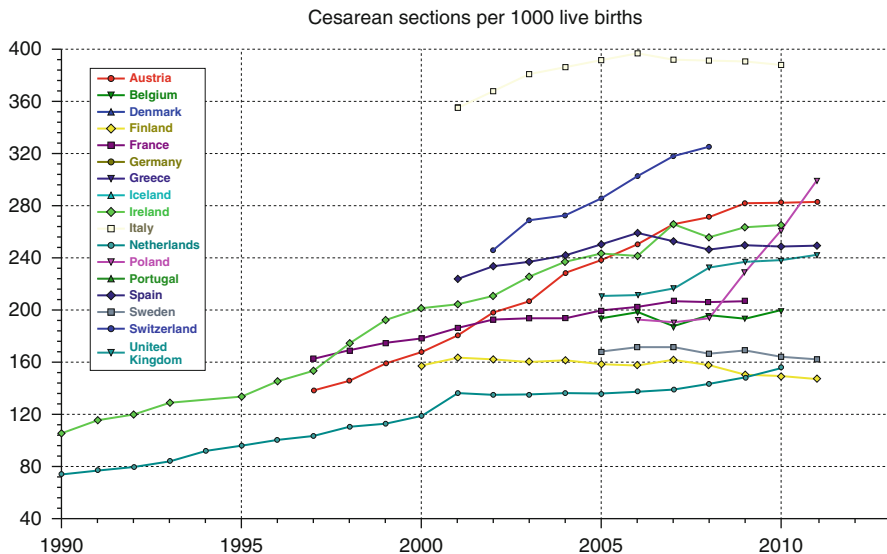


Fig. 14.1 Rate of planned cesarean deliveries. Reproduced with permission, Copyright European Health for All Database (online database) Copenhagen, WHO Regional Office for Europe 2014. *Source:* WHO/Europe, European HFA Database, July 2013

an epidural analgesia to epidural anesthesia for a cesarean section remains uncertain. This chapter is devoted to possible techniques and practical aspects around laboring women with an epidural catheter in situ in need of a cesarean section.

Generally, it is difficult to predict the mode of delivery, and clinical prediction models have been developed [5]. Data from nine birth clinics in the Netherlands [6] demonstrated that nulliparity, previous cesarean delivery, and induced labor were the three most important risk factors for cesarean delivery. Epidural analgesia was of minor importance, and when the interaction of epidural analgesia and oxytocin augmentation was included as a factor in this analysis, it was associated with a reduced risk of cesarean delivery indicated by fetal distress (OR 0.48, 95 % CI 0.31–0.73). Further on, the interaction was not a significant factor for cesarean delivery due to failure to progress (OR 0.93, 95 % CI 0.60–1.42). Interestingly, they found a significant difference between the involved birth clinics indicating an obstetrician-dependent variation. An increasing number of cesareans are performed due to unforeseen complications during delivery. This can, to some extent, be explained by more extensive monitoring and a lower threshold for intervention during delivery. Fear of litigation and criticism by colleagues may be another reason. In the UK the cesarean delivery rate has increased to 25 % [7]. A recently published analysis discusses the impact of socioeconomic status and communication skills which both are inversely correlated with cesarean section rate in the UK [8].

14.2 Definition of Urgency

In 2001, Banerjee and colleagues published a study from the region of West Sussex in the UK showing that the percentage of cesarean section performed on the basis of “fetal distress” increased from 6.4 % in 1980 to 13 % in 1999 without any changes in perinatal morbidity [9]. Extensive use of electronic fetal monitoring, with limited sensitivity and reliability, seems to have led to an increase in the cesarean section rate rather than the expected opposite [10]. The use of the term “fetal distress” is stigmatizing and may lead to a demand for general anesthesia in cases where it is unnecessary. Most cesarean sections during labor are unplanned rather than urgent. Obstetric complications such as dystocia, failure to progress, and non-reassuring fetal heart rates will often lead the obstetrician to decide on a cesarean delivery, and in these circumstances there will generally be ample time to use a regional technique such as a spinal anesthesia or a top-up of a well-functioning epidural in situ. Urgent causes for a cesarean section such as hemorrhage or cord prolapse are more seldom, and in these cases the time from decision to delivery will matter more.

The definition of “emergency” during labor is difficult. The obstetricians’ distress, combined with poor communication or substandard services, may also lead to emergency situations. An urgent (grade 1) cesarean section implies that the section should be performed immediately. According to the “Organisational Standards for Maternity Services,” published in 1995 by the Royal College of Obstetricians and Gynaecologists, a maximum time from decision to delivery of 30 min should be the gold standard for an urgent cesarean section. This time rule was acknowledged by the UK society of anesthesiologists. This definition of urgency has been heavily debated in the literature, especially the arbitrary time standard of 30 min between decision and delivery [11–13]. In the NICE Cesarean Section guidelines, published in 2011, it is stated that the 30 min rule should be used as an audit standard, but that it should not be used to judge performance in individual cases [14]. Clearly, factors other than the decision-to-delivery interval such as the pathological condition of the fetus are far more important prognostic factors. Also, transport time from the delivery suite to the operation theater accounts for approximately half the time spent in an urgency setting, and great care should be put into organizational aspects in all hospitals. Internal audits looking at every aspect of urgency and regular training in teamwork are highly recommended [15].

14.3 Extension from Analgesia to Anesthesia

The best way to avoid an urgent (grade 1) cesarean delivery is to be well prepared. A thorough multidisciplinary communication between obstetrician, midwife, and anesthetist is mandatory. As a general rule, the anesthetist in charge of the labor ward should have a continuously updated knowledge of the parturients in the delivery unit. When in doubt he or she should make an antenatal risk estimation of the parturient in question. Parturients at risk should receive an epidural catheter

as early as possible, and it should be ensured that it is well functioning in ample time in order to perform a top-up for a cesarean section, if needed. The American Society of Anesthesiologists task force on obstetric anesthesia has even recommended that in high-risk patients, such as women with preeclampsia, anticipated difficult airways, or twin gestations, the parturient should have an epidural catheter inserted before they go into labor [16]. The same recommendation applies for parturients planning a vaginal delivery after a previous cesarean. In these cases, the neuraxial catheter can be used either for labor analgesia or for anesthesia in case of an operative delivery.

When administering an epidural early in labor, great care should be taken in order to ensure its efficacy and reliability. If the laboring woman is in mild or moderate pain, this may be a difficult task. The use of low or ultralow doses of local anesthesia as in modern epidural labor analgesia will make an estimation of efficacy even more challenging. Clearly, evaluation by degree of motor blockade is of little use. The reduction of temperature sensibility is probably the best-suited method of testing, ensuring that the changes in temperature sensation are evenly distributed on both sides. Changes in sensitivity to pinprick, if used by an experienced hand, are an excellent option. As labor progresses an efficacy estimation will be easier, where a satisfied parturient with good progression of labor is the best guarantee for a well-functioning epidural. One must remember that even well-functioning epidurals may change to poor functioning ones during the progress of labor, leading to a non-satisfactory anesthesia in case of a cesarean section. In a prospective audit from Bristol, UK, over a 5-year period, the rate of failure to achieve a pain-free operation was 24 % with epidural top-up and 18 % with the combined spinal-epidural technique [4]. A failure to ensure a pain-free operation will, of course, lead to pain and discomfort but may also lead to a conversion to general anesthesia. Having a tight follow-up on the epidurals in laboring women at risk is therefore advisable. If changes in pain perception and discomfort are apparent, a replacement of the epidural should be considered as early as possible.

14.3.1 Speed of Onset

Several studies of urgent cases have demonstrated that the decision-to-delivery times are comparable between a top-up technique and the induction of general anesthesia. In an audit performed at the Royal Women's Hospital in Melbourne, Australia, encompassing 444 code green (grade 1) emergency cesarean sections, mean decision-to-delivery time was 17 (SD 6) min for general anesthesia, 19 (SD 9) min for an epidural extension, and 26 (SD 6) min for a spinal anesthesia [17]. Lim and colleagues from Singapore reported an identical decision-to-delivery time between general anesthesia and top-up of epidurals, 7.7 (SD 3.0) min [18]. The choice of local anesthetics, as well as the possible combination with different adjuvants like opioids, epinephrine and bicarbonate, is important and will be discussed later in this chapter. Another issue is the timing of the top-ups when

the decision to perform a cesarean has been made. In cases of emergency, should the anesthetist start the top-up procedure during transportation from the labor ward to the operation theater in order to win time? In many places, a top-up is commenced in the delivery room, enabling a possibility to assess the degree of anesthesia immediately when arriving in the operation theater. A further top-up is then performed if the block is insufficient, or one may consider a change from an epidural top-up to a spinal anesthesia if the block is unilateral or nonexistent before the administered volume of local anesthesia makes this procedure unsafe. The practice of starting a top-up in the labor ward is controversial [19]. We still recommend this practice if precautions are taken in order to avoid a large dose of local anesthesia and treatment of possible hypotension is available during transport.

14.3.2 Type of Local Anesthetics and Adjuvants

The choice of local anesthetics and possible adjuvants varies between countries and institutions. The grade of urgency may also influence on the choices made. 2-Chloroprocaine 3 % or lidocaine 2 % with or without additives seems to be the most frequently used solutions for epidural top-ups. The onset time is short and the efficacy versus side effect profile favorable. The possible additives for lidocaine are epinephrine, bicarbonate, and lipophilic opioids. Alkalinization of a local anesthetic with bicarbonate will shift more of the local anesthetic from the ionized to the lipid-soluble non-ionized form, thus facilitating transfer through biological membranes. It will increase the speed of onset and may also prolong the duration and quality of the block [20, 21]. An alkalinized solution should be prepared immediately before the top-up in order to maximize the effect and reduce the risk of precipitation and possible degradation of epinephrine in the solution. It should not be used together with the longer acting amides where precipitation may occur at very low concentrations of bicarbonate [22]. Epinephrine will have a double effect, both as a local vasoconstrictor, thus reducing the systemic uptake of the solution, and as a direct α_2 agonist in the spinal cord enhancing its analgesic effect [23]. Adding a lipophilic opioid like fentanyl or sufentanil to an epidural solution has been shown to have a synergetic effect with the local anesthetic enabling the use of low-dose local anesthesia technique for epidural analgesia in labor [24]. As the low-dose combination solutions for epidural analgesia are the standard around the world, whether adding additional opioids in the top-up solution is a matter of discussion and controversy. Studies comparing an addition of an opioid to local anesthesia alone in the top-up solutions have shown little effect on time to onset and quality of blocks, but more side effects like pruritus and nausea [25]. The commonest adjunct to local anesthetics, fentanyl, has been extensively studied. Observational retrospective data and one randomized controlled trial indicated that fentanyl may have a negative effect on breast feeding [26, 27]. The impact of these observations is controversial, but avoiding large epidural bolus doses of lipophilic opioids, as part of extending the epidural to surgical anesthesia, is recommended [28].

As examples of mixtures and efficacy, Allam et al. found a time from start of top-up till surgical readiness to be half using a mixture of 1.8 % lidocaine, 0.76 % bicarbonate, and epinephrine 5 µg/ml (1:200,000) compared to 0.5 % levobupivacaine [29]. Median time to reach a T5 block for touch and T4 for cold sensation was 7 min (range 5–17) for the lidocaine mixture and 14 min (range 9–31) for levobupivacaine [30]. Balaji et al. compared a mixture of lidocaine 2 %, epinephrine 5 µg/ml, and fentanyl 5 µg/ml with plain levobupivacaine 0.5 % [31]. Median onset time for the lidocaine mixture was 15 min vs 18 min for levobupivacaine when preparation time for the solutions was included in the sum (145 s for lidocaine vs 60 for levobupivacaine). They also found statistically significantly more inadequate blocks in the levobupivacaine group. 2-Chloroprocaine without preservatives or additives is as fast as lidocaine with additives, without the time-consuming preparation of mixture and the possible logistic problems [32] (Fig. 14.2).

However, when mixtures are introduced as a standard routine in institutions, the preparation time may be shortened [33].

In some countries like the UK, bupivacaine and levobupivacaine are standard [19, 31]. The newer amide derivatives like levobupivacaine and ropivacaine have advantages compared to bupivacaine, due to reduced systemic toxicity. Ropivacaine 0.75 % is comparable to bupivacaine 0.5 % in terms of time to reach a satisfactory block level, the reduced need of analgesic supplementation [34], and the degree of motor block. The longer duration of amides blocks (bupivacaine, levobupivacaine, and ropivacaine) reduces the need of intraoperative analgesic supplementation compared to lidocaine and 2-chloroprocaine. Sng et al. found equal time from top-up till readiness to surgery defined as loss of cold sensation to T4 between a

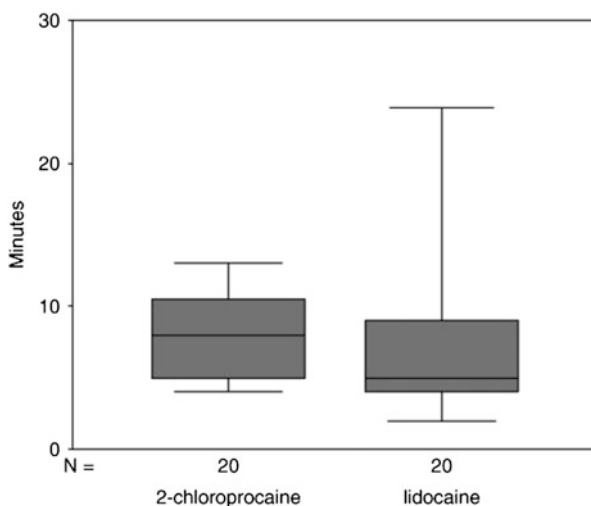


Fig. 14.2 Similar onset time of 2-chloroprocaine and lidocaine + epinephrine for epidural anesthesia for elective cesarean section. Reproduced with permission from *Acta Anaesthesiologica Scandinavica*, 2006; 50 (3): 358–363. Copyright © 2006, John Wiley and Sons

Table 14.1 Local anesthetics for epidural top-up: Summary of recommendations

| Type of local anesthetic | Concentration of LA | Possible adjuvants | Time to onset of block (min) | Comments |
|--------------------------|---------------------|-------------------------------------------------------|------------------------------|-------------------------------------------------------------------------------------|
| Lidocaine | 20 mg/ml | Epinephrine fentanyl/ sufentanil Bicarbonate | 7–15 | More adjuvants equal longer time to prepare, dependent on hospital logistics |
| 2-Chloroprocaine | 30 mg/ml | Fentanyl/ sufentanil | 8–12 | Not available in all countries. Fast offset, possible need of perioperative top-ups |
| Bupivacaine | 5 mg/ml | Fentanyl/ sufentanil | 20 | More need of supplemental analgesia |
| Levobupivacaine | 5 mg/ml | Fentanyl/ sufentanil | 15–18 | More need of supplemental analgesia |
| Ropivacaine | 7.5 mg/ml | Fentanyl/ sufentanil | 10–15 | Less motor block |

mixture of 2 % lidocaine + epinephrine + fentanyl, 0.75 % ropivacaine, and 0.5 % levobupivacaine [35]. A meta-analysis by Hillyard et al. concluded that there was a significant increase in the need of supplemental analgesia perioperatively when using bupivacaine or levobupivacaine compared to lidocaine or ropivacaine (RR 2.03, 95 % CI 1.22–3.39, $P = 0.07$) [36].

In summary, a mixture of 2 % lidocaine and epinephrine 2.5–5 $\mu\text{g/ml}$ is the local anesthetic mixture of choice. If available, 3 % 2-chloroprocaine without preservatives or additives is a good alternative. When choosing one of the amides, ropivacaine 0.75 % or levobupivacaine 0.5 % is the preferred solution in many countries. The use of levobupivacaine may result in more need of rescue analgesia during the operation, when compared to ropivacaine. Although probably somewhat slower in onset time compared to lidocaine and 2-chloroprocaine, ropivacaine has the advantage of acting longer, thus reducing the possible need of additional top-up during surgery and rescue analgesia in the immediate postoperative period (Table 14.1).

14.3.3 Volume of Solution

The volume needed to achieve a satisfactory block when topping up a well-functioning labor epidural will vary. Factors like height and obesity will influence, as well as previous infusion speed of the epidural analgesia, timing of bolus doses, and efficacy of the labor analgesia being administered. It is therefore advised to divide the top-up in incremental boluses and try to evaluate the speed of onset, hemodynamic changes, and the level of block as thorough as possible between the individual top-up doses. The vasodilatation induced by the sympathetic block should be opposed by phenylephrine, unless the patient has pregnancy-induced

hypertension. Normally, a total volume of between 15 and 20 ml of the chosen solution is needed in order to obtain a surgical block. If possible, one should start top-up with 5–10 ml of the solution in the labor ward and then assess the sensory block when arriving to the operating theater. If the block progresses bilaterally and cephalad, one should add another 5–10 ml in order to bring the sensory level of block to T4 for pinprick sensation/temperature or a T5 level to touch [37, 38]. If the block is unsatisfactory, one has the possibility to adjust the epidural or convert to a spinal anesthesia at an early period.

14.3.4 Inadequate Block

One known problem with the epidural top-up technique is the poorly functioning labor epidural, resulting in a non-satisfactory anesthesia for a cesarean section. In a prospective audit performed over a 5-year period in Bristol, UK, the failure to achieve a pain-free cesarean operation was 24 % with epidural top-up and 18 % with the combined spinal/epidural technique [4]. In a report from Canada encompassing 895 cases, the failure rate was 14 %. Out of the 120 cases of inadequate anesthesia, more than 80 % were successful after pulling the epidural catheter back 1 cm [39]. If managed by a subspecialist in obstetric anesthesia, the failure rate was reduced. In a meta-analysis including 13 trials and more than 8,500 cases published in 2012, Bauer et al. identified three risk factors for failed conversion: an increasing number of administered boluses during labor, greater urgency for a cesarean delivery, and a non-obstetric anesthesiologist providing the care. Other factors, such as high BMI, duration of epidural analgesia, and cervical dilatation at the time of epidural placement, had no impact [40].

As mentioned before in this chapter, the best way to avoid a substandard functioning epidural anesthesia after a period of epidural labor analgesia is to ensure that the epidural analgesia is well functioning. Careful evaluation and regular checkups are mandatory, especially in a high-risk parturient. Breakthrough pain, one-sided effect, and the need of additional boluses during labor are all signs of a suboptimal placement of the epidural catheter. Partly, unilateral analgesic blocks may turn out to be sufficient when injecting higher volume of an anesthetic concentration, but if the epidural analgesia during labor is not satisfactory, measures should be made to correct the placement of the catheter. One option is to pull out the catheter 1–2 cm and reevaluate as soon as possible. If one is uncertain of the effect of this strategy, a replacement of the epidural catheter is warranted, the sooner the better.

14.4 Alternatives to Epidural Top-Up

Spinal anesthesia is the preferred method in planned and urgent cesarean delivery and represents standard practice in many units, even in patients with an established labor epidural. Some case reports and case series reported high blocks when spinal

anesthesia was given to patients with ongoing epidural analgesia [41]. Even if this occurs rarely, it has been heavily debated [42, 43]. Combined spinal-epidural technique allows reducing the spinal dose in order to reduce the risk of high block. The spinal dose can be reduced to avoid a high block, and an insufficient block can be extended by an epidural bolus. The recommended dose of a single-shot spinal is controversial, but based on our experience bupivacaine 8–10 mg + fentanyl 20 µg or sufentanil 4 µg is safe. In cases of real urgency, adding an opioid to the solution may not be optional or practical. In such cases, a larger dose of local anesthesia should be considered in order to ensure a well-functioning block. If one uses an isobaric solution of bupivacaine, a dose of 12–13 mg is indicated [44]. For hyperbaric solutions of bupivacaine, 8–10 mg is normally sufficient [45]. In case of recent large epidural injection, the spinal dose can be greatly reduced. Documented success with de novo bupivacaine doses of 7 mg, 6.6 mg or 5 mg supports this, given that the combined spinal-epidural technique is used [46–48]. In this context, however, spinal induced hypotension is common and phenylephrine should always be given prophylactically.

General anesthesia will be the only option if fetal bradycardia or maternal emergencies necessitate prompt action. However, top-up technique can be used in a well-functioning epidural. During the time elapsed between epidural injection and sufficient surgical anesthesia, the patient should be prepared for surgery. Even if this preparation takes only a few minutes the block will often be sufficient, and if not, the anesthetist should be ready to give general anesthesia immediately. Concomitantly with preparation to cesarean delivery the anesthetist can inform the patient briefly about the procedure, ask the patient about relevant comorbidity, and, most importantly, assess airways to identify risk of difficult airway management. If difficult airway management is likely, the anesthetist in charge should consider calling for more experienced personnel and airway management equipment. A reevaluation of performing the cesarean delivery in regional anesthesia, rather than general, may even be compulsory.

14.5 Conclusions

An extension of an epidural analgesic block for labor analgesia in order to achieve an anesthetic block for a cesarean section is an easy, fast, and well-advised technique. The rate of success, i.e., achievement of a well-functioning anesthetic block with good operating conditions, hemodynamic stability, and a satisfied patient, is dependent upon preparedness and the technique used for the extension. High-risk parturients should receive a well-functioning epidural as early as possible in labor. Lidocaine 2 % with epinephrine, 2-chloroprocaine 3 %, or ropivacaine 0.75 % is the recommended local anesthetic that should be used for a top-up of an epidural. Inadequate blocks must be recognized early and measures be taken in order to avoid risks or unbearable suffering during surgery.

References

1. Betran AP, Merialdi M, Lauer JA, Bing-Shun W, Thomas J, Van Look P et al (2007) Rates of caesarean section: analysis of global, regional and national estimates. *Paediatr Perinat Epidemiol* 21(2):98–113
2. World Health Organization. European Health for all database. 23-4-2014. Ref Type: Internet Communication
3. Norwegian Institute of Public Health. Pregnancy, birth and infancy. 8-4-2014. Ref Type: Internet Communication
4. Kinsella SM (2008) A prospective audit of regional anaesthesia failure in 5080 Caesarean sections. *Anaesthesia* 63(8):822–832
5. Hueston WJ (1994) Development of a cesarean delivery risk score. *Obstet Gynecol* 84(6):965–968
6. Schuit E, Kwee A, Westerhuis ME, Van Dessel HJ, Graziosi GC, Van Lith JM et al (2012) A clinical prediction model to assess the risk of operative delivery. *BJOG* 119(8):915–923
7. NHS Maternity Statistics – England, 2012–2013 (2014) <http://www.hscic.gov.uk/searchcatalogue?productid/413418&q/caesarean&topics/40%2fHospital+care&sort/4Relevance&size/410&page/41#top>. 8-4-2014. Ref Type: Internet Communication
8. Essex HN, Green J, Baston H, Pickett KE (2013) Which women are at an increased risk of a caesarean section or an instrumental vaginal birth in the UK: an exploration within the Millennium Cohort Study. *BJOG* 120(6):732–743
9. Banerjee A, Hollinshead J, Williams E (2001) Delivery by caesarean section. Increased numbers of caesareans do not match diagnoses of fetal distress. *BMJ* 323(7318):930–931
10. Costantine MM, Saade GR (2012) The first cesarean: role of “fetal distress” diagnosis. *Semin Perinatol* 36(5):379–383
11. Tuffnell DJ, Wilkinson K, Beresford N (2001) Interval between decision and delivery by caesarean section—are current standards achievable? Observational case series. *BMJ* 322(7298):1330–1333
12. Wee HY, Quek SC (2001) Delivery by caesarean section. Effective system of mobilisation is used in Singapore. *BMJ* 323(7318):931
13. Colvin JR, Peden CJ (2006) Raising the Standard: a compendium of audit recipes for continuous quality improvement in anaesthesia. [http://www.rcoa.ac.uk/2012\(3\):1-402](http://www.rcoa.ac.uk/2012(3):1-402). Available from: http://www.rcoa.ac.uk/system/files/CSQ-ARB-2012_1.pdf
14. Caesarean section (2012) <http://publications.nice.org.uk/caesarean-section-cg132>
15. Leung TY, Lao TT (2013) Timing of caesarean section according to urgency. *Best Pract Res Clin Obstet Gynaecol* 27(2):251–267
16. American Society of Anesthesiologists Task Force on Obstetric Anesthesia (2007) Practice guidelines for obstetric anesthesia: an updated report by the American Society of Anesthesiologists Task Force on Obstetric Anesthesia. *Anesthesiology* 106(4):843
17. Popham P, Buettner A, Mendola M (2007) Anaesthesia for emergency caesarean section, 2000–2004, at the Royal Women’s Hospital, Melbourne. *Anaesth Intensive Care* 35(1):74–79
18. Lim Y, Shah MK, Tan HM (2005) Evaluation of surgical and anaesthesia response times for crash caesarean sections—An audit of a Singapore hospital. *Ann Acad Med Singapore* 34(10):606
19. Regan KJ, O’Sullivan G (2008) The extension of epidural blockade for emergency Caesarean section: a survey of current UK practice. *Anaesthesia* 63(2):136–142
20. Lam DTC, Ngan Kee WD, Khaw KS (2001) Extension of epidural blockade in labour for emergency Caesarean section using 2% lidocaine with epinephrine and fentanyl, with or without alkalinisation. *Anaesthesia* 56(8):777–798
21. Capogna G, Celleno D, Costantino P, Muratori F, Sebastiani M, Baldassini M (1993) Alkalinization improves the quality of lidocaine-fentanyl epidural anaesthesia for caesarean section. *Can J Anaesth* 40(5):425–430

22. Chassard D, Berrada K, Bouletreau P (1996) Alkalinization of local anesthetics: theoretically justified but clinically useless. *Can J Anaesth* 43(4):384–393
23. Niemi G (2005) Advantages and disadvantages of adrenaline in regional anaesthesia. *Best Pract Res Clin Anaesthesiol* 19(2):229–245
24. Dahl V, Hagen I, Koss KS, Nordentoft J, Raeder JC (1999) Bupivacaine 2.5 mg/ml versus bupivacaine 0.625 mg/ml and sufentanil 1 microg/ml with or without epinephrine 1 microg/ml for epidural analgesia in labour. *Int J Obstet Anesth* 8(3):155–160
25. Malhotra S, Yentis SM (2007) Extending low-dose epidural analgesia in labour for emergency Caesarean section – a comparison of levobupivacaine with or without fentanyl. *Anaesthesia* 62(7):667–671
26. Torvaldsen S, Roberts CL, Simpson JM, Thompson JF, Ellwood DA (2006) Intrapartum epidural analgesia and breastfeeding: a prospective cohort study. *Int Breastfeed J* 1:24
27. Beilin Y, Bodian CA, Weiser J, Hossain S, Arnold I, Feierman DE et al (2005) Effect of labor epidural analgesia with and without fentanyl on infant breast-feeding: a prospective, randomized, double-blind study. *Anesthesiology* 103(6):1211–1217
28. Reynolds F (2010) The effects of maternal labour analgesia on the fetus. *Best Pract Res Clin Obstet Gynaecol* 24(3):289–302
29. Allam J, Malhotra S, Hemingway C, Yentis SM (2008) Epidural lidocaine-bicarbonate-adrenaline vs levobupivacaine for emergency Caesarean section: a randomised controlled trial. *Anaesthesia* 63(3):243–249
30. Russell IF (2004) A comparison of cold, pinprick and touch for assessing the level of spinal block at caesarean section. *Int J Obstet Anesth* 13(3):146–152
31. Balaji P, Dhillon P, Russell IF (2009) Low-dose epidural top up for emergency caesarean delivery: a randomised comparison of levobupivacaine versus lidocaine/epinephrine/fentanyl. *Int J Obstet Anesth* 18(4):335–341
32. Bjørnstad E, Iversen O, Raeder J (2006) Similar onset time of 2-chloroprocaine and lidocaine + epinephrine for epidural anesthesia for elective Cesarean section. *Acta Anaesthesiol Scand* 50(3):358–363
33. Hemingway C, Woolnough M, Richards N, Yentis S (2008) Preparation times for pH-adjusted lidocaine/adrenaline epidural top-up mixture. *Int J Obst Anesth* 17(Suppl):S30
34. Sanders RD, Mallory S, Lucas DN, Chan T, Yeo S, Yentis SM (2004) Extending low-dose epidural analgesia for emergency Caesarean section using ropivacaine 0.75%. *Anaesthesia* 59(10):988–992
35. Sng BL, Pay LL, Sia AT (2008) Comparison of 2% lignocaine with adrenaline and fentanyl, 0.75% ropivacaine and 0.5% levobupivacaine for extension of epidural analgesia for urgent caesarean section after low dose epidural infusion during labour. *Anaesth Intensive Care* 36(5):659–664
36. Hillyard SG, Bate TE, Corcoran TB, Paech MJ, O’Sullivan G (2011) Extending epidural analgesia for emergency Caesarean section: a meta-analysis. *Br J Anaesth* 107(5):668–678
37. Russell IF (2001) Editorial: assessing the block for caesarean section. *Int J Obstet Anesth* 10(2):83–85
38. Inadequate Regional Block (2011) <http://www.oaanaes.ac.uk/content.asp?ContentID=411>. Ref Type: Internet Communication
39. Campbell DC, Tran T (2009) Conversion of epidural labour analgesia to epidural anesthesia for intrapartum Cesarean delivery. *Can J Anaesth* 56(1):19–26
40. Bauer ME, Kountanis JA, Tsen LC, Greenfield ML, Mhyre JM (2012) Risk factors for failed conversion of labor epidural analgesia to cesarean delivery anesthesia: a systematic review and meta-analysis of observational trials. *Int J Obstet Anesth* 21(4):294–309
41. Furst SR, Reisner LS (1995) Risk of high spinal anesthesia following failed epidural block for cesarean delivery. *J Clin Anesth* 7(1):71–74
42. Wilson MJA (2005) When using spinal anaesthesia for caesarean section after the epidural has failed, the normal dose of spinal anaesthetic should be used. *Int J Obstet Anesth* 14(1):53–55

43. Stocks GM (2005) When using spinal anaesthesia for caesarean section after the epidural has failed, the normal dose of spinal anaesthetic should be used. *Int J Obstet Anesth* 14:55–57
44. Carvalho B, Durbin M, Drover DR, Cohen SE, Ginosar Y, Riley ET (2005) The ED50 and ED95 of intrathecal isobaric bupivacaine with opioids for cesarean delivery. *Anesthesiology* 103(3):606–612
45. Leo S, Sng BL, Lim Y, Sia AT (2009) A randomized comparison of low doses of hyperbaric bupivacaine in combined spinal-epidural anesthesia for cesarean delivery. *Anesth Analg* 109(5):1600–1605
46. Langesaeter E, Rosseland LA, Stubhaug A (2008) Continuous invasive blood pressure and cardiac output monitoring during cesarean delivery: a randomized, double-blind comparison of low-dose versus high-dose spinal anesthesia with intravenous phenylephrine or placebo infusion. *Anesthesiology* 109(5):856–863
47. Vercauteren MP, Coppejans HC, Hoffmann VL, Saldien V, Adriaensen HA (1998) Small-dose hyperbaric versus plain bupivacaine during spinal anesthesia for cesarean section. *Anesth Analg* 86(5):989–993
48. Ben-David B, Miller G, Gavriel R, Gurevitch A (2000) Low-dose bupivacaine-fentanyl spinal anesthesia for cesarean delivery. *Reg Anesth Pain Med* 25(3):235–239

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15.1 Definition and Causes of Postpartum Pain

Pain experienced during childbirth is a given knowledge shared and experienced by women worldwide. However, much less attention is given to the period that women face once the child is born. This postpartum period, also called the puerperium, extends until 6–8 weeks postdelivery. It is this pain, experienced hours, days, and at times even months postpartum which can immobilize a new mother. With the knowledge that acute postoperative pain may lead to chronic pain, more attention is needed to focus on analgesia directly postpartum [1]. Therefore, we will focus our attention in this chapter on the postpartum period and the complications that can be encountered.

15.2 Short-Term and Long-Term Postpartum Pain

Pain is defined as an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage [2]. Acute pain is defined as pain of short duration; it is often a symptom with underlying cause. Chronic pain is defined as pain lasting for an extended period or persisting

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pain which exceeds the expected healing time [1]. There are different factors that contribute to pain. Not only patient's characteristics before the event but also sensory components play an important role. Inflammatory, nociceptive, and neuropathic pain are all factors that contribute, not only to any postsurgical pain but also to postpartum pain.

It is essential for a new mother to be able to function on a daily basis postpartum. Pain control is not only important for the mother's comfort, it is also important for the mother–baby relationship. Being able to breastfeed and to function as a nurturing mother for your new baby is of extreme importance. Pain may hinder the breastfeeding process which in return may lead to decreased breast milk production [3]. Certain analgesics may affect the alertness of the mother and the newborn. Therefore, one can imagine the hesitance of inexperienced medical staff in providing breastfeeding mothers with analgesics.

Different causes for postpartum pain have been identified. Some occur due to the physiological changes the woman's body endures during pregnancy, and some are due to iatrogenic factors. These factors will be discussed in more detail below.

15.3 Types of Pain Postpartum

15.3.1 After Pain

This is a common phenomenon in the postpartum period, characterized as lower back pain in the midline region. This type of pain is associated with contractions of the uterus as it is returning to its size before pregnancy. This type of pain lasts for 2–3 days postpartum. Due to the involvement of a contracting uterus, this type of pain may be exaggerated by breastfeeding due to the release of oxytocin [4].

15.3.2 Perineal Pain

Perineal pain following vaginal delivery is a common experience affecting women. Perineal pain is experienced in 42 % of women within the first 2 weeks postpartum [5]. Risk factors for perineal pain include macrosomia, nulliparity, malposition, and the use of forceps and episiotomy. Perineal pain leads to discomfort of the mother which decreases mobility and as such negatively influences the mother's ability to care for her child, which may lead to mental exhaustion and depression. Exhaustion and depression may not only have an effect on the mother–child relationship, it may also affect the mother–father relationship. Perineal tear wounds can cause other complications including incontinence, sexual dysfunction, chronic pain, and embarrassment. Risk factors identified for perineal wound complications include high tobacco use, fourth degree lacerations, use of postpartum antibiotics, use of forceps, and vacuum delivery [6].

Spontaneous perineal tears can be classified as follows: first degree tears involve the perineal skin and subcutaneous tissue and second degree tears involve the

perineal muscles and perineal body. Third degree tears include the anal sphincter. Fourth degree tears extend through the sphincter into the rectal mucosa [7]. The amount of pain is associated with the extent of perineal injury sustained.

An episiotomy is a surgical incision of the perineum which facilitates the delivery of the newborn during the last stage of labor or delivery. The routine use of episiotomy has been restricted since significant higher infection rates and longer healing periods have been reported in women who have received an episiotomy [8]. However, at times a perineal tear may be prevented by performing this procedure. Even though, infection rates and healing periods may be increased in women who have received an episiotomy, there seems to be no difference in the risk of pain. Carroli and colleagues compared restrictive episiotomy use with routine episiotomy use in a Cochrane review and came to the conclusion that there is no difference in postpartum pain when comparing an episiotomy versus a spontaneous tear [9]. Also no studies have shown a different outcome in postpartum pain when comparing restrictive versus routine episiotomy [9].

Regardless of a spontaneous perineum tear or an episiotomy procedure during delivery, the majority of women experience perineal pain the first day postpartum. The more complex the perineal tear, the worse the perineal pain. Studies have shown that first degree tears require less analgesia the first days postpartum than do the more complicated tears [10, 11]. Third and fourth degrees are associated with much more pain [12]. Macarthur et al., reported an increase in perineal pain on the first day postvaginal delivery. In a prospective cohort study, 92 % of the 447 women included experienced perineal pain on day 1 postvaginal delivery. By 6 weeks, this number had decreased to only 6 % [12].

A cross-sectional community survey conducted among women by Williams and colleagues showed that 32.6 % of 2,064 women still experienced some degree of perineal pain 1 year postpartum [13].

15.3.2.1 Treatment and Prevention of Painful Perineal Tears

Perineal Massage The use of warm compresses and lubricants to massage the perineum may prevent tears during labor. This is due to the relaxation of muscle and increased tissue circulation which all lead to thinning and stretching of the vaginal and perineal tissues and therefore may benefit the mother during labor [14]. Studies have also reported a reduction in 16 % of episiotomies, when these women start perineal massage from week 35 of gestation. These women also reported less ongoing perineal pain postpartum [15].

Cooling Once perineal trauma has occurred, cooling the perineum provides pain relief as well as a reduction in tissue swelling. Ice packs and gel pads have been reported as an adequate tool to relieve perineal pain postpartum. However, a combination of localized cooling and analgesia seems to be the most effective [16].

15.3.2.2 Analgesia After Episiotomy Repair and Cervical Lacerations

Pain medication is often used for acute and chronic pain. However, since each person may respond differently to medication, restrictive use of medication is preferred if the patient is a breastfeeding mother. This is due to all the maternal and newborn side effects caused by the various medications. However, pain relief is essential for a mother to function adequately. Therefore, the use of certain analgesics is at times unavoidable. The first choice of effective and safe analgesia, for both the breastfeeding mother and the newborn, are analgesics of the nonopioid kind, due to fewer side effects.

Paracetamol is a nonopioid kind of analgesic with antipyretic effects. It has an onset of action of 30–60 min. Its anti-inflammatory aspect is limited. Paracetamol is effective in postpartum perineal pain and studies have shown that women require less additional analgesia once paracetamol is used. For the newborn no adverse effects have been reported, and paracetamol is compatible with breastfeeding [7, 17].

The second step to control pain is the use of Non-Steroidal-Anti-Inflammatory-Drugs (NSAIDs); these drugs inhibit in a nonselective manner, COX1 and COX2 enzymes, and thus the conversion of arachidonic acid into prostaglandin and thromboxane [18]. This type of analgesic is effective in the reduction of swelling and pain after an episiotomy [19]. NSAIDs are also effective for after pain due to uterine contractions [4]. Aspirin, Ibuprofen, Diclofenac, Ketorolac, and Naproxen are all examples of NSAIDs. Aspirin has shown to be potentially toxic and is therefore not compatible with breastfeeding mothers, whereas Ibuprofen and Diclofenac are compatible. Ketorolac and Naproxen are considered compatible; however, there have been cases of adverse effects in children [17].

If pain control cannot be achieved with nonopioid analgesics, then low doses of intravenous or intramuscular morphine are preferred [3]. Morphine has a low passage into milk and a low oral bioavailability in the newborn, whereas Meperidine/Pethidine may affect the newborns alertness [20, 21]. Codeine and Oxycodone should also be avoided since they lead to central nervous system depression in the mother and newborn alike. Consumption of as little as 0.03 mg/kg Oxycodone daily has been reported to lead to neonatal lethargy [22].

15.3.3 Postdural Puncture Headache

Epidural anesthesia is a procedure where an epidural catheter is placed in the epidural space. The catheter is used to relieve the pain during the early stages of labor, by injecting local anesthetics in the epidural space. However, with this technique any discontinuity of the dura may result in postdural puncture headache. Incidence rates of 0.2–6.6 % have been reported for accidental dural punctures, with half of these leading to postdural puncture headaches [23]. Young women, pregnancy, and low body mass index increase the risk for postlumbal puncture headache. Most pregnant women, because they are young and are more often exposed to epidural anesthesia, are at the highest risk [24]. An older person has a lower risk due to a less stretchable dura mater caused by atherosclerosis and because of other anatomical differences in the elderly [25].

Postdural puncture headache is defined as a type of headache that worsens within 15 min of sitting or standing and is relieved within 15 min of lying down [26]. Other diagnoses must be ruled out before the diagnosis of postdural puncture headache can be made, for it is a diagnosis of exclusion. [24] Typically, this type of headache is characterized by its often dull and bilateral form. However, throbbing headaches have been described. The pain distributes over the frontal and occipital areas with radiation to the neck [24]. It is experienced in an upright position, with head movements and jugular compression. The pain disappears rapidly once a supine position is assumed. Accompanying symptoms may include nausea, vomiting, neck pain, and problems with vision [27]. The extremes of pain and accompanying symptoms may lead to complete immobilization.

A “wet tap” occurs when dural puncture causes spinal fluid loss. This in turn leads to a decrease of intracranial pressure once spinal fluid loss exceeds spinal fluid production. Even though the true mechanism is not fully understood, thoughts are that loss of CSF may lead to traction on the intracranial structures, more specifically the meninges. Once in an upright position this may lead to headaches. Failure of the leak to close may lead to adhesions and continuing central spinal fluid loss and increased risk of infections [24].

Fahkran et al., reviewed postmyelogram CTs and reported an increase of negative intracranial pressure due to the central spinal fluid loss. This leads to the dilation of intracranial venous structures as a compensatory response. According to Fahkran et al., these venous structures are pain sensitive and dilation of these structures leads to orthostatic headache [28].

Postdural puncture headache seldom develops immediately. Often the pain develops within 7 days, with an average of 2 days, and the pain disappears within 2 weeks. However, cases have been reported where postdural headaches have taken several months to years to resolve.

15.3.3.1 Treatment of Postdural Puncture Headache

Since postdural puncture headaches develop a few days postpartum, patients should be warned before discharge. Since more than 85 % of headaches resolve with conservative methods, information should be provided about symptoms and supporting treatment that can be applied at home. Treatment initially consists of bed rest and simple analgesics and opioids. However, if the headache persists for more than 72 h, medical intervention may be indicated [25].

Caffeine Sechzer reported the relief of postdural puncture headache in patients receiving caffeine. Caffeine is a cerebral vasoconstrictor and blocks adenosine receptors which are thought to play a role in the pathogenesis of headaches [25]. However, recurrence of headaches was often noticed when caffeine was given to patients. Caffeine can be given orally or intravenously [29].

Epidural Blood Patch This type of intervention is of mechanical form. Blood of the patient is taken from a vein, often the arm, and injected in the epidural space.

This “patch” of blood prevents leakage of central spinal fluid and allows a seal to form over the dura. This procedure has a success rate of 70–98 % [25].

Epidural Dextran-40 Epidural blood patch is the recommended intervention in persisting postdural puncture headache; there are cases reported where this intervention has had no effect. There are also contraindications for the use of autologous epidural blood patch in, for example, patients with leukemia [30]. Souron and colleagues reported in 1999 the use of Dextran-40 as a successful alternative treatment for postdural puncture headache in cases where an epidural blood patch was not effective or was contraindicated. Souron and colleagues reported no severe complications with the use of Dextran-40 [31]. However, since Dextrans were initially introduced as colloid plasma volume expanders, and increased anaphylaxis or anaphylactoid reactions were noticed, their clinical use has declined over the years [32].

Surgical Repair of the Dura This intervention is often used once nonsurgical therapies have failed. The dura is closed with sutures or metallic clips, preferably by a surgeon experienced in spinal surgery [33].

15.3.4 Musculoskeletal Pain

Due to pregnancy, the female body endures many changes which may lead to musculoskeletal discomfort during pregnancy and postpartum period. For example, fluid retention may lead to nerve entrapment, and increase in body weight will put strain on joints. Also due to hormonal release certain joints become more mobile and this may lead to pain.

15.3.5 Lower Back Pain

Even though complications due to epidural anesthesia are rare, short-term acute back pain may be due this type of analgesia received by many women during labor. Local bruising, an epidural hematoma, and epidural infections may all be causes of lower back pain postepidural anesthesia in the acute phase. A meta-analysis conducted by Ruppen and colleagues reported 1 in 183,000 women to have an epidural hematoma postepidural anesthesia. For epidural infections they reported rates of 1 in 145,000 women [34]. Chronic back pain, however, is not due to epidural anesthesia [18].

15.3.6 Pelvic Floor Disorders and Pain

The pelvic floor consists of the levator ani and coccygeus muscles and urethral and anal sphincter muscles. The sacral segments S2–S4 innervate the levators and

coccygeus muscles and urogenital diaphragm. The fusion of S2–S4 into the pudendal nerve provides innervation of the sphincters. Pelvic floor disorders are common in women with a history of vaginal delivery. During the delivery process the descending fetal head can damage the surrounding structures. This may lead to incontinence over time.

During pregnancy, 14 % of women experience pelvic pain. The pelvic area becomes more mobile and asymmetrical in the joints which may lead to pain. These movements of the pelvic joints are said to be caused by the production of the hormone relaxin. Relaxin in combination with other hormones leads to laxity of the ligaments in the body including the pelvic girdle; this then leads to a larger range of movement of the pelvic joints and may cause pain [35].

In 4 % of women, pelvic pain during pregnancy persists until several months to years postpartum [36, 37]. Pelvic girdle pain is defined as pain between the posterior iliac crest and gluteal fold, in the vicinity of the sacroiliac joints. It generally arises in relation to pregnancy, trauma, arthritis, and osteoarthritis and can occur with or without lower back pain [35]. The pain is experienced on a daily basis and is worsened by walking, lifting and changing position [36]. Risk factors are previous lower back pain or trauma to the pelvis. Nonrisk factors include contraceptives, high BMI, age, and smoking [35].

There is an association between cesarean section and chronic pelvic pain. Possible causes may be myofascial pain and neuroma formation at the site of incision [38]. The type of surgical technique used also contributes to chronic pelvic pain after cesarean delivery. Pfannenstiel incision may lead to the entrapment of lower abdominal wall nerves and lead to neuropathy. However, cesarean delivery has a low incidence of chronic pain compared with other types of surgical procedures. Oxytocin may play a protective role and provides antihyperalgesic effects during labor and delivery [1].

15.3.7 Knee Pain

The increase in body weight due to pregnancy has an impact on knee joints and ligaments. This pain is a common condition in pregnant women and commonly improves a few months postpartum without medical intervention.

15.3.7.1 Treatment of Musculoskeletal Pain

For the treatment of chronic lower back pain, NSAIDs have been reported to be more effective than paracetamol due to their analgesic and anti-inflammatory effects. There seems to be no difference in the effects of NSAIDs compared to paracetamol in acute lower back pain [39]. Paracetamol remains the first choice, followed by NSAIDs in the treatment of pain, due to the side effects of NSAIDs [35, 40].

For postpartum pelvic girdle pain, the effects of physical therapy remain unclear [35]. Stabilizing exercises have shown to be effective and decrease pain caused by the pelvic girdle postpartum. These exercises are based on training of the deep local

muscles. These include the transverse abdominal wall muscles and the superficial muscles, global muscles, which include the gluteus maximus, latissimus dorsi, oblique abdominal, erector spinae and hip adductors, and abductor muscles [41].

Also, positive effects have been reported with the injection of slow-release corticosteroids to the insertion site of the sacrospinous ligament. Torstensson and colleagues reported a significant decrease in pain in women treated with triamcinolone compared with saline [42].

15.4 Relationship Between Postpartum Pain, Postpartum Depression, and Maternal Breastfeeding

Postpartum depression is seen in 10–20 % of mothers. It develops within the first month postpartum and is associated with a period of mood swings, insomnia, crying spells, and feelings of inadequacy as a parent. The Edinburgh Postnatal Depression Scale is designed to detect postpartum depression. This screening tool is used to predict maternal mood, 4–8 weeks ahead of time [43]. Chronic pain and postpartum depression are often seen in relation to one another and are associated with general pains including back pain and headaches. Eisenach and colleagues [44] conducted a prospective, longitudinal cohort study in which they determined the association between postpartum pain and postpartum depression. They reported that 10 % of the 652 women included in the study experienced pain 8 weeks postvaginal delivery. Of these women, 60 % experienced pain on a daily basis. The severity of acute pain experienced was reported to be associated with postpartum depression 8 weeks postdelivery. An increase of 8.3 % in the Edinburgh depression scale was noted with every point increase of the acute pain score [44]. Unfortunately, 25 % of women with a history of postpartum depression have a recurrence in their next pregnancy [45].

Difficulties encountered during breastfeeding are another issue for women postpartum Akman et al. found a significantly higher Edinburgh depression Scale score in women who discontinued breastfeeding within 4 months postpartum [46].

Many women decide to breastfeed; however, due to breastfeeding difficulties only a small number manage to endure the full recommended 6 months. Wagner et al. [47] conducted a prospective study, in which they interviewed 4,179 primiparas about their breastfeeding concerns. These included milk quantity, signs of inadequate intake, latching problems, and pain.

Early in pregnancy milk, ducts in the breast start to enlarge. In the first period of the puerperium, usually the first 3–5 days, breast enlargement and pain can be caused by edema, the accumulation of milk, and swelling of breast tissue. Women often also complain of extremely sensitive nipples; this starts during pregnancy and peaks several days postpartum. It is felt in the first few suckles of the feed and usually persists during the first few weeks. Later, breast engorgement is mostly due to accumulation of milk.

15.5 Chronification of Acute Pain

Since the development of chronic pain from acute pain has been well established, studies have also reported predictors for the development of chronic pain from acute pain. For the development of chronic pain postsurgery, preoperative pain and acute pain postsurgery have been reported as predictors of chronic pain development [48]. In the case of postpartum pain, studies have also shown predictors for the development of chronic pain postpartum. Vermelis and colleagues concluded in their review that acute labor pain can be a predictor of chronic pain development. The presence of previous chronic pain and high pain scores postpartum are predictors for the development of chronic pain postvaginal delivery. In the case of a cesarean section, general anesthesia is an additional predictor of chronic pain development [49].

The development of chronic pain from acute pain is a complex process. The pain neuromatrix are regions in the brain which are activated during pain perception. These regions include the primary and secondary somatosensory cortex, insula, anterior cingulate cortex, amygdala, prefrontal cortex, and the thalamus [50]. Chronic pain often leads to the activation of the brain regions involved in cognitive and emotional processing. Eventually, chronic pain leads to a functional reorganization of the central and peripheral nervous system. This reorganization has been observed in both the somatosensory and the motor system. These changes have an effect on how both painful and nonpainful stimuli are processed. This is called central sensitization. Central sensitization is when the nervous system goes into a state of persistent reactivity. This persistent reactivity leads to a state of continuous pain even though the initial trigger for injury has disappeared. Allodynia and hyperalgesia are observed. Allodynia is the perception of pain on a nonpainful stimuli, and hyperalgesia is an extreme perception of pain on less painful stimulus [51]. Many factors including depression, anxiety, and other comorbidities have an effect on the reorganization of the central nervous system [52].

15.6 Chronic Pain Postpartum

The laboring process leads to tissue damage, which in turn leads to acute postvaginal delivery inflammatory pain. A continuing inflammatory process can also lead to chronic pain [53]. Chronic postpartum pain can be caused by neuropathic pain, due to direct nerve injury. This nerve damage can lead not only to sensory loss but also to spontaneous pain, dysesthesia, hyperalgesia, and allodynia. Therefore, treatment of acute pain could be essential in the prevention of chronic pain [54]. An effective method is when a combination of analgesia is used. This multimodal approach has proven not only to reduce side effects, but also to be effective in relieving pain. The combination of diclofenac with tramadol has been reported to prevent postoperative hyperalgesia. Also the use of NSAIDs causes an opioid-sparing effect and can therefore reduce the morphine-related side effects [55].

There is a high prevalence of morbidity among women postpartum; among others, bowel problems, urinary incontinence, perineal pain, extreme exhaustion, backache, and headaches are all common. A decrease in headaches during pregnancy and an increase postpartum have been reported to be related to the increased estrogen levels during pregnancy and the withdrawal of the hormone postpartum [56]. All these different types of pain have been reported. Unfortunately, the differentiation of pain developed postpartum and already preexisting pain often does not occur.

Eisenach and colleagues followed 1,169 women who experienced pain immediately postpartum. At 2 months, only 9.8 % of the women reported to experience pain; by 12 months this number was decreased to 3 women [57]. In another study, Eisenach and colleagues reported delivery mode to be related to acute pain postpartum. The use of forceps and higher degree of perineal lacerations and cesarean section were all associated with higher pain scores. The prevalence of persisting pain 8 weeks after delivery was 10 % in the vaginal delivery group, and 9.2 % women in the cesarean delivery group. The women described the pain to interfere with their daily activities since the pain was present daily or constantly. The painful locations postvaginal delivery included the birth canal, back, and pelvic. The scar was mentioned as painful in the cesarean section group. Also an association was reported with the severity of acute pain postpartum and persisting pain experienced 8 weeks after delivery [44]. Not only scar tissue has been reported as a cause for pain postcesarean section but also nerve entrapment and pelvic adhesions [58].

15.7 Conclusion

Chronic pain postsurgery is believed to be a manifestation of neuropathic pain or an ongoing inflammatory process caused by tissue injury or inflammation [53]. Studies report a higher incidence of chronic pain in women [58]. To establish chronic pain from already preexisting pain, a differentiation should be made between pain already present before labor and pain developed postpartum. There is a low incidence of chronic pain in obstetric medicine, once preexisting pain has been excluded [44]. Even though there is an association between acute pain during delivery and postdelivery and the development of chronic pain postpartum, there are no studies that differentiate among the different causes of pain and the development of chronic pain postpartum. The question remains whether the complete abolishment of pain during the laboring and postdelivery process may eventually lead to the reduction of chronic pain. However, with the current state of knowledge and childbirth occurring early in a woman's life, it is important to minimize pain and educate the new mother with what she can expect during the postpartum period. Early recognition and treatment is necessary to reduce the psychosocial, medical, and financial consequences. Unfortunately, there is no standard method for the reduction of pain, and since each mother and every delivery process is unique every case should be viewed on a case-by-case basis.

References

1. Landau R, Bollag L, Ornter C (2013) Chronic pain after childbirth. *Int J Obstet Anesth* 22:133–145
2. IASP Task force on Taxonomy (1994) Part III: pain terms, a current list with definitions and notes on usage. In: Merskey H, Bogduk N (eds) *Classification of chronic pain*, 2nd edn. IASP, Seattle, WA, pp 209–214
3. Montgomery A et al (2012) ABM clinical protocol #15: analgesia and anesthesia for the breastfeeding mother, revised 2012. *Breastfeed Med* 7(6):547–553
4. Deussen AR, Ashwood, P, Martis R (2011) Analgesia for relief of pain due to uterine cramping/involution after birth. *Cochrane Database Syst Rev* (5):CD004908
5. Carroli G, Mignini L (2009) Episiotomy for vaginal birth. *Cochrane Database Syst Rev* (1):CD000081. doi:[10.1002/14651858](https://doi.org/10.1002/14651858)
6. Stock L, Basham E, Gossett DR et al (2013) Factors associated with wound complications in women with obstetric anal sphincter injuries (OASIS). *Am J Obstet Gynecol* 208:327.e1–327.e6
7. Chou A et al (2013) Paracetamol/acetaminophen (single administration) for perineal pain in the early postpartum period. *Cochrane Database Syst Rev* 1:CD008407
8. Larsson PG et al (1991) Advantage or disadvantage of episiotomy compared with spontaneous perineal laceration. *Gynecol Obstet Invest* 31(4):213–216
9. Carroli G, Belizan J, Stamp G (1998) Episiotomy policies in vaginal births. (Cochrane Review). In: *Cochrane Library Oxford, Issue 2; Update Software (Updated quarterly)*
10. Andrews V, Thakar R, Sultan AH, Jones PW (2008) Evaluation of postpartum perineal pain and dyspareunia – a prospective study. *Eur J Obstet Gynecol Reprod Biol* 137:152–156
11. Klein MC, Gauthier RJ, Robbins JM et al (1994) Relationship of episiotomy to perineal trauma and morbidity, sexual dysfunction, and pelvic floor relaxation. *Am J Obstet Gynecol* 171(3):591–598
12. Macarthur AJ, Macarthur C (2004) Incidence, severity, and determinants of perineal pain after vaginal delivery: a prospective cohort study. *Am J Obstet Gynecol* 191:1199–1204
13. Williams A, Herron-Marx S, Carolyn H (2007) The prevalence of enduring postnatal perineal morbidity and its relationship to perineal trauma. *Midwifery* 4:392–403
14. Albers LL (2003) Reducing genital tract trauma at birth: launching a clinical trial in midwifery. *J Midwifery Womens Health* 48:105–110
15. Beckmann MM, Garrett AJ (2006) Antenatal perineal massage for reducing perineal trauma. *Cochrane Database Syst Rev* (1):CD005123
16. East CE et al (2012) Localized cooling for relieving pain from perineal trauma sustained during childbirth. *Cochrane Database Syst Rev* 5:CD006304
17. Hutchinson S et al (2013) Use of common migraine treatments in breast-feeding women: a summary of recommendations. *Headache* 53(4):614–627
18. Aitkenhead AR, Smith G, Rowbotham DJ (2007) *Textbook of anaesthesia*, 5th edn. Elsevier, Edinburgh, pp 75–76
19. Yildizhan R et al (2009) Comparison of the efficiency of diclofenac and indomethacin suppositories in treating perineal pain after episiotomy of laceration: a prospective, randomized, double-blind clinical trial. *Arch Gynecol Obstet* 280(5):735–738
20. Wittels C, Scott DT, Sinatra RS (1990) Exogenous opioids in human breast milk and acute neonatal neurobehavior: a preliminary study. *Anesthesiology* 73:864–869
21. Feilberg VL et al (1989) Excretion of morphine in human breast milk. *Acta Anaesthesiol Scand* 33:426–428
22. Lam J et al (2012) Central nervous system depression of neonates breastfed by mothers receiving oxycodone for postpartum analgesia. *J Pediatr* 160(1):33–37
23. Heesen M, Klohr S, Rossaint R et al (2013) Can the incidence of accidental dural puncture in laboring women be reduced? A systemic review and meta-analysis. *Minerva Anesthesiol* 79(10):1187–1197

24. Turnbull DK, Sherperd DB (2003) Post-dural puncture headache: pathogenesis, prevention and treatment. *Br J Anaesth* 91(5):718–729
25. Ahmed SV, Jayawarna C, Jude E (2006) Post lumbar puncture headache: diagnosis and management. *Postgrad Med J* 82(973):713–716
26. Headache Classification Subcommittee of the International Headache Society (2004) The International classification of headache disorders: 2nd edition. *Cephalalgia* 24(Suppl 1):9–160
27. Fernandez E (1990) Headaches associated with low spinal fluid pressure. *Headache* 30(3):122–128
28. Fahkram S, Palfey S, Thomas A et al (2014) Incidental findings of CSF leakage in patients without spontaneous intracranial hypotension and development of post-dural puncture headache. *Eur Radiol* 24(4):827–833
29. Sechzer PH (1979) Post-spinal anesthesia headache treated with caffeine. Part II: intracranial vascular distention, a key factor. *Curr Ther Res Clin Exp* 26:440
30. Bel I, Moreno LA, Gomar C (2006) Epidural dextran-40 and paramethasone injection for treatment of spontaneous intracranial hypotension. *Can J Anaesth* 53(6):591–594
31. Souron V, Hamza J (1999) Treatment of postdural puncture headaches with colloid solutions: an alternative to epidural blood patch. *Anesth Analg* 89(5):1333–1334
32. Zinderman CE, Landow L, Wise RP (2006) Anaphylactoid reactions to Dextran 40 and 70: reports to the United States Food and Drug Administration, 1969 to 2004. *J Vasc Surg* 43(5):1004–1009
33. Schievink WI (2006) Spontaneous spinal cerebrospinal fluid leaks and intracranial hypotension. *JAMA* 295(19):2286–2296
34. Ruppen W et al (2006) Incidence of epidural hematoma, infection and neurological injury in obstetric patients with epidural analgesia/anesthesia. *Anesthesiology* 105(2):394–399
35. Vleeming A et al (2008) European guidelines for the diagnosis and treatment of pelvic girdle pain. *Eur Spine J* 17:794–819
36. Hansen A et al (2005) Postpartum pelvic pain—the “pelvic joint syndrome”: a follow-up study with special reference to diagnostic methods. *Acta Obstet Gynecol Scand* 84(2):170–176
37. Albert H, Godskesen M, Westergaard J (2001) Prognosis in four syndromes of pregnancy-related pelvic pain. *Acta Obstet Gynecol Scand* 80(6):505–510
38. Almeida EC et al (2002) Cesarean section as a cause of chronic pelvic pain. *Int J Gynaecol Obstet* 79:101–104
39. Roelofs PD et al (2008) Non-steroidal anti-inflammatory drugs for low back pain. *Cochrane Database Syst Rev* (1):CD000396
40. Pohjolainen T et al (2000) Treatment of acute low back pain with the COX-2-selective anti-inflammatory drug Nimesulide. *Spine* 12:1579–1585
41. Stuge B et al (2004) The efficacy of a treatment program focusing on specific stabilizing exercises for pelvic girdle pain after pregnancy. *Spine* 29(10):E197–E203
42. Torstensson T, Lindgren A, Kristiansson P (2013) Improved function in women with persistent pregnancy-related pelvic pain after a single corticosteroid injection to the ischiadic spine: a randomized double-blind controlled trial. *Physiother Theory Pract* 29(5):371–378
43. Dennis CL (2004) Can we identify mothers at risk for postpartum depression in the immediate postpartum period using the Edinburgh Postnatal Depression Scale? *J Affect Disord* 78(2):163
44. Eisenach JC et al (2008) Severity of acute pain after childbirth, but not type of delivery, predicts persistent pain and postpartum depression. *Pain* 140(1):87–94
45. Wisner KL, Parry BL, Piontek CM (2002) Postpartum depression. *N Engl J Med* 347:194–199
46. Akman I et al (2008) Breastfeeding duration and postpartum psychological adjustment: role of maternal attachment styles. *J Paediatr Child Health* 44(6):369–373
47. Wagner EA et al (2013) Breastfeeding concerns at 3 and 7 days postpartum and feeding status at 2 months. *Pediatrics* 132(4):e865–e875
48. Gramke HF et al (2009) Predictive factors of postoperative pain after day-case surgery. *Clin J Pain* 25:455–460

49. Vermelis JM et al (2010) Prevalence and predictors of chronic pain after labor and delivery. *Curr Opin Anaesthesiol* 23(3):295–299
50. May A (2009) Plasticity in the developing brain: implications for rehabilitation. *Dev Disabil Res Rev* 15:94–101
51. Farmer MA, Baliki MN, Apkarian AV (2012) A dynamic network perspective of chronic pain. *Neurosci Lett* 520(2):197–203
52. Henry DE, Chiodo AE, Yang W (2011) Central nervous system reorganization in a variety of chronic pain states: a review. *PM R* 3(12):1116–1125
53. Kehlet H, Jensen TS, Woolf C (2006) Persistent postsurgical pain: risk factors and prevention. *Lancet* 367:1618–1625
54. Hirose M et al (1996) The effect of postoperative analgesia with continuous epidural bupivacaine after caesarean section on the amount of breastfeeding and infant gain. *Anesth Analg* 82:1166–1169
55. Lavand'homme P (2006) Postcaesarean analgesia: effective strategies and association with chronic pain. *Curr Opin Anaesthesiol* 19(3):244–248
56. Thompson JF et al (2002) Prevalence and persistence of health problems after childbirth: associations with parity and method of birth. *Birth* 29(2):83–94
57. Eisenach JC et al (2013) Resolution of pain after childbirth. *Anesthesiology* 118(1):143–151
58. Lavand'homme P (2013) Chronic pain after childbirth. *Curr Opin Anaesthesiol* 26(3):273–277

Marc Van de Velde

16.1 Introduction

The benefits of breastfeeding for the mother's and the infant's short- and long-term well-being have been extensively described and are unchallenged by the medical community [1]. The fetus benefits from improved maternal/infant bonding, protection against infectious disease by improving infant immunity, and better nutritional status [1, 2]. The mother has better involution of the uterus, less postpartum blood loss, reduced risk of breast and ovarian cancer, reduced bone demineralization, less risk of developing type II diabetes, and natural contraception [1, 2]. The recommendation of the World Health Organization (WHO) states clearly that all women should breastfeed their infants exclusively until 6 months of age [3].

Unfortunately, these goals are not achieved [4]. In the USA, at 6 months only 44 % is breastfed and only 15 % is breastfed exclusively [4]. Many factors might influence breastfeeding success including social and economical factors, hospital and workplace factors and drugs used during labor and delivery. Also analgesia used to relieve labor pain might have an effect, but published data are often conflicting.

16.2 Importance of Breastfeeding

Breastfeeding is optimal for infant nutrition and improves maternal postpartum health. Human milk feeding reduces infant mortality and infectious complications [5–7]. Human milk may also protect against sudden infant death syndrome,

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diabetes, lymphoma, allergies, and chronic digestive diseases. Breast milk feeding also seems to improve neurodevelopment in infants as opposed to formula feeding [8]. Less infant hospitalizations in the first year of life were reported when women gave breastfeeding to their offspring [9].

Also the mother confers benefits from breastfeeding her infant. In the immediate postpartum period, the risk of postpartum hemorrhage is reduced, involution of the uterus is enhanced, return to prepregnancy weight is facilitated, natural contraception is achieved, and bone remineralisation is improved [1, 6]. Long-term effects include risk reduction of ovarian and breast cancer and a decreased risk for developing type II diabetes [1, 6].

However, in the last few years papers have emerged which failed to demonstrate positive effects. In three large cohorts, including 40,000 children, a beneficial effect of breastfeeding on visual development could not be substantiated [10]. Furthermore, the conventional wisdom that breastfeeding protects against allergy and asthma could not be confirmed in several recent studies [11, 12]. Furthermore, Freeman and coworkers investigated the relationship between breastfeeding duration and infant health during the first year of life and did not demonstrate a significant reduction of infant illness visits with prolonged breastfeeding [13].

Despite these critical studies, the value and importance of breastfeeding the infant is unchallenged. Therefore, worldwide prolonged and exclusive breastfeeding is encouraged and firmly recommended [3, 14, 15]. Unfortunately, this goal is not achieved [4]. In the USA, at 6 months, only 44 % is breastfed and only 15 % is breastfed exclusively [4]. Many factors might influence breastfeeding success including social and economical factors and workplace factors. Breastfeeding problems can also be due to infant issues (e.g., palate structure, not sucking well) or maternal issues (e.g., inverted nipples, lack of education). Lactation initiation is also strongly influenced by hospital policies and lactation support [16]. Immediate peripartum factors play a significant role: type of delivery, the use of general anesthesia or regional anesthesia during C-section, nulliparity versus multiparity, drugs used during labor and delivery, and bottle feeding supplementation. Also the type and strategy of labor analgesia have been discussed as a factor that might affect breastfeeding success. Both parenteral and neuraxial analgesia have been implicated.

16.3 Systemic Opioids

Systemic labor analgesia is often achieved using opioids most commonly morphine, pethidine, fentanyl, remifentanyl, butorphanol, and nalbuphine [17]. Systemic opioid analgesia is reported to be used in 34–42 % of parturients in the USA in 2001 [18]. In up to 43 % of units in the UK and 77 % of units in Norway pethidine is the most commonly used opioid [19, 20]. Almost 50 % of units in the UK also offer remifentanyl PCA to their laboring patients [19], a trend that is also present in the Netherlands. In Belgium in 40 % of units opioid PCA (usually remifentanyl of fentanyl) is offered to patients when neuraxial analgesia is contraindicated

[21]. Pain relief is mild and at best incomplete [17] and primarily seems to be heavy sedation [22].

Maternally administered systemic opioids have neonatal effects ranging from respiratory depression, impaired muscle tone, and neonatal sedation to subtle neurobehavioral changes. These may slow breastfeeding initiation and for the worse affect suckling efforts by the child resulting in nipple trauma and pain, deterring women from continuing breastfeeding [17, 23].

Most opioids are detected in colostrum and breast milk. Wittels et al. demonstrated that morphine and its metabolite are excreted in colostrum and breast milk [24]. More studies are however required to determine the effects of morphine labor analgesia on the neonate. Pethidine is metabolized in its active metabolite by mother, fetus, and neonate and both the native drug as well as the active metabolite accumulate in fetal tissue. Accumulation also occurs in colostrum and breast milk, which has been shown to cause impaired breastfeeding behavior and success [25, 26]. Nissen et al. demonstrated that pethidine administered less than 5 h prior to delivery significantly affected suckling behavior and delayed lip and mouth movements of the neonate [26]. Despite this body of evidence and clear expert advice no longer to use pethidine for labor analgesia, it is still used in many institutions around the world. Intravenous fentanyl used during labor is excreted in colostrum and breast milk. However, given the low initial use of colostrum, the rapid decline of fentanyl levels, and the low bioavailability of fentanyl, the amount of fentanyl transferred to the neonate must be extremely low [27, 28]. Remifentanyl used during labor demonstrates a large transplacental passage [29, 30], but the effects on breastfeeding success have not adequately been studied. In one study [31], 6 % of neonates exposed intrauterine to remifentanyl demonstrated breastfeeding difficulties.

In conclusion, opioids are weak analgesics and produce sedation. Especially, pethidine might induce breastfeeding difficulties, but all opioids carry that potential side effect [32].

16.4 Nitrous Oxide and Inhalational Analgesia

Nitrous oxide has been used extensively as a labor analgesic by inhalation of 50/50 oxygen/nitrous oxide mixture (entonox) and is still the most popular analgesic in labor in the UK [32]. In an excellent review, Rooks concluded that nitrous oxide seems to be safe for the mother and the child and does not affect the labor process [33]. It readily crosses the placenta but is also immediately and easily excreted by the newborn's lungs [32–34]. The effects of nitrous oxide labor analgesia on breastfeeding and suckling behaviors are therefore considered to be minimal or nonexistent by most experts.

Similar effects have been described for inhalational analgesia using volatile agents: they hold a greater potential for maternal depression, but since they are also rapidly excreted by the newborn, the potential of neonatal depression seems low [32].

16.5 Neuraxial Analgesia

The use of regional anesthesia has become increasingly popular for labor analgesia; however, its effect on breastfeeding has been questioned. The most important concern regarding epidural analgesia and breastfeeding is that epidural drugs, especially opioids, cross the placenta and decrease neurobehavioral scores, which may have an impact on breastfeeding.

Neuraxial analgesia has direct and indirect effects on fetus and neonate. Direct effects occur when neuraxially administered drugs are absorbed systemically and reach a detectable plasma level, which may result in placental transfer and effects on the fetus. Since lidocaine absorption is rapid, it produced drowsiness in the mother and the neonate when prolonged infusions were given throughout labor. Long-acting local anesthetics are more slowly absorbed and systemic effects are rare. As mentioned before, all opioids can affect the neonate and impair breastfeeding. Also when large doses are administered epidurally, systemic absorption can be significant and transfer to the fetus is a real possibility.

Indirect effects of neuraxial analgesia can be both positive and negative. The fetal stress response to the labor process is a massive catecholamine surge during the final stages of labor, preserving regional blood flow to essential organ systems such as the brain and the heart. This fetal stress response is not affected by effective neuraxial analgesia [32]. The maternal stress response to labor pain is a combination of the release of catecholamines and cortisol and maternal hyperventilation. Maternal hyperventilation negatively affects the fetus by various mechanisms [35–38] including

- A leftward shift of the oxygen dissociation curve (due to maternal respiratory alkalosis), impairing oxygen transfer to the fetus.
- A gradual development of metabolic acidosis in the mother to compensate for the respiratory alkalosis, which is transferred to the fetus.
- In between contractions, periods of hypoventilation occur resulting in desaturation.
- Clinically relevant uterine vasoconstriction.

The release of maternal stress hormones induces maternal lipolysis and hyperglycemia, which in turn aggravate fetal acidosis [39–41]. The above-described negative effects of labor pain and maternal stress on the fetus are obtunded or reversed by effective analgesia [32, 42–44]. Neuraxial analgesia can cause significant negative effects as well such as hypotension, potentially impairing uteroplacental perfusion and fetal well being [45, 46]. The balance between positive and negative effects will determine the final outcome on fetus and neonate.

Numerous small, nonrandomized, retrospective, and observational trials have been published looking at breastfeeding success following neuraxial labor analgesia. Many of these studies do not distinguish between types of analgesia or confuse systemic and neuraxial analgesia. Furthermore, many confounding variables (such as social class, education, tradition, duration of labor, maternal exhaustion, age,

parity, and postpartum lactation support) make interpretation of the results of these low-level quality studies even more difficult. Additionally, nonstandardization of breastfeeding evaluations and outcome parameters confounds the issue even further [47]. Unfortunately, there is a paucity of well-designed, prospective, randomized studies investigating the true effect of neuraxial labor analgesia on successful breastfeeding [32, 47]. The available low-level quality data make firm recommendations for clinical practice difficult, but it seems that the effects of neuraxial labor analgesia on breastfeeding success are minimal or nonexistent, with the possible exception of high-dose epidural opioids. A summary of relevant studies is given in Table 16.1.

Several studies indicated that epidural analgesia might influence breastfeeding success in a negative way. Torvaldsen S et al. reported on 1,280 women giving birth to a singleton pregnancy in Australia in 1997 [48]. The authors concluded that breastfeeding success was much smaller in those women who had epidurals during labor. The study was heavily criticized in the scientific literature. An excellent editorial by William Camann in the *International Journal of Obstetric Anesthesia* summarized nicely the limitations [49]. First of all, the title is misleading: it is a retrospective trial and a secondary analysis and not a prospective, randomized study. Second, no patient charts were examined and data were gathered by sending out surveys at 1, 8, 16 and 24 weeks postpartum to women relying on their recall. Analgesia used during labor was self-reported by the patients. The so-called epidural group was an amalgamate of pure epidural analgesia or epidural analgesia combined with other forms of pain relief (in most instances parenteral pethidine). Also included in the epidural group were all the women who underwent Cesarean delivery either using pure epidural anesthesia or spinal anesthesia with or without initial labor epidural analgesia. Moreover, all women who underwent pure epidural analgesia (without other forms of analgesia) were women that underwent Cesarean delivery. So the authors did not discriminate between labor analgesia (and weaker solutions of local anesthetic) and anesthesia for Caesarean section, nor did they consider the type of delivery as an important factor for breastfeeding success. The authors also failed to describe the exact epidural solutions used and relied on personal communication with one single anesthetist to conclude that all patients received a similar solution. This study is therefore considered to be misleading, and conclusions regarding breastfeeding and epidural analgesia cannot be drawn.

Volmanen et al. performed a retrospective cohort study in which 164 nulliparous women received a questionnaire in the postpartum period [50]. Only 60 % of women returned the questionnaire. Primary outcome variable was full breastfeeding at 12 weeks postpartum. Epidural analgesia was associated with less women giving full breastfeeding to their infants. However, it seems that local policies might influence this.

Dozier et al. performed a secondary analysis of two previous cohort studies (one prospective and one retrospective) in which 727 primiparous, singleton pregnancies were studied for the primary outcome variable "breastfeeding cessation at 30 days postpartum," comparing any form of epidural analgesia in labor with no analgesia or other forms of analgesia. The two studies were done in three community

Table 16.1 Overview of different studies evaluating the effects of anesthesia/analgesia on breastfeeding success

| Study | Type of study | Number | Control group(s) | Intervention group (s) | Primary breastfeeding (BF) outcome parameter(s) | Result |
|------------------------------------------------------------------|---------------------------------------------------------------------------------------------|------------------------------------------|--------------------------------------|--------------------------------------|-------------------------------------------------|---------------------------------------------------------------------------------------------------------------------|
| Rajan Midwifery 1994; 10, 87– 103 | Secondary analysis of a subset of an earlier trial using a postal questionnaire | 1,149 parturients | No epidural | Mixed types of epidural analgesia | BF success at 6 weeks postpartum | Epidural analgesia had no effect on BF success |
| Halpern et al. Birth 1999; 26, 83–88 | Prospective, nonrandomized | 171 term parturients, mixed parity | No analgesia or opioid analgesia | Epidural analgesia | BF success at 6 weeks | No effects of epidural analgesia on BF success |
| Albani et al. Minerva Anesthesiol 1999; 65, 625– 630 | Prospective observational study | 1,920 parturients | No analgesia | Epidural analgesia | BF at discharge | No difference between epidural analgesia and no analgesia |
| Riordan et al. J Hum Lact 2000; 16, 7–12 | Prospective, nonrandomized study in multiparous women | 129 mothers | No analgesia Parenteral pethidine | Epidural analgesia | Initial suckling behavior BF duration | Parenteral pethidine or epidural analgesia impair initial suckling behavior No effect on BF duration |
| Baumgarder et al. J Am Board Fam Med 2003; 16, 7–13 | Prospective observational cohort study | 231 parturients | No epidural analgesia | Epidural analgesia | BF success at 24 h postpartum | BF success similar between groups In the epidural group more bottle feeding was occasionally used |

| | | | | | | |
|-----------------------------------------------------------------|------------------------------------------------------------------------------|-----------------------------|-------------------------------------------------|------------------------------------------------------|---------------------------------------|--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Henderson et al. Aust J Obstet Gynaecol 2003; 43, 372–377 | Secondary analysis of prospective trial, observational study, nonrandomized | 992 nulliparous women | No analgesia or narcotic analgesia (n = 302) | Epidural analgesia (n = 690) | % women still BF at 2 and 6 months | 2 months: No analgesia 78 % Pethidine 68 %, epidural 62 % 6 months: No analgesia 52 % Pethidine 44 %, epidural 38 % Hazard ratio for epidural was 1.44 (1.04–1.99) |
| Radzysinski JOGNN 2003; 32, 322–331 | Nonrandomized, observational study | 56 parturients and neonates | No analgesia | Epidural analgesia | Suckling behavior, BF success at 24 h | No effects of epidural analgesia |
| Volmanen et al. IJOA 2004; 13, 25–29 | Retrospective, nonrandomized cohort using a postpartum, mailed questionnaire | 99 parturients | No analgesia | Epidural analgesia | BF success at 12 weeks | Epidural analgesia reduced BF success |
| Chang et al. J Hum Lact 2005; 21, 305–314 | Prospective, nonrandomized, cohort study | 115 mixed parity women | No analgesia | Epidural analgesia | BF initiation BF at 4 weeks | Initiation and duration of BF is NOT affected by epidural analgesia |
| Jordan et al. BJOG 2005; 112, 927–934 | Retrospective, cohort study | 425 parturients | Nitrous oxide or pethidine | Epidural analgesia with or without epidural fentanyl | BF success at discharge | Epidural analgesia did not affect BF success but epidural fentanyl did have a negative impact |

(continued)

Table 16.1 (continued)

| Study | Type of study | Number | Control group(s) | Intervention group (s) | Primary breastfeeding (BF) outcome parameter(s) | Result |
|---------------------------------------------------|--------------------------------------------------------------------------|---------------------------------------------------------|------------------|-----------------------------------------------------------------------------------------------------------------------------------------------------|-------------------------------------------------|------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Beilin et al. Anesthesiology 2005; 103, 1211–1217 | Prospective, randomized trial evaluating the effect of epidural fentanyl | 177 multiparous women | No fentanyl | Two groups: Low dose fentanyl < 150 mcg High dose fentanyl > 150 mcg | BF at 1 day and 6 weeks | 1 day: trend towards less BF with high dose, but not significant 6 weeks: the incidence of not BF was 2 % (no fentanyl); 6 % (low fentanyl) and 19 % (high fentanyl); $p = 0.002$ Lactation nurse: no difference noted |
| Torvaldsen et al. Int Breastfeeding J 2006; 1: 24 | Retrospective, secondary analysis of nonrandomized cohort | 1,280 parturients; vaginal delivery and C-section mixed | No analgesia | Epidural analgesia combined with various additional strategies of pain relief (pethidine, ...). Pure epidural analgesia only in C-section patients. | BF success at 24 weeks | Epidural group more likely to stop BF by 24 weeks. Hazard ratio of 2.02 (1.53–2.67) |

| | | | | | | |
|------------------------------------------------------|-------------------------------------------------------------------------------------------------------------------------------------------------------------|-----------------------------------------------------------------------------------------------|------------------------------------------------------------------------------------------------------------------------------|-------------------------------------------------------------------------------------------|--------------------------------------------------------------------------------------------------------------------------------|---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Wiklund et al. Midwifery 2009; 25, e31– e38 | Retrospective, cohort study | 1,170 | No epidural analgesia (but paracervical or pudendal block possible); parenteral opioids were not administered | Epidural analgesia (some also paracervical or pudendal block before epidural) | BF initiation within 4 h postpartum Use of artificial milk during hospitalisation BF success at discharge | Epidural analgesia resulted in significantly less babies being breast fed during the first 4 postpartum hours (OR 3.79, $p < 0.0004$), had a higher chance of receiving artificial milk supplementation (OR 2.19, $p < 0.0012$) and were more likely not to be breast fed at discharge (OR 1.79, $p < 0.043$) |
| Wieczorek et al. IJOA 2010; 19, 273–277 | Prospective, observational, cohort study | 87 parturients | No analgesia or parenteral pethidine | All epidural analgesia | BF success at 6 weeks | 95 % BF success at 6 weeks |
| Wilson et al. Anesthesia 2010; 65, 145– 153 | Secondary analysis of previous trial and additional inclusion of nonepidural patients. Questionnaires sent 12 months after delivery | 1,054 epidural subjects and 351 matched parturients without epidural analgesia | No analgesia or parenteral pethidine | Three different strategies of epidural analgesia | Time to initial BF % initiating BF Duration of BF | Epidural analgesia had no negative effects Pethidine was associated with less initiation of BF |

(continued)

Table 16.1 (continued)

| Study | Type of study | Number | Control group(s) | Intervention group (s) | Primary breastfeeding (BF) outcome parameter(s) | Result |
|----------------------------------------------------|-------------------------------------------------------|-----------------------------------------|----------------------------------------|----------------------------------|---------------------------------------------------------------------------|-------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Gizzo et al. Breastfeeding Med 2012; 7, 262–268 | Nonrandomized, prospective, observational study | 128 nulliparous, term parturients | No analgesia | Epidural analgesia | Neonatal reactivity, suckling behavior, duration of initial episode of BF | No effect on neonatal reactivity and suckling behavior. More neonates in the epidural group had shorter initial breastfeeding: 62 % vs. 29 % in the control group |
| Dozier et al. Mat Child Health J 2013; 17, 689–698 | Retrospective, secondary analysis of 2 cohort studies | 727 primiparous patients 3 hospitals | No analgesia or nonneuraxial analgesia | Any epidural for labor analgesia | BF cessation at 30 days | Increased risk of cessation with a hazard ratio of 1.26 (1.10–1.44) |
| Bai et al. J Midwif Womens Health 2013; 58, 25–32 | Prospective, nonrandomized, observational study | 1,280 mixed parity women | No analgesia Opioid analgesia | Epidural analgesia | BF duration | No effect of epidural analgesia on BF duration; opioid analgesia did reduce BF duration |

hospitals in the USA [51]. Epidural analgesia was associated with an increased risk of breastfeeding cessation at 30 days [HR 1.26 (1.10–1.44)]. Additional risk factors to cease breastfeeding were identified and these included low income, low social class, low confidence in breastfeeding success, and younger age. Especially, the combination of epidural analgesia and IV oxytocin was associated mostly with breastfeeding cessation. Of note, this study demonstrates an association, but a causative effect of epidural analgesia was not proven!

Henderson et al. performed a secondary analysis of a prospective randomized study comparing epidural analgesia, intramuscular pethidine, and no analgesia in a group of primiparous Australian women [52], but did not do an intention to treat analysis. Intrapartum analgesia (both narcotic or epidural analgesia) ($p = 0.04$), smoking ($p < 0.001$), younger age ($p < 0.001$), lower levels of education ($p < 0.001$), and mode of delivery (C-section; $p = 0.025$) were all associated with reduced rates of breastfeeding at 2 and 6 months postpartum. This study can be criticized since the analysis was not performed based on an intention to treat but based on the actual analgesia used. Since there was a high crossover to epidural analgesia, results become unreliable. Again association was demonstrated but causation was not proven. Wiklund et al. performed a retrospective study in 1,170 women [53]. Half of the study population received epidural analgesia and half did not receive epidural analgesia, but could receive pudendal or paracervical blocks. Parenteral opioids were not administered in any study subject. Epidural analgesia resulted in significantly fewer babies being breast fed during the first 4 postpartum hours (OR 3.79, $p < 0.0004$), had a higher chance of receiving artificial milk supplementation (OR 2.19, $p < 0.0012$), and were more likely not to be breast fed at discharge (OR 1.79, $p < 0.043$).

Beilin et al. evaluated the effect of adding an opioid (fentanyl) to the epidural mixture for labor analgesia by prospectively randomizing 177 multiparous women who had previously breastfed at least one child to three study groups [54]. All women received epidural analgesia with either no fentanyl, a low-dose fentanyl, or a high-dose fentanyl (>150 mcg). Breastfeeding was assessed at the first postpartum day and at 6 weeks postpartum. A lactation consultant saw all women. Slightly more women reported breastfeeding difficulty at the first postpartum day, but this did not reach significance (Fig. 16.1). This was also not reported by the lactation consultant (Table 16.2) However, at 6 weeks, more women (19 % vs. 6 % and 2 %) had ceased breastfeeding their child in the high-dose fentanyl group (Figs. 16.1 and 16.2). Of note, the failure rate at 6 weeks was low even in the high-dose fentanyl group.

In a nonrandomized, prospective, observational study 129 primiparous women receiving either epidural analgesia or no analgesia were evaluated for neonatal reactivity, initial suckling behavior, and length of first breastfeed [55]. No differences in neonatal activity and suckling behavior were identified. Neonates from mothers receiving epidural analgesia more frequently had shorter initial breastfeeds (Fig. 16.2). Unfortunately, no information is available on the duration of breastfeeding. The authors concluded that the effects of epidural analgesia on breastfeeding are minimal.

Fig. 16.1 Breastfeeding difficulty at 24 h and breastfeeding failure at 6 weeks postpartum when using no, low, or high doses of fentanyl epidurally [54]

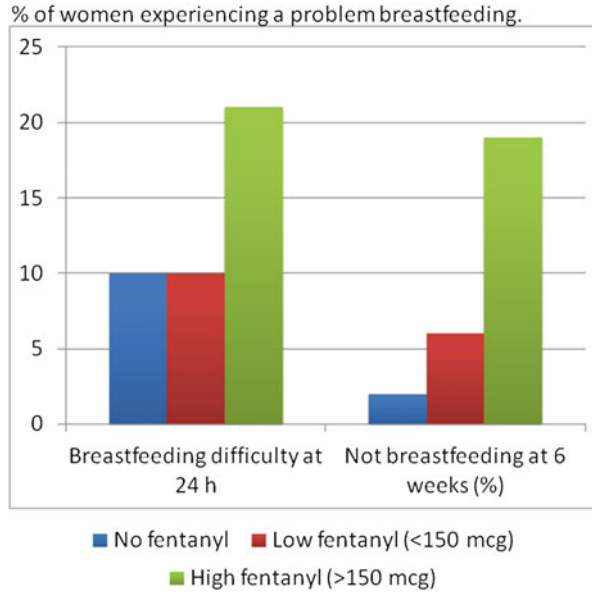


Table 16.2 Evaluation of the effects of no, low, or high doses of epidural fentanyl on breastfeeding success [54]

| Outcomes | No fentanyl group (n = 60) | Intermediate-dose fentanyl group (n = 59) | High-dose fentanyl group (n = 58) |
|------------------------------------------|----------------------------|-------------------------------------------|-----------------------------------|
| Apgar score—1 min | 9 (7–9) | 9 (8–10) | 9 (8–9) |
| Apgar score—5 min | 9 (8–10) | 9 (8–10) | 9 (8–10) |
| Supplemental bottle feed | 71 % | 75 % | 67 % |
| 5 mg oxycodone with 325 mg acetaminophen | 62 % | 49 % | 64 % |
| Duration of epidural analgesia, min | 304 (39–868) | 306 (30–1,091) | 268 (38–775) |
| Total fentanyl in labor, µg | 0 (0–100) | 70 (20–350) | 200 (75–395)* |
| Fentanyl cord, pg/ml | 0 (0–82) | 54 (0–323) | 122 (0–533)* |
| Total bupivacaine in labor, mg | 77.5 (39–175) | 57.5 (24.5–352.5) | 45 (17–86)* |
| Bupivacaine cord, ng/ml | 11.4 (0.1–60.7) | 8.7 (0.1–58.7) | 9.8 (0.1–87) |
| NACS score | 35 (24–40) | 34 (19–40) | 32 (20–40)* |
| BF difficulty 24 h postpartum-mother | 10 % | 10 % | 21 % (p = 0.09) |
| BF difficulty 24 h postpartum-nurse | 40 % | 40 % | 40 % |
| Not BF at 6 weeks | 2 % | 6 % | 19 %* |

This table is based on data from Beilin et al. [54]

BF breast feeding, NACS Neurologic and Adaptive Capacity Scoring System

*p < 0.05 versus groups “No fentanyl” and “Intermediate dose fentanyl”

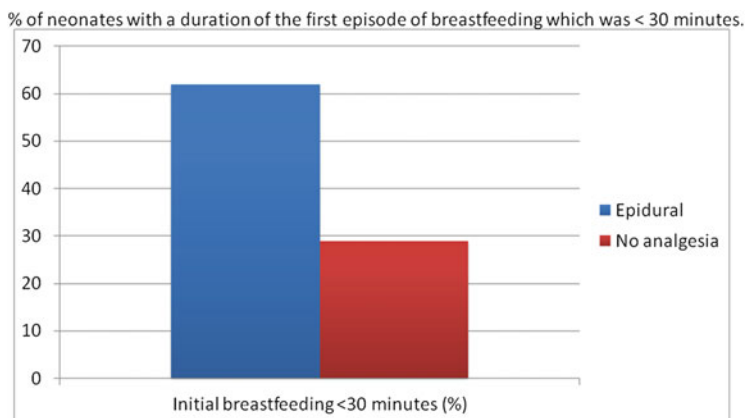


Fig. 16.2 Initial breastfeeding duration <30 min in neonates whose mothers received epidural labora analgesia or no labor analgesia in an observational study by Gizzo et al.

Bai et al. studied 1,280 women of mixed parity in four community hospitals in Hong Kong [56] in this observational prospective study. Bivariate analysis revealed that the induction of labor, parenteral opioid analgesia, and having an emergency Cesarean section shortened the duration of breastfeeding, while receiving epidural analgesia did not affect breastfeeding. When correction for known confounding variables was performed, no intrapartum intervention affected breastfeeding success or duration.

Chang et al. prospectively studied 115 mixed parity women in a nonrandomized, cohort study comparing epidural analgesia with no analgesia [57]. The primary outcome parameters, breastfeeding initiation and breastfeeding success at 4 weeks, were unaffected by the use of epidural analgesia.

Halpern et al. prospectively studied a cohort of nonrandomized women of mixed parity who received either epidural analgesia during labor or no epidural analgesia [58]. The primary outcome variable was breastfeeding success at 6 weeks postpartum. Epidural analgesia did not affect early and late breastfeeding success. Radzynski evaluated in a nonrandomized observational study 56 multiparous women who received either low-dose epidural analgesia or no analgesia [59]. No negative effect of epidural analgesia on initial suckling behavior and early breastfeeding success was observed.

Wieczorek et al. studied a cohort of 99 women using a prospective, observational design [60]. All women were multiparous and received epidural analgesia. Successful breastfeeding at 6 weeks postpartum was reported in 95 % of mothers.

In a secondary analysis of the COMET study data, Wilson et al. reported that various strategies of epidural analgesia had no effect on initial breastfeeding success, time to initial breastfeeding, and duration of breastfeeding as compared to women receiving no analgesia or parenteral pethidine [61]. Parenteral pethidine was associated with less initial breastfeeding success.

Rajan performed a postal questionnaire study to a subset of patients from a previous study and looked at breastfeeding success 6 weeks postpartum [62]. Epidural analgesia had no influence. Also Albani et al. could not demonstrate a difference between epidural analgesia and no analgesia in a prospective, observational study in 1,920 parturients [63]. Riordan et al. studied prospectively, but in a nonrandomized fashion, 129 multiparous women comparing no analgesia, parenteral pethidine and epidural analgesia and found that initial suckling behavior was affected by pethidine, and epidural analgesia, but this did not result in reduced BF success [23]. Baumgarder et al. and Jordan et al. came to similar conclusions: breastfeeding success was not affected by epidural analgesia [64, 65]. However, Jordan et al. noted a negative effect of epidural fentanyl which was dose dependent [65].

16.6 Conclusion

Parenteral opioid labor analgesia negatively affects breastfeeding. The verdict on neuraxial labor analgesia is less clear. There is a paucity of well-designed, randomized, and prospective trials. Retrospective or nonrandomized prospective cohort studies indicate that neuraxial analgesia does not significantly affect breastfeeding success, provided the doses of epidural opioids are not excessively high. There is a need for large, prospective, randomized, and well-designed trials!

References

1. Devroe S, Decoster J, Van de Velde M (2009) Breastfeeding and epidural analgesia during labour. *Curr Opin Anesthesiol* 22:327–329
2. Dalal PG, Bosak J, Cheston B (2014) Safety of the breastfeeding infant after maternal anesthesia. *Pediatr Anesth* 24:359–371
3. World Health Organization (2003) Global strategy for infant and young child feeding. World Health Organization, Geneva
4. Breastfeeding report card – United States (2011) August 11, 2011
5. Shinya I (2000) Drug-therapy for breastfeeding women. *N Engl J Med* 343:118–126
6. Halpern SH (1999) Effect of labor analgesia on breastfeeding success. *Birth* 26:2
7. Quigley MA, Kelly YJ, Sacker A (2007) Breastfeeding and hospitalization for diarrheal and respiratory infection in the United Kingdom Millennium Cohort study. *Pediatrics* 119:e837–e842
8. Lucas A, Morley R, Cole TJ, Lister G, Leeson-Payne C (1992) Breast milk and subsequent intelligence quotient in children born preterm. *Lancet* 339:261–264
9. Chertok IR, Shoham-Vardi I (2008) Infant hospitalization and breastfeeding post-Caesarean delivery. *Br J Nurs* 17:786–991
10. Rudnicka AR, Christopher GO, Richards M, Wadsworth MEJ, Strachan DP (2008) *Am J Clin Nutr* 87:1392–1399
11. Duncan JM, Sears MR (2008) Breastfeeding and allergies: time for a change in paradigm? *Curr Opin Allergy Clin Immunol* 8:398–405
12. Kramer MS, Matush L, Vanilovich I et al (2007) Effect of prolonged and exclusive breast feeding on risk of allergy and asthma: cluster randomised trial. *Br Med J* 335:815–820

13. Freeman K, Bonuck KA, Trombley M (2008) Breastfeeding and infant illness in low-income, minority women: a prospective cohort study of the dose-response relationship. *J Hum Lact* 24:14–22
14. American Academy of Pediatrics Section on Breastfeeding (2012) Policy statement: breastfeeding and human milk. *Pediatrics* 129:e827–e841
15. Maternal and Child Nutrition (2008) National Institute for health and care excellence (NICE) guidance PH1, pp 11–12
16. DiGirolamo AM, Grummer-Strawn LM, Fein SB (2008) Effect of maternity-care practices on breastfeeding. *Pediatrics* 122(S2):S43–S49
17. Anderson D (2011) A review of systemic opioids commonly used for labour pain relief. *J Midwifery Womens Health* 56:222–239
18. Bucklin BA, Hawkins JL, Andeson JR, Ullrich FA (2005) Obstetric anesthesia workforce survey: twenty year update. *Anesthesiology* 103:645–653
19. Saravanakumar K, Garstang JS, Hasan K (2007) Intravenous patient controlled analgesia for labour: a survey of UK practice. *Int J Obstet Anest* 16:221–225
20. Tveit TO, Halvorsen A, Rosland JH (2009) Analgesia for labour: a survey of Norwegian practice – with a focus on parenteral opioids. *Acta Anaesthesiol Scand* 53:794–799
21. Lavand'homme P, Roelants F (2009) Patient controlled intravenous analgesia as an alternative to epidural analgesia during labor: questioning the use of the short acting opioid remifentanyl. Survey in the French part of Belgium (Wallonia and Brussels). *Acta Anaesthesiol Belg* 60:75–82
22. Olofsson C, Ekblom A, Ekman-Ordeberg G, Granstrom L, Irestedt L (1996) Lack of analgesic effect of systemically administered morphine or pethidine on labour pain. *Br J Obstet Gynecol* 103:968–972
23. Riordan J, Gross A, Angeron J, Krumwiede B, Melin J (2000) The effect of labor pain relief medication on neonatal suckling and breastfeeding duration. *J Hum Lact* 16:7–12
24. Wittels B, Scott DT, Sinatra RS (1990) Exogenous opioids in human breast milk and acute neonatal behavior: a preliminary study. *Anesthesiology* 73:864–869
25. Wittels B, Glosten B, Faure EA et al (1997) Postcesarean analgesia with both epidural morphine and intravenous patient controlled analgesia: neurobehavioral outcomes among nursing neonates. *Anesth Analg* 85:600–606
26. Nissen E, Widstrom AM, Lilja G et al (1997) Effects of routinely given pethidine during labour on infants' developing breastfeeding behavior: effects of dose-delivery time interval and various concentrations of pethidine/norpethidine in cord plasma. *Acta Paediatr* 86:201–208
27. Steer PL, Biddle CJ, Marley WS, Mantz RK, Sulik PL (1992) Concentration of fentanyl in colostrum after an analgesic dose. *Can J Anaesth* 39:231–235
28. Leuschen MP, Wolf LJ, Rayburn WF (1990) Fentanyl excretion in breast milk. *Clin Pharm* 9:336–337
29. Ngan Kee WD, Khan KS, Ma KC, Wong AS, Lee BB, Ng FF (2006) Maternal and neonatal effects of remifentanyl at induction of general anesthesia for cesarean delivery: a randomized, double-blind, controlled trial. *Anesthesiology* 104:14–20
30. Kan RE, Hughes SC, Rosen MA, Kessin C, Preston PG, Lobo EP (1998) Intravenous remifentanyl: placental transfer, maternal and neonatal effects. *Anesthesiology* 88:1467–1474
31. Evron S, Glezerman M, Sadan O, Boaz M, Ezri T (2005) Remifentanyl: a novel systemic analgesic for labor pain. *Anesth Analg* 100:233–238
32. Reynolds F (2010) The effects of maternal labour analgesia on the fetus. *Best Pract Res Clin Obstet Gynaecol* 24:289–302
33. Rooks JP (2011) Safety and risks of nitrous oxide labor analgesia: a review. *J Midwifery Womens Health* 56:557–565
34. Rosen MA (2002) Nitrous oxide for relief of labor pain: a systematic review. *Am J Obstet Gynecol* 186:S110–S126
35. Hugh R (1986) Maternal hyperventilation and the fetus. *J Perinat Med* 14:3–17

36. Motoyama EK, Rivard G, Acheson F, Cook CD (1966) Adverse effect of maternal hyperventilation on the foetus. *Lancet* 1:286–288
37. Thalme B, Belfrage P, Raabe N (1974) Lumbar epidural analgesia in labour. Acid-base balance and clinical condition of mother, fetus and newborn child. *Acta Obstet Gynecol Scand* 53:27–35
38. Minnich ME, Brown M, Clark RB (1990) Oxygen desaturation in women in labor. *J Reprod Med* 35:693–696
39. Lederman RP, Lederman E, Work BA Jr, McCann DS (1978) The relationship of maternal anxiety, plasma catecholamines and plasma cortisol to progress in labor. *Am J Obstet Gynecol* 132:495–500
40. Ohno H, Yamashita K, Yahata T, Doi R, Kawamura M (1986) Maternal plasma concentrations of catecholamines and cyclic nucleotides during labor and following delivery. *Res Commun Chem Pathol Pharmacol* 51:183–194
41. Segal S, Wang SY (2008) The effect of maternal catecholamines on the caliber of gravid uterine microvessels. *Anesth Analg* 106:888–892
42. Cascio M, Pygon B, Bennett C, Ramanathan S (1995) Effects of intrathecal fentanyl on plasma catecholamine levels in term laboring parturients. *Anesthesiology* 83:A493
43. Eberle R, Kinsella SM, Arrison E (1995) Maternal plasma catecholamine concentrations after labor analgesia with intrathecal sufentanil. *Anesthesiology* 83:A971
44. Griffin R, Reynolds F (1995) Maternal hypoxaemia during labour and delivery: the influence of analgesia and effect on neonatal outcome. *Anaesthesia* 50:151–156
45. Van de Velde M, Teunkens A, Hanssens M, Vandermeersch E, Verhaeghe J (2004) Intrathecal opioids and fetal heart rate abnormalities: a double-blind, double placebo-controlled trial comparing two forms of combined spinal epidural analgesia with epidural analgesia in labor. *Anesth Analg* 98(4):1153–1159
46. Leighton BL, Halpern SH (2002) The effects of epidural analgesia in labor, maternal and neonatal outcome: a systematic review. *Am J Obstet Gynecol* 186:S69–S77
47. Szabo AL (2013) Intrapartum neuraxial analgesia and breastfeeding outcomes: limitations of current knowledge. *Anesth Analg* 116:399–405
48. Torvaldsen S, Roberts CL, Simpson JM, Thompson JF, Ellwood DA (2006) Intrapartum epidural analgesia and breastfeeding: a prospective cohort study. *Int Breastfeed J* 1:24
49. Camann W (2007) Labor analgesia and breast feeding: avoid parenteral narcotics and provide lactation support. *Int J Obstet Anest* 16:199–201
50. Volman P, Valanne J, Alahuhta S (2004) Breastfeeding problems after epidural analgesia for labour: a retrospective cohort study of pain, obstetrical procedures and breast-feeding practices. *Int J Obstet Anest* 13:25–29
51. Dozier AM, Howard CR, Brownell ER, Wissler RN, Glantz JL, Ternullo SR, Thevenet-Morrison KN, Childs CK, Lawrence RA (2013) Labour epidural anesthesia, obstetric factors and breastfeeding cessation. *Matern Child Health J* 17:689–698
52. Henderson JJ, Dickinson JE, Evans SF, McDonald SJ, Paech MJ (2003) Impact of intrapartum epidural analgesia on breastfeeding duration. *Aust N Z J Obstet Gynaecol* 43:372–377
53. Wiklund I, Norman M, Uvnas-Moberg K, Ransjo-Arvidson AB, Andolf E (2009) Epidural analgesia: breastfeeding success and related factors. *Midwifery* 25:e31–e38
54. Beilin Y, Bodian CA, Weiser J, Hossain S, Arnold I, Feierman DE, Martin G, Holzman I (2005) Effect of labor epidural analgesia with and without fentanyl on infant breastfeeding. *Anesthesiology* 103:1211–1217
55. Gizzo S, Di Gangi S, Saccardi C, Patrelli TS, Paccagnella G, Sansone L, Barbara F, D’Antona D, Nardelli GB (2012) Epidural analgesia during labor: impact on delivery outcome, neonatal well-being and early breastfeeding. *Breastfeed Med* 7:262–268
56. Bai DL, Wu KM, Tarrant M (2013) Association between intrapartum interventions and breastfeeding duration. *J Midwifery Womens Health* 58:25–32
57. Chang ZM, Heaman MI (2005) Epidural analgesia during labor and delivery: effects on the initiation and continuation of effective breastfeeding. *J Hum Lact* 21:305–321

58. Halpern SH, Levine T, Wilson DB, MacDonnell J, Katsiris SE, Leighton BL (1999) Effect of labor analgesia on breastfeeding success. *Birth* 26:83–88
59. Radzyminski S (2003) The effect of ultra low dose epidural analgesia on newborn breastfeeding behaviors. *J Obstet Gynecol Neonatal Nurs* 32:322–331
60. Wiczorek PM, Guest S, Balki M, Shah V, Carvalho JCA (2010) Breastfeeding success rate after vaginal delivery can be high despite the use of epidural fentanyl: an observational cohort study. *Int J Obstet Anest* 19:273–277
61. Wilson MJA, MacArthur C, Cooper GM, Bick D, Moore PAS, Shennan A (2010) Epidural analgesia and breastfeeding: a randomized controlled trial of epidural techniques with and without fentanyl and a non-epidural comparison group. *Anesthesia* 65:145–153
62. Rajan L (1994) The impact of obstetric procedures and analgesia/anaesthesia during labour and delivery on breast feeding. *Midwifery* 10:87–103
63. Albani A, Addamo P, Renghi A, Voltolin G, Peano L, Ivani G (1999) The effect on breastfeeding rate of regional anesthesia technique for cesarean and vaginal childbirth. *Minerva Anestesiol* 65:625–630
64. Baumgarder DJ, Muehl P, Fischer M, Pribbenow B (2003) Effect of labor epidural anesthesia on breastfeeding of healthy full-term newborns delivered vaginally. *J Am Board Fam Pract* 16:7–13
65. Jordan S, Emery S, Bradshaw C, Watkins A, Friswell W (2005) The impact of intrapartum analgesia on infant feeding. *BJOG* 112:927–934

Daniela Perrotta

17.1 Physiology of Transition from Intrauterine to Extrauterine Life

The transition from a fetus to a newborn is the most complex physiologic adaptation that occurs in human experience.

All organ systems are involved at some level, but the major immediate adaptations are the establishment of air breathing concurrently with changes in pressures and flows within the cardiovascular system. Other essential adaptations are striking changes in endocrine function, substrate metabolism, and thermogenesis (Table 17.1).

Preterm deliveries cause particular difficulties for transition and expose the preterm infant to lung injury from mechanical ventilation.

17.2 Endocrine Adaptations to Birth

17.2.1 Cortisol

Cortisol is the major regulatory hormone for terminal maturation of the fetus and for neonatal adaptation at birth [1].

The “cortisol surge” is initiated with the switch from maternal-transplacental-derived corticosteroids to the ability of the fetal adrenal to synthesize and release cortisol under fetal hypothalamic control.

Cortisol increases further during labor to peak at high levels of about 200 mg/mL several hours after term delivery. The increase in fetal cortisol throughout late

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Table 17.1 Essential components for a normal neonatal transition

| |
|------------------------------------------------------------------------------|
| Clearance of fetal lung fluid |
| Surfactant secretion and breathing |
| Transition of fetal to neonatal circulation |
| Decrease in pulmonary vascular resistance and increased pulmonary blood flow |
| Endocrine support of the transition |

Table 17.2 Some effects of cortisol on factors contributing to a normal fetal-to-newborn transition

| |
|-----------------------------------------|
| Lung maturation: anatomy and surfactant |
| Clearance of fetal lung fluid |
| Increased beta receptor density |
| Gut functional maturation |
| Maturation of thyroid axis |
| Control energy substrate metabolism |
| Regulate catecholamine release |

gestation supports multiple physiologic changes that facilitate normal neonatal adaptation. This normal increase in cortisol supports an integrated transition following birth (Table 17.2). Cesarean section without labor at term blunts the postnatal rise in cortisol, and the cortisol responses to preterm birth are also attenuated because of the unresponsiveness and immaturity of the adrenal gland [2].

17.2.2 Catecholamines

The catecholamine surge is primarily responsible for the increase in blood pressure following birth, for the adaptation of energy metabolism with the support of the primary substrates for metabolism after birth (glucose and fatty acids), and for initiating thermogenesis from brown fat. The preterm secretes more catecholamines because the organ systems are less responsive: higher concentration thresholds for response and lower responses. Cesarean section of the unlabored fetus depresses catecholamine release. Catecholamine release at birth can be viewed as the “gas” that drives the adaptive responses. However, fetal exposure to cortisol is the “carburetor” that is the potent regulator of the responses of the newborn to catecholamines. Antenatal corticosteroid treatments decrease catecholamine levels in preterm infants compared with unexposed infants [3].

17.2.3 Thyroid Hormones

The thyroid axis matures in late gestation in parallel to the increase in cortisol with increased thyroid-stimulating hormone (TSH), T3 and T4 levels [4]. Following term birth, TSH quickly peaks and decreases, and T3 and T4 increase in response primarily to the increased cortisol, to cord clamping, and to the cold stimulus of birth.

17.3 Metabolic Adaptations

17.3.1 Energy Metabolism

Fetal energy needs are supported primarily by the transplacental transfer of glucose to the fetus [5]. Although the fetal liver is capable of gluconeogenesis from early gestation, this process is minimal during normal fetal homeostasis. Rather, as term approaches, glucose and other substrates are being stored as glycogen and fat in anticipation of birth in the high insulin and low glycogen fetal environment. With delivery and cord clamping, the maternal glucose supply is removed, and plasma glucose levels normally fall over the early hours after birth. The glucose and free fatty acid levels are accompanied by a fall in insulin and by a marked surge in plasma glucagon levels, the normal glucose homeostatic hormones. However, the large catecholamine release and increase in cortisol are probably the major acute regulators of plasma glucose and free fatty acid levels in the immediate newborn period.

17.3.2 Thermoregulation

Fetal body temperature is about 0.5 °C above the maternal temperature. Although fetus produces heat from metabolism, that heat is effectively dissipated across the placenta and fetal membranes. At birth, the sympathetic release resulting from the redundant stimuli of increased oxygenation, ventilation, cord occlusion, and a cold stimulus to the skin activates thermogenesis by brown adipose tissue. This thermogenic potential response has developed during late gestation by an increase in brown adipose tissue around the kidney and in the intrascapular areas of the back to become about 1 % of fetal weight at term [6].

The preterm is at a major disadvantage for thermoregulation following birth, as brown adipose tissue has not developed in quantity or response potential for a cold stress.

17.4 Cardiovascular Adaptations

Profound changes in the cardiovascular system occur after delivery in response to removal of the low resistance placenta as the source of fetal gas exchange and nutrition. The major changes are an increase in the cardiac output and transition of fetal circulation to an adult type of circulation. Increased cardiac output is required to provide for increase in basal metabolism, work of breathing, and thermogenesis. In the close-to-term fetus, the combined ventricular output is about 450 mL/kg/min, with the right ventricular output accounting for two-third of the cardiac output and the left ventricle ejecting one-third of the cardiac output [7].

Soon after birth, the circulation changes from “parallel” to “series,” where the right ventricular output equals the left ventricular output. The cardiac output nearly doubles after birth to about 400 mL/kg/min for the right ventricle and the same amount for the left ventricle. This increase in cardiac output is proportional to the rise in oxygen consumption. The organs experiencing increased blood flow after birth are lungs, heart, kidney, and the gastrointestinal tract [8].

Although the precise mechanisms mediating increased cardiac output after birth are not known, the increase in cortisol and vasoactive hormones, which include catecholamines, the rennin-angiotensin system, vasopressin, and thyroid hormone, contribute to support the blood pressure and cardiovascular function [7].

In the fetus, the relatively well-oxygenated blood from the placenta is delivered via the umbilical cord and ductus venosus. The ductus venosus blood enters the right atrium from the inferior vena cava and is directed preferentially to the left atrium by the foramen ovale and subsequently delivered mainly to the brain and the coronary circulation by the fetal left ventricle. The right ventricle is the predominant ventricle in the fetus, and most of the right ventricular output goes to the descending aorta via the ductus arteriosus because small amount of blood enters the pulmonary circulation. With birth and removal of the low resistance placenta, pulmonary circulation blood flow increases. Shortly after birth, functional closure of the ductus arteriosus begins. The main mechanisms contributing to the high pulmonary vascular resistance in the fetal lung are the low oxygen tension and the low pulmonary blood flow, which suppress the synthesis and release of nitric oxide (NO) and prostaglandin I₂ from the pulmonary endothelium [8].

Fetal exposure to hypoxia will increase the already high pulmonary vascular resistance and hyperoxia will decrease pulmonary vascular resistance and increase fetal pulmonary blood flow [9]. With delivery, ventilation, and oxygenation, NO and PGI₂ increase with a rapid fall in pulmonary vascular resistance.

The cardiovascular transition at birth is also modulated by corticosteroids.

The normal oxygen saturation of fetal blood in the left atrium is about 65 %. During labor, the human fetus tolerates oxygen saturations as low as 30 % without developing acidosis [10]. After birth, the preductal saturation in healthy term infants gradually increases to about 90 % at 10 min of age [11]. This knowledge is important to avoid unnecessary administration of supplemental oxygen during resuscitation.

17.5 Lung Adaptations

17.5.1 Fetal Lung Fluid

The most essential adaptation to birth is the initiation of breathing, but the airspaces of the fetal lung are filled with fetal lung fluid.

Fetal lung fluid is secreted by the airway epithelium as a filtrate of the interstitial fluid of the lung by the active transport of chloride [12]. Consequently, the chloride content of fetal lung fluid is high and protein content is very low.

Production and maintenance of the normal volume of fetal lung fluid are essential for normal lung growth. The electrochemical gradient for the production of fetal lung fluid is substantial and can overdistend the airspaces.

The endocrine adaptations that begin before delivery are critical to fluid clearance. Cortisol, thyroid hormones, and catecholamines all increase and shut down the active chloride-mediated secretion of fetal lung fluid and activate the basal Na^+ , K^+ , ATPase of type II cells on the airway epithelium. Sodium in fetal lung fluid enters the apical surfaces of type II cells and is pumped into the interstitium. Water and other electrolytes follow sodium passively, thus removing fluid from the airways.

The frequent clinical scenario in which retained lung fluid contributes to poor respiratory adaptation is the operative delivery of infants who were not in labor. These infants do not increase their oxygen saturations as quickly as vaginally delivered term infants [11], and there is an increased incidence of transient tachypnea of the newborn and other respiratory morbidities.

17.5.2 Breathing at Birth

The essential component to neonatal adaptation to birth is the maintenance of adequate respiratory effort. The stimuli changing the fetal breathing pattern virtually instantaneously to continuous breathing remain incompletely defined and probably are redundant, as are the stimuli for other adaptations to birth.

The net effect is that the normal fetal-to-neonatal transition results in the rapid onset of vigorous breathing because of the combined stimuli of cord clamping (and the probable removal of rapidly catabolized prostaglandins that suppress breathing), diffuse tactile and cold stimuli that act centrally, and changes in PCO_2 and PO_2 levels in the blood. The newborn will not initiate breathing if hypoxia is severe. Remarkably, in the absence of hypoxia, virtually all term infants will effectively initiate breathing [13].

17.5.3 Surfactant and Lung Adaptation

The adequate development of the fetal lung to support gas exchange is the essential adaptation in preparation for birth. During the last third of gestation, the fetal lung septates into about four million distal saccules (respiratory bronchioles and alveolar ducts) derived from the 17 generations of airways by about 32 weeks and then further separates to form alveoli [14].

In parallel, the lung parenchymal tissue mass decreases relative to body weight such that the potential gas volume of the airways and alveoli increases greatly. Concurrently, from about 22 weeks' gestational age, surfactant lipid and the lipophilic proteins SP-B and SP-C begin to be synthesized and aggregated into lamellar bodies in the maturing type II cells.

The lamellar bodies are the storage and secretory packets for the essential biophysically active components of surfactant. As the lung matures, more and more of the lamellar bodies are released into fetal lung fluid and subsequently mix with amniotic fluid or are swallowed. By term, type II cells in the fetal lung contain much more surfactant than does the adult lung, and this large pool of surfactant is poised for release before and at delivery.

As delivery approaches, fetal lung fluid secretion ceases and its volume may decrease. Simultaneously, surfactant is secreted into the fetal lung fluid with labor, which will increase the surfactant concentration in the fetal lung fluid [15].

Subsequently, the initiation of ventilation following birth causes alveolar stretch and therefore deformations of type II cells, which works as secretion signal. The large increase in catecholamines following delivery probably further stimulates surfactant secretion.

The preterm lung has several disadvantages for transition to air breathing. The structurally immature lung has less potential gas volume relative to body weight and metabolic needs, and secretion of fetal lung fluid may not cease before and after delivery, which will delay clearance of fetal lung fluid. Further, the amount of surfactant stored in type II cells is low, and, thus, less surfactant can be secreted in response to birth. The result is a lower concentration of surfactant to form a surface film and stabilize the lung.

17.5.4 Injury of the Preterm Lung

The transition from a fetus to a newborn requires the initiation of breathing, clearance of fluid from airways, and ventilation of the distal airspaces. Healthy newborns inflate their lungs at birth by generating large negative pressure breaths, which pull the lung fluid from the airways into the distal airspaces. The infant continues to clear lung fluid with subsequent inflations [13].

Many preterm or asphyxiated term infants do not have adequate spontaneous respirations at birth and require positive-pressure ventilation. Premature infants have immature lungs that are more difficult to ventilate because of inadequate surfactant to decrease surface tension and maintain functional residual capacity (FRC). The initial ventilation of the preterm lung will occur before much of the endogenous surfactant is secreted, and surfactant therapy cannot practically be given before the initiation of ventilation. The movement of fluid at the air interface across epithelial cells generates high surface forces that distort the cells and injure the epithelium of the small airways [16].

Continuous positive airway pressure or PEEP should minimize the movement of fluid in the airways, and surfactant will lower the pressure required to move fluid into the small airways and decrease the injury from fluid movement [17].

17.6 Neonatal Resuscitation

The following guidelines are an interpretation of the evidence presented in the *2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations* [18].

They apply primarily to newly born infants undergoing transition from intrauterine to extrauterine life, but the recommendations are also applicable to neonates who have completed perinatal transition and require resuscitation during the first few weeks to months following birth.

17.6.1 Overview

Neonatal resuscitation skills are essential for all healthcare providers who are involved in the delivery of newborns. The transition from fetus to newborn requires intervention by a skilled individual or team in approximately 10 % of all deliveries. Less than 1 % require extensive resuscitative measures [18, 19].

The newly born infants who do not require resuscitation can generally be identified by a rapid assessment of the following three characteristics:

- Term gestation?
- Crying or breathing?
- Good muscle tone?

If the answer to all three of these questions is “yes,” the baby does not need resuscitation and should not be separated from the mother.

The baby should be dried, placed skin-to-skin with the mother, and covered with dry linen to maintain temperature. Observation of breathing, activity, and color should be ongoing.

If the answer to any of these assessment questions is “no,” the infant should receive one or more of the following four categories of action in sequence:

- (1) Initial steps in stabilization (provide warmth, clear airway if necessary, dry, stimulate)
- (2) Ventilation
- (3) Chest compressions
- (4) Administration of epinephrine and/or volume expansion.

Approximately 60 s (“the Golden Minute”) are allotted for completing the initial steps, reevaluating, and beginning ventilation if required (see Fig. 17.1). The decision to progress beyond the initial steps is determined by simultaneous assessment of two vital characteristics: respirations (apnea, gasping, or labored or unlabored breathing) and heart rate (whether greater than or less than 100 beats per minute).

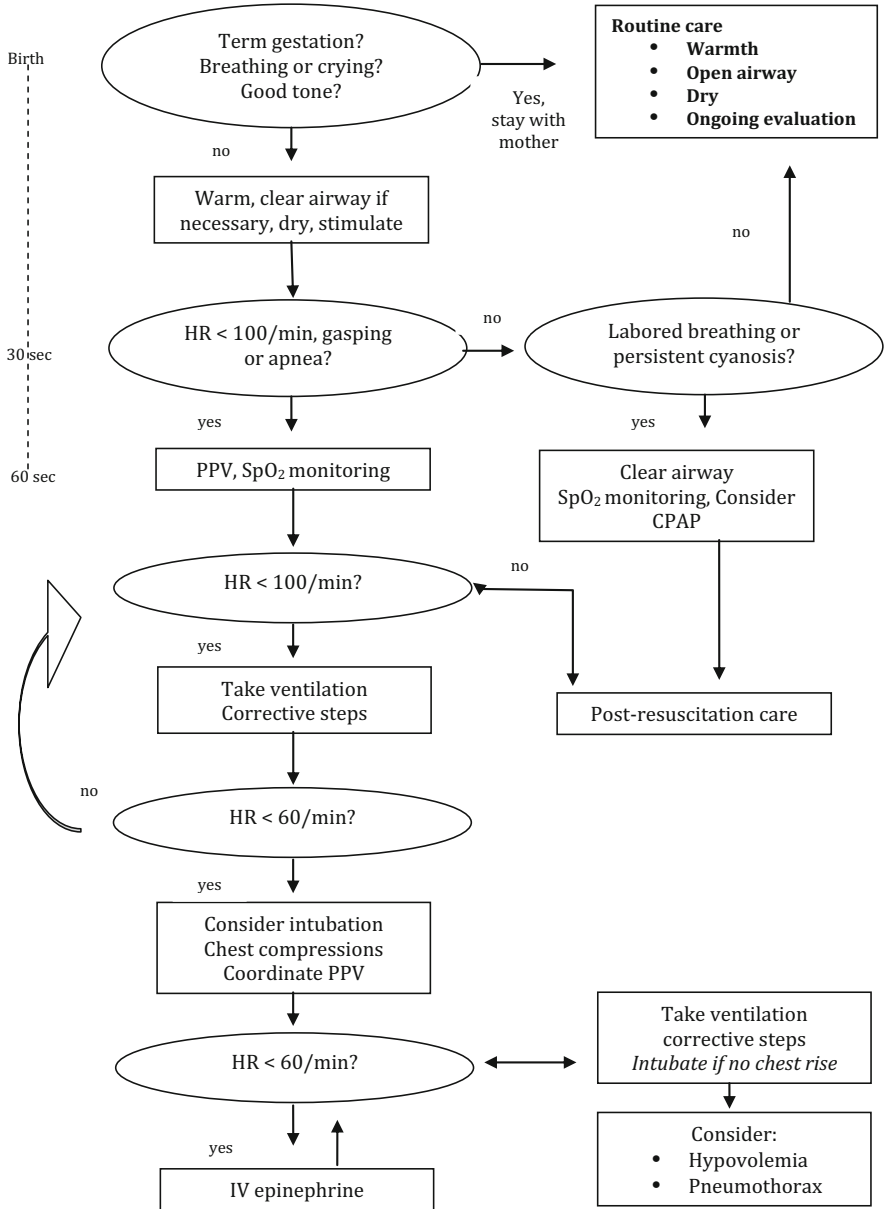


Fig. 17.1 Algorithm neonatal resuscitation

Assessment of heart rate should be done by intermittently auscultating the precordial pulse. When a pulse is detectable, palpation of the umbilical pulse can also provide a rapid estimate of the pulse and is more accurate than palpation at other sites [20, 21].

Once positive-pressure ventilation or supplemental oxygen administration is begun, assessment should consist of simultaneous evaluation of three vital characteristics: heart rate, respirations, and the state of oxygenation, the latter optimally determined by a pulse oximeter.

17.6.2 Anticipation of Resuscitation Need

Anticipation, adequate preparation, accurate evaluation, and prompt initiation of support are critical for successful neonatal resuscitation.

With careful consideration of risk factors, the majority of newborns who will need resuscitation can be identified before birth.

The communication between the people caring for the mother and those responsible for resuscitation of the newly born is very important, and it should include details of ante partum and intrapartum maternal medical conditions and treatment as well as specific indicators of fetal condition (fetal heart rate monitoring, lung maturity, and ultrasonography).

If a preterm delivery (less than 37 weeks of gestation) is expected, special preparations will be required [22].

Preterm babies have immature lungs that may be more difficult to ventilate and are also more vulnerable to injury by positive-pressure ventilation (PPV). Preterm babies also have immature blood vessels in the brain that are prone to hemorrhage; thin skin and a large surface area, which contribute to rapid heat loss; increased susceptibility to infection; and increased risk of hypovolemic shock related to small blood volume.


17.6.3 Apgar Score

The Apgar score (Table 17.3) describes the condition of the newborn infant immediately after birth [23] and, when properly applied, is a tool for standardized assessment. It also provides a mechanism to record fetal-to-neonatal transition. An Apgar score of 0–3 at 5 min may correlate with neonatal mortality but alone does not predict later neurologic dysfunction. The Apgar score is affected by gestational age, maternal medications, resuscitation, and cardiorespiratory and neurologic conditions. Low 1- and 5-min Apgar scores alone are not conclusive markers of an acute intrapartum hypoxic event.

17.6.3.1 Limitations of the Apgar Score

It is important to recognize the limitations of the Apgar score. The Apgar score is an expression of the infant's physiologic condition, has a limited time frame, and includes subjective components. In addition, the biochemical disturbance must be significant before the score is affected. Elements of the score such as tone, color, and reflex irritability partially depend on the physiologic maturity of the infant. The healthy preterm infant with no evidence of asphyxia may receive a low score only because of immaturity [24].

Table 17.3 Apgar score

| Score | Condition of the newborn | | | |
|--------------------------|--------------------------|-----------------------------|----------------------------------|-----------------------------------------------------------------------------------|
| 0–3 | Severely depressed | | | |
| 4–6 | Moderately depressed | | | |
| 7–10 | Excellent condition | | | |
| | 0 points | 1 point | 2 points | Points totaled |
| Activity (muscular tone) | Absent | Arms and legs flexed | Active movement |  |
| Pulse | Absent | Below 100 bpm | Over 100 bpm | |
| Reflex irritability | Flaccid | Some flexion of extremities | Active motion (cough, pull away) | |
| Skin color | Blue, pale | Body pink, extremities blue | Completely pink | |
| Respiration | Absent | Slow, irregular | Vigorous cry | |

A number of factors may influence an Apgar score, including but not limited to drugs, trauma, congenital anomalies, infections, hypoxia, hypovolemia, and pre-term birth. The incidence of low Apgar scores is inversely related to birth weight, and a low score is limited in predicting morbidity or mortality [25]. Accordingly, it is inappropriate to use an Apgar score alone to establish the diagnosis of asphyxia.

17.6.4 Umbilical Cord Clamping

For healthy term infants delaying cord clamping for at least 1 min *or* until the cord stops pulsating following delivery improves iron status through early infancy [26].

For preterm babies in good condition at delivery, delaying cord clamping for up to 3 min results in increased blood pressure during stabilization, a lower incidence of intraventricular hemorrhage, and fewer blood transfusions.

There are limited data on the hazards or benefits of delayed cord clamping in the nonvigorous infant [18].

17.6.5 Initial Measures

17.6.5.1 Temperature Control

Babies are born small and wet. They get cold very easily, especially if they remain wet and in a draught.

Whatever the situation, it is important that the baby does not get cold at this stage. If intervention is required in a term or near-term baby, dry him, remove the wet towels, and cover the newborn with dry towels.

Significantly, preterm babies are best placed, without drying, into food-grade plastic wrapping under a radiant heater. This process will provide significant stimulation and will allow time to assess tone, breathing, and heart rate. Reassess these observations regularly every 30 s or so throughout the resuscitation process, but it is the heart rate which is the key observation. The first sign of any improvement in the baby will be an increase in heart rate. Consider the need for help; if needed, ask for help immediately.

A healthy baby will be born blue but will have good tone, will cry within a few seconds of delivery, and will have a good heart rate within a few minutes of birth (the heart rate of a healthy newborn baby is about 120–150/min). A less healthy baby will be blue at birth, will have less good tone, may have a slow heart rate (less than 100/min), and may not establish adequate breathing by 90–120 s. An ill baby will be born pale and floppy, not breathing, and with a slow, very slow, or undetectable heart rate.

A pulse oximeter is probably the best way of assessing heart rate and oxygenation in the delivery room. With practice, it is possible to attach a pulse oximeter probe and to obtain a useful reading of heart rate and oxygen saturation about 1–2 min after delivery.

17.6.5.2 Clear the Airway

Before the baby can breathe effectively, the airway must be open. The best way to achieve this is to place the baby on his back with the head in the neutral position. Most newborn babies have a relatively prominent occiput, which will tend to flex the neck if the baby is placed on his back on a flat surface. This can be avoided by placing some support under the shoulders of the baby, but be careful not to overextend the neck. If the baby is very floppy (i.e., has no or very little tone) it may also be necessary to apply chin lift or jaw thrust. These maneuvers are effective for the majority of babies requiring airway stabilization at birth.

Airway suction immediately following birth should be reserved for babies who have obvious airway obstruction to spontaneous breathing or who require PPV. In these situations, direct visualization and suction of the oropharynx should be performed.

When Meconium is present, its aspiration before delivery, during birth, or during resuscitation can cause severe meconium aspiration syndrome (MAS). Historically a variety of techniques have been recommended to reduce the incidence of MAS. Suctioning of the oropharynx before delivery of the shoulders was considered routine until a randomized controlled trial demonstrated it to be of no value [27].

Elective and routine endotracheal intubation and direct suctioning of the trachea were initially recommended for all meconium-stained newborns until a randomized controlled trial demonstrated that there was no value in performing this procedure in babies who were vigorous at birth [28]. Although depressed infants born to mothers with meconium-stained amniotic fluid (MSAF) are at increased risk to

develop MAS, tracheal suctioning has not been associated with reduction in the incidence of MAS or mortality in these infants.

In the absence of randomized, controlled trials, there is insufficient evidence to recommend a change in the current practice of performing endotracheal suctioning of nonvigorous babies with meconium-stained amniotic fluid (Class IIb, LOE C). However, if attempted intubation is prolonged and unsuccessful, bag-mask ventilation should be considered, particularly if there is persistent bradycardia [18].

17.6.5.3 Assessment of Oxygen Need and Administration of Oxygen

There is substantial evidence that blood oxygen levels in infants do not always reach typical extrauterine values until 10 min post birth. Cyanosis can appear for up to several minutes after birth but is a poor indicator of oxygen saturation. Optimal management of oxygen is critical, given both a lack and excess of oxygenation can be detrimental to the infant's health.

Pulse oximetry is not routinely recommended following delivery but should be used when a resuscitation is expected, when PPV will be needed for more than a few breaths, in the setting of sustained cyanosis, or when supplementary oxygen is used.

The probe should be applied to a preductal site on the infant (right upper extremity), so that values can be compared to published data [18].

The target oxygen saturation level is as follows:

- 60–65 % at 1 min
- 65–70 % at 2 min
- 70–75 % at 3 min
- 75–80 % at 4 min
- 80–85 % at 5 min
- 85–95 % at 10 min

In order to achieve the target oxygen saturation and avoid hypo- or hyperoxemia, resuscitation should begin with room air or blended room air and oxygen (proceed to higher concentrations of supplemental oxygen only as clinically indicated). In the case of bradycardia (HR < 60/min) that persists after 90 s of resuscitation with room air or low concentrations of supplementary oxygen, the oxygen concentration should be increased until the infant's heart rate is greater than 60/min.

Following the above measures, commence PPV if the infant is apneic, is gasping, or has a pulse <100/min. Continue PPV until these signs of distress are correct.

17.6.5.4 Assisted Ventilation

Initial breaths inflate the lungs and create the FRC. This can occur spontaneously or with assisted inflation by PPV.

In infants requiring assisted ventilation, the improving heart rate and the chest rise are the main indicators of proper ventilation.

As a general guideline, use the minimal inflation necessary and provide assisted ventilation at a rate of 40–60 breaths/min to reach or preserve a heart rate greater than 100/min.

The use of CPAP and PEEP or endotracheal intubation and mechanical ventilation in preterm infants with spontaneous breathing but respiratory distress is recommended.

17.6.5.5 Laryngeal Mask Airways

Use of a laryngeal mask is recommended if the facemask ventilation and tracheal intubation are unsuccessful in newborn delivered at or later than 34 weeks gestation and weighing more than 2,000 g.

17.6.5.6 Endotracheal Intubation

It may be needed at various steps of resuscitation, in part depending on the skillset of available providers.

Initial suctioning of depressed newborns with meconium-stained amniotic fluid should be considered,

- When the bag-mask ventilation is not effective or is performed for a lengthy period
- When chest compressions are required
- In the setting of certain congenital abnormalities.

End-tidal CO₂ detection confirms proper tube placement.

17.6.5.7 Chest Compression

Almost all babies needing help at birth will respond to successful lung inflation with an increase in heart rate followed quickly by normal breathing. However, in some cases chest compression is necessary.

Chest compression should be started only when the lungs have been aerated successfully.

In babies, the most efficient method of delivering chest compression is to grip the chest in both hands in such a way that the two thumbs can press on the lower third of the sternum, just below an imaginary line joining the nipples, with the fingers over the spine at the back.

Compress the chest quickly and firmly, reducing the antero-posterior diameter of the chest by about one-third.

The ratio of compressions to inflations in newborn resuscitation is 3:1 (90 compressions and 30 breaths/min).

Compressions should be continued until spontaneous heart rate is at least 60 or more beats per minute.

17.6.5.8 Drugs

Epinephrine

Newborn infant bradycardia typically results from an oxygenation or ventilation deficiency. If the heart rate is less than 60/min after adequate ventilation, 100 % supplementary oxygen and chest compressions, epinephrine or volume expansion may be required.

The epinephrine is recommended to be administered intravenously. The suggested intravenous dose is 0.01–0.03 mg/kg (0.1 mL/kg of 1:10,000 solution). If endotracheal tube administration is performed, the recommended dosage is 0.05–0.1 mg/kg at the same concentration [18].

Volume expansion

Very rarely, the heart rate cannot increase because the baby has lost significant blood volume. If this is the case, the use of isotonic crystalloid solution or blood is suggested at the recommended dosage of 10 mL/kg.

17.7 Post-Resuscitation Care

17.7.1 Hypoglycemia

Newborns with hypoglycemia have an increased risk of brain injury following hypoxic-ischemic events. IV glucose infusion should be considered following resuscitation.

17.7.2 Therapeutic Hypothermia

Term or near-term infants, with evolving moderate to severe hypoxic-ischaemic encephalopathy, should be treated with therapeutic hypothermia.

Whole body cooling and selective head cooling are both appropriate strategies 304–308. Cooling should be initiated and conducted under clearly defined protocols with treatment in neonatal intensive care facilities and the capabilities for multidisciplinary care. Treatment should be consistent with the protocols used in the randomized clinical trials, i.e., commence within 6 h, continue for 72 h and rewarm over at least 4 h [18].

17.7.3 Withholding and Discontinuing Resuscitation

Resuscitation might be withheld if early death is almost certain due to gestational age, extremely low birth weight, or certain devastating congenital anomalies.

Parental desires should be considered and supported in the case of unclear prognosis. Cessation of resuscitative efforts can be considered in a pulseless newborn who does not regain a pulse after at least 10 min of resuscitative efforts [18].

References

1. Liggins GC (1994) The role of cortisol in preparing the fetus for birth. *Reprod Fertil Dev* 6:141–150
2. Watterberg K (2011) Fetal and neonatal adrenalcortical physiology. In: Polin R, Fox W, Abman S (eds) *Fetal and neonatal physiology*, 4th edn. Elsevier, Philadelphia, PA, pp 1995–2004
3. Kallio J, Karlsson R, Toppari J et al (1998) Antenatal dexamethasone treatment decreases plasma catecholamine levels in preterm infants. *Pediatr Res* 43:801–807
4. Fisher DA (2008) Thyroid system immaturities in very low birth weight premature infants. *Semin Perinatol* 32:387–397
5. Ward Platt M, Deshpande S (2005) Metabolic adaption at birth. *Semin Fetal Neonatal Med* 10:341–350
6. Power G, Blood A (2011) Thermoregulation. In: Polin R, Fox W, Abman S (eds) *Fetal and neonatal physiology*, 4th edn. Elsevier, Philadelphia, PA, pp 615–624
7. Heymann MA, Iwamoto HS, Rudolph AM (1981) Factors affecting changes in the neonatal systemic circulation. *Annu Rev Physiol* 43:371–383
8. Gao Y, Raj J (2010) Regulation of the pulmonary circulation in the fetus and newborn. *Physiol Rev* 90:1291–1335
9. Teitel DF, Iwamoto HS, Rudolph AM (1990) Changes in the pulmonary circulation during birth-related events. *Pediatr Res* 27:372–378
10. Garite TJ, Dildy GA, McNamara H et al (2000) A multicenter controlled trial of fetal pulse oximetry in the intrapartum management of nonreassuring fetal heart rate patterns. *Am J Obstet Gynecol* 183:1049–1058
11. Dawson JA, Kamlin CO, Vento M et al (2010) Defining the reference range for oxygen saturation for infant after birth. *Pediatrics* 125:e1340–e1347
12. Jain L, Eaton DC (2006) Physiology of fetal lung fluid clearance and the effect of labor. *Semin Perinatol* 30:34–43
13. Alvaro R, Rigatt H (2011) Breathing in fetal life and onset and control of breathing in the neonate. In: Polin R, Fox W, Abman S (eds) *Fetal and neonatal physiology*, 4th edn. Elsevier, Philadelphia, PA, pp 980–992
14. Burri PH (2006) Structural aspects of postnatal lung development-alveolar formation and growth. *Biol Neonate* 89:313–322
15. Faridy EE, Thliveris JA (1987) Rate of secretion of lung surfactant before and after birth. *Respir Physiol* 68:269–277
16. Jobe AH, Ikegami M (1998) Mechanisms initiating lung injury in the preterm. *Early Hum Dev* 53:81–94
17. Davis PG, Colin JM, Louise SO (2009) Non-invasive respiratory support of preterm neonates with respiratory distress: continuous positive airway pressure and nasal intermittent positive pressure ventilation. *Semin Fetal Neonatal Med* 14:14–20
18. Perlman JM, Wyllie J, Kattwinkel J et al (2010) Neonatal resuscitation: 2010 international consensus on cardiopulmonary resuscitation and emergency cardiovascular care science with treatment recommendations. *Circulation* 122:S516–S538
19. Barber CA, Wyckoff MH (2006) Use and efficacy of endotracheal versus intravenous epinephrine during neonatal cardiopulmonary resuscitation in delivery room. *Pediatrics* 118:1028–1034
20. Owen CJ, Wyllie JP (2004) Determination of heart rate in baby at birth. *Resuscitation* 60:213–217
21. Kamlin CO, Dawson JA, O'Donnell CP et al (2008) Accuracy of pulse oximetry measurement of heart rate of newborn infants in delivery room. *J Pediatr* 152:756–760
22. Kattwinkel J (ed) (2011) *Textbook of neonatal resuscitation*, 6th edn. American Academy of Pediatrics, Elk Grove Village
23. Papile LA (2001) The Apgar score in the 21st century. *N Engl J Med* 344:519–520

24. Catlin EA, Carpenter MW, Brann BSIV et al (1986) The Apgar score revisited: influence of gestational age. *J Pediatr* 109:865–868
25. Hegyi T, Carone T, Anwar M et al (1998) The Apgar score and its components in the preterm infant. *Pediatrics* 101:77–81
26. Zaramella P, Freato F, Quaresima V et al (2008) Early versus late cord clamping: effects on peripheral blood flow and cardiac function in term infants. *Early Hum Dev* 84:195–200
27. Vain NE, Szyld EG, Prudent LM et al (2004) Oropharyngeal and nasopharyngeal suctioning of meconium-stained neonates before delivery of their shoulders: multicentre, randomized controlled trial. *Lancet* 364:597–602
28. Wiswell TE, Gannon CM, Jacob J et al (2000) Delivery room management of the apparently vigorous meconium-stained neonate: results of the multicenter, international collaborative trial. *Pediatrics* 105(1 pt 1):1–7

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Epidural analgesia is considered the most effective method of pain relief during labor. Advances, in both drugs and equipment, have meant a great improvement in its safety and effectiveness. However, epidural analgesia may fail in its main objective, which is to provide effective analgesia or anesthesia and some of these failures are due to technical problems.

In this chapter we will discuss the:

- 18.1. Definition of ineffective epidural during labor: its causes and possible solutions
- 18.2. Epidural catheter dislodgement
- 18.3. Unilateral analgesia
- 18.4. Subdural block
- 18.5. Breakthrough pain (BP) during labor analgesia: causes and solutions.

These points will be analyzed from a technical and practical point of view in relation to analgesia during labor.

18.1 Definition of Ineffective Epidural During Labor: Causes and Possible Solutions

The failure in providing adequate analgesia is obviously an unpleasant and unsatisfactory experience and can also be a source of litigation and claims. This risk of failed analgesia due to technical or other reasons should be clearly discussed with the parturient before the procedure.

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The vast majority of unsatisfactory epidural blocks occur in obstetric anesthesia, being due in most cases to the fact that the tip of the catheter and therefore the local anesthetic solution are not in the desired site or to the existence of anatomical abnormalities in the epidural space [1].

One of the main difficulties is to know and define exactly what is meant by failure of analgesia. Definitions given for a failed epidural cover a spectrum ranging from insufficient analgesia to catheter migration out of the epidural space.

18.1.1 Definition of Ineffective Epidural During Labor

Failed neuraxial analgesia can be described as an intrathecal or epidural injection of local anesthetic that results in the absence of nerve block, unilateral analgesia, and patched or inadequate density of block despite apparently adequate spread. In obstetrics, a failed block is defined as the neuraxial technique (epidural, spinal, or combined spinal epidural), which results in inadequate analgesia or anesthesia or absence of blocking after administration of a suitable dose [2].

According to Eappen et al. [3] failed epidural in obstetrics is defined as any reason requiring catheter replacement after the catheter was secured to the back with adhesive tape or a greater than three-dermatomal segment discrepancy between the analgesic level as assessed by temperature (ice) sensation in a patient complaining of pain after the initial bolus of epidural bupivacaine. Initial studies in relation to failed analgesia considered indirect signs of failure, the catheter replacement rate. More recently, the degree of pain relief is assessed with a visual analogue scale (VAS) 30 min after initiation of neuraxial analgesia [4, 5], to define failed analgesia.

18.1.2 Epidemiology

Failure rates currently reported in the literature range from 0.9 % to 24 % [6, 7] and are more frequent than generally recognized.

Pan et al. [8] evaluated the failure rate in a series of 19,259 neuraxial procedures performed over 3 years. The overall failure rate was 12 % due to various reasons, technical or not. According to these authors, the failure rate after a combined spinal epidural (CSE) was significantly lower than after an epidural (10 % vs. 14 %). Excluding the initial failures, only 6.8 % of catheters inserted for analgesia in labor required replacement due to insufficient analgesia. Their replacement rate is higher than the rate published by Peach et al. (4.7 %) [6] but less than the rate published by Eappen et al. (13.1 %) [3] or by Crawford (15.4 %) [9].

18.1.3 Block Assessment and Diagnosis

The key to achieve an adequate block is to make an early diagnosis of failure, in order to solve the problem as soon as possible.

To evaluate the appropriate extension of neuraxial blockade, the use of cold solutions or a sharp stimulus starting from the lateral thigh and sliding cephalic and caudal on both sides is recommended.

18.1.4 Factors Associated with Failure of Neuraxial Analgesia

According to Agaram et al. [10] the experience of the people who perform the technique, the method used to localize the epidural space, the appearance of paresthesias when inserting the catheter, the catheter insertion difficulty, the volume of local anesthetic used (<12 mL or >12 mL), the total dose of local anesthetic used, and the use of fentanyl may be factors associated with a higher failure rate of analgesia. However, after analyzing these factors, none were significantly associated with the incidence of failed epidural. The best predictors of failure of epidural analgesia, according to these authors, were cervical dilation above 7 cm, the existence of previous opioid use, a history of prior failed epidural analgesia, and the performance of the technique by a resident [10].

It is desirable to systematize the factors that have been associated with the failed or inadequate analgesia and broadly classify them into [1]:

- 18.1.4.1. Technical factors
- 18.1.4.2. Delivery factors
- 18.1.4.3. Patient factors
- 18.1.4.4. Pharmacological factors

18.1.4.1 Technical Factors

- (a) *Anatomical reasons*: The existence of anatomical barriers (longitudinal band of tissue between the dura mater and the ligamentum flavum) or the placement of the catheter tip in the anterior or in the paravertebral epidural space may explain the block of isolated roots, the unilateral blocks, or the asymmetric blocks (See Sect. 18.3.1).
- (b) *Patient's position when performing the block*: The position of the patient can affect the placement of the needle, as it can modify the relationship between the osseous and soft tissue. Furthermore, opening of the posterior interlaminar space by spinal flexion modifies the position of spinal content. When the patient assumes a flexed position with the head down, it results in an anterior movement of the thoracic spinal cord, while at the lumbar level, the spinal cord and cauda equina move posteriorly [11].

Technically, there is no clear evidence that the sitting position or the lateral position is related to a higher or lower rate of failures or unilateral analgesia, although there is a clinical report that documented a higher incidence of technique difficulties when the parturient is on her side [12]. Moreover, the sitting position, favoring the distention of the epidural plexus, makes vascular puncture more likely [13].

- (c) *Midline or paramedian approach*: Catheter placement is faster with the paramedian approach and its success rate is less related to patients' spine flexion. The risk of accidental vascular puncture has no relation to these approaches in obstetric patients [14, 15].
- (d) *Identification of epidural space*: According to Beilin et al., the frequency of adequate analgesia is higher when using saline in the loss of resistance technique to identify the epidural space. Parturients in which air was used demanded additional analgesia more frequently (36 %) than with saline (19 %) [16].

Anesthesiologists who use saline suggest that when using air, it's easier to cause a pneumocephalus with the subsequent headache, and that air bubbles could be responsible of an incomplete blockade preventing the spread of local anesthetic [2].

In 2009 a meta-analysis compared the loss of resistance with saline versus air. It included 4,422 patients; four clinical trials were in obstetric patients and one in the nonpregnant population.

No significant differences in any outcome were found, other than a 1.5 % reduction in the incidence of post-dural puncture headache in the *saline* group [17].

In any case, it is important to remember that the ligamentum flavum is not continuous in all patients, and therefore, the existence of midline gaps may alter the loss of resistance perception when the midline approach is used. To avoid it, the use of ultrasound may be useful, as proved in obese patients [18].

- (e) *Location, catheter insertion, and fixation*: Even when the epidural space has been correctly identified, the catheter may not follow a straight line during its insertion. Clinical or radiological transforaminal catheter migration has been described only in obstetric patients. It is recommended to insert the catheter at least 4 cm into the epidural space [1]. Catheter fixation devices can significantly reduce the rate of migration and thus reduce the rate of analgesic failure [19].
- (f) *Equipment*: Failure can also occur due to catheter obstruction, preventing the proper spread of local anesthetics. The most commonly used epidural catheters are the ones with three side orifices. Morrison and Buchan [20] demonstrated that although multi-orifice catheters may be less safe due to the fact that the orifices may be located outside of the epidural space (in the subdural space, for example), the need of replacement was less frequent.

Occasionally, there may be manufacture catheter defects, such as absence of marks that hinder proper placement, accidental disconnection of any part, or air obstruction of the system. As little as 0.3–0.7 mL of air in the antibacterial filter can cause obstruction of the epidural infusion system [21]. Exceptionally, there may be a knot in the catheter, which is a rare cause of obstruction.
- (g) *Experience of the people who perform the block*: Agaram's series show that the experience of the people who perform the technique is related to analgesic failure [10]. In Pan's series [8], almost all blocks are performed by residents

and non-obstetric anesthesiologists, which would explain the high percentage of failed blocks, unlike the results found by Peach et al. [6].

Supervision by an experienced anesthesiologist is recommended. In difficult cases, we suggest that the blockade is performed by an experienced obstetric anesthesiologist.

- (h) *CSE versus epidural*: The CSE failure rate is as low as 0.16 % [22], significantly lower than with the epidural technique.

18.1.4.2 Delivery Factors

- (a) *Patient's position during dilation*: Maternal position has little effect on the appearance of an asymmetric block. Authors who have studied this factor found only a small, two or three dermatomes difference between both sides when the patient remained in the lateral position. It is more likely that the relationship between the epidural space and its surrounding structures (connective tissue, fat, blood vessels) has more influence in analgesia quality than maternal posture [23, 24].
- (b) *Labor progress*: As labor progresses, pain becomes more intense. When investigating the reason for an inadequate analgesia, the anesthesiologist must always consider the stage of labor of the parturient. In the last stages, it may be necessary to administer a bolus of a higher and more concentrated dose of local anesthetic (5–15 mL of 0.25 % bupivacaine with or without opioid). The use of opioid is specially recommended when back pain is secondary to a fetal occipito-posterior position [1].

18.1.4.3 Patients Factors

- (a) *Spinal deformities or previous back surgery*: The most severe deformities are associated with failed or patched blocks, as occurs after spinal surgery, due mainly to tissue adhesions or bone grafts that may affect the distribution of the local anesthetic. Disk pathology may also interfere with the diffusion, as shown in a series of 600 epidurograms. In up to 33 % of patients with uncomplicated disk prolapse, contrast did not reach the affected root [2].
- (b) *The midline barrier* (See Sect. 18.3.1).
- (c) *Differences between patients*: The demographic characteristics of the study population and expectations of different groups of women in labor may be different as far as analgesia for labor is concerned. Catheter migration occurs more often in women with higher body mass index [2]. Younger patients, with a higher body mass index and a longer labor, have higher risk of failure in the extension of the epidural block for cesarean section [25]. To avoid failure in these patients, it is convenient to emphasize the role of ultrasound. Ultrasound measurement of the epidural space depth before epidural placement technique decreases the rate of epidural catheter replacements for failed labor analgesia and reduces the number of epidural attempts when performed by first-year residents and compared to attempts without ultrasound guidance [26].

18.1.4.4 Pharmacological Factors

Among the pharmacological factors, there may also be numerous causes of failure.

- (a) *Local anesthetic dose vs. volume*: The influence of dose, concentration, and volume on the spread of epidural analgesia has been studied extensively. In general, the main determinant of the effect of local anesthetics is the dose, while the volume plays a minor role [1]. The effect of volume is more pronounced during bolus application. During labor, when two equivalent doses, in mg, of local anesthetic with different volume are administered, the spread of the block is greater with higher volume [27].
- (b) *Addition of opioids*: The addition of small doses of opioids can reduce the dose of local anesthetic with an improvement in the quality of analgesia during labor and potentially reduce motor block. The concept of “low-dose local anesthetics” for analgesia is only feasible when opioids are used.
- (c) *Addition of epinephrine*: The effects of epinephrine on opioids and local anesthetics are additive. Minimum local anesthetic concentration (MLAC) of bupivacaine is reduced by 29 % in laboring parturients with the use of epinephrine.
- (d) *Test dose*: Before administering a bolus of local anesthetic, the anesthesiologists must first administer a test dose to exclude intravascular or intrathecal catheter placement. If analgesia remains unsatisfactory with a negative test dose and after the first dose of local anesthetic, replacement of the catheter is recommended.
- (e) *Drug delivery systems*: The different epidural drug delivery systems are also important [27], such as patient-controlled epidural analgesia (PCEA) associated or not with continuous infusion and/or programmed intermittent boluses.

These factors, although mainly related to pharmacokinetics, may be related to a failed or incomplete analgesia and catheter replacement.

These strategies, the use of epidural drug delivery systems, the administration of larger volumes (in slightly extended blocks), addition of opioids, and/or the use of more concentrated doses (in patchy analgesia associated with fetal presentation pain), are known as “pharmacological optimization” strategies and help in making decisions (resite the epidural catheter).

In summary, some technical factors may contribute to increase the primary or secondary success rate of epidural analgesia.

- Epidural catheters may be misplaced from the beginning or may migrate from their initial location due to the patient’s movements. Likewise, the catheters may deviate from what is strictly midline during insertion.
- The optimum depth at which the epidural catheter should be left is 5 cm.
- The recommended method to identify the epidural space is the loss of resistance with saline.

- There is a growing evidence base for the use of ultrasound to locate the epidural space in obese patients.
- Dose is the main determinant in continuous epidural analgesia, where concentration and volume play a subordinate role.

Proposed Solutions The key to manage an inadequate block is early detection. Any woman with a neuraxial block for labor pain should be evaluated in the first 30 min of the first dose and the level should be checked with the correct stimulus (Table 18.1).

Table 18.1 Epidural failure causes and solutions

| Situation | Problem | Causes and questions | Symptoms | Solutions |
|-----------------------------------------------|------------------------------------------------|-----------------------------------------------------------------------------------------------------------------------------------|---------------------------------------------------------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------|
| Ineffective analgesia Limited extension | Catheter dislodgment/migration | Vein | – No block – Block regression | Catheter replacement |
| | | Transforaminal, paravertebral | Very limited block | |
| | | Out of epidural space (subcutaneous) | No block | |
| Unilateral analgesia | No analgesia on one side | – Anatomical reasons (Plica, surgery, etc.) – Catheter location (depth > 6 cm?) | Adequate level only on one side | – Pharmacologic optimization – Withdraw catheter 1 cm + bolus + maternal position – Catheter replacement/consider CSE |
| Subdural block | Patchy block | – Onset 10–20 min? – Sensitive block? – Sympatholysis? – Horner, syndrome? | – Intermediate onset – Asymmetric sensitive block – No sacral block | Catheter replacement |
| Breakthrough pain Inadequate block density | Pain in previously adequate epidural analgesia | – Labor progress too fast? – Abnormal fetal presentation? – Catheter >5 cm into epidural space? – Too diluted solutions? | Correct block assessment | – Opioids plus local anesthetic boluses – Occasional use of concentrated solute – Catheter replacement (role of CSE) – Team communication |

CSE Combined spinal epidural

- (a) Excessive catheter insertion into the epidural space: Not more than 5 cm of catheter should be left in the epidural space. In an attempt to solve inadequate analgesia, multi-orifice catheters can be withdrawn up to 3–5 cm. If pain persists after another dose is administered, it is time for catheter replacement in another space. Even if the catheter was initially placed at the right distance, it may migrate due to muscles and vertebrae movements.
- (b) Evaluation of the infusion system, if it is connected or if it is occluded, should be considered when managing a failed block.
- (c) If there is no block at all, a new block should be performed.

18.2 Catheter Dislodgement/Migration

The epidural space is a compartmentalized and complex structure, which can interfere with the proper placement of the catheter when a block is attempted [1].

Epidural catheters may be incorrectly placed from the start or later dislodged. A catheter initially located in the epidural space may migrate into a vein, into the subdural or subarachnoid space. This may occur either with intermittent bolus administration or continuous infusion.

18.2.1 Venous Migration

If an epidural catheter migrates into a vein during continuous epidural infusion of a diluted solution of local anesthetic, it is unlikely that the patient has symptoms of local anesthetic toxicity, but the block will gradually disappear. When a woman in labor with a continuous infusion of local anesthetic begins to complain of an unexpected and progressive pain, you should suspect a migration of the catheter into a vein.

18.2.2 Subdural or Subarachnoid Migration

Migration of an epidural catheter into the subarachnoid or subdural space during an infusion would give a slow rise of the block with a high motor component, considering doses of local anesthetics of 0.125 % at a rate of 10–15 mL/h. If more concentrated solutions are used, the safety margin is lower. An unexpectedly high level of anesthesia indicates administration of an excessive dose of local anesthetic or the migration to subdural or intradural space.

18.2.3 Transforaminal Migration

Transforaminal migration of the tip of the catheter and an asymmetrical distribution of the block are situations often described. The epidural catheter can leave the

epidural space through the intervertebral foramen, either above or below the place of insertion.

A high transforaminal rate of catheter migration (4.5 %) was described when catheters were inserted more than 5 cm into the epidural space [2]. It seems that after these migrations were diagnosed, only 50 % of cases were resolved successfully with catheter replacement. This may be due to the fact that the misplaced catheter could open a new path, which favors catheter dislodgement and failed analgesia [28].

18.2.4 Catheter Dislodgement

Catheter dislodgement out of the epidural space after the correct initial positioning can occur due to the patient's movements [29]. In addition to the patient's movements, changes in epidural pressure and the cerebrospinal fluid (CSF) oscillations can contribute to the dislodgement of an epidural catheter initially in place. This should be suspected when the block disappears after a period of adequate analgesia, and a differential diagnosis should be done with the catheter's migration to a vein. In both cases, the recommended approach is the catheter replacement, preferably with the aid of ultrasound, especially in obese patients or difficult backs.

18.3 Unilateral Block

Unilateral block is described as a correct cranial and caudal extension of the block but limited to only one half of the body. Its incidence appears to be somewhat lower if a paramedian approach is performed, since the catheter seems to be more directly addressed to the epidural space in this way. When it appears, it may be due to several causes:

18.3.1 Plica Mediana Dorsalis and Other Anatomical Changes

It has been radiologically demonstrated that in some individuals the epidural space may have a midline fibrous. This band of connective tissue is called "plica median dorsalis" and may just consist of a few sagittal fibers of connective tissue or form a real membrane. It is possible that the removal of 1 cm of catheter, the administration of a fractionated additional dose of local anesthetic, and the change of position help to avoid the plica in some cases. To overcome this barrier, you can try the pharmacological optimization with larger volumes of local anesthetic and if this measure is not effective, catheter replacement is recommended.

In 48 autopsies, on patients between 20 and 88 years old, 2 % of cadavers showed a complete or incomplete connective tissue band in the midline of the epidural space [30]. This connective tissue band covered at least two epidural

spaces and as in most cases it was an incomplete barrier, the unilateral blockade could be avoided by administering larger volumes of local anesthetic.

Unilateral blocks are described more frequently in patients with scoliosis, even when they are asymptomatic, because the catheter itself heads in the opposite direction to where scoliosis is [28]. Unilateral blocks have also been described after spinal surgery due to the development of fibrosis after the surgery.

18.3.2 Catheter Location

The orifices in the catheter can be placed above or laterally into the epidural space, spreading the local anesthetic more on one side than another, thereby favoring the occurrence of unilateral block [31].

The multi-orifice catheters present advantages over single-orifice type. It has been suggested that to reduce the rate of asymmetrical blocks and analgesic failures, the distance of catheter insertion should be of 4 cm.

When managing a unilateral block, you can try placing the patient in the lateral position on the affected side for a uniform distribution, together with the administration of larger volumes of local anesthetic. It is sometimes effective, but frequently a motor block of the already blocked side is obtained, the other side remaining with an unsatisfactory sensitive block.

In patients with unilateral block, a randomized control study found no significant difference in the success rate when patients were placed in the lateral position if the catheter was withdrawn before or after the administration of a local anesthetic bolus [23]. In both cases, the success rate of analgesia bolus is around 75 %. In that situation, epidural catheter replacement must be considered. The administration of 5–15 mL of diluted local anesthetic, to try to get a satisfactory analgesia, is recommended in unilateral, asymmetrical, patchy, or incomplete blocks. Complementary opioids may also be useful.

The CSE technique, which is an alternative to the classical administration of 5–15 mL of diluted local anesthetic, is recommended to try to get a satisfactory analgesia in unilateral, asymmetrical, or patchy epidural for labor analgesia [32]. Several studies have associated this technique with a lower incidence of unilateral blocking [22] and catheter replacement [8, 22]. This may be because the target of the subdural puncture is clearly the output of CSF through the needle and, therefore, a correct positioning of the catheter in the midline [32].

In 2014, Hessen et al. [32] published a meta-analysis in order to determine whether the CSE had lower incidence of unilateral block or catheter replacement during labor. These authors found that CSE, compared to the epidural technique, was not associated with a significantly lower risk of repositioning of the catheter. However, the risk of unilateral blockade was significantly lower in parturients who received CSE, even though there was considerable heterogeneity between studies. None of the studies analyzed in this meta-analysis had as its primary objective the replacement rate of catheters.

Nowadays, we cannot say that CSE provides a better quality of analgesia, although data suggest a lower rate of unilateral blocks [22, 32].

18.4 Subdural Block

The subdural space is a potential space located between the dura mater and the pia-arachnoid membrane extending from the second sacral vertebra to the cranial cavity.

Subdural block incidence is low, ranging between 1:1,850 [6] and 1:10,500 [33]. The report of Jenkins, comprising 145,550 epidurals, gives an intermediate incidence of 1:4,200 [34], although other studies document a somewhat different incidence.

Unlike the subarachnoid block, the maximum height of subdural block appears to be unrelated to the volume of local anesthetic injected, but, nevertheless, it often reaches the cervical nerve roots and even intracranial nerves. A subdural block may extend cranially, affecting arms and face, but without involvement of sacral dermatomes [24].

The onset of subdural blockade is slower than the subarachnoid block (usually 10–20 min) and characteristically patchy while motor block and hypotension are virtually absent. The incidence of hypotension is less frequent than in the high spinal block, as the subdural injection brings a sympathetic block of lesser extent and intensity. However, it can lead to hypotension and apnea seriously threatening the parturient. A Horner's syndrome is not uncommon.

However, in addition to life-threatening situations, accidental injection of local anesthetic into the subdural space may result in a failed or patchy block. It has been suggested that when local anesthetics are injected through the catheter into the anterior subdural space, there is an exaggerated block extension, while injection in the posterior subdural space leads to a more restricted extension [35].

If diagnosed, the attitude should be the immediate removal of the subdural catheter and replacement with an epidural one.

Collier, the author with more publications on subdural blocks, described almost 30 years ago the clinical criteria for subdural block [36]: moderate hypotension, slow symptoms progression, progressive respiratory difficulty, and complete recovery within 2 h.

Lubenow et al. [37], a few years later, defined two major criteria (negative aspiration test and unexpectedly extensive sensory block) and three minor criteria (prolonged onset, variable motor block despite the use of small amounts of local anesthetic, and wide sympatholysis).

The presence of two major and one minor criteria is highly suggestive of subdural block. More recently, Hoftman and Ferrante [38] analyzed a wide extensive range of cases of subdural blocks published and documented with radiological images, which define the most characteristic clinical presentation; 75 % of the cases reviewed by Hoftman had at least one of Colliers criteria, but only one-third had

two or more criteria and 25 % had none. Lubenow criteria diagnosed correctly 71 % of cases and failed in 29 %.

In summary: the subdural block is rare. The key for its diagnosis is clinical suspicion and the existence of diagnostic criteria (two major and one minor). The administration of local anesthetic through a catheter placed at the subdural space may result in an inappropriate or ineffective sensitive block and may also put the parturient's life at serious risk. The recommended approach is to remove the catheter and replace it, after informing the parturient.

18.5 Breakthrough Pain

Parturients receiving epidural analgesia may experience BP requiring the use of additional medication.

We can define two types of breakthrough pain (BP): “primary breakthrough pain” as the moment when a woman first requests analgesia during labor. “Secondary breakthrough pain” can be defined as the moment when previously used analgesia becomes ineffective [39].

The BP may have multiple causes. Clearly, the technical causes such as catheter misplacement, migration, and dislodgement may give inadequate analgesia.

There is no single pattern of providing adequate neuraxial analgesia for labor, although for certain patients and specific clinical situations, some methods provide advantages over others. The choice of suitable solutions and an adequate drug delivery system have an important role in achieving proper analgesia and a low incidence of BP and parturient unsatisfaction [40].

In order to develop preventive strategies for BP, there has been an attempt to develop predictive scales that could be applied previously to the technique. Among the risk factors included in the scales, we can highlight: nulliparity, epidural catheter placement at an earlier cervical dilation, neonatal weight, and technique performed (epidural vs. CSE) [39].

A close and frequent communication with the obstetric team is important for the performance of safe and effective neuraxial analgesia for labor and for the prevention and treatment of BP, among other situations. In addition, the role of new treatment modalities during labor is essential.

18.5.1 PCEA

Compared to continuous epidural infusion, PCEA increases maternal satisfaction, reduces the team's workload, reduces the incidence of BP, and also reduces local anesthetic consumption and motor block intensity [41, 42].

These results can be explained because the injection pressure generated when administering a bolus results in a more uniform distribution of the solution into the epidural space (cadaveric studies) [43]. The negative point is that the ideal PCEA regimen has not yet been determined.

In terms of volume and frequency of PCEA boluses, it seems that programs with high volumes and extended lockout intervals give better quality of analgesia [44].

18.5.2 Basal Infusion

The evidence available today is confusing and inconclusive when recommending or not a basal infusion. Nevertheless according to Halpern and Carvalho [45], the addition of a basal infusion appears to improve the quality of analgesia and less intervention is required for clinical pain. Probably, the use of high rates (above 5 mL/h) improves the efficiency of the PCEA. (The efficiency of the analgesia is measured by the ratio of boluses administered/boluses required [46].)

18.5.3 Novel Drug Delivery Systems

Recently, more sophisticated drug delivery systems have been developed, which may be an additional tool for higher and better quality analgesia in obstetrics.

One of these novel methods is the computer-integrated patient-controlled epidural analgesia (CI-PCEA), which automatically titrates the background infusion rate based on the individual parturient's needs. Sia et al. compared a CI-PCEA with a continuous infusion regimen, observing that the incidence of BP was reduced without increasing consumption of local anesthetic [47].

CI-PCEA was also compared to a PCEA continuous infusion and a higher maternal satisfaction but with similar local anesthetic consumption and incidence of BP was observed [48].

The fact that the analgesic and local anesthetic needs (MLAC) vary throughout the progress of labor [49] may justify that the use of an adjustable basal infusion gives higher maternal satisfaction rates and reduces the BP incidence, without increasing local anesthetic consumption.

18.5.4 Continuous Infusion Versus Mandatory Boluses

The use of a PCEA with a basal infusion compared to PCEA with intermittent automatic or programmed bolus appears to reduce local anesthetic consumption and the incidence of BP. This may be due, as already mentioned, to the fact that injection pressure generated when administering a bolus results in a more uniform distribution of the solution into the epidural space. This pressure does also favor local anesthetic output in multi-orifice catheters, and therefore, a more uniform distribution in the epidural space [40].

In pharmacodynamic terms of penetration of local anesthetic into the nerve and in terms of diffusion gradients, one can determine the generation or reversal of analgesia (or motor block) [27].

18.5.5 Role of CSE in Breakthrough Pain

Some authors have studied the intermittent automatic bolus after having performed a CSE. The pressure of the bolus can facilitate a certain transfer of anesthetic solution into the dural space through the orifice [50].

The possibility of taking advantage of this intentional transfer of local anesthetic through the dural hole is something to investigate in more depth.

When considering neuraxial analgesia in a parturient with high BP risk, the CSE may be the technique of choice (nulliparous, early cervical dilation, and high estimated fetal weight) [51].

The use of neostigmine or clonidine with CSE can reduce BP, although its use is not as common as the use of opioids combined with local anesthetics [52].

Practical Points Having assessed the nature of pain, the correct extension of neuraxial blockade, and the progress of labor, BP is generally treated with an epidural bolus of bupivacaine 0.125 %, about 10–15 mL, administered in increments of 5 in 5 mL. Occasionally, we may choose to use more concentrated solutions of local anesthetic (bupivacaine 0.25 %), especially in cases of abnormal fetal presentation or dysfunctional labor. In this case, it may also be necessary to increase the concentration of the basal solution. This often results in a satisfactory perineal analgesia for labor. Sometimes, more analgesia is required for labor, especially in instrumented delivery. In this case, the administration of 5–12 mL of Lidocaine 1 % or 2 % generally provides adequate sacral analgesia [40].

Summary

- It is important to recognize failure or BP risk factors before performing a neuraxial block for analgesia during labor.
- When evaluating a possible failed neuraxial block, we must discard technical factors associated with failure of the technique.
- When we have to replace the catheter, the CSE technique should be considered.
- If the patient needs an additional bolus, we have to consider the concomitant use of opioids. More concentrated solutions are used only occasionally.
- The role of new epidural drug delivery systems is increasing.

References

1. Hermanides J, Hollmann MW, Stevens MF, Lirk P (2012) Failed epidural: causes and management. *Br J Anaesth* 109:144–154
2. Bimbach DJ, Ranasinghe JS (2008) Anesthesia complications in the birthplace: is the neuraxial block always to blame? *Clin Perinatol* 35:35–52
3. Eappen S, Blinn A, Segal S (1998) Incidence of epidural catheter replacement in parturients: a retrospective chart review. *Int J Obstet Anesth* 7:220–225
4. Polley L, Columb MO, Naughton N et al (2002) Effect of epinephrine on the minimum local analgesic concentration of epidural bupivacaine in labor. *Anesthesiology* 96:1123–1128

5. Columb MO, Lyons G (1995) Determination of minimum local anaesthetic concentration of epidural bupivacaine and lidocaine in labor. *Anesth Analg* 81:833–837
6. Paech MJ, Godkin R, Webster S (1998) Complications of obstetric epidural analgesia and anesthesia: a prospective analysis of 10,995 cases. *Int J Obstet Anesth* 7:5–11
7. Beilin Y, Bernstein H, Zucker-Pinchoff B (1995) The optimal distance that a multiorifice epidural catheter should be threaded into the epidural space. *Anesth Analg* 81:301–304
8. Pan PH, Bogard TD, Owen MD (2004) Incidence and characteristics of failures in obstetrical neuraxial analgesia and anesthesia: a retrospective analysis of 19,259 deliveries. *Int J Obstet Anesth* 13:227–233
9. Crawford JS (1972) The second thousand epidural blocks in an obstetric hospital practice. *Br J Anaesth* 44:1277–1286
10. Agaram R, Douglas MJ, McTaggart RA et al (2009) Inadequate pain relief with labor epidurals: a multivariate analysis of associated factors. *Int J Obstet Anesth* 18:10–14
11. Lee RA, van Zundert AA, Botha CP et al (2010) The anatomy of the thoracic spinal canal in different postures: a magnetic resonance imaging investigation. *Reg Anesth Pain Med* 35:364–369
12. Coppejans HC, Hendrickx E, Goossens J et al (2006) The sitting versus right lateral position during combined spinal–epidural anesthesia for cesarean delivery: block characteristics and severity of hypotension. *Anesth Analg* 102:243–247
13. Bahar M, Chanimov M, Cohen ML (2004) The lateral recumbent head-down position decreases the incidence of epidural venous puncture during catheter insertion in obese parturients. *Can J Anaesth* 51:577–580
14. Griffin RM, Forum SRP (1984) A comparison between the midline and paramedian approaches to the extradural space. *Anaesthesia* 39:584–586
15. Podder S, Kumar N, Yaddanapudi LN, Chari P (2004) Paramedian lumbar epidural catheter insertion with patients in the sitting position is equally successful in the flexed and unflexed spine. *Anesth Analg* 99:1829–1832
16. Beilin Y, Arnold I, Telfeyan C et al (2000) Quality of analgesia when air versus saline is used for identification of the epidural space in the parturient. *Reg Anesth Pain Med* 25:596–599
17. Schier R, Guerra D, Aguilar J et al (2009) Epidural space identification: a meta-analysis of complications after air versus liquid as the medium for loss of resistance. *Anesth Analg* 109:2012–2021
18. Balki M, Lee Y, Halpern S, Carvalho JC (2009) Ultrasound imaging of the lumbar spine in the transverse plane: the correlation between estimated and actual depth to the epidural space in obese parturients. *Anesth Analg* 108:1876–1881
19. Clark MX, O'Hare K, Gorringer J, Oh T (2001) The effect of the Lockit epidural catheter clamp on epidural migration: a controlled trial. *Anaesthesia* 56:865–870
20. Morrison LMM, Buchan AS (1990) Comparison of complications associated with single-holed and multiholed extradural catheters. *Br J Anaesth* 64:183–185
21. Lin CC (2003) Air-locked epidural filter. *Anesthesiology* 99:515
22. Miro M, Guasch E, Gilsanz F (2008) Comparison of epidural analgesia with combined spinal–epidural analgesia for labor: a retrospective study of 6497 cases. *Int J Obstet Anesth* 17:15–19
23. Beilin Y, Zahn J, Bernstein H et al (1998) Treatment of incomplete analgesia after placement of an epidural catheter and administration of local anesthetic for women in labor. *Anesthesiology* 88:1502–1506
24. Wong C (2009) Epidural and spinal analgesia/anesthesia for labor and vaginal delivery. In: Chestnut's obstetric anesthesia principles and practice. Mosby-Elsevier, Philadelphia, PA, pp 430–492
25. Orbach-Zinger S, Avramovich A, Ilgiaeva N et al (2006) Risk factors for failure to extend labor epidural analgesia to epidural anesthesia for cesarean section. *Acta Anaesthesiol Scand* 50:1014–1018
26. Vallejo MC, Phelps AL, Singh S, Orebaugh SL, Sah N (2010) Ultrasound decreases the failed labor epidural rate in resident trainees. *Int J Obstet Anesth* 19:373–378

27. Capogna G, Stirparo S (2013) Techniques for the maintenance of epidural labour analgesia. *Curr Opin Anaesthesiol* 26:261–267
28. Collier CB (1996) Why obstetric epidurals fail: a study of epidurograms. *Int J Obstet Anesth* 5:19–31
29. Hamilton CL, Riley E, Cohen S (1997) Changes in the position of epidural catheters associated with patient movement. *Anesthesiology* 86:778–784
30. Blomberg R (1986) The dorsomedian connective tissueband in the lumbar epidural space of humans. An anatomical study using epiduroscopy in autopsy cases. *Anesth Analg* 65:747–752
31. Asato F, Goto F (1996) Radiographic findings of unilateral epidural block. *Anesth Analg* 83:519–522
32. Heesen M, Van de Velde M, Klöhr S et al (2014) Meta-analysis of the success of block following combined spinal-epidural vs epidural analgesia during labour. *Anaesthesia* 69:64–71
33. Scott DB, Tunstall ME (1995) Serious complications associated with epidural/spinal blockade in obstetrics: a two-year prospective study. *Int J Obstet Anesth* 4:133–139
34. Jenkins JG (2005) Some immediate serious complications of obstetric epidural analgesia and anaesthesia: a prospective study of 145,550 epidurals. *Int J Obstet Anesth* 14:37–42
35. Collier CB (2003) Accidental subdural injection during obstetric epidural block, it's commoner than you think! Epidurogram evidence. *Int J Obstet Anesth* 12:201
36. Collier C (1982) Total spinal or massive subdural block? *Anaesth Intensive Care* 10:92–93
37. Lubenow T, Keh-Wong E, Kristof K et al (1988) Inadvertent subdural injection: a complication of an epidural block. *Anesth Analg* 67:175–179
38. Hoftman N, Ferrante M (2009) Diagnosis of unintentional subdural anesthesia/analgesia: analyzing radiographically proven cases to define the clinical entity and to develop a diagnostic algorithm. *Reg Anesth Pain Med* 34:12–16
39. Hess PE, Pratt SD, Lucas TP et al (2001) Predictors of breakthrough pain during labor epidural analgesia. *Anesth Analg* 93:414–418
40. Loubert C, Hinova A, Fernando R (2011) Update on modern neuraxial analgesia in labour: a review of the literature of the last 5 years. *Anaesthesia* 66:191–212
41. van der Vyver M, Halpern S, Joseph G (2002) Patient controlled epidural analgesia versus continuous infusion for labour analgesia: a meta-analysis. *Br J Anaesth* 89:459–465
42. Hawkins JL, Arens JF, Bucklin BA et al (2007) Practice guidelines for obstetric anesthesia: an updated report by the American society of anesthesiologists task force on obstetric anesthesia. *Anesthesiology* 106:843–863
43. Hogan Q (2002) Distribution of solution in the epidural space: examination by cryomicrotome section. *Reg Anesth Pain Med* 27:150–156
44. Wong CA, Ratliff JT, Sullivan JT et al (2006) A randomised comparison of programmed intermittent epidural bolus with continuous epidural infusion for labor analgesia. *Anesth Analg* 102:904–909
45. Halpern SH, Carvalho B (2009) Patient-controlled epidural analgesia for labor. *Anesth Analg* 108:921–928
46. Stratmann G, Gambling DR, Moeller-Bertram T et al (2005) A randomized comparison of a five-minute versus fifteen-minute lockout interval for PCEA during labor. *Int J Obstet Anesth* 14:200–207
47. Sia AT, Lim Y, Ocampo CE (2006) Computer-integrated patient-controlled epidural analgesia: a preliminary study on a novel approach of providing pain relief in labour. *Singapore Med J* 47:951–956
48. Sng BL, Sia AT, Lim Y et al (2009) Comparison of computer-integrated patient-controlled epidural analgesia and patient-controlled epidural analgesia with a basal infusion for labour and delivery. *Anaesth Intensive Care* 37:46–53

49. Capogna G, Celleno D, Lyons G, Columb M, Fusco P (1998) Minimum local analgesic concentration of extradural bupivacaine increases with progression of labour. *Br J Anaesth* 80:11–13
50. Chua SM, Sia AT (2004) Automated intermittent epidural boluses improve analgesia induced by intrathecal fentanyl during labour. *Can J Anaesth* 51:581–585
51. Goodman SR, Smiley RM, Negron MA et al (2009) A randomized trial of break-through pain during combined spinal-epidural versus epidural labor analgesia in parous women. *Anesth Analg* 108:246–251
52. Boogmans T, Vertommen J, Valkenborgh T et al (2014) Epidural neostigmine and clonidine improves the quality of combined spinal epidural analgesia in labour: a randomised, double-blind controlled trial. *Eur J Anaesthesiol* 31:190–196

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19.1 Introduction

Anticoagulants are administered during pregnancy and postpartum to treat or prevent thrombotic events. For two decades, this practice has been supported by numerous studies, clinical evaluation, and diagnostic scoring of the peri- and postpartum thrombotic risk. Guidelines have since emerged to manage its treatment and prevention [1–4]. However, gaps persist in our knowledge of the condition, and there is evidence that anticoagulants can be more precisely monitored and targeted according to the individual risk [5]. Multidisciplinary management of the peripartum period aims to prevent both hemorrhagic and thrombotic risks, to avoid epidural hematoma, an unusual but severe complication of regional analgesia, as well as severe postpartum hemorrhage, and the occurrence or recurrence of a thrombotic event [6]. Managing analgesia during labor and delivery is a complex challenge due to the unique pharmacokinetic characteristics of drugs at the end of pregnancy and the unpredictability of spontaneous labor [6, 7]. A thorough multidisciplinary protocol should detail the neuraxial labor analgesia management during the peripartum period in these patients.

In this review, we discuss the therapeutic planning and peripartum management in patients administered anticoagulants, with a focus on the following: evaluating the risk of hemorrhage in affected patients; evaluating the epidural hematoma risk

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after administering labor analgesia; risk of thrombosis occurrence or recurrence following thromboprophylaxis discontinuation; hemostasis recovery to enable safe regional analgesia; and guidelines in neuraxial analgesia and labor management in patients administered anticoagulants. Furthermore, the recommended indications for initiating thromboprophylaxis and antithrombotic treatment during pregnancy and postpartum are discussed in the Appendix.

19.2 Hemorrhagic Risk Evaluation in Patients Receiving Thromboprophylaxis

Anticoagulation increases the risk of severe hemorrhage. Knol et al. conducted a case-control study of 88 patients receiving a high dose of low molecular weight heparin (LMWH), comprising nadroparin once daily during early pregnancy at $175 \text{ IU kg}^{-1} \text{ day}^{-1}$ initiated at pregnancy confirmation or following a peripartum venous thromboembolism (VTE) episode [8]. The risk of postpartum hemorrhage (PPH) was compared between treated subjects and non-treated paired controls. They found that the risk of severe hemorrhage was twice as high in treated subjects compared to control subjects undergoing vaginal delivery [30 % vs. 18 %, respectively (OR 1.9, 95 % CI 1–1.1)] and three times higher after cesarean section [12 % vs. 4 %, respectively (OR 2.9, 95 % CI 0.5–19.4)] [8]. Similarly, in a 5 year retrospective Australian cohort study, Chan et al. reported three occurrences of severe PPH among 31 patients administered anticoagulants to treat peripartum thrombosis; they subsequently recommended careful therapeutic management during the peripartum period [9]. In a retrospective consecutive cohort of 72 thromboembolic events treated during pregnancy, Blanco-Molina et al. noted that the immediate postpartum period was associated with a 5.6 % risk of severe hemorrhage [10].

However, other studies have not reported any significant risk of severe hemorrhage. Galambosi et al. did not observe any significant difference in the incidence of severe hemorrhage (0.15 %) in 648 patients administered LMWH compared to 626 paired controls [11]. Limmer et al., in a case-control study of cesarean deliveries, found that women who received anticoagulants during pregnancy had a greater incidence of wound complications compared to those who did not (30 % vs. 8 %, $p < 0.001$), but there was no increased risk of hemorrhage [12]. In a meta-analysis, Romualdi evaluated 18 studies of 981 patients who experienced an acute thrombotic event during pregnancy; 822 were treated with LMWH and 79 with unfractionated heparin. Severe hemorrhage occurred in 1.41 % (95 % CI 0.60–2.41 %) of patients during pregnancy and 1.90 % (95 % CI 0.80–3.60 %) after delivery [4].

19.3 Epidural Hematoma Risk Evaluation

The incidence of spinal epidural hematoma in pregnant anticoagulated patients after labor analgesia is unknown. Most of the estimated rates and recommendations are based on case reports, national drug monitoring surveys, and expert opinion. In the obstetric setting, epidural hematoma is apparently infrequent: less than 1 per 150,000–275,000 patients after epidural puncture and less than 1 per 220,000–250,000 patients after spinal anesthesia in a large obstetric series describing regional anesthesia [13]. Bateman et al. identified seven cases of epidural hematoma requiring laminectomy in a nationwide survey of 62,450 orthopedic and obstetric neuraxial anesthesia events [14]. The frequency after epidural catheter insertion for perioperative anesthesia or analgesia varied from 1 event per 22,189 placements to 1 event per 4,330 placements. The risk was significantly lower for obstetric epidurals at $0-4.6 \times 10^{-5}$ ($p = 0.003$) [13]. In the orthopedic series, four of the seven patients who developed epidural hematomas were receiving anticoagulant treatment according to the recommended guidelines [14]. Furthermore, in older, coagulopathy, or anticoagulated patients, the incidence has been estimated as high as 1 per 1,500 epidural blocks and 1 per 3,600 spinal anesthesia procedures in patients administered unfractionated heparin; the incidence was 1 per 3,000 after epidural and 1 per 40,000 after spinal anesthesia in patients receiving LMWH [15].

Moen et al. estimated the risk of epidural hematoma in a 10 year retrospective analysis of severe neurologic complications after 1,260,000 spinal and 450,000 epidural (including 200,000 parturients for labor analgesia) neuraxial blockades in Sweden [16]. A total 127 adverse events were identified, with 33 identified as spinal hematoma. The incidence of neuraxial hematoma was estimated at 1 per 156,000 after spinal anesthesia and 1 per 18,000 after epidural anesthesia, and hemorrhage occurred less frequently in obstetric patients (1:200,000) than in female orthopedic patients undergoing knee arthroplasty (1:3,600) [16]. The study identified several risk factors for epidural hematoma after neuraxial regional anesthesia including the lack of treatment guidelines, administration of antithrombotic agents, female gender, and difficulty during the procedure.

Detection of this rare but catastrophic complication must be instituted in all obstetric and obstetric anesthesia units [17, 18]. Clinical symptoms of epidural hematoma, mainly the absence of regional block reversion and interscapular pain, should be monitored as a component of the routine systematic postpartum patient assessment. Multidisciplinary management of the anticoagulated parturient should focus on the detection of this complication as well as its prevention by respecting a sufficient delay to recover coagulation function before regional anesthesia and analgesia.

19.4 Risk of Thrombosis After Discontinuing Antithrombotic Treatment

Discontinuing preventive or curative anticoagulant treatment exposes the patient to potential occurrence or recurrence of a thrombotic event. Since 2010, venous thromboprophylaxis during pregnancy has been recommended in RCOG guidelines, and maternal mortality has since decreased [19]. Strict evaluation of the global thrombotic risk in each patient should guide the clinician in determining the optimal time to discontinue anticoagulant therapy to minimize the risks of both hemorrhage and thrombosis. Van Lennep et al. analyzed the risk of thrombosis in patients designated as intermediate and high risk based on the Dutch thrombosis registry and reported a thrombosis recurrence rate of 5.5 % (95 % CI 2.4–12.3) [20]. The thrombotic events only occurred in high-risk women at an antepartum incidence of 1.8 % (95 % CI 0.4–9.2) and postpartum incidence of 7 % (95 % CI 2.9–16.7). Accordingly, they recommended changing the LMWH dose during pregnancy. In another study, Kamel et al. reported that the 6 weeks postpartum period had the highest risk of a primary arterial or venous thrombotic event; they recommended continuing thromboprophylaxis during this period [21, 22].

Hematologists, obstetricians, and anesthesiologists have all devised scoring systems and algorithms to better assess each patient's risk and enable better prevention [1, 2]. However, risk evaluation is complex and is currently based on minimal evidence; therefore, a multidisciplinary algorithm may be more effective [23]. Individual risk assessment and multidisciplinary cooperation may be required to minimize the thrombosis risk [1].

19.5 Recovery of a Normal Hemostatic Function Allowing Safe Regional Analgesia

The recovery of normal hemostatic function after discontinuing anticoagulants determines the time when neuraxial anesthesia can be safely performed. This duration is based primarily on our knowledge of the pharmacokinetic characteristics of the individual agents concerned. Most professional consensus estimates the time interval between cessation of the medication and neuraxial blockade at two and half times the drug elimination half-life [7]. Clinicians must consider both the altered pharmacokinetics of LMWH during pregnancy and the decrease in anti-Xa activity beginning in the second trimester of pregnancy, as anti-Xa activity is used for monitoring [24–28].

A preventive LMWH dose (less than 100 IU kg⁻¹) generally allowed a 10–12 h delay, whereas a curative dose (more than 100 IU kg⁻¹) requires a 24 h delay before performing regional anesthesia [2, 6, 7, 27]. Anti-Xa activity has been validated for monitoring the LMWH efficacy [24–28]. A residual activity below 0.1 IU mL⁻¹ indicates complete elimination of the drug.

For subcutaneous unfractionated heparin (UFH), a 12–15-h delay has been recommended, whereas intravenous heparin requires a mere 4–6 h delay. Anti-Xa

activity is also used to monitor UFH activity. In cases of heparin overdose or emergency cesarean section, protamin can be administered [7].

Anti-vitamin K is administered during the second and third trimesters primarily in patients with mechanical heart valves and is monitored using the international normalized ratio (INR). The INR therapeutic range is 3–4, and depending on the specific drug, the recommended delay is long, lasting from 2 to 3 days. In cases of emergency cesarean section or premature delivery, vitamin K and prothombin complex or fresh frozen plasma can be administered to counteract the medication and recover normal hemostasis [29].

In cases of heparin induced thrombopenia and heparin or LMWH hypersensitivity, danaparoid can be administered to treat or prevent thrombosis. The route of administration can be subcutaneous or intravenous. Danaparoid activity is monitored with APTT and anti-Xa activity based on the specific dosage. The delay following cessation of danaparoid therapy has been estimated at 24 h [1, 30].

In 2012, based on level 1C evidence, because of a significant transplacental transfer, the ACCP recommended limiting the use of fondaparinux, oral thrombin, and anti-Xa inhibitors in pregnant women with severe heparin hypersensitivity (including Heparin Induced Thrombopenia) who cannot receive danaparoid [1].

After discontinuing anticoagulant treatment, biological assessment of hemostasis recovery should be planned to allow neuraxial block during painful labor. However, this assessment is hampered by the unpredictability of the first phase of labor and the delay needed to obtain diagnostic results. Point of care assessment has been suggested to verify hemostasis at the time of parturient admission. Recent studies have established thromboelastography and thromboelastometry reference values in pregnant women [31, 32], Thromboelastometric HEPTM test (ROTEM® TEM international Munich Germany) is able to discriminate the hypocoagulable state due to heparin and may be helpful for a patient receiving LMWH or UFH. However, these new methods have not yet been scientifically assessed for gauging the epidural hematoma and hemorrhage risks in patients receiving anticoagulants.

19.6 Neuraxial Analgesia and Labor Management in the Anticoagulated Patient

Regional analgesia is the most efficient and safe pain management option during labor. Guidelines for regional anesthesia management in the anticoagulated patient have been developed and regularly updated [1, 2, 6, 7, 33, 34]. These guidelines are based on case reports or cohort surveys. While an algorithm may help anesthetists, the decision to perform spinal or epidural analgesia in an anticoagulated parturient is individually tailored according to the hematoma risk and the benefits of regional analgesia. Therefore, multidisciplinary management may be recommended to adjust induction of labor and anticoagulation arrest delay (Fig. 19.1).

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| <p><u>Safe duration before performing regional anaesthesia</u></p> <p>Epidural or spinal anaesthesia or analgesia should be performed as follows:</p> <ul style="list-style-type: none"> ◆ If intravenous UFH is required patients with a very high risk of thrombosis recurrence, avoid regional anaesthesia and discontinue UFH 4 hours before delivery. ◆ Regional anaesthesia can be performed: ◆ Twelve hours after the last subcutaneous UFH dose; measure aPTT if the duration is shorter. ◆ Twelve hours after the last LMWH dose $\leq 100 \text{ UI kg}^{-1}$. ◆ After a 24 hours delay following the last curative or intermediate dose of LMWH $> 100 \text{ UI kg}^{-1}$. |
| <p><u>Benefit-risk analysis</u></p> <ul style="list-style-type: none"> ◆ Avoid regional anaesthesia if the therapeutic window will expose the patient to an acute thrombotic event. ◆ Promote regional anaesthesia or analgesia with an adapted therapeutic window if regional anaesthesia/analgesia eases delivery or maternal disease management. |
| <p><u>Regional anaesthesia in the anticoagulated parturient</u></p> <ul style="list-style-type: none"> ◆ Assess the hemostatic function ◆ Choose a senior anesthetist ◆ Choose median over lateral epidural access when possible ◆ Use ultrasound guidance, if possible ◆ Choose spinal over epidural when possible ◆ Strict neurologic monitoring during the immediate postpartum period ◆ Avoid postoperative local analgesia, which may mask neurologic deficiency |

Fig. 19.1 Guidelines for regional anaesthesia in an anticoagulated parturient [1, 2, 6, 7, 33, 34]. *UFH* unfractionated heparin, *LMWH* low molecular weight heparin

This multidisciplinary management algorithm is built on the following principles:

- In low thrombotic risk patients receiving anticoagulants to prevent placental vascular disease, anticoagulants should be stopped at the end of pregnancy and labor analgesia performed after the anticoagulation period.
- In the high thrombotic risk patients (mechanical heart valves, lupus, combined or antithrombin deficiency, arterial thrombosis, deep vein thrombosis < 3 months previously, and a history of pregnancy or postpartum thrombosis), antithrombotic drugs should not be stopped or should be discontinued for a minimal period; alternative analgesia should be chosen and explained to the parturient.
- The unpredictability of labor presents a challenge. Some clinicians choose to administer unfractionated intravenous heparin and induce labor to conduct a

medical guided delivery. The ACCP 2012 guidelines recommend partitioning the LMWH dose twice daily to reduce residual anti-Xa activity. After administering a therapeutic dose of UFH, a delay of 4 h is recommended. After administering a therapeutic dose of LMWH, 24 h interruption is recommended to allow delivery [1, 2, 6, 7, 33, 34].

- For intermediate risk patients, coagulation status should be optimized and monitored to ensure normal hemostasis recovery at the time of spinal or epidural catheter placement and removal [6, 35]. After administering an intermediate LMWH dose, the delay for heparin discontinuation is estimated at 12 h. At the end of pregnancy, the cervical maturation and obstetrical conditions for inducing labor are the most important parameters guiding LMWH discontinuation and allowing labor analgesia. These obstetrical conditions must be strictly and frequently monitored to optimize the therapeutic strategy.

19.7 Conclusion

Labor analgesia and optimized and safe delivery in the anticoagulated parturient requires multidisciplinary management to adjust the dose regimen, time drug discontinuation, and safely resume antithrombotic treatment postpartum in a manner that balances thrombotic complications, hemorrhage risk, and parturition.

Appendix

Guidelines for Thromboprophylaxis and Antithrombotic Treatment During Pregnancy and the Postpartum Period

Antithrombotic prophylaxis and anticoagulant treatment are prescribed during the peri- and postpartum periods to prevent venous, arterial, or placental thrombosis occurrence and recurrence. In low risk patients with no familial or individual history of previous thrombosis, the venous thrombosis incidence is low during all three trimesters at 1–2 per 1,000, increasing only during the postpartum [1, 2, 21, 22, 36, 37]. Perinatal practitioners are particularly concerned with detecting any risk factor that may justify thromboprophylaxis [1, 2].

Thrombosis risk factors are detailed in the 2012 ACCP guidelines [1]:

- Patients at extreme risk or those with special conditions require multidisciplinary preconception and pregnancy management. Relevant conditions include: antiphospholipid syndrome with antiprothrombinase, myeloproliferative disorders, Budd Chiari disease or digestive venous thrombosis, inflammatory systemic disease, antithrombin deficiency, known complex thrombophilia, and pulmonary hypertension.
- High-risk conditions include: previous pulmonary embolism, previous proximal idiopathic DVT, previous contraceptive, pregnancy, or postpartum (estrogenic)

induced proximal DVT, proximal DVT within the previous 2 years, and current long-term anticoagulation therapy.

- The intermediate risk conditions are: non-idiopathic or non-estrogenic DVT at least 2 years previously, distal estrogenic DVT, and previous estrogenic or cerebral vein thrombosis.
- Low risk conditions include: previous recurrent superficial vein thrombosis and/or major venous insufficiency, previous non-estrogenic distal DVT, previous ovarian DVT, previous non-estrogenic cerebral vein thrombosis, and previous familial DVT before age 45 years in an immediate relative.

Concurrent risk factors increase the final risk level. If two or more of the following additional risk factors are present, then the risk level category increases: aged over 35 years, multiparity, long distance travel, BMI >30 kg/m² before pregnancy, prolonged bed rest, medical assisted procreation, previous severe placental vascular disease, multiple pregnancies, major venous insufficiency, protein C or protein S deficiency, and heterozygous and homozygous factor V Leiden (FVL) or prothrombin mutation (PTG20210A). The following additional risk factors increase the postpartum final risk level: Cesarean section, severe postpartum hemorrhage, and severe preeclampsia [21, 22, 36–38].

Evaluation scores have been devised to detect excessive thrombotic risk and guide the thromboprophylaxis regimen based on antithrombotic or anticoagulant treatments available during pregnancy and postpartum [38, 39]. Screening for thrombophilia can be performed in the clinical risk evaluation and may reveal additional risk in the form of genetic polymorphisms [1, 40].

The thromboprophylaxis regimen should be tailored according to the evaluated risk level.

General principles for thromboprophylaxis in pregnant and postpartum patients include the following:

- Contention socks are always recommended throughout pregnancy and the postpartum period.
- Low Molecular Weight Heparin (LMWH) recommended for thrombosis prevention and cure; its safety and efficiency have been validated in large cohort series [1, 2, 33–35].
- The LMWH or UFH dose must be tailored according to the patient weight in classes of <50, 50–100, 100–130, and >130 kg [1, 25].
- The LMWH or UFH dose must be monitored and increased to counteract inflammatory syndrome occurring at the end of pregnancy [26–28].
- The LMWH pharmacokinetics allows once daily administration during pregnancy and postpartum except in obese patients [28, 41].
- Peripartum therapeutic management is easier when a half dose is administered twice daily 1 week before delivery [1].
- Postpartum hemostasis changes are maximal during the first 3 days and persist for 6–8 weeks following delivery.

Targeted thromboprophylaxis can be performed as follows [1]:

- Low risk patient: Postpartum LMWH at 50–60 IU kg⁻¹ daily immediately after delivery for 6 weeks.
- Intermediate risk patient: Pregnancy and postpartum LMWH at 50–60 IU kg⁻¹ daily from pregnancy diagnosis, until delivery, and continued an additional 6 weeks.
- High-risk patient: Pregnancy and postpartum prevention or treatment [1, 2] with LMWH at 100–200 IU kg⁻¹ daily from pregnancy diagnosis, until delivery, and continued an additional 6 weeks.

The platelet count should be monitored twice weekly for the 3 first weeks of therapy and then once monthly. Monitor clinical safety using aPTT or anti-Xa activity.

Key Messages

- Anticoagulants are currently used to prevent or treat pregnancy and postpartum related thrombosis (Appendix)
- The anticoagulated parturient has an increased risk of hemorrhage after delivery
- Anticoagulation in the obstetric context seems to have a lower risk of epidural hematoma after neuraxial anesthesia or analgesia compared to other non-obstetric patients
- For a safe delivery, anticoagulants must be discontinued at least when first stage of labor begins
- Neuraxial analgesia setting may respect the optimal delay of two and half times the drug elimination half-life after anticoagulation stop
- The management of the anticoagulated parturient requires a multidisciplinary approach and individual assessment of the hemorrhage and thrombosis risk balance.

References

1. Bates SM, Greer IA, Middeldorp S et al (2012) VTE, thrombophilia, antithrombotic therapy, and pregnancy: antithrombotic therapy and prevention of thrombosis. American College of Chest physicians evidence-based clinical practice guidelines (9th edn). *Chest* 141(2 Suppl): e691S–e736S
2. Benhamou D, Mignon A, Aya G et al (2005) Prophylaxis of thromboembolic complications in obstetrics and gynaecology. *Ann Fr Anesth Reanim* 24:911–920
3. Chauleur C, Gris J-C, Seffert P et al (2012) Latest developments on risk factors and prophylaxis of thromboembolic disease in obstetrics. *Gynecol Obstet Fertil* 40:301–307
4. Romualdi E, Dentali F, Rancan E et al (2013) Anticoagulant therapy for venous thromboembolism during pregnancy: a systematic review and a meta-analysis of the literature. *J Thromb Haemost* 11:270–281

5. Middeldorp S (2013) Thrombosis in women: what are the knowledge gaps in 2013? *J Thromb Haemost* 11(Suppl 1):180–191
6. Horlocker TT, Wedel DJ, Rowlingson JC et al (2010) Regional anesthesia in the patient receiving antithrombotic or thrombolytic therapy: American society of regional anesthesia and pain medicine evidence-based guidelines (3rd edn). *Reg Anesth Pain Med* 35:64–101
7. Gogarten W, Vandermeulen E, Van Aken H et al (2010) Regional anaesthesia and antithrombotic agents: recommendations of the European society of anaesthesiology. *Eur J Anaesthesiol* 27:999–1015
8. Knol HM, Schultinge L, Veeger NJGM et al (2012) The risk of postpartum hemorrhage in women using high dose of low-molecular-weight heparins during pregnancy. *Thromb Res* 130:334–338
9. Chan N, Merriman E, Hyder S et al (2012) How do we manage venous thromboembolism in pregnancy? A retrospective review of the practice of diagnosing and managing pregnancy-related venous thromboembolism at two major hospitals in Australia and New Zealand. *Intern Med J* 42:1104–1112
10. Blanco-Molina A, Trujillo-Santos J, Criado J et al (2007) Venous thromboembolism during pregnancy or postpartum: findings from the RIETE registry. *Thromb Haemost* 97:186–190
11. Galambosi PJ, Kaaja RJ, Stefanovic V et al (2012) Safety of low-molecular-weight heparin during pregnancy: a retrospective controlled cohort study. *Eur J Obstet Gynecol Reprod Biol* 163:154–159
12. Limmer JS, Grotegut CA, Thames E et al (2013) Postpartum wound and bleeding complications in women who received peripartum anticoagulation. *Thromb Res* 132:e19–e23
13. Horlocker T, Kopp S (2013) Epidural hematoma after epidural blockade in the United States: it's not just low molecular heparin following orthopedic surgery anymore. *Anesth Analg* 116:1195–1197
14. Bateman BT, Mhyre JM, Ehrenfeld J et al (2013) The risk and outcomes of epidural hematomas after perioperative and obstetric epidural catheterization: a report from the multi-center perioperative outcomes group research consortium. *Anesth Analg* 116:1380–1385
15. Lumpkin MM (1998) FDA public health advisory. *Anesthesiology* 88:27A–28A
16. Moen V, Dahlgren N, Irestedt L (2004) Severe neurological complications after central neuraxial blockades in Sweden 1990–1999. *Anesthesiology* 101:950–959
17. Meikle J, Bird S, Nightingale JJ et al (2008) Detection and management of epidural haematomas related to anaesthesia in the UK: a national survey of current practice. *Br J Anaesth* 101:400–404
18. Toner A, Prabhu P (2009) Reliable detection of epidural haematomas. *Br J Anaesth* 102:140–141
19. Touqmatchi D, Cotzias C, Girling J (2012) Venous thromboprophylaxis in pregnancy: the implications of changing to the 2010 RCOG guidelines. *J Obstet Gynaecol* 32:743–746
20. Roeters van Lennep JE, Meijer E, Klumper FJCM, Middeldorp JM et al (2011) Prophylaxis with low-dose low-molecular-weight heparin during pregnancy and postpartum: is it effective? *J Thromb Haemost* 9:473–480
21. Kamel H, Navi BB, Sriram N et al (2014) Risk of a thrombotic event after the 6-week postpartum period. *N Engl J Med* 370:1307–1315
22. Virkus RA, Løkkegaard ECL, Lidegaard Ø et al (2013) Venous thromboembolism in pregnancy and the puerperal period: a study of 1210 events. *Acta Obstet Gynecol Scand* 92:1135–1142
23. Wu P, Poole TC, Pickett JA et al (2013) Current obstetric guidelines on thromboprophylaxis in the United Kingdom: evidence based medicine? *Eur J Obstet Gynecol Reprod Biol* 168:7–11
24. Ellison J, Thomson AJ, Conkie JA et al (2001) Thromboprophylaxis following caesarean section—a comparison of the antithrombotic properties of three low molecular weight heparins—dalteparin, enoxaparin and tinzaparin. *Thromb Haemost* 86:1374–1378

25. Lebaudy C, Hulot JS, Amoura Z et al (2008) Changes in enoxaparin pharmacokinetics during pregnancy and implications for antithrombotic therapeutic strategy. *Clin Pharmacol Ther* 84:370–377
26. Fox NS, Laughon SK, Bender SD et al (2008) Anti-factor Xa plasma levels in pregnant women receiving low molecular weight heparin thromboprophylaxis. *Obstet Gynecol* 112:884–889
27. Gibson PS, Newell K, Sam DX et al (2013) Weight-adjusted dosing of tinzaparin in pregnancy. *Thromb Res* 131:e71–e75
28. Patel JP, Green B, Patel RK et al (2013) Population pharmacokinetics of enoxaparin during the antenatal period. *Circulation* 128:1462–1469
29. Levine M, Pizon AF, Padilla-Jones A et al (2014) Warfarin overdose: a 25-year experience. *J Med Toxicol* 10:156–164
30. Bradbrook ID, Magnani HN, Moelker HC et al (1987) ORG 10172: a low molecular weight heparinoid anticoagulant with a long half-life in man. *Br J Clin Pharmacol* 23:667–675
31. Karlsson O, Sporrang T, Hillarp A et al (2012) Prospective longitudinal study of thromboelastography and standard hemostatic laboratory tests in healthy women during normal pregnancy. *Anesth Analg* 115:890–898
32. Oudghiri M, Keita H, Kouamou E et al (2011) Reference values for rotation thromboelastometry (ROTEM®) parameters following non-haemorrhagic deliveries. Correlations with standard haemostasis parameters. *Thromb Haemost* 106:176–178
33. Nathan N (2006) Recommandations pour la pratique clinique: Blocs périmédullaires de l'adulte. Bloc périmédullaire en présence d'une anomalie de l'hémostase. Eds SFAR 2006. www.SFAR.org
34. Butwick AJ, Carvalho B (2010) Neuraxial anesthesia in obstetric patients receiving anticoagulant and antithrombotic drugs. *Int J Obstet Anesth* 19:193–201
35. Hunt BJ, Gattens M, Khamashta M et al (2003) Thromboprophylaxis with unmonitored intermediate-dose low molecular weight heparin in pregnancies with a previous arterial or venous thrombotic event. *Blood Coagul Fibrinolysis* 14(8):735–739
36. Jacobsen AF, Skjeldestad FE, Sandset PM (2008) Incidence and risk patterns of venous thromboembolism in pregnancy and puerperium—a register-based case-control study. *Am J Obstet Gynecol* 198:233.e1–233.e7
37. Lindqvist PG, Bremme k, Hellgren M, Working Group on Hemostatic Disorders (Hem-ARG) (2011) Swedish society of obstetrics and gynecology. Efficacy of obstetric thromboprophylaxis and long-term risk of recurrence of venous thromboembolism. *Acta Obstet Gynecol Scand* 90:648–653
38. Chaleur C, Cochery-Nouvellon E, Mercier E et al (2008) Analysis of the venous thromboembolic risk associated with severe postpartum haemorrhage in the NOHA First cohort. *Thromb Haemost* 100:773–779
39. Chaleur C, Quenet S, Varlet M-N et al (2008) Feasibility of an easy-to-use risk score in the prevention of venous thromboembolism and placental vascular complications in pregnant women: a prospective cohort of 2736 women. *Thromb Res* 122:478–484
40. Clark P, Walker ID, Govan L et al (2008) The GOAL study: a prospective examination of the impact of factor V Leiden and ABO(H) blood groups on haemorrhagic and thrombotic pregnancy outcomes. *Br J Haematol* 140(2):236–240
41. Bombeli T, Raddatz-Mueller P, Fehr J (2001) Coagulation activation markers do not correlate with the clinical risk of thrombosis in pregnant women. *Am J Obstet Gynecol* 184(3):382–389

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The most frequent complication of neuraxial labor analgesia is post-dural puncture headache (PDPH). This is also the major focus of this chapter. But less common complications, such as nerve damage or epidural hematoma, are at least as important and must be part of the differential diagnosis in patients with postpartum neurological symptoms.

20.1 Post-dural Puncture Headache

PDPH is a potentially severe complication of epidural labor analgesia. It is usually the result of accidental dural puncture (ADP) and can be a debilitating condition. PDPH intensifies the burden of the young mother who is expected to care for her newborn child. Headache has been reported as one of the most frequent reasons for complaints in a closed claims analysis [1].

20.1.1 Incidence

It must be emphasized that tension headache, migraine, and preeclampsia are the most common causes of postpartum headache. In a retrospective review of 95 women with postpartum headache persisting for longer than 24 h after delivery, the most frequent diagnoses were tension-type headache (39 %), preeclampsia (24 %), PDPH (16 %), and migraine (11 %) [2].

ADP is dependent on the experience of the anesthesiologist, and this must be accounted for in teaching and training [3–5]. Most authors report an incidence of around 1–1.5 % [6, 7]. ADP is not always immediately recognized, and up to 25–

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40 % are only diagnosed by the occurrence of PDPH [6]. The loss of cerebrospinal fluid (CSF) after ADP results in a PDPH in approximately half of the parturients [7]. This is reflected by an incidence of PDPH in labor analgesia of 0.5–1.5 % [6–9].

20.1.2 Pathophysiology

Total CSF production is approximately 500 ml/day or 0.35 ml/min [10]. The total volume is around 150 ml, where half is intracranial and the other half spinal. Loss of CSF through a dural lesion leads to intracranial hypotension. Subsequently, this reduces the CSF “cushion” of the brain. When the patient is in a vertical position, the reduced volume of CSF allows the brain to descend, straining the sensitive meningeal structures [10]. A second mechanism is postulated on the basis of the Monroe–Kellie doctrine, which states that the total volume of brain + CSF + intracranial blood is constant. Therefore, a reduction in CSF leads to vasodilation and to a compensatory increase in cerebral blood volume [10]. Cerebral vasodilation is acknowledged as a common cause of headache.

20.1.3 Risk Factors

Risk factors for the development of PDPH following an intended or ADP are well known [4, 5, 11]:

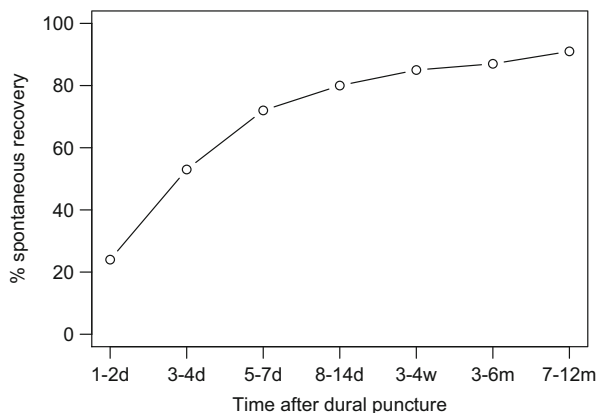
- Female gender: odds ratio 2.25 [11]
- Young age: odds ratio 2.21 for <50 years [11]
- Larger needle, 16 vs. 18G epidural needle: odds ratio 6.5 [5]
- Bevel orientation: odds ratio 2.16 [11]
- Cutting bevel of the needle
- Vaginal vs. Cesarean delivery: odds ratio 4.55 [5].

20.1.3.1 Needle Type and Size

PDPH following a dural puncture with a small, non-cutting needle is usually mild to moderate and resolves spontaneously within a couple of days and is shown in Fig. 20.1 [11–13].

However, if PDPH is due to an ADP with an epidural needle, the symptoms are substantially more severe and have been described to be like “hot molten metal in the head” [14]. The probability of spontaneous resolution is significantly lower in ADP with an epidural needle compared to intended dural puncture with a small gauge spinal needle [12]. The CSF loss is obviously related to the size of the lesion of the dura mater. According to the Hagen–Poiseuille equation, resistance is inversely proportional to the fourth power of the radius. Hence, it is not surprising that larger needles are associated with a higher incidence of PDPH. Simply taking the radius of the needle into consideration, then the radius of a 16G and an 18G needle is 0.825 mm and 0.635 mm, respectively. The fourth power of these is

Fig. 20.1 Cumulative spontaneous recovery of PDPH. Representation of the cumulative spontaneous recovery rate of PDPH. Graph drawn on the basis of data from Turnbull et al. [13]. *d* days, *w* weeks, *m* months



0.463 mm⁴ and 0.163 mm⁴ for 16G and 18G, respectively. The use of smaller (18G) epidural needles should be advocated in order to reduce the risk of PDPH should an ADP occur [12]. The incidence of PDPH was 3.5 % and 0.8 % in a historical cohort comparing the use of 16G and 18G needles, respectively [8]. In another study, the relative risk of PDPH was 2.2 (95 % CI 1.4–2.6) for a 16G compared to an 18G needle [5].

20.1.3.2 Direction of the Bevel

It has been advocated to direct the bevel of the needle parallel to the longitudinal fibers of the dura [3]. This, however, has been challenged because the parallel orientation of dural fibers has not been confirmed in microscopic investigations [15], but clinical investigations confirm a reduced incidence of PDPH when the bevel of the needle is parallel to the longitudinal axis [10]. This seems to be of importance in spinal anesthesia but not for epidurals. Rotation of a Tuohy needle after identification of the epidural space can in itself lead to a large tear in the dura mater and should be avoided [16, 17]. Rotation of the epidural needle should, therefore, be discouraged.

20.1.3.3 Mode of Delivery

Another important risk factor for PDPH following ADP is the mode of delivery. There is a significant difference between women with an ADP who undergo a Cesarean delivery before entering the second stage of labor compared to vaginal delivery [18, 19]. A strong correlation between the length of time spent pushing and the risk of PDPH has been reported [4, 19]. PDPH occurred in more than 80 % of patients who delivered vaginally, compared to 17 % of those who did not push. This leads to a risk ratio of more than 4 to develop PDPH after an ADP followed by vaginal delivery as shown in Fig. 20.2.

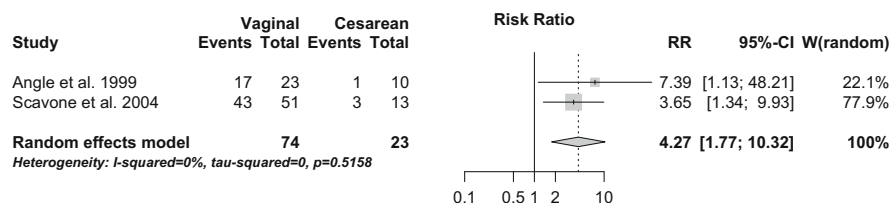


Fig. 20.2 Risk ratio by mode of delivery to develop PDPH following an ADP. Funel plot of the risk ratio to develop PDPH following an ADP in women who deliver vaginally or by Cesarean section. Data according to Angle et al. [18] and Scavone et al. [19]. Numbers are n for events and risk ratio and 95 % confidence interval, respectively

20.1.4 Diagnosis

PDPH is described by the Headache Classification Committee of the International Headache Society as follows [20]:

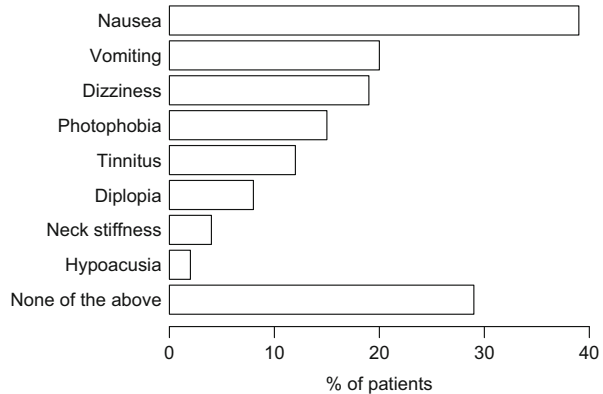
7.2.1 Post-dural (post-lumbar) puncture headache [G97.0] ICD-10: G44.820

- A. *Headache that worsens within 15 min after sitting or standing and improves within 15 min after lying, with at least one of the following and fulfilling criteria C and D:*
1. *Neck stiffness*
 2. *Tinnitus*
 3. *Hypacusia*
 4. *Photophobia*
 5. *Nausea*
- B. *Dural puncture has been performed*
- C. *Headache develops within 5 days after dural puncture*
- D. *Headache resolves either:*
1. *Spontaneously within 1 week*
 2. *Within 48 h after effective treatment of the spinal fluid leak (usually by epidural blood patch)*

This definition has been criticized, as it does not account for a duration of more than 1 week and because PDPH is accompanied by neck stiffness or auditory or visual symptoms in only 70 % of patients [11]. Therefore, an adaption of the classification has been proposed [11] and is already present in the current beta-version of the Third International Classification of Headache Disorders (ICHD-3) [21]:

Headache occurring within 5 days of a lumbar puncture, caused by cerebrospinal fluid (CSF) leakage through the dural puncture. It is usually accompanied by neck stiffness and/or subjective hearing symptoms. It remits spontaneously within 2 weeks, or after sealing of the leak with autologous epidural lumbar patch.

Fig. 20.3 Accompanying symptoms in patients with PDPH. Frequency of accompanying symptoms in patients with PDPH following dural puncture with a spinal needle. Data according to Amorim et al. [11]



Diagnostic criteria:

- A. Any headache fulfilling criterion C
- B. Dural puncture has been performed
- C. Headache has developed within 5 days of the dural puncture
- D. Not better accounted for by another ICHD-3 diagnosis.

Atypical headache, which is nonpostural, was found to be present in 5.6 % of patients (95 % CI 1.7–9.4 %) [22]. In these patients, fronto-temporo-occipital headache was the most common complaint [22]. This was frequently combined with stiffness and pain in the cervico-thoracic or lumbar spine [22]. As comprised in the above definition, PDPH occurs within 5 days of the dural puncture, two-thirds already occur within 48 h and almost 90 % within the first 3 days (Fig. 20.3) [10].

20.1.4.1 MRI

Magnetic resonance imaging (MRI) in patients with PDPH usually shows diffuse meningeal enhancement with signs of brain sagging [13]. Enhanced MRI can also be used to visualize the lumbar CSF leak. In the presence of typical symptoms of PDPH, imaging is usually not necessary. However, if the symptoms are atypical or persist after one—and definitely after two—epidural blood patches, then the diagnosis PDPH should be reevaluated [11, 12, 23].

20.1.5 Differential Diagnosis

A correct differential diagnosis is obviously important, but it can be quite challenging to achieve. The most important differential diagnoses are:

- Tension headache, migraine [2]
- Late-onset preclampsia [24]

- Withdrawal of caffeine [25]
- Cerebral venous sinus thrombosis [26, 27]
- Meningitis
- Intracranial (subdural) hemorrhage [28].

20.1.6 Prevention

In case of ADP, several potential measures to prevent PDPH have been presented. These include prophylactic epidural blood patch (EBP), epidural morphine, and adrenocorticotrophic hormone (ACTH).

20.1.6.1 Prophylactic EBP

As the incidence of PDPH is high after ADP for labor epidural analgesia, the option of prophylactic EBP has been investigated. Autologous blood is injected either through the epidural needle after being pulled back into the epidural space or through an epidural catheter positioned in the epidural space after the ADP. Although initial studies have shown promising effects, further randomized trials and a meta-analysis of the available data could not confirm any benefit in using prophylactic EBP [4, 18, 29–31].

20.1.6.2 Epidural Morphine

Epidural morphine has the potential to reduce the incidence of PDPH following ADP. One randomized controlled trial resulted in a significant reduction of PDPH from 48 % (95 % CI 28–68 %) in the control group to 12 % (95 % CI 3–32 %) in the intervention group [32]. There was no EBP in the morphine group (0 %, 95 % CI 0–16 %), compared to 24 % (95 % CI 10–45 %) in the control group [32]. Although these are promising results, the safety of 3 mg epidural morphine—in the presence of an ADP—is currently unclear [4].

20.1.6.3 ACTH

The use of ACTH and its analog has been investigated for the prophylaxis of PDPH. A single study has reported a significant reduction in PDPH [33]. So far, this is the only data in favor of ACTH. Further studies need to confirm this finding and determine the safe dosage before this treatment can be recommended [4].

20.1.7 Therapy

20.1.7.1 Conservative

Bed Rest

Since the symptoms of PDPH lessen in the horizontal position, the patient will naturally seek this position for relief. However, prophylactic and therapeutic bed rest has proven to be of no value [9, 10, 12, 34].

Fluids

Excessive fluid intake has no preventive or therapeutic value [12, 34, 35], and the increased diuresis can in turn increase symptoms due to more frequent mobilization of the mother.

Analgesics

Analgesic therapy with acetaminophen, nonsteroidal anti-rheumatic drugs (NSARD), or opioids cannot mend PDPH but can decrease the severity of symptoms [13]. Such supportive therapy can be useful until more invasive therapy—such as epidural blood patch—is considered or performed.

Caffeine

Caffeine causes cerebral vasoconstriction and could, therefore, theoretically alleviate PDPH [36]. Clinical studies could not confirm a clinically relevant effect, and the prophylactic or therapeutic use of caffeine has mostly been abandoned [4, 13, 37, 38].

Caffeine is also found in breast milk and can—dependent on dosing—lead to neonatal irritation. This could lead to a vicious circle where the neonate is hyperactive, which further reduces the time for resting for the mother with PDPH.

Sumatriptan

Serotonin receptor agonists lead to cerebral vasoconstriction. They are one of the essentials in migraine therapy. Evidence for the use of Sumatriptan for therapy of PDPH is scarce [39], and it cannot be recommended as a first-line therapy [8, 40, 41].

20.1.7.2 Epidural Blood Patch

The EBP is the only therapy that is effective and supported by evidence and, therefore, it represents the gold standard [42]. More than 80 % of patients who had an ADP with an epidural needle receive an EBP [43, 44]. The most common side effect of EBP is back pain. This occurs in more than 80 % of patients, and the median time from EBP until back ache is 27 h (95 % confidence interval 20–35 h) [45].

Contraindications

Contraindications to perform epidural analgesia are equally true for the EBP: refusal by the patient, infection at the site of puncture, coagulopathy, and severe systemic infection.

Technique

The procedure should be performed with the patient in the lateral position, as the PDPH renders the sitting position uncomfortable. At least two operators are requested, as one person has to take a sterile blood sample, while the other locates the epidural space. This is done either at the site of dural tap or one level below the previous insertion. It is self-evident that an experienced anesthesiologist should

locate the epidural space. He/she should be prepared for the presence of CSF in the epidural space [13]. For decades, the amount of blood to be injected has been a matter of debate. A recent study was designed to determine the optimal volume to be injected, and although the confidence intervals overlap, the authors concluded the optimal target volume to be 20 ml [45]. There is agreement that the injection of blood should be terminated once the patients feel a painful pressure in their back [13, 45, 46]. There is a relationship between the amount of blood injected into the epidural space and the epidural pressure that is generated, but interestingly, there does not seem to be a direct correlation between the injected volume and success rates [47].

We recommend using several sterile syringes, each with a volume of 5 ml, to draw the blood sample. The syringes should be filled at the same speed the second operator injects the blood through the Tuohy needle. This strategy avoids clotting of the blood in larger syringes or in the Tuohy needle. After the procedure, the patient should rest horizontally in his/her bed for 2 h [48].

Timing

Less satisfactory results were observed if the EBP was performed within the first 48 h after onset of symptoms. Although the initial success rate seems to be similar, the recurrence of symptoms is more frequent [43, 49]. This has led to a recommendation to postpone EBP to 48 h after initiation of PDPH. There might, however, be a substantial selection bias in these recommendations. Women with early and severe PDPH generally suffer more severe symptoms; therefore, it is expected to find a lower success rate of EBP in this group [12, 49]. There is no contraindication to perform an EBP within the first 48 h in women with severe PDPH.

Success Rate

Historically, EBP was reported to have a very high success rate exceeding 90 %. These numbers, however, overestimate the true success. First, ADP was frequently not considered separate from dural puncture with a spinal needle. Primary relief of symptoms is comparable in patients with PDPH following spinal anesthesia and those with ADP [49]. However, recurrence of PDPH is significantly higher following ADP (31 % vs. 5 %) [49]. Permanent relief of PDPH following ADP is between 30 % [12, 44] and 58 % [49] after a first EBP and partial or permanent relief in >85 % after a second EBP [43–45].

Alternatives

Alternatives for EBP have been investigated. These include the injection of saline or hydroxyethyl starch. ACTH was also investigated for its therapeutic value. Epidural injections of saline have not been proven to be effective [31]. Hydroxyethyl starch was used with some success in patients with systemic infection and might be an alternative in specific situations [50]. Unlike for prophylaxis for PDPH, there is no therapeutic value for ACTH [51]. Therefore, it is currently not recommended [38].

Intrathecal Catheter There is a theoretical advantage in threading a catheter into the intrathecal space should an ADP occur. This would temporarily close the lesion in the dura, and the presence of the catheter can trigger an inflammatory reaction that in turn stimulates healing [4, 5, 30]. Initial studies have been very promising and have shown marked reductions in PDPH, with 92 % and 6 % when a catheter is left intrathecally for 24 h and by resiting the epidural, respectively [52]. First meta-analysis has shown a reduction in EBP, while the risk of PDPH remained unchanged [53]. Subsequent work has not confirmed these results [5, 30]. In the randomized trial published by Russel et al., the authors report on another important result: resiting the epidural leads to a second ADP in 9 % of the patients [5]. As a consequence, it might be advantageous to insert an intrathecal catheter in the case of ADP. Especially in the case of difficult identification of the epidural space, this seems to be the better option [4]. However, it is of utmost importance to label the catheter accordingly and to inform every person potentially caring for this patient about the spinal catheter.

20.2 Rare Complications

The third national audit program in the United Kingdom investigated complications of neuraxial anesthesia [54]. The results were quite reassuring, especially for obstetric neuraxial anesthesia [54]. Complication rates were estimated on a “pessimistic” and on an “optimistic” basis. The pessimistic calculation included complications that were unlikely to be associated with neuraxial anesthesia, while the optimistic calculation did not. Permanent harm occurred “pessimistically” in 1.2 (95 % CI 1.0–3.2) and “optimistically” in 0.3 (95 % CI 0–1.7) per 100,000 patients [54]. Paraplegia or death did not occur in the obstetric cohort, and there were one epidural abscess and two cases of nerve injury [54]. Similar numbers were found in Sweden [55] and calculated in a meta-analysis with an incidence of long-lasting neurological injury of 1 in 240,000 obstetric neuraxial blocks [56].

20.2.1 Epidural Abscess

The epidural abscess is a very rare complication. The source of the infection is usually either contamination by the anesthesiologist or hematological spreading. In both cases, the most commonly identified organism was *Staphylococcus aureus* [57]. There are reports of the same type of bacteria isolated from an epidural abscess and from a nasal swab of the involved anesthesiologist [57]. But there are others where the infective organism was brought by hematological spreading, because the same type was identified vaginally [57].

There is no reason not to follow the aseptic rules of the operating room in neuraxial labor analgesia [58–62]. The use of sterile gloves, surgical hat, and face mask should be routine. Wearing a sterile gown is recommended by some national

societies [60, 61]. Unfortunately, the adherence of anesthesiologists to aseptic guidelines is rather poor [58].

The leading symptoms of epidural abscess are back pain and fever possibly with neurological symptoms. This triad was, however, only present in 13 % of patients and even leukocytosis was only present in 68 % [63]. The interval from the epidural procedure until the onset of symptoms is usually 4–10 days [64]. The diagnosis is confirmed by MRI and treatment is usually surgical combined with parenteral antibiotic treatment [63]. Minimal delay between diagnosis and treatment is important to ensure a positive outcome.

20.2.2 Epidural Hematoma

The use of low molecular weight heparin (LMWH) during pregnancy is increasing, and this implies a higher possibility of drug-induced coagulopathy when the parturient requires labor analgesia [65]. Different international guidelines all recommend an interval of 12 h following prophylactic and 24–36 h following therapeutic doses of LMWH [66–68]. Parturients with thrombocytopenia should be evaluated based on the underlying etiology. HELLP (hemolysis, elevated liver enzymes, and low platelets) syndrome is frequently accompanied by a disturbed function of the platelets, and in these the temporal dynamics of thrombocytopenia is more important than the absolute number. Platelets in idiopathic thrombocytopenia have a normal function. Therefore, lower values might still be acceptable for neuraxial procedures [68].

Impairment of coagulation can occur during or after delivery and, thus, after placement of the epidural. This might be the case in coagulopathy accompanying severe hemorrhage or in patients with severe preeclampsia, such as HELLP syndrome. Therefore, it is important to keep in mind that removal of an epidural catheter bears the same bleeding risk as placing it [69].

Symptoms of spinal hematoma are sensory and motor deficits, as well as back pain. Interestingly enough, sensory and motor deficits are more frequent and are, therefore, more important than back pain [69]. This fact underlines the advantages of preserved motor function with low-dose epidural labor analgesia. The importance for a high degree of alertness for sensory and motor deficits developing after delivery cannot be emphasized enough.

20.2.3 Subdural Hematoma

Cranial subdural hematoma is a very rare complication of epidural labor analgesia. It occurs after ADP, and the leading clinical symptom is persistent headache [57]. To reiterate, a persistent PDPH should lead to a high suspicion of possible cranial subdural hematoma [57]. Delayed treatment or protracted symptoms can lead to intracranial pathologies such as subdural hemorrhage or sinus vein thrombosis [2, 13, 70–72].

20.2.4 Spinal Cord Trauma

A direct trauma to the spinal cord results from needle injury. The conus medullaris usually terminates at the level of L1 or L2 [73]. Thus, the recommended level for neuraxial labor analgesia is below L2. Unfortunately, estimation of this level is far less than accurate and has been shown to be incorrect in more than 70 % of patients [73]. This estimation might be even more inaccurate in pregnant patients where hyperlordosis and weight gain might further impede identification of landmarks [64, 74]. The use of ultrasound can obviously increase the accuracy in detection of the correct lumbar level. But ultrasound is not always available, and clinical routine demands for an easier method. A recent investigation in pregnant women has confirmed that selection of the intervertebral space immediately caudal to the intercrystal line avoids L2/3 in 96 % of the patients [74].

20.2.5 Accidental Intravenous Injection

Accidental intravenous injection of amide local anesthetics has been a major safety issue in obstetric anesthesia and led to the withdrawal of 0.75 % bupivacaine in 1979. Today, very low concentrations of local anesthetics are used for labor analgesia and accidental intravenous injection of a small dose is very unlikely to pose a relevant threat to the patient. However, it is important to confirm the correct connection of epidural local anesthetic to the epidural route.

20.3 Postpartum Neurological Complications Due to Obstetrical Factors

Postpartum neurological complications after spinal or epidural analgesia or anesthesia are far more common due to obstetrical factors [69]. Complications usually arise from compression of a single nerve or nerve plexus. The incidence of neurological deficit is higher in patients with epidural analgesia. This is most probably a selection bias, as patients with prolonged labor experience more pain and the request for epidural analgesia is higher [57]. On the other hand, there is a possible contribution of neuraxial analgesia, as the parturient might be limited in mobility and the second stage of labor might be prolonged [64]. Although intrinsic obstetric neurologic complications are usually unilateral, they can also occur bilaterally [64]. If parturients with neuraxial labor analgesia experience postpartum neurological symptoms, then the first and sometimes only conclusion is to suspect a complication of neuraxial analgesia. Some anesthesiologists understand this as distrust in their neuraxial techniques, but in fact it is of great importance to address the most serious complications first. Symptoms of spinal cord or nerve root compressions due to an epidural hematoma can be difficult to distinguish from peripheral neurological complications (Table 20.1). Unless postpartum neurological complications are clearly identified as obstetric palsy, spinal hematoma should

Table 20.1 Nerve lesions and their typical sensory and motor deficit according to Wong [64]

| Sensory deficit | Motor deficit | Nerve |
|--------------------------------------------------------------------|-----------------------------------------------------|---------------------------------|
| Anterolateral thigh | None | Lateral femoral cutaneous nerve |
| Anterior thigh, medial leg, foot | Hip flexion, knee extension, patellar reflex | Femoral nerve |
| Medial thigh, knee | Thigh adduction | Obturator nerve |
| Lateral leg, dorsum of foot | Foot drop, possibly: foot plantar flexion (S1 root) | Lumbosacral plexus |
| Buttocks, posterior thigh, anterolateral lower leg, dorsum of foot | Knee flexion, foot drop | Sciatic nerve |
| Anterolateral lower leg, dorsum of foot | Foot drop | Common perineal nerve |
| Sole of foot | Foot plantar flexion | Posterior tibial nerve |

be actively excluded. Hence bilateral symptoms or atypical symptoms should be investigated by MRI in due time [69]. Recovery from spinal hematoma is best if there is a minimal delay of decompressive surgery.

20.3.1 Peripheral Lesion

20.3.1.1 Meralgia Paresthetica

Meralgia paresthetica is also known as “Bernhardt–Roth syndrome”. This is the most common obstetric palsy [64]. Irritation of the lateral femoral cutaneous nerve (L2, L3) in the region of the inguinal ligaments is the etiology of this neurologic syndrome [57]. Risk factors are obesity, external pressure, or prolonged flexion of the hips [64]. Meralgia paresthetica can also present during pregnancy due to increased lumbar lordosis [64]. The lateral femoral cutaneous nerve innervates the lateral and anterior side of the thigh. The clinical signs can be either irritation (i.e., burning and painful sensations) or a loss of function with numbness in the respective area [57]. Meralgia paresthetica is an important differential diagnosis to regional anesthesia-induced neuropathies [57]. Diagnosis can be confirmed by forceful palpation along the inguinal ligament, which reproduces the symptoms [57]. Relief of symptoms after infiltration with a local anesthetic in this region confirms the diagnosis [57].

20.3.1.2 Peroneus Nerve

Compression injury is the most frequent cause of common peroneal neuropathy. Compression can occur against the fibular head, e.g., due to a prolonged lithotomy positioning. Another cause is hyperflexion of the knees with compression around the tibia by the parturient’s hands [75] or prolonged squatting [64].

The superficial peroneal portion of the nerve is sensory and the lesion leads to a sensory deficit of the L5 dermatome, i.e., lateral leg and dorsal part of the foot [57,

64]. The deep peroneal nerve is a motor nerve and the leading symptom of its lesion is foot drop [57, 64].

20.3.2 Intrapelvic Lesion

The passage of the fetal head through the pelvis can compress neuronal structures and lead to neuronal damage [76]. Of possibly greatest importance are the lumbosacral trunk, the obturator nerve, and the femoral nerve as shown in Fig. 20.4 [76]. A lesion of the lumbosacral trunk involves nerve roots from L4 and L5. Typically, the contribution of L4 to the femoral nerve is not contained in the lumbosacral trunk. Hence, a lesion of the lumbosacral trunk spares the femoral nerve, and typically the patellar reflex is unaffected. A weakness of plantar flexion of the foot hints at compression of the first sacral root (S1).

20.3.2.1 Lumbosacral Plexus

All fibers of the L5 root and some of the L4 root travel through the lumbosacral trunk. While the psoas muscle protects the intrapelvic part of this nerve trunk, the last part before exiting the pelvis is in close contact with bone. At this site, the S1 root joins the lumbosacral trunk to form the sciatic nerve [77].

Compression of the lumbosacral trunk can lead to neuronal damage; foot drop is the most prominent sign. The clinical signs are very similar to a root lesion of L5. Nomenclature is far from standardized and contains “maternal birth palsy”, “post-partum foot drop”, and “maternal obstetrical sciatic paralysis” [77]. Compression neuropathy and, therefore, lumbosacral plexopathy are usually used by neurologists [77].

Neurological examination, such as nerve conduction studies, can localize the lesion distal to the dorsal root ganglion [77]. Another possibility for differentiation between L5 radiculopathy and lumbosacral plexopathy is the tibialis anterior muscle: the dual innervation (L4/L5) leads to moderate weakness in radiculopathy, while it is severely weakened in lumbosacral plexopathy [77].

Risk factors for lumbosacral plexopathy seem to be a cephalopelvic disproportion as well as prolonged labor and failure to descend [77]. Notably, all patients described in a recent series were delivered by secondary Cesarean section [77]. Intrapartum complaints on radiating leg pain were mentioned as a possible warning sign, but the authors also emphasized that epidural labor analgesia masks these signs [77]. Modern neuraxial labor analgesia with dilute local anesthetics and new delivery systems—such as programmed intermittent epidural boluses (PIEB)—have the potential to dramatically reduce motor block [78]. In such a setting, unilateral foot drop in women with prolonged labor might be a warning sign of lumbosacral plexopathy.

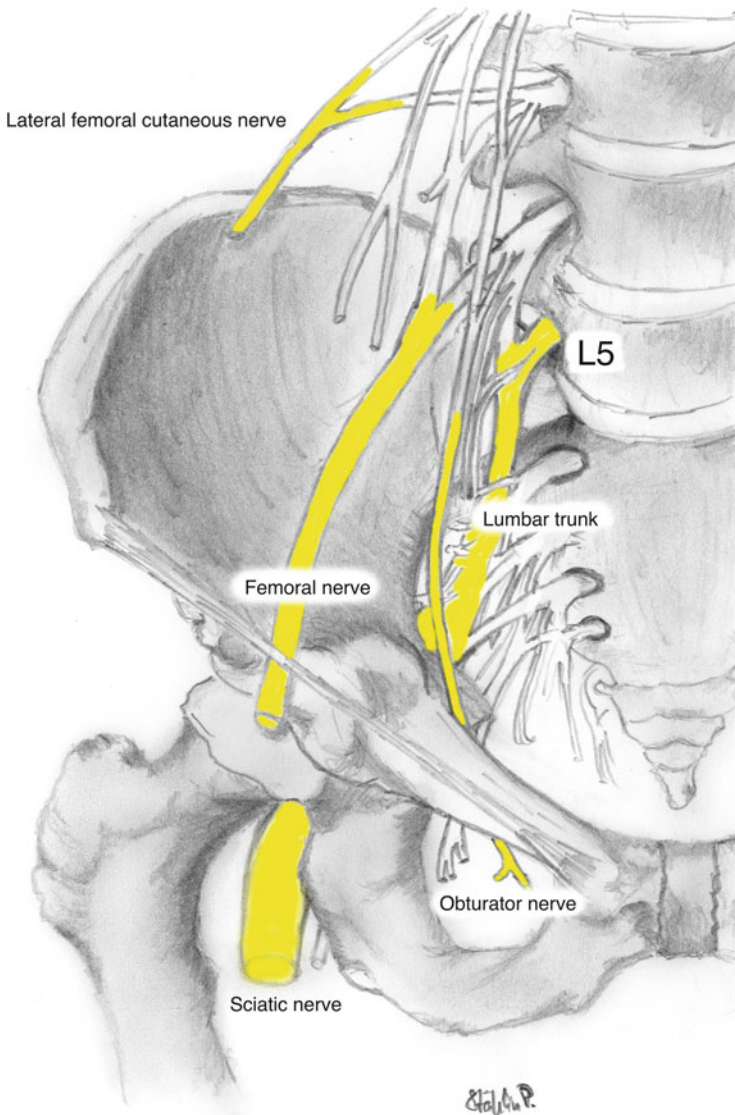


Fig. 20.4 Pelvic nerves possibly affected by delivery. Scheme of the pelvic nerves possibly affected by child birth. The most important are shown in *yellow*. These are: lateral femoral cutaneous nerve, femoral nerve, obturator nerve and lumbosacral plexus. The L5 root of the lumbosacral plexus is highlighted

20.3.2.2 Obturator Nerve

Again, compression by the fetal head is the origin of this neuropathy. Symptoms include hypesthesia over the upper inner thigh and weakness of hip adduction and rotation [57]. The latter might present itself in an abnormal gait [64].

20.3.2.3 Femoral Nerve

Causes of femoral nerve neuropathy are identical to neurology of the lumbar trunk: direct compression of the femoral nerve by the fetal head [57]. The lesion is caused by a stretch injury of the intrapelvic segment [64]. The leading clinical sign is the absence of patellar reflex and hypesthesia over the inner region of the thigh and medial lower limb. Postpartum femoral nerve neuropathy leads to a weakness in hip flexion and knee extension [64].

20.3.3 Therapy and Prognosis

Physiotherapy and patience are the most important therapies. Neurological complications associated with labor and delivery generally have a good prognosis. In most patients, symptoms resolve after a median duration of 6–8 weeks to a couple of months [64]. Neurophysiological investigations can shed more light on the extent of the neuronal damage and can be helpful for individual prognosis.

References

1. Davies JM, Posner KL, Lee LA et al (2009) Liability associated with obstetric anesthesia: a closed claims analysis. *Anesthesiology* 110:131–139
2. Stella CL, Jodicke CD, How HY et al (2007) Postpartum headache: is your work-up complete? *Am J Obstet Gynecol* 196:318.e1–318.e7
3. Reynolds F (1993) Dural puncture and headache. *BMJ* 306:874–876
4. Gaiser RR (2013) Postdural puncture headache: a headache for the patient and a headache for the anesthesiologist. *Curr Opin Anaesthesiol* 26:296–303
5. Russell IF (2012) A prospective controlled study of continuous spinal analgesia versus repeat epidural analgesia after accidental dural puncture in labour. *Int J Obstet Anesth* 21:7–16
6. Van de Velde M, Schepers R, Berends N et al (2008) Ten years of experience with accidental dural puncture and post-dural puncture headache in a tertiary obstetric anaesthesia department. *Int J Obstet Anesth* 17:329–335
7. Choi PT, Galinski SE, Takeuchi L et al (2003) PDPH is a common complication of neuraxial blockade in parturients: a meta-analysis of obstetrical studies. *Can J Anaesth* 50:460–469
8. Sprigge JS, Harper SJ (2008) Accidental dural puncture and post dural puncture headache in obstetric anaesthesia: presentation and management: a 23-year survey in a district general hospital. *Anaesthesia* 63:36–43
9. Darvish B, Gupta A, Alahuhta S et al (2011) Management of accidental dural puncture and post-dural puncture headache after labour: a Nordic survey. *Acta Anaesthesiol Scand* 55:46–53
10. Turnbull J, Bell R (2014) Obstetric anaesthesia and peripartum management. *Best Pract Res Clin Obstet Gynaecol* 28:593–605
11. Amorim JA, Gomes de Barros MV, Valença MM (2012) Post-dural (post-lumbar) puncture headache: risk factors and clinical features. *Cephalalgia* 32:916–923
12. Paech MJ (2012) Iatrogenic headaches: giving everyone a sore head. *Int J Obstet Anesth* 21:1–3. doi:10.1016/j.ijoa.2011.11.004
13. Turnbull DK, Shepherd DB (2003) Post-dural puncture headache: pathogenesis, prevention and treatment. *Br J Anaesth* 91:718–729
14. Weir E (2000) The sharp end of the dural puncture. *BMJ* 320:127

15. van Zundert AAJ, Reina MA, Lee RA (2013) Prevention of post-dural puncture headache (PDPH) in parturients. Contributions from experimental research. *Acta Anaesthesiol Scand* 57:947–949
16. Jordan MJ (1993) Dural puncture. Rotating needle increases risk. *BMJ* 306:1339
17. Bromage PR (1995) Rotation of the epidural needle: a caution. *Anesth Analg* 81:209–210
18. Scavone BM, Wong CA, Sullivan JT et al (2004) Efficacy of a prophylactic epidural blood patch in preventing post dural puncture headache in parturients after inadvertent dural puncture. *Anesthesiology* 101:1422–1427
19. Angle P, Thompson D, Halpern S, Wilson DB (1999) Second stage pushing correlates with headache after unintentional dural puncture in parturients. *Can J Anaesth* 46:861–866
20. International Headache Society Classification ICHD-II. <http://www.ihs-classification.org/enklassifikationteil.nonvascular.html>. Accessed 7 Sept 2014
21. Headache Classification Committee of the International Headache Society (IHS) (2013) The international classification of headache disorders, 3rd edn (beta version). *Cephalalgia* 33:629–808
22. Loures V, Savoldelli G, Kern K, Haller G (2014) Atypical headache following dural puncture in obstetrics. *Int J Obstet Anesth* 23:246–252
23. Rucklidge MW (2014) All patients with a postdural puncture headache should receive an epidural blood patch. *Int J Obstet Anesth* 23:171–174
24. Douglas KA, Redman CW (1994) Eclampsia in the United Kingdom. *BMJ* 309:1395–1400
25. Hampl KF, Stotz G, Schneider MC (1994) Postoperative transient hemihypaesthesia and severe headache associated with caffeine withdrawal. *Anaesthesia* 49:266–267
26. Jungmann V, Werner R, Bergmann J et al (2009) Postpartum cerebral venous sinus thrombosis after epidural anaesthesia. *Anaesthesist* 58:268–272
27. Stocks GM, Wooller DJ, Young JM, Fernando R (2000) Postpartum headache after epidural blood patch: investigation and diagnosis. *Br J Anaesth* 84:407–410
28. Schmidt A, Nolte H (1992) Subdurale und epidurale Hämatomme nach rückenmarknahen Regionalanästhesien. Eine Literaturübersicht. *Anaesthesist* 41:276–284
29. Agerson AN, Scavone BM (2012) Prophylactic epidural blood patch after unintentional dural puncture for the prevention of postdural puncture headache in parturients. *Anesth Analg* 115:133–136
30. Apfel CC, Saxena A, Cakmakkaya OS et al (2010) Prevention of postdural puncture headache after accidental dural puncture: a quantitative systematic review. *Br J Anaesth* 105:255–263
31. Boonmak P, Boonmak S (2010) Epidural blood patching for preventing and treating post-dural puncture headache. *Cochrane Database Syst Rev*. 20(1):CD001791. doi: [10.1002/14651858.CD001791.pub2](https://doi.org/10.1002/14651858.CD001791.pub2)
32. Al-metwalli RR (2008) Epidural morphine injections for prevention of post dural puncture headache. *Anaesthesia* 63:847–850
33. Hakim SM (2010) Cosyntropin for prophylaxis against postdural puncture headache after accidental dural puncture. *Anesthesiology* 113:413–420
34. Arevalo-Rodriguez I, Ciapponi A, Munoz L et al (2013) Posture and fluids for preventing post-dural puncture headache. *Cochrane Database Syst Rev* 7:CD009199
35. Dieterich M, Brandt T (1988) Incidence of post-lumbar puncture headache is independent of daily fluid intake. *Eur Arch Psychiatry Neurol Sci* 237:194–196
36. Choi A, Laurito CE, Cunningham FE (1996) Pharmacologic management of postdural puncture headache. *Ann Pharmacother* 30:831–839
37. Halker RB, Demaerschalk BM, Wellik KE et al (2007) Caffeine for the prevention and treatment of postdural puncture headache: debunking the myth. *Neurologist* 13:323–327
38. Basurto Ona X, Uriona Tuma SM, Martínez García L et al (2013) Drug therapy for preventing post-dural puncture headache. *Cochrane Database Syst Rev* 2:CD001792
39. Hodgson C, Roitberg-Henry A (1997) The use of sumatriptan in the treatment of postdural puncture headache. *Anaesthesia* 52:808

40. Connelly NR, Parker RK, Rahimi A, Gibson CS (2000) Sumatriptan in patients with postdural puncture headache. *Headache* 40:316–319
41. Baysinger CL, Pope JE, Lockhart EM, Mercaldo ND (2011) The management of accidental dural puncture and postdural puncture headache: a North American survey. *J Clin Anesth* 23:349–360
42. Malhotra S (2014) All patients with a postdural puncture headache should receive an epidural blood patch. *Int J Obstet Anesth* 23:168–170
43. Banks S, Paech M, Gurrin L (2001) An audit of epidural blood patch after accidental dural puncture with a Tuohy needle in obstetric patients. *Int J Obstet Anesth* 10:172–176
44. Williams EJ, Beaulieu P, Fawcett WJ, Jenkins JG (1999) Efficacy of epidural blood patch in the obstetric population. *Int J Obstet Anesth* 8:105–109
45. Paech MJ, Wong CA, Doherty DA et al (2011) The volume of blood for epidural blood patch in obstetrics: a randomized, blinded clinical trial. *Anesth Analg* 113:126–133
46. Safa-Tisseront V, Thormann F, Malassiné P et al (2001) Effectiveness of epidural blood patch in the management of post-dural puncture headache. *Anesthesiology* 95:334–339
47. Pratt SD, Kaczka DW, Hess PE (2014) Observational study of changes in epidural pressure and elastance during epidural blood patch in obstetric patients. *Int J Obstet Anesth* 23:144–150
48. Martin R, Jourdain S, Clairoux M, Tétrault JP (1994) Duration of decubitus position after epidural blood patch. *Can J Anaesth* 41:23–25
49. Kokki M, Sjövall S, Keinänen M, Kokki H (2013) The influence of timing on the effectiveness of epidural blood patches in parturients. *Int J Obstet Anesth* 22:303–309
50. Vassal O, Baud MC, Bolandard F et al (2013) Epidural injection of hydroxyethyl starch in the management of postdural puncture headache. *Int J Obstet Anesth* 22:153–155
51. Rucklidge MWM, Yentis SM, Paech MJ (2004) Synacthen depot for the treatment of postdural puncture headache. *Anaesthesia* 59:138–141
52. Ayad S, Demian Y, Narouze SN, Tetzlaff JE (2003) Subarachnoid catheter placement after wet tap for analgesia in labor: influence on the risk of headache in obstetric patients. *Reg Anesth Pain Med* 28:512–515
53. Heesen M, Klöhr S, Rossaint R et al (2013) Insertion of an intrathecal catheter following accidental dural puncture: a meta-analysis. *Int J Obstet Anesth* 22:26–30
54. Cook TM, Counsell D, Wildsmith JAW, Royal College of Anaesthetists Third National Audit Project (2009) Major complications of central neuraxial block: report on the Third National Audit Project of the Royal College of Anaesthetists. *Br J Anaesth* 102:179–190
55. Moen V, Dahlgren N, Irestedt L (2004) Severe neurological complications after central neuraxial blockades in Sweden 1990–1999. *Anesthesiology* 101:950–959
56. Ruppen W, Dery S, McQuay H, Moore RA (2006) Incidence of epidural hematoma, infection, and neurologic injury in obstetric patients with epidural analgesia/anesthesia. *Anesthesiology* 105:394–399
57. Loo CC, Dahlgren G, Irestedt L (2000) Neurological complications in obstetric regional anaesthesia. *Int J Obstet Anesth* 9:99–124
58. Ioscovich A, Davidson EM, Orbach-Zinger S et al (2014) Performance of aseptic technique during neuraxial analgesia for labor before and after the publication of international guidelines on aseptic technique. *Isr J Health Policy Res* 3:9
59. McKenzie AG, Darragh K (2011) A national survey of prevention of infection in obstetric central neuraxial blockade in the UK. *Anaesthesia* 66:497–502
60. Association of Anaesthetists of Great Britain and Ireland (2010) Best practice in the management of epidural analgesia in the hospital setting. Royal College of Anaesthetists, pp. 1–15. Churchill House, London
61. American Society of Anesthesiologists Task Force on Infectious Complications Associated with Neuraxial Techniques (2010) Practice advisory for the prevention, diagnosis, and management of infectious complications associated with neuraxial techniques: a report by the American Society of Anesthesiologists Task Force on infectious complications associated with neuraxial techniques. *Anesthesiology* 112:530–545

62. Hebl JR (2006) The importance and implications of aseptic techniques during regional anesthesia. *Reg Anesth Pain Med* 31:311–323
63. Grewal S, Hocking G, Wildsmith JAW (2006) Epidural abscesses. *Br J Anaesth* 96:292–302
64. Wong CA (2010) Nerve injuries after neuraxial anaesthesia and their medicolegal implications. *Best Pract Res Clin Obstet Gynaecol* 24:367–381
65. Abramovitz S, Beilin Y (2003) Thrombocytopenia, low molecular weight heparin, and obstetric anesthesia. *Anesthesiol Clin North America* 21:99–109
66. Gogarten W, Vandermeulen E, Van Aken H et al (2010) Regional anaesthesia and antithrombotic agents: recommendations of the European society of anaesthesiology. *Eur J Anaesthesiol* 27:999–1015
67. Horlocker T, Kopp S (2013) Epidural hematoma after epidural blockade in the United States. *Anesth Analg* 116:1195–1197
68. Working Party: Association of Anaesthetists of Great Britain & Ireland, Obstetric Anaesthetists' Association, Regional Anaesthesia UK (2013) Regional anaesthesia and patients with abnormalities of coagulation: the Association of Anaesthetists of Great Britain & Ireland The Obstetric Anaesthetists' Association Regional Anaesthesia UK. *Anaesthesia* 68:966–972
69. Moen V, Irested L (2008) Neurological complications following central neuraxial blockades in obstetrics. *Curr Opin Anaesthesiol* 21:275–280
70. Zeidan A, Farhat O, Maaliki H, Baraka A (2006) Does postdural puncture headache left untreated lead to subdural hematoma? Case report and review of the literature. *Int J Obstet Anesth* 15:50–58
71. Fiala A, Furgler G, Baumgartner E, Paal P (2012) Delayed subdural haematoma complicated by abducens nerve palsy and cortical vein thrombosis after obstetric epidural anaesthesia. *Br J Anaesth* 108:705–706
72. Borum SE, Naul LG, McLeskey CH (1997) Postpartum dural venous sinus thrombosis after postdural puncture headache and epidural blood patch. *Anesthesiology* 86:487–490
73. Broadbent CR, Maxwell WB, Ferrie R et al (2000) Ability of anaesthetists to identify a marked lumbar interspace. *Anaesthesia* 55:1122–1126
74. Srinivasan KK, Deighan M, Crowley L, McKeating K (2014) Spinal anaesthesia for caesarean section: an ultrasound comparison of two different landmark techniques. *Int J Obstet Anesth* 23:206–212
75. Bunch K, Hope E (2014) An uncommon case of bilateral peroneal nerve palsy following delivery: a case report and review of the literature. *Case Rep Obstet Gynecol* 2014:1–4. doi:[10.1155/2014/746480](https://doi.org/10.1155/2014/746480)
76. Besmer I, Schüpfer G, Hodel D, Jöhr M (2001) Postpartum neurologic complications following delivery with peridural analgesia. Case report with literature review. *Anaesthesist* 50:852–855
77. Katirji B, Wilbourn AJ, Scarberry SL, Preston DC (2002) Intrapartum maternal lumbosacral plexopathy. *Muscle Nerve* 26:340–347
78. Capogna GG, Camorcia MM, Stirparo SS, Farcomeni AA (2011) Programmed intermittent epidural bolus versus continuous epidural infusion for labor analgesia: the effects on maternal motor function and labor outcome. A randomized double-blind study in nulliparous women. *Anesth Analg* 113:826–831

Stephen H. Halpern and Rahul Garg

21.1 Introduction

Since biblical times, the labor process has been recognized as being one of the most painful human experiences. Early treatments varied widely, according to the cultural and religious practices of the time. In the middle ages, treatments such as amulets, magic girdles, and readings from the Christian liturgy were considered to be appropriate treatment. More invasive pharmacologic treatments such as the use of soporific sponges (a mixture of biologically active plants, inhaled or ingested) were sufficiently potent to cause unconsciousness. Of interest, bloodletting was used until the middle of the nineteenth century to cause swooning and thus pain relief [1].

Physicians and midwives that wished to relieve labor pain had to overcome a number of obstacles. Pain, although severe, was known to be self-limited and was not thought to be inherently dangerous to the health of the mother and newborn. In contrast, many treatments of the day carried significant risks to both. It is small wonder that a non- interventional approach was preferred.

Over the last 100 years, pain relief options have become safer and more effective. It became clear that medications that are given to the mother may influence the course of labor and may depress the baby at the time of delivery. Regional analgesia became an important method of providing effective pain relief. However, questions persisted about the effect on the progress of labor and subtle changes in the newborn. Often, fears of harm are based on poorly designed studies that were more likely to demonstrate the researchers' biases than truth.

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In this chapter, we will review the evidence base for providing labor analgesia. We will begin with a definition of “evidence-based medicine.” We will then discuss how to formulate a clinical question and to formulate a plan for best practice. Finally, we will discuss some of the topics that have a clear evidence base and areas for future research.

21.2 Evidence-Based Medicine

21.2.1 Definition

Evidence-based medicine is “the conscientious, explicit, and judicious use of current best evidence in making decisions about the care of individual patients” [2]. This approach must take the available clinical expertise and experience into account. In addition, patient preferences and expectations must be integrated into the process.

21.2.2 How to Use an Evidence-Based Approach

This approach can be broken down into four well-defined steps.

21.2.2.1 Ask a Clinical Question

Often, one is faced with a patient with a clinical condition that requires treatment. When considering labor analgesia, one is faced with a number of choices each with different advantages and disadvantages depending on the patient’s expectations, skills, and preferences of the healthcare providers, resources available and other considerations. The “PICO” format is often used as a template to help formulate the question. When considering labor analgesia, the Population must be considered. Are the subjects nulliparous, multiparous, or mixed? Are they healthy or are there obstetric or medical factors that place the patients at risk for adverse outcome? The Setting (private vs. public, academic vs. community) should also be considered. The Intervention is usually the experimental treatment. Examples might be method of initiation of analgesia (*combined spinal/epidural*, *epidural* alone), timing of the analgesia (early in labor or later), or drug used (ropivacaine, bupivacaine). The Comparator is the control. It is rare for *placebo* to be used as a comparator in this setting except for some non-pharmacologic treatments such as *transcutaneous electrical nerve stimulation* (TENS) or *intra-dermal sterile water* injections [3, 4]. In other trials, the control is almost always at least thought to be active. It could be *parenteral opioid* analgesia, a different form of regional block, or a different mode of maintaining analgesia. The main Outcomes should be clearly defined. Often, when drugs are compared, the main outcome is a measure of quality of analgesia. Sometimes, the main outcome is a particular side effect (operative delivery, motor block, nausea) or benefit (cord pH, maternal satisfaction). An example of how the

Table 21.1 The table illustrates how to use the “PICO” format to answer a clinical question

| Item | Example (from Wong et al. [5]) |
|-------------------|---------------------------------------------------------------------------------------------------------------------------------------------------|
| Population | Healthy nulliparous patients requesting epidural analgesia for pain relief |
| Intervention | Intrathecal fentanyl, followed by an epidural test dose before 4 cm dilation. Standard epidural infusion and patient controlled bolus maintenance |
| Comparison group | Parenteral opioid before 4 cm dilation, followed by epidural analgesia with standard infusion and patient controlled bolus maintenance |
| Outcome (primary) | Incidence of cesarean section |

The question in this case is: Is there harm in initiating epidural analgesia early in labor?

PICO format could be used to help formulate a treatment plan is shown in Table 21.1.

When designing a clinical trial, the best type of study (randomized controlled trial, cohort study, etc.) will depend on the clinical question and feasibility. Therefore, the “*PICOT*” format (with the “T” for Type of study) is often used to formulate research questions.

21.2.2.2 Search for the Best Evidence

Once the clinical question has been formulated, the next step is to search for the most reliable information available. A hierarchy of evidence has been formulated, with information at the highest level being (theoretically) the least susceptible to bias. In general, the hierarchy of evidence is shown in Table 21.2.

The type of information available will depend on the exact question. For example, the question posed in Table 21.1 describes two treatments (early vs. late epidural analgesia) and asks about common treatment harms (cesarean section). In that case, the most reliable information, as shown in Table 21.2, is a systematic review of randomized controlled trials. However, questions concerning diagnostic tests (e.g. will a test dose before epidural labor analgesia prevent harm?), or prognosis (e.g. what is the natural history of dural puncture headache with a large gauge needle?), may require other types of information. A summary of the hierarchy of evidence, depending on the clinical question, has recently been published [7]. However, the hierarchy in Table 21.2 pertains to the most common issues in labor analgesia therapy.

21.2.2.3 Critically Appraise and Combine the Evidence

Fortunately, clinicians rarely have to rely on individual studies to formulate a treatment plan. Many topics related to pain relief in labor have recently been systematically reviewed and are available in evidence-based guidelines [8, 9]. These are examples of guidelines that were created using recognized methodology by experts in the field and tested for validity by clinicians. In addition to making recommendations, the strength of the recommendations, using a modification of Table 21.2, is also reported. These guidelines are updated periodically to take into account new information.

Table 21.2 The hierarchy of evidence (adapted from [6])

| Level | Type of information |
|-------|--------------------------------------------------------------------------------|
| 1a | A systematic review of well-designed, homogeneous randomized controlled trials |
| 1b | Single large randomized controlled trial |
| 1c | All or none trial |
| 2a | A systematic review of homogeneous cohort studies |
| 2b | Individual cohort study or low quality RCT |
| 2c | Outcome studies |
| 3a | Systematic review of case controlled studies |
| 3b | Individual case controlled studies |
| 4 | Case series or seriously flawed studies of other designs |
| 5 | Expert opinion |
| 6 | Nonhuman (animal/in vitro) studies |

21.2.2.4 Determine the Best Treatment for Your Patient

While randomized controlled trials and systematic reviews can often be used as a guide to treatment, they do not give the whole picture. Factors such as the expertise of the clinician, expectations of the patient, and the resources available must also be considered when treating individual patients. For example, epidural analgesia initiated with a low concentration of *local anesthetic* may reduce the incidence of instrumental vaginal delivery [10], but it may not be the best treatment for a patient with rapidly progressing labor.

21.3 Topics in Analgesia for Labor with Systematic Review or Large RCT Support (Level 1)

There have been many randomized controlled trials that help guide practice in providing labor analgesia for our patients. Some are quite large and definitive, while others are small and yield a less precise estimate of effect. Taken together in a systematic review, a consistent pattern often emerges. Table 21.3 summarizes some of the questions that have been thoroughly studied and have level 1 evidence to support recommendations.

21.4 Conclusions

The optimal provision of analgesia in labor requires application of evidence-based medicine, “the conscientious, explicit, and judicious use of current best evidence in making decisions about the care of individual patients.” This involves four steps:

- (1) Asking a clinical question; the “PICO” format can be used as a template where the clinician considers the *Population*, the *Intervention*, the *Comparator*, and the *Outcomes* when formulating a question.

Table 21.3 Topics in analgesia for labor

| Clinical question | Reference # | Level of evidence | Best evidence | Recommendation |
|---------------------------------------------------------------------------------------------------------------------------------------------------------------------|-------------|-------------------|------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Compared to other forms of analgesia, how does epidural analgesia affect the progress of labor? | [11] | 1A | <ul style="list-style-type: none"> – No effect on cesarean section rate – May increase the risk of operative vaginal delivery rate – No effect on length of first stage of labor – Prolongation of second stage of labor by about 13 min | <ul style="list-style-type: none"> – Epidural analgesia provides the most effective analgesia compared to other forms – Fear of an increased incidence of cesarean section is unfounded – There may be an increased incidence of operative vaginal delivery |
| Compared to opioid analgesia, what is the efficacy of epidural analgesia and incidence of major side effects? | [12] | 1A | <ul style="list-style-type: none"> – Epidural analgesia provided superior analgesia during the first and second stages of labor and superior maternal satisfaction with analgesia – There was no difference in the incidence of long term back pain – There was a higher incidence of maternal fever and hypotension in the epidural group – There was a higher incidence of naloxone use and low 1 min Apgar scores in the opioid group | |
| Does administration of epidural analgesia early in labor increase the incidence of cesarean section or operative vaginal delivery compared to later administration? | [13] | 1A | There was no difference in the incidence of cesarean section or operative vaginal delivery when epidural analgesia was administered in the latent phase of labor compared to the active phase of labor | There is no need to delay epidural analgesia until the active phase of labor |

(continued)

Table 21.3 (continued)

| Clinical question | Reference # | Level of evidence | Best evidence | Recommendation |
|-----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|-------------|-------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| What are the effects of higher concentrations (>0.1 % bupivacaine) of local anesthetic compared to lower concentrations (≤0.1 % bupivacaine) when used for maintenance of epidural labor analgesia? | [10] | 1A | <ul style="list-style-type: none"> – No difference in maternal pain scores, maternal nausea, hypotension, or fetal heart rate abnormalities – No effect on cesarean section rate – Higher operative vaginal delivery rate – Increased incidence of lower limb motor block, with fewer patients able to ambulate – Increased incidence of urinary retention – Prolonged second stage of labor | <ul style="list-style-type: none"> – Low concentrations of local anesthetic are equally effective but cause fewer adverse effects than higher concentrations – Initial concentrations of bupivacaine should be less than 0.1 % |
| What are the advantages and disadvantages of combined spinal–epidural compared to an epidural alone for initiation of labor analgesia? | [14] | 1A | <ul style="list-style-type: none"> – CSE has a faster onset of analgesia (~3 min), but increases the risk of pruritis – No differences in maternal satisfaction, hypotension, mode of delivery, or neonatal outcome | Overall there is little difference between the two methods of analgesia, and it is not possible to recommend either method as superior |
| How does patient-controlled epidural analgesia (PCEA) compare with continuous epidural infusion alone for maintenance of analgesia? | [15] | 1A | Patient controlled analgesia required fewer unscheduled clinician interventions, used less local anesthetic, and caused less lower limb muscle weakness than continuous infusion | Where possible, patient controlled analgesia is preferred for maintenance of labor analgesia compared to continuous infusion alone |
| What is the best strategy for maintaining | [8, 16] | 1A | – The addition of a continuous infusion to PCEA | A continuous infusion should be |

(continued)

Table 21.3 (continued)

| Clinical question | Reference # | Level of evidence | Best evidence | Recommendation |
|---------------------------------------------------------------------------------------------------------------------------------------------------|-------------|-------------------|-----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------|
| epidural analgesia with PCEA? | | | <p>provides better analgesia and reduces clinician workload compared to PCEA alone</p> <ul style="list-style-type: none"> – There are a large number of regimens that specify different bolus doses, lockout intervals, and infusion rates, but there is insufficient evidence to show one is superior | added to PCEA regimens |
| What is the efficacy of intermittent mandatory boluses and PCEA compared to continuous background infusion and PCEA for maintenance of analgesia? | [17] | 1A | <ul style="list-style-type: none"> – Intermittent mandatory boluses may reduce the dose of local anesthetic, reduce second stage of labor duration, and increase maternal satisfaction – Too few patients studied to determine effect on clinician workload and other outcomes | This is a new and promising mode of maintenance of epidural labor analgesia, but more studies are required to make definitive recommendations on its use |
| Are there clinically important differences between the use of bupivacaine or ropivacaine for epidural analgesia? | [18, 19] | 1A 1B | <ul style="list-style-type: none"> – Low concentrations of both local anesthetics provide effective labor analgesia – Bupivacaine has a higher incidence of motor block compared to ropivacaine after prolonged usage – There is no significant difference between the two | There is insufficient information to recommend either ropivacaine or bupivacaine as superior for routine labor analgesia |

(continued)

Table 21.3 (continued)

| Clinical question | Reference # | Level of evidence | Best evidence | Recommendation |
|--------------------------------------------------------------------------------------------------------------------------------------|-------------|-------------------|------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| | | | agents in maternal satisfaction, mode of delivery, or neonatal outcomes – Currently, ropivacaine is more costly than bupivacaine – Overall there is little difference between the two agents in clinically used concentrations for epidural analgesia | |
| What is the effect of systemic opioids for labor analgesia? | [20, 21] | 1A 1B | – Systemic opioids provide minimal analgesia for labor – They are associated with maternal nausea, vomiting, and sedation – There is insufficient evidence for the superiority of particular opioids in terms of analgesia – Pethidine is associated with a higher incidence of drowsiness and nausea compared to other opioids | – Systemic opioids are less effective than regional techniques and are associated with adverse maternal effects – They may be considered if regional techniques are contraindicated |
| Compared to no analgesia or placebo what is the effect of nitrous oxide on labor pain, progress of labor, and maternal side effects? | [22] | 1A | – Nitrous oxide provides some pain relief during the first and second stages of labor – There was no effect on the progress of labor, incidence of cesarean section, or incidence of operative vaginal delivery | Nitrous oxide is a reasonable alternative for labor analgesia in institutions equipped to limit exposure to healthcare personnel |

(continued)

Table 21.3 (continued)

| Clinical question | Reference # | Level of evidence | Best evidence | Recommendation |
|-----------------------------------------------------------------------------------------------------------------------------------------------------|-------------|-------------------|-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|---------------------------------------------------------------------------------------------------------------------------|
| | | | <ul style="list-style-type: none"> – There is an increased incidence of nausea, vomiting, drowsiness, and dizziness | |
| What is the effect of transcutaneous electrical nerve stimulation (TENS) compared to placebo or standard care for the treatment of labor analgesia? | [23] | 1A | <ul style="list-style-type: none"> – TENS does not reduce the severity of labor pain or increase satisfaction with labor pain management – No difference in the incidence of cesarean section or operative vaginal delivery – No difference in neonatal outcomes | TENS has a limited role to play in treatment of labor analgesia |
| What is the effect of sterile water papule injections compared to placebo or standard care for the treatment of labor analgesia? | [24] | 1A | <ul style="list-style-type: none"> – No evidence of analgesic efficacy – No significant adverse effects or differences in maternal or neonatal outcomes | Further study is required to determine whether or not intradermal sterile water papules are effective for labor analgesia |
| What is the effect of acupuncture or acupressure compared to placebo or standard care for the treatment of labor analgesia? | [25] | 1A | <ul style="list-style-type: none"> – Some pain reduction reported compared to placebo, standard care, or no treatment – May reduce the need for pharmacologic intervention – No significant adverse effects reported – There were no studies in the analysis that had a low probability of bias | Insufficient data to determine the role of acupuncture or acupressure in the treatment of labor pain |

- (2) Searching for the best evidence; this will depend on the exact question formulated. There are established hierarchies of evidence based on study design which guide clinicians in determining the most suitable evidence base.
- (3) Critically appraising and combining the evidence; systematic reviews, meta-analyses, and evidence-based guidelines can provide clinicians with useful combined results and recommendations from a broad evidence base.
- (4) Determining the best treatment for specific patients taking into consideration their unique characteristics or clinical situations.

There are a number of topics in labor analgesia which have been extensively studied, with high level evidence available to support clinical practice. Neuraxial regional analgesia remains the most effective available modality for labor pain relief. Epidural analgesia does not increase the risk of cesarean delivery, although the second stage of labor may be prolonged, and there may be an increased risk of instrumental delivery. Epidural analgesia may be provided early in labor without affecting labor outcome. Although systemic opioids and nitrous oxide have some analgesic efficacy and may be considered if neuraxial techniques are contraindicated, they are less effective and can cause significant maternal adverse effects. There is little evidence to suggest that non-pharmacological techniques of analgesia (e.g., TENS, acupuncture, sterile water injections) are efficacious.

When initiating neuraxial analgesia, there is little difference between a combined spinal-epidural technique and epidural technique alone. Low concentrations of local anesthetic (e.g., $\leq 0.1\%$ bupivacaine) should be used for maintenance of analgesia to reduce the risks of motor block and instrumental delivery. Either ropivacaine or bupivacaine used at low concentrations can be safely and effectively used for epidural analgesia. PCEA along with background infusion is an effective and safe maintenance strategy. There is developing evidence that intermittent mandatory boluses may be superior to continuous infusion when combined with PCEA for maintenance of epidural analgesia; however, further research is required in this area.

References

1. Mander R (1998) Analgesia and anaesthesia in childbirth: obscurantism and obfuscation. *J Adv Nurs* 28:86–93
2. Sackett DL, Rosenberg WM, Gray JA, Haynes RB, Richardson WS (1996) Evidence based medicine: what it is and what it isn't. *BMJ* 312:71–72
3. Chao AS, Chao A, Wang TH et al (2007) Pain relief by applying transcutaneous electrical nerve stimulation (TENS) on acupuncture points during the first stage of labor: a randomized double-blind placebo-controlled trial. *Pain* 127:214–220
4. Bahasadri S, Ahmadi-Abhari S, Dehghani-Nik M, Habibi GR (2006) Subcutaneous sterile water injection for labour pain: a randomised controlled trial. *Aust N Z J Obstet Gynaecol* 46:102–106
5. Wong CA, Scavone BM, Peaceman AM et al (2005) The risk of cesarean delivery with neuraxial analgesia given early versus late in labor. *N Engl J Med* 352:655–665

6. Centre for Evidence Based Medicine, University of Oxford (2013) <http://www.cebm.net/?o=1025> . Retrieved 18 April 2014
7. Howick J, Chalmers I, Glasziou P et al (2011) The Oxford level of evidence 2. <http://www.cebm.net/index.aspx?o=5653> . Retrieved 10 Jan 2014
8. American Society of Anesthesiologists Task Force on Obstetric Anesthesia (2007) Practice guidelines for obstetric anesthesia: an updated report by the American society of anesthesiologists task force on obstetric anesthesia. *Anesthesiology* 106:843– 863
9. Obstetric Anaesthesia Special Interest Group (2008) Obstetric anaesthesia: scientific evidence, 1st edn. <http://db.wikipaces.com/file/view/Obstetric+Anaesthesia+Scientific+Evidence.pdf> . Retrieved 24 April 2014
10. Sultan P, Murphy C, Halpern S, Carvalho B (2013) The effect of low concentrations versus high concentrations of local anesthetics for labour analgesia on obstetric and anesthetic outcomes: a meta-analysis. *Can J Anaesth* 80:840– 853
11. Anim-Somuah M, Smyth RM, Jones L (2011) Epidural versus non- epidural or no analgesia in labour. *Cochrane Database Syst Rev* 12:CD000331
12. Leighton BL, Halpern SH (2002) The effects of epidural analgesia on labor, maternal, and neonatal outcomes: a systematic review. *Am J Obstet Gynecol* 186:S69–S77
13. Wassen MM, Zuijlen J, Roumen FJ et al (2011) Early versus late epidural analgesia and risk of instrumental delivery in nulliparous women: a systematic review. *BJOG* 118:655– 661
14. Simmons SW, Taghizadeh N, Dennis AT, Hughes D, Cyna AM (2012) Combined spinal-epidural versus epidural analgesia in labour. *Cochrane Database Syst Rev* 10:CD003401
15. van der Vyver M, Halpern S, Joseph G (2002) Patient-controlled epidural analgesia versus continuous infusion for labour analgesia: a meta-analysis. *Br J Anaesth* 89:459– 465
16. Halpern SH, Carvalho B (2009) Patient-controlled epidural analgesia for labor. *Anesth Analg* 108:921– 928
17. George RB, Allen TK, Habib AS (2013) Intermittent epidural bolus compared with continuous epidural infusions for labor analgesia: a systematic review and meta-analysis. *Anesth Analg* 116:133– 144
18. Halpern SH, Walsh V (2003) Epidural ropivacaine versus bupivacaine for labor: a meta-analysis. *Anesth Analg* 96:1473– 1479
19. Halpern SH, Breen TW, Campbell DC et al (2003) A multicenter, randomized, controlled trial comparing bupivacaine with ropivacaine for labor analgesia. *Anesthesiology* 98:1431– 1435
20. Douma MR, Verwey RA, Kam-Endtz CE, van der Linden PD, Stienstra R (2010) Obstetric analgesia: a comparison of patient-controlled meperidine, remifentanyl, and fentanyl in labour. *Br J Anaesth* 104:209– 215
21. Ullman R, Smith LA, Burns E, Mori R, Dowswell T (2010) Parenteral opioids for maternal pain relief in labour. *Cochrane Database Syst Rev* 9:CD007396
22. Klomp T, van Poppel M, Jones L et al (2012) Inhaled analgesia for pain management in labour. *Cochrane Database Syst Rev* 9:CD009351
23. Dowswell T, Bedwell C, Lavender T, Neilson JP (2009) Transcutaneous electrical nerve stimulation (TENS) for pain relief in labour. *Cochrane Database Syst Rev* 2:CD007214
24. Derry S, Straube S, Moore RA, Hancock H, Collins SL (2012) Intracutaneous or subcutaneous sterile water injection compared with blinded controls for pain management in labour. *Cochrane Database Syst Rev* 2:CD009107
25. Smith CA, Collins CT, Crowther CA, Levett KM (2011) Acupuncture or acupressure for pain management in labour. *Cochrane Database Syst Rev* 7:CD009232

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22.1 Introduction

The possibilities of pain control that are now within the reach of medicine allow analgesia in childbirth, i.e., they offer the chance of giving birth painlessly, thus overcoming the Biblical curse par excellence “I will greatly increase your pain in childbirth” (Genesis 3:16), and today this option is available to many women, although it should be recalled that the aforementioned biblical sentence was the guideline for conduct which passed from generation to generation right up to the mid-nineteenth century. Despite the weight of this cultural tradition, the use of anesthetics in childbirth was introduced at the request of women but was received with great reluctance by the medical community. Doctors were forced to offer it to their patients while questioning whether the benefits justified the potential risks. Aside from the cultural atavism, scientific arguments still persisted since there were unresolved issues especially regarding the effects of the drugs on newborns as well as the relationship between pain and labor [1].

In our opinion, all this raises several significant bioethical issues which will be dealt with in the following sections and focus on two main points: the woman’s personal choice of option in this respect and justice in accessing this, both in the context of protecting the future newborn as a general ethical and legal duty, and the professional obligation of healthcare staff that fits into the principles of non-maleficence and beneficence.

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22.2 About Pain and Suffering

Since it is clear that “pain is an unpleasant emotional experience,” interestingly, the pains of childbirth have been presented as joyful and a condition to being a “true mother” by linking this pain to the positive idea of bearing children, of giving life, and opposing this productive (and therefore “justified” and “natural”) pain to other forms of undesirable pain due to this being unproductive, which has been called useless pain.

According to Lévinas [2], all suffering is brute datum of consciousness, which carries a certain “psychological content,” as is the case with any feeling. However, this can be considered as something unacceptable, not only because it is related to the excessive intensity of a sensation that exceeds the measurement of sensitivity and the means for capturing and apprehending, but one which penetrates as a suffering that one undergoes by “disregarding physical and psychophysiological conditions”; in its pure phenomenology, the passivity of suffering is passive in a way that is deeper than our senses can perceive, which already becomes an activity of acceptance, which turns all this into perception. ./. . . In suffering, sensitivity is vulnerability, and is more passive than acceptance; it is a test, and is more passive than the experience, an evil to be exact. In fact evil cannot be described through passivity, but instead suffering is understood based on evil. Suffering is pure pain, extreme passivity, impotence, abandonment, and loneliness.¹ Here lies the fundamental ethical problem posed by “senseless” pain and the inevitable ethical problem and main priority of medication and analgesia appears as a duty. One considers whether the evil of suffering is not also a form of integration within an order and a sense, the possibility of giving life, producing positively and that is where the notion of “useful suffering” and even happily suffering is anchored—such as “love sickness”—or even a condition of demonstrating love itself and this creates a possibility of demanding retribution for past ills for the happiness of others: the child “owes” the mother not only his life but also the pain caused by his birth will create a duty of gratitude and filial love for life.

22.3 Characteristics of Pain in Childbirth

The contractions of the uterus in the first phase of childbirth cause a sharp characteristic cramping pain that increases in intensity as the contraction increases and then decreases when the uterus relaxes. In the second phase, new sources of pain appear in the sensitive structures of the pelvis and perineum distention [4].

¹ The International Association for the Study of Pain (IASP) defines pain as “an unpleasant sensory and emotional experience with actual or potential tissue damage or described in terms of this harm.” International Association for the Study of Pain, Subcommittee on Taxonomy [3] and size-S221.

The pain of uterine contractions is a pain similar to renal colic pain. But if several women who had had renal colic pain cramps and had also given birth (with wanted or unwanted pregnancy) were asked, it is likely that all would agree that the worst pain is renal colic pain followed by the pain that accompanied childbirth after an unwanted pregnancy and would consider the pain of childbirth from a wanted pregnancy the least unpleasant. This is because experiencing pain is more tied to the emotional and sensory experience than the intensity of pain. Not all people have the same threshold of pain, and therefore, the experience of pain is personal and nontransferable and very difficult to explain from a subjective viewpoint; thus, a pain of identical intensity produces an unpleasant sensory and emotional experience in some women and not in others.

22.4 Analgesia in Childbirth: Uncomplicated Versus Complicated Delivery

Since the second half of the twentieth century in childbirth pathology or complicated deliveries, one has always attempted to relieve the pain of the mother by means of different types of analgesia/anesthesia available in each period. In uncomplicated deliveries, a feeling of pain has been inherent to the evolution of childbirth.

If we take as an example, among many other possible valid ones, the situation in Spain's public health system in the 1980s, uncomplicated deliveries were generally attended by midwives and only if this became complicated, that is to say, if there were signs of risk for the fetus or mother, only then did the obstetrician and medical anesthesiologist become involved. Anesthesia was given to make patients fall asleep using Pentothal in low doses and maintaining spontaneous ventilation in vaginal birth or general anesthesia with mechanical ventilation if a C-section was performed. In the previous decade, there were some pioneers of epidural analgesia in labor,² but it was only at the end of the 1970s—with the arrival of anesthesiologists who had fled the Uruguayan and Argentine dictatorships and who were highly trained in this analgesic technique in childbirth—when it became popular in our country, thus achieving a better analgesia during delivery with a lower risk for the woman in labor. Thus, the use of “Pentothal” at birth was dropped completely. In Catalonia, epidural analgesia was used in uncomplicated deliveries in the public health system from the 1980s (Hospital Clinic, Hospital de Sant Pau, Hospital de Granollers, Hospital de Mataró, Hospital General de L'Hospitalet, . . .), but it only became widespread at the beginning of the following decade.

² Dr. Fernando Vidal, as well as being a pioneer, was a master in techniques of local anesthesia for several years in the training of specialists in Anesthesiology in Catalonia.

The involvement of anesthesiologists in analgesia in childbirth led to obstetrics specialists deciding on the optimal time to provide analgesia, and the anesthesiologist gave the minimal doses of local anesthetics needed to achieve adequate analgesia which provided comfort to the mother during the process of childbirth so as not to obstruct the process or affect the fetus. Currently, decisions are made within the framework of a protocol agreed between the midwife, anesthesiologist, and obstetrician.

In the private health sector, from the 1960s onward obstetricians started controlling the evolution of childbirth in which the midwife took part and where the analgesia/anesthesia was given by anesthesiologist as well as medical care during delivery. The so-called modified Bedoya method was generally used and was based on giving intravenous Pentotal with low concentrations during complete dilation and during delivery could be associated with sodium hydroxybutyrate (GammaOH) or not.

It is worth highlighting another major issue of bioethics, namely the existence of frequent conflicts of interest arising when a doctor practiced medicine in private and in public hospitals simultaneously, since if the epidural analgesia in childbirth became widespread in public hospitals and since these same obstetricians also had their private surgeries, they feared they might lose clientele as many women went to a private obstetrician precisely to have “a painless delivery,” i.e., delivery with analgesia. For many years now, the chance to receive analgesia in uncomplicated deliveries has been included in the list of services provided to all women in labor in the public health sector as another health benefit.

22.5 Autonomy? The Claim of Pain: From the Good Sufferer to the Trend of Natural Childbirth

From the above, one can see that women who give birth are clearly excluded from any discussion, and the patient's right to informed consent yields, in practice, when it comes up against technical criteria, probably due to separating women from their own childbirth. Certain trends have arisen which assert the leading role of women in this process as their own and as something natural so they demand that the relationship and link between the mother and the newborn child be respected from the first moment of life and not always dictated by technique.

There is surely some abuse felt in the modernization and coldness of the operating room and in the sedation that leads the woman in labor to miss joyful moments, moments of transcendence that are so important in life as the birth of a child, yet this has led to opposition which, in our opinion, involves other serious dangers: the myth of natural childbirth that is perfectly acceptable if no complications arise, yet should they arise, safety criteria for the mother and the future child must be applied. One must not forget the chilling figures of mortality in childbirth in times and places when the assistance of qualified professionals is not available. In contrast, according to the Spanish Society of Gynecology and

Obstetrics, in 2013 maternal mortality in Spain reached 4.7 women per 100,000 live births and perinatal mortality 8.3 deaths for every 1,000 births.³

However today, especially in high cultural and economic circles, many women request the so-called “natural childbirth,” “home birth,” “birth under water,” and all sorts of alternatives which can leave them completely defenseless and this can clearly endanger both mother and infant should any complications arise. This trend is part of a broader back-to-nature view, which also includes valuing alternative medicines so that they are no longer considered purely supplementary. This can be seen in many other areas of health relations in our societies, but it is quite striking in the field of obstetrics, as it shares space with many other similar fashions or contrary to these as the ones linked to biotechnology such as the proliferation of private umbilical cord banks, etc.

In a different socioeconomic and cultural context, there has also been and there are women who have considered suffering in childbirth as a duty and this compliance carries both social recognition and obligations of affection from the child [5]. This is the case of something called “the good sufferer” by Cañas Romero et al., in a study which investigated the reasons why epidural analgesia had not been used in 15.4 % of births attended in the period analyzed. In this work, most of the mothers interviewed had received primary school education, and although 90 % had been informed about epidural analgesia, only in 18 % of the cases this information had been provided by the anesthesiologist. 13 % of mothers did not want epidural analgesia and, in addition, their opinion was reinforced for future births. The study does not analyze the reason why epidural analgesia was rejected since it was not designed specifically for this. Several reasons might explain this phenomenon. Firstly, given their social level, there could be a lack of information or the information was “not adequate” for pregnant women. Secondly, in accordance with the authors’ opinion on the traditional concept of the good mother or good sufferer, the influence received from their family environment must not be overlooked when it comes to decision making, without forgetting that pain can be, unconsciously at times, a strategy to obtain attention and recognition from others.

22.6 Conclusions

The fundamental bioethical principles involved in analgesia in childbirth are:

- (a) The principle of autonomy linked to women’s informed choice about whether they want analgesic or not, which must be respected though not absolutely as each condition of childbirth must be considered together with the safety and the welfare of the future child.

³ According to the WHO, maternal mortality has been estimated between 2 and 15 % per 100,000 live births in developed countries.

- (b) The principle of justice since this option should be available to all women regardless of their economic situation, which requires this be provided not only in the private healthcare sector but also as part of the universal coverage in the free public health sector.
- (c) The principle of non-maleficence is shown in the aforementioned conflict of interest which led to the nonuse of analgesic techniques for all women in labor who requested it, causing a clearly avoidable and unnecessary prejudice.

References

1. Celesia C (2004) Brief history of analgesia in obstetrics. *Magazine of the Hospital Materno Infantil Ramón Sardá* 2004, 23 (No month). ISSN 1514-9838. Available at: <http://www.redalyc.org/articulo.oa?id=91223307>. Date of reference: 20 March 2014
2. Lévinas (1998) *Entre Nous: essays on thinking-of-the-other*. Columbia University Press, New York, NY, 256 pp. ISBN: 978-0-231-07911-2
3. International Association for the Study of Pain, Subcommittee on Taxonomy (1986) Classification of chronic pain. *Pain* 3:S3–S12
4. Fernández MA, Ros J, Villalonga A (2000) Failures in obstetric epidural analgesia and its causes. *Rev ESP Anesthesiol Reanim* 47:261
5. Echevarria Moreno M (2010) Analgesia del parto: ¿qué más podemos hacer? *Rev Soc Esp Dolor* 17(1):1–2. ISSN 1134-8046. Available at: http://scielo.isciii.es/scielo.php?script=sci_arttext&pid=S1134-80462010000100001&lng=es&nrm=iso

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23.1 Introduction

Teaching and learning in anesthesiology is a demanding and complex task because the majority of the teaching and learning takes place in clinical settings, where the safety and comfort of the patient must be balanced against the responsibility for the education of the trainee. Teaching and learning epidural for labor analgesia can be especially demanding because the patient may be in severe pain and therefore be unwilling to participate in a teaching encounter. Also, the clinical setting in a delivery room where relatives are present may not be a comfortable environment for teaching and learning. Epidural for labor analgesia can therefore be difficult to teach and learn in a clinical setting and is a challenge to both the teacher and the trainee [1, 2].

Simulation-based training seems to have a place in learning this advanced clinical procedure before clinical training is initiated [2, 3]. Along with training advanced clinical procedures simulation is the ideal place for training communication skills with the patient and with the inter-professional team. In addition, simulation-based training can provide the opportunity for trainees to apply medical expertise skills in context and train with the team.

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For optimal learning the trainee must focus on clear learning goals and be willing to learn and be guided by feedback from the educator. The educator must on the other hand be able to promote a positive learning climate with clear learning goals and supportive feedback to facilitate the trainees' self-directed learning [2, 4, 5].

23.2 Trends in Medical Education

In this section, three major trends in medical education are described: the seven physician roles, simulation-based training, patient safety and training of teams.

23.2.1 The Seven Roles

The approach to medical competence has changed over the last decade. Previously, the focus was mainly on teaching trainees the required clinical knowledge and procedural skills to practice as a specialist. Now it is generally accepted that physicians must possess a defined body of knowledge, clinical skills, procedural skills, and professional attitudes to ensure proficiency. One of the first attempts to describe this is the CanMEDS competency framework [6]. It consists of seven roles: Medical Expert, Communicator, Collaborator, Manager, Scholar, Professional, and Health Advocate. The framework has been adopted in Denmark, the Netherlands, and Australia [7–9].

The medical domain has adapted another way of looking at competence from aviation by dividing it into technical skills and nontechnical skills. Technical skills are the skills related to the role of the medical expert. Nontechnical skills (NTS) can be defined as “the cognitive, social, and personal resource skills that complement the technical skills, and contribute to safe and efficient task performance” [10]. NTS for anesthesiologists is described in the Anaesthetists' NonTechnical Skills (ANTS) [11]. NTS include skills in situation awareness, decision making, communication, teamwork, leadership, and management of factors such as disturbances and stress. Although NTS is a different framework than the CanMEDS, NTS can be seen as a means to describe some of the roles (e.g., communicator, collaborator, manager, and professional).

23.2.2 Simulation-Based Training

The technological evolution has made it possible to develop tools to be used at all stages of professional development—from novice to expert level. Simulation-based training ranges from basic skill trainers and simulated patients to advanced surgical and patient simulators which can be used to train complex medical situations for individuals or teams. The rationale for simulation-based training is to improve patient safety (see below), to provide training opportunities that might not be

available in the clinical setting, and to facilitate learning at the individual and team level. Simulation provides the link between knowing and doing. Simulation-based training is followed by debriefing. This combination seems to improve reflection and improvement of competence of the individual [12].

23.2.3 Patient Safety: Training of Teams

Studies have documented that a range of adverse events occur in more than 10 % of hospital admissions [13]. Research has established the link between NTS, performance, and adverse events [14]. The role of communication in the operation room has been studied extensively.

The good news is that many of these are preventable as NTS are trainable skills. Neily et al. found that training of NTS (implementation of team training, introduction of briefing and debriefing) reduced the mortality rate by 11 % when compared to a control group [15].

Most tasks are handled by teams, which might not have worked together previously. We are trained in “silos of care” in the different professions and specialities. In order to increase patient safety, team training activities should be implemented in the organization as described by Neily et al. [15].

23.3 Setting Learning Goals for Labor Analgesia

To learn the theory and procedural skills in the placement of an epidural for labor analgesia, the trainee first needs to understand the complexity of the task. Learning to provide epidural labor analgesia is not just learning how to place the epidural but a complex task that involves theoretical knowledge, technical skills, and NTS. A precise description of the task and specific learning goals for the procedure must be established before training can begin. The learning goals must include learning goals on the theory, which is mandatory for the trainee to be accomplished before practical training can begin.

The majority of learning goals can be taken directly from the curriculum, but data from patient safety and quality databases can help focus on where to improve. Furthermore questionnaires administered to the trainee can be used to identify the difficult aspects of labor analgesia.

23.3.1 Learning the Medical Expertise Skills: Theory and Procedural Skills

The learning goals addressing the theoretical knowledge must be clear and meaningful in relation to the procedural skills, and it is mandatory for the trainee to have a basic theoretical knowledge prior to training procedural skills. Examples of learning goals are listed in Table 23.1.

Table 23.1 Learning goals

| |
|--------------------------------------------------------------------------------|
| <i>Theoretical learning goals</i> |
| Understand the anatomy |
| Know the risks and complications of epidural placement and how to manage these |
| Know the different local anesthetics and how to use these |
| Know dosing of local anesthetics |
| Know the contraindications for epidural placement |
| <i>Learning goals for technical skills</i> |
| Know the equipment |
| Use correct aseptic technique |
| Use a safe method for placing the epidural |
| Test the epidural for correct placement |
| Use correct medication |
| <i>Learning goals for nontechnical skills</i> |
| Leadership (take leadership, guide the team) |
| Communication (closed loop, information) |
| Team working (coordination, support) |
| Task management (plan, prepare, prioritize) |
| Situation awareness (recognize, understand) |
| Decision making (identify options, reevaluate) |

23.3.2 NTS Training and Team Training

As described in Sect. 23.2, the seven roles of the physician are important to become proficient in labor analgesia. It is important to understand that insertion of an epidural catheter for labor analgesia is not only a procedural skill, but it also involves roles other than that of the medical expert such as a communicator and collaborator [6].

NTS are critical for good anesthetic practice and must be addressed.

Communication with the parturient and the relatives is important. A specific learning goal must be included to address this. Placing epidurals for labor analgesia is inter-professional teamwork. The team players are the anesthetist, a midwife, and sometimes students and a nurse. One can also see the parturient and her relatives as part of the team. Good teamwork is essential if the procedure should be encountered as a successful experience by the parturient. Therefore NTS must be trained for the team to be successful and the tasks, roles, and mutual expectations must be known by all team members. It is important for the trainee to learn how to manage this part of the procedure, and the first step is to address the competencies in learning goals. Examples can be seen in Table 23.1.

23.4 Methods for Learning Labor Analgesia

In this section the different methods to be used in the learning process will be addressed, especially the methods used for procedural skill training and simulation-based training will be discussed.

23.4.1 Theoretical Knowledge

Lectures and educational sessions in the department are usually used to support the trainee in the learning process. The trainee may be introduced to relevant reading material and theoretical questions, and learning goals may be discussed with the clinical teacher to facilitate learning before training of the procedural skills. This may help to increase the confidence of the trainee in advance of skill training. An e-learning programme can be developed, which makes it easier to plan and be more flexible for the trainee. It provides an opportunity to repeat if necessary.

23.4.2 Training of Procedural Skills

To teach and learn the procedural skills in epidurals for labor analgesia is a challenge in the clinical setting. Therefore, the trainee may benefit from basic technical skill training in a simulator before training in the clinical environment begins.

Several central neural blockade simulators exist from the simple “greengrocer’s” model to lifelike high-fidelity devices [16, 17]. However, no simulator is demonstrated to be superior in teaching technical skills. The important thing is for the trainee to be able to train in the procedural skills—an epidural insertion technique—in a safe environment without the risk of damaging the patient. Simulation training of procedural skills is shown to improve knowledge and skills in both simple procedures and more complex procedures such as laparo- and endoscopic procedures [18]. A three-step teaching technique can be used where the educator first shows the procedure in the simulator without explaining while the trainee observes. Afterward the educator repeats the procedure and at the same time explains all the steps systematically to the trainee. Finally the trainee does the procedure explaining all the steps to the educator. The last step is repeated until the trainee feels confident.

An important task in placement of an epidural is a correct aseptic technic, but teaching epidural catheter placement tends to overlook this important step. Video-assisted teaching where a teaching video highlights key steps of a thorough aseptic technique is demonstrated to significantly improve aseptic practice and can be recommended. It is likely that good aseptic technique habits taught early in training may instill a higher level of practice over longer periods of time.

23.4.3 Training of NTS

Communication with parturient and relatives can be trained using simulated patients and role-playing different scenarios with increasing difficulty from information about inserting an epidural catheter to information about an adverse event.

Teamwork in relation to epidural insertion for labor analgesia is difficult to train in a clinical setting. The parturient is in pain, the atmosphere may be tense, and

training teamwork along with technical skills in this situation may not be acceptable for the parturient. Several of the seven roles that physicians must possess to provide effective patient-centered care can be trained using simulation-based training. Furthermore simulation can provide a safe learning environment where patients are not harmed and provides the possibility for repetitive practice. Simulation-based training of applying the medical skills and of teamwork skills using a variety of different scenarios seems to have proven to be effective for obstetric teams [19]. Simulation-based training is educationally effective and can complement medical education in patient care settings under the right conditions [20]. The most important factor for learning is feedback [20].

A structured debriefing is a mandatory part of full-scale simulation and can be directed by the learning goals [12]. Providing feedback is a complex process and a structured approach is recommended. Often a three-phased structure is used consisting of a description, an analysis, and an application phase [21]. Here the trainees are asked to critically reflect on actions in the simulation and explore alternatives and areas for development. Often the trainees are asked to write a learning plan for future development in the clinical setting to improve transfer to the clinical tasks.

23.4.4 Teaching and Learning in the Clinical Setting

Teaching and learning in the clinical setting is challenging, but a well-prepared trainee will be able to handle the procedure. Throughout theoretical learning and training in the simulator, the trainee may benefit from observing the teacher in the clinical setting and be able to reflect. This may prepare the trainee for the transition from the simulated to the clinical setting. After theoretical preparation and training in a simulator, the trainee can begin training in the clinical setting. The educator must supervise the trainee and training must be guided by the learning goals. In the clinical setting it is vital to provide feedback, which is a cornerstone of effective clinical teaching. With feedback the trainees' strengths are reinforced and errors can be corrected. It is also important to encourage reflection and self-directed learning. Learning and success can be guided by CUSUM scoring and assessment [22]. The trainee should be confident and skilled in all aspects before performing the procedure alone.

The role of the educator is described in Sect. 23.6.

23.5 Evaluation/Assessment of Learning

Assessment of learning (or competence) is one aspect that does not get enough attention in teaching labor analgesia. Often it is only a matter of counting how many epidurals for labor analgesia the trainees have performed. Not all the trainees

receive adequate training before performing their first labor epidural and seldom formal assessment is performed [23].

Just as it is important to decide and describe which learning objectives to include in postgraduate training (knowledge, skills, and attitudes), it is also important to decide how these can be assessed. The first question to consider is “What is the purpose of our assessment?” Is it a formative assessment designed to promote further learning or a summative assessment aiming to evaluate whether the trainee has the necessary competence to perform a given medical activity unsupervised.

Ideally both types of assessments should be used, formative assessment during the specialist training to guide learning and summative assessment at specific time points. This could be one of the milestones in the training [24].

Assessment plays a major role in how trainees learn. Assessment drives learning [25, 26], which means that if both the role of the medical expert and the roles of the communicator and collaborator are assessed, the trainee will focus on all aspects.

23.5.1 Assessment of Learning/Competence

The Miller’s pyramid is often used to illustrate the different layers of competence. The attainment of each level assumes the attainment of the lower levels. The lowest level of the pyramid (the base) is knowledge, the “knows” level. Competence at this level can be tested using written exams such as multiple-choice questions (MCQ). The next level of competence is “the knows how” level, where the ability to apply knowledge can be assessed by case-based assessment. The third level is the performance level, the “shows how” level. Here objective structured clinical examinations (OSCE) and clinical exams are used as assessment methods. The tip of the pyramid is the action, “the does” level. Assessment of competence in the clinical setting is done using direct observation practical skills (DOPS) or competence cards where the clinical performance of the trainee is in focus [2, 27]. The assessment of competence should include not only the role as medical expert but also the roles of collaborator, communicator, manager, and professional. Ideally the assessment of several roles should be included in one assessment of a work situation. The assessment should include all three levels of competence: (1) knowledge and skills, (2) ability to apply knowledge and skills, and (3) attitudes and personal abilities.

Assessment in the clinical environment is quite challenging, but it is important in order to secure patient care of high quality. Assessment should reflect what trainees/doctors do in actual patient care. As mentioned, DOPS and other types of checklist for systematic observation and assessment can be used to evaluate trainees’ skills in labor analgesia. This should be used in combination with an experience log to ensure a minimum number of procedures to obtain a given level of routine (experience). The most important aspect for the trainee, however, is to receive constructive feedback in order to improve their skill. Feedback is found to be the cornerstone of

clinical training; however, clinicians can be reluctant to give honest feedback and the quality of feedback can be poor [28]. A set of clearly defined criteria makes it, according to Norcini, easier to provide guidance based on observed performance [25]. If the performance of the trainee is marginal, it is very important to develop a plan of action to develop the trainees' skills and reassess later.

23.5.2 Evaluation of Effect of Programs

Evaluation of the training program is compulsory. Kirkpatrick's four-level model of evaluations is a way to structure evaluation of a training program [29]. Level one, the reaction level, measures the participants/trainees satisfaction with the training/intervention. Level two, the learning level, describes the degree of change in the trainee's knowledge, skills, and attitudes. It is measured as described in the previous section of the Miller's pyramid. There are several examples of the effect of simulation-based training on knowledge and skills [30]. Level three is the behavioral or organizational level that illustrates the impact on the clinical practice: Do they follow the guidelines? Are there any changes of routines in the organization? Finally, level four represents the patients' outcome level. It describes the benefit for the patients, such as a lower incidence of dural puncture. It is both challenging and resource demanding to evaluate a program at all four levels. A large study population is needed in order to show differences at the outcome level. One example is the study of Draycott et al. showing improvement in perinatal outcome after simulation-based team training [19].

23.6 The Educator and the Learning Environment

23.6.1 The Teacher and the Teaching Models

Teaching epidural labor analgesia is demanding and the educator must be more than a medical expert to succeed. Nevertheless, many clinicians in teaching hospitals have received no formal training in how to teach and base teaching on their own learning experiences [35]. The roles of the medical teacher are described: (1) the information provider, (2) the role model, (3) the facilitator, (4) the assessor, (5) the curriculum course planner, and (6) the resource material creator [31]. For successful teaching institutions need to provide support and training for their clinical teachers. The skills that make the clinical teacher excellent are outlined in the box below [32].

Skills that Makes a Clinical Teacher Excellent

- Passion for teaching
- Clear, organized, accessible, supportive, and compassionate
- Able to establish rapport; provide direction and feedback; exhibit integrity and respect for others
- Demonstrate clinical competence
- Utilize planning and orientation strategies
- Possesses a broad repertoire of teaching methods
- Engage in self-evaluation and reflection
- Draw upon multiple forms of knowledge; target teaching to the learners' level of knowledge

In the following box a simple framework for daily teaching is described by Neher [5]. To give feedback and facilitate learning the clinical teacher must be a content expert and able to create a positive learning environment as mentioned in step 1. Also, the teacher must have good listening skills with enthusiasm for teaching and enthusiasm in general [33].

The educators' most important task is to facilitate learning and stimulate the trainees' reflection on competence. The trainee needs to set new learning objectives and continuously develop competence in order to achieve proficiency.

Another major issue is faculty development in order to train the use of the assessment methods [35, 34]. Without faculty development the assessment of the trainee in the simulated and the clinical setting is not likely to happen with sufficient quality. In case of major deficiencies the action taken needs to be based on reliable and valid assessments.

The Five Important Steps to Facilitate Learning

- Step 1 Create a safe learning environment. Let the trainee manage and lead the procedure
- Step 2 Encourage the trainee to explain and speak out loud during the procedure. Encourage or gently correct during the procedure
- Step 3 Guide the trainee to learn and understand general rules
- Step 4 Reinforce what I have done well
- Step 5 Correct mistakes. Encourage self-assessment

23.7 Conclusion

In conclusion, a systematic approach to teaching and learning labor analgesia is important. Clear learning goals addressing knowledge, skills, and attitudes within the seven roles of a physician should be described. Practical skill training is

valuable before a procedure is performed on a patient. Simulation-based training seems to be valuable to train the team. The integration of the training into the curriculum is important. Structured workplace-based learning and assessment is recommended. Faculty development to ensure the quality of the assessment and the feedback is of utmost importance.

23.8 Recommendations

For optimal teaching and learning the following steps are recommended:

- Description of clear learning objectives for knowledge, skills, and attitudes within the seven roles of a physician
- Practical skill training of procedures before the procedure is performed in the clinical setting
- Simulation-based training for the multi-professional team
- Development of structured assessment tools to stimulate learning and reflection
- Training of the faculty in providing feedback and assessing learning
- Develop a safe learning environment.

References

1. Spencer J (2003) Learning and teaching in the clinical environment. *BMJ* 326(7389):591–594
2. Ramani S, Leinster S (2008) AMEE guide no. 34: teaching in the clinical environment. *Med Teach* 30(4):347–364
3. Friedman Z, Siddiqui N, Katznelson R, Devito I, Bould MD, Naik V (2009) Clinical impact of epidural anesthesia simulation on short- and long-term learning curve: high- versus low-fidelity model training. *Reg Anesth Pain Med* 34(3):229–232
4. Litzelman DK, Stratos GA, Marriott DJ, Skeff KM (1998) Factorial validation of a widely disseminated educational framework for evaluating clinical teachers. *Acad Med* 73(6):688–695
5. Neher JO, Gordon KC, Meyer B, Stevens N (1992) A five-step “Microskills” model of clinical teaching. *J Am Board Fam Pract* 5(4):419–424
6. Frank JR (2005) The CanMEDS 2005 physician competency framework: better standards, better physicians, better care. The Royal College of Physicians and Surgeons of Canada, Ottawa. www.RCPSC.medical.org
7. Ringsted C, Hansen TL, Davis D et al (2006) Are some of the challenging aspects of the CanMEDS roles valid outside Canada? *Med Educ* 40:807–815
8. <https://sundhedsstyrelsen.dk/en/news/2013/~media/39D3E216BCBF4A9096B286EE44F03691.ashx>
9. Royal Australian College of Surgeons, Nine RACS competencies. www.surgeons.org/becoming-a-surgeon/surgical-education-training/competencies
10. Flin RH, O'Connor P, Crichton M (2008) Safety at the sharp end: a guide to non-technical skills. Ashgate, Aldershot
11. Fletcher GCL, McGeorge P, Flin RH, Glavin RJ, Maran NJ (2002) The role of non-technical skills in anaesthesia: a review of current literature. *Br J Anaesth* 88:418–429
12. Rudolph JW, Simon R, Raemer DB et al (2008) Debriefing as formative assessment: closing the performance gaps in medical education. *Acad Emerg Med* 15:1010–1016

13. Baker GR, Norton PG, Flintoft V et al (2004) The Canadian adverse event study: the incidence of adverse events among hospital patients in Canada. *Can Med Assoc J* 170:1678–1686
14. Mazzocco K, Petetti DB, Fong KT et al (2009) Surgical team behaviours and patient outcomes. *Am J Surg* 197:678–685
15. Neily J, Mills PD, Young-Xu Y et al (2010) Association between implementation of a medical team training program and surgical mortality. *JAMA* 304:1693–1700
16. Leighton BL (1989) A greengrocer's model of the epidural space. *Anesthesiology* 70:368–369
17. Vaughan N, Dubey VN, Wee MY, Isaacs R (2013) A review of epidural simulators: where are we today? *Med Eng Phys* 35(9):1235–1260
18. Sturm LP, Windsor JA, Cosman PH, Cregan P, Hewett PJ, Maddern GJ (2008) A systematic review of skills transfer after surgical simulation training. *Ann Surg* 248(2):166–179
19. Draycott T, Sibanda T, Owen I et al (2006) Does training in obstetric emergencies improve neonatal outcome? *Br J Obstet Gynaecol* 113:177–182
20. Issenberg SB, McGaghie WC, Petrusa ER et al (2008) Features and uses of high fidelity medical simulations that lead to effective learning: a BEME systematic review. *Med Teach* 27:10–28
21. Steinwachs B (1992) How to facilitate a debriefing. *Simul Gaming* 23:186–195
22. Young A, Miller JP, Azarow K (2005) Establishing learning curves for surgical residents using cumulative summation (CUSUM) analysis. *Curr Surg* 63(3):330–334
23. Watterson LM, Hyde S, Bajenov S, Kennedy SE (2007) The training environment of junior anaesthetic registrars learning epidural labour analgesia in Australian teaching hospitals. *Anaesth Intensive Care* 35(1):38–45
24. Ten Cate O (2005) Entrustability of professional activities and competency-based training. *Med Educ* 39:1176–1177
25. Norcini J, Burch V (2007) Workplace-based assessment as an educational tool: AMEE guide no 31. *Med Teach* 29:855–871
26. Grant J (2002) Learning needs assessments: assessing the need. *BMJ* 324:156–159
27. Ringsted C, Østergaard D, van der Vleuten CP (2003) Implementation of a formal in-training assessment programme in anaesthesiology and preliminary results of acceptability. *Acta Anaesthesiol Scand* 47:1196–1203
28. Holmboe ES, Yepes M, Williams F, Huet SJ (2004) Feedback and the mini clinical evaluation exercise. *J Gen Intern Med* 19:558–561
29. Kirkpatrick DL (1998) Evaluating training programs. Beret-Koehler, San Francisco, CA
30. Birch L, Jones N, Doyle PM et al (2007) Obstetric skills drills: evaluation of teaching methods. *Nurse Educ Today* 27:915–922
31. Schwartz AJ (2010) Resident/fellow evaluation of clinical teaching: an essential ingredient of effective teacher development and educational planning. *Anesthesiology* 113(3):516–517
32. Crosby RMHJ (2000) AMEE guide no 20: The good teacher is more than a lecturer – the twelve roles of the teacher. *Med Teach* 22(4):334–347
33. Surkin G, Wagner E, Harris I, Shiffer R (2008) What makes a good clinical teacher in medicine? A review of the literature. *Acad Med* 83:452–466
34. Holmboe ES, Fiebach NH, Galaty LA, Huot S (2001) Effectiveness of a focused educational intervention on resident evaluation from faculty a randomized controlled trial. *J Gen Intern Med* 16:427–434
35. Van der Hem-Stokroos NH, Daelmans HF, Van der Vleuten CP, Haarman HJ, Scherphier AL (2004) The impact of multi-faceted educational structuring on learning effectiveness in a surgical clerkship. *Med Educ* 38:879–886

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24.1 Introduction

On Saturday May 14, 1853, 1 month after the childbirth of Queen Victoria under chloroform, the prestigious journal *The Lancet* [1], wrote: “intense astonishment, therefore, has been excited throughout the profession by the rumour that Her Majesty during her last labour was placed under the influence of chloroform, an agent which has unquestionably caused instantaneous death in a considerable number of cases. Doubts on this subject cannot exist. . . we could not imagine that anyone had incurred the awful responsibility of advising the administration of chloroform to Her Majesty during a perfectly natural labour with a seventh child.”

The thoughts of the age are better elucidated by the story of the visit of the Prime Minister, Mr. Gladstone, to the Queen [2]. On this occasion, Gladstone congratulated the Queen on the newly born, and then asked her how she liked chloroform. The Queen replied “very well, Mr. Gladstone.” He replied “the bishops are not pleased Madam.” “Then let the bishops have the babies, Mr. Gladstone!” she answered.

Queen Victoria’s request for what we nowadays might call an “off-label intervention” may be viewed as early evidence of maternal participation and choice in medical decision making, one of the most important components of childbirth humanization.

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24.2 Humanization of Childbirth

Childbirth is considered as one of the most important events in a woman's life, and it can, in turn, affect the rest of her life, both physically and emotionally.

Humanization of childbirth is an exclusive approach to make childbirth a positive and satisfying experience for both the women, and their families as a whole [3, 4]. This strategy is used to empower women and their care providers by taking into consideration values such as the women's emotional state, their values, beliefs, and sense of dignity and autonomy during childbirth.

Humanized birth cannot perhaps be limited to a specific definition, nor can it be seen as a long list of tasks that need to be performed. It is a process, a transition for every woman, professional, person, and family: humanized care cannot be defined as a specific action or methodology. In all cases it affects mother and family and it continues after birth, and may be different, depending on the culture and place.

Humanization of childbirth includes: continuous emotional and psychological care and support during pregnancy and postpartum, the avoidance of unnecessary medical intervention, and the empowering of women by allowing them to actively participate in the decision making with regard to their own experience [3]. The literature describes the specific characteristics of a humanized birth as follows: one which promotes the active participation of women regarding decision making, and other aspects of their own care, one which takes advantage of the expertise of both physicians and non-physicians, and allows them to work together as equals, and one which involves the use of evidence-based technology and medical intervention [3–6].

During the past decades, giving birth has involved increasingly medicalized procedures in most countries [7, 8]. Pregnancy and birth were conceptualized as pathological processes that require intensive monitoring and intervention by a physician. Medical interventions in childbirth such as the use of electronic fetal monitoring, amniotomy, induced labor, episiotomy, and elective cesarean section deliveries continue to increase [9–11]. These procedures reinforce the perception of the mother's role as patient and can reduce her sense of control over her body [7, 8].

Epidural analgesia has usually been included in the list of these medical interventions, due to its frequent association with painful, long, and dystocic labors, usually requiring medical intervention. The inclusion of epidural analgesia in the list of medical interventions reinforcing the perception of the mother's role as a patient rather than a laboring women may also be due to the typical side effects of the "old fashioned" epidural, such as the occurrence of numbness of the legs, difficulty to void, absence of the feeling of uterine contraction, the absence of the feeling of the urge to push, nausea and pruritus, difficulties in ambulation. These features occurred quite often in the past and were due to the common use of relatively high concentrations of local anesthetic solutions given by continuous epidural administration. With the introduction in clinical practice of the ultralow local anesthetic solution and of the programmed intermittent analgesic techniques, almost all the typical side effects of labor epidural observed in the past are now no more an issue, since a painless but "spontaneous" vaginal delivery is now really possible.

Table 24.1 Barriers and facilitators in humanized childbirth

| | |
|--------------------------|--------------------------------------------------------------------------------|
| <i>A. Barriers</i> | |
| 1. Rules and regulations | |
| | Prevention from having a companion during labor, postpartum, in operation room |
| | Banning of children from the mother's room |
| 2. Physical structure | |
| | Common labor and delivery room |
| | Common postpartum room |
| 3. Contingence factors | |
| | University-affiliated hospitals |
| | Lack of midwifery autonomy |
| | Malpractice litigation |
| | Physician's training and skills |
| | Overcharge of work |
| <i>B. Facilitators</i> | |
| 1. Rules and regulations | |
| | Preventing unnecessary medical intervention |
| | Getting the women's consent |
| 2. Physical structure | |
| | Labor and delivery room and other facilities |
| | Contingence factors |
| | Midwifery system |

For this reason epidural analgesia, if performed with the new techniques and new drugs, should no longer be included in the list of barriers to childbirth humanization, but in the facilitators.

In addition, from the professionals' point of view, humanized birth should not be perceived as a restriction in using medical intervention, but as something that involves all aspects of care that provide a good physical and psychological status for the patient. Humanized birth can be married to medical intervention just by explanation, by communication, and by maintaining confidence.

In Table 24.1 [12] some examples of the barriers and facilitators faced by the humanized birth practice are reported: all the care provided around the time of birth that promote the physical and psychological health of women and respect their desires and needs can be defined as humanized care.

Humanized childbirth emphasizes the need for access to a continuous pool of emotional and physical support during the pregnancy, labor, and postpartum stages. The importance of a companion is prominent and the presence of family members during childbirth experiences has been recommended by the WHO as one of the main aspects of humanized care [13]. The benefits of continuous one-on-one support by a companion during labor have also been noted by a Cochrane systematic review [14] and other research reviews in the past [15].

24.3 Pain, Suffering, and Epidural Analgesia

After the health of mother and baby, labor pain is the greatest concern of women, their partners, and their caregivers. Nurses and doctors promise little or no pain when their medications are used, and they feel frustrated and disappointed if a woman has pain. Most are also extremely uncomfortable with her expressions of pain during labor such as moans, crying, tension, and frustration because they don't know how to help her, except to give her medication.

When staff believes that labor pain equals suffering, they communicate that belief to the woman and her partner, and, instead of offering pain relief and support and guidance for comfort, they offer pain medication only.

However, there is a distinction between pain and suffering.

Pain has been defined as “an unpleasant sensory or emotional experience associated with actual or potential tissue damage” or described in terms of such damage [16]. The emphasis is on the physical origins of pain. Suffering describes negative emotional reactions [17] and includes any of the following factors: perceived threat to body and/or psyche, helplessness and loss of control, distress, inability to cope with the distressing situation, and fear of death of mother or baby. Therefore, one can have pain without suffering and suffering without pain. There are many occasions in life when we have been in pain but did not fear damage or death to ourselves or others. This is because the person has enough knowledge, attention to other matters or goals, companionship, reassurance, touch, self-help measures, feelings of safety, and other positive factors to keep him/her from interpreting the painful experience as suffering.

On the other hand, we can suffer without pain. Acute worry or anguish about oneself or a loved one, death of a loved one, cruel or insensitive treatment, deep shame, extreme fear, loneliness, depression, and other negative emotions do not necessarily include real or potential physical damage but certainly cause suffering. Therefore, all suffering is not caused by pain.

Unfortunately, very often the goal of anesthesiology has been to remove pain, on the assumption that when there is little or no pain, there will be no suffering.

Caregivers who want to practice humanized childbirth must recognize that if a parturient has an epidural, she still needs emotional support and assistance with measures to enhance labor progress and effective pushing. The absence of pain, usually accomplished so effectively by epidural analgesia, does not mean absence of suffering. With the assumption that pain and suffering are the same, after the pain is eliminated, the woman's emotional needs are often neglected even if it has been reported that the balance of coping and distress-related thought for women with epidurals was virtually identical to that of women with no analgesia [18].

Even without pain women may be distressed [17] by many things, including the length of labor, side effects such as itching and nausea, being left alone by supporters when the woman was “comfortable,” helplessness, passivity, worries over the baby's well-being (e.g., with the sudden and dramatic reactions of staff on the occurrence of fetal heart rate changes), or feeling incompetent, for example,

when unable or afraid to push effectively despite clear instructions to push long and hard.

The point is that women may suffer even if they have no pain, and their need for continuing companionship, reassurance, kind treatment, assistance, and attention to their discomforts and their emotional state remains as important to the satisfaction and positive long-term memory of the woman independently of the presence of epidural analgesia [19].

The lack of attention to a woman's emotional needs when laboring with an epidural is fixed in the erroneous and widespread assumption that pain equals distress and that abolishing pain means a stress-free and positive birth experience is ensured [20]. Furthermore, there is little systematic documentation of women's emotional responses when laboring with an epidural.

For some women who had desired a natural childbirth, the decision to have an epidural is accompanied by disappointment in themselves or disillusionment over being prepared for the degree of pain. Prenatal classes and appointments with the caregiver should provide correct information and advice for flexibility, if needed. Most women today, however, plan in advance to use an epidural, but often the staff and anesthesiologist have control over when they may have it. If told it is too early in labor for an epidural, or that the anesthesiologist is not immediately available, the woman may be upset.

The time from when the decision is made until the woman has adequate pain relief may be stressful, because the woman does not want to cope any longer, yet she has no choice. The wait can seem quite long if the anesthesiologist is needed elsewhere, or if the woman must undergo various procedures, such as admission, obstetric examination, fetal monitoring, and administration of IV fluids, before the epidural is given. The wait is short if the anesthesiologist is readily available and all pre-anesthesia procedures are completed. The partner may feel ineffective and frustrated, because the parturient does not want to continue using his comforting techniques. The partner is further troubled by the distress of standing by watching the woman he loves becoming progressively more distressed [21].

Antenatal childbirth classes or any other informal talk during the onset of labor may be the place and the time to inform the couple about the difficulty of this period and help them plan to continue with their coping techniques until the epidural takes effect.

Administering the epidural anyway takes some minutes, depending on the skill and experience of the anesthesiologist, the technical difficulties encountered during the procedure, and the woman's cooperation. The woman must lie or sit and sometimes the position may be uncomfortable, especially during contractions. She needs help to remain still and calm, acknowledgment that it is a difficult position, and congratulation on how well she is doing. Not surprisingly, if the epidural procedure takes several attempts or takes a long time, the woman and her partner become frustrated or frightened.

Within minutes after the epidural is placed, the contraction pain begins to subside and usually disappears within 15 min. The woman's mood improves markedly, and she is delighted that her pain has gone. She often becomes talkative,

optimistic, and very grateful to the anesthesiologist. Of course, if pain relief is incomplete, she is disappointed and becomes impatient for adjustments to correct the problem. Her partner matches her relief, very grateful that she is comfortable and acting more like the woman he knows. He now shifts his focus to his own needs for a break, food, or sleep. Once the woman is comfortable, she no longer needs the intense support and close physical contact she needed before the epidural. Nevertheless, she may feel suddenly unimportant when her partner turns on the TV, goes to get a meal or takes a break. The dynamic in the room is almost as if she were not in labor. It may surprise and disappoint her that her care team becomes less attentive and less concerned about her emotional well-being. Although the nature of the partner's support changes as long as the woman is awake and alert, he/she should converse with the woman and nurse, answer questions, and help her with grooming and comfort, to ensure that the woman does not feel alone or less important. It is not unusual for a situation requiring quick action to arise during an otherwise calm period created by the epidural; or the doctor comes in to make a clinical decision (e.g., to start oxytocin or rupture the membranes); or the midwife decides to catheterize the woman or to have her change position.

Epidural analgesia may also affect the companion's attitudes. In one study [22] fathers whose partners did not receive epidural analgesia felt their presence as troublesome and unnecessary while the presence of maternal epidural analgesia increased paternal feelings of helpfulness threefold and was associated with a greater involvement and less anxiety and stress.

With modern epidural analgesia the awareness of uterine contractions is not usually lost, but this may not be the case should a more profound analgesia be needed and/or provided, contractions can be no more perceived. In this situation the partner or the midwife can encourage the women to feel her abdomen for contractions and fetal movements and teach her about bearing-down efforts in the second stage.

After the epidural takes effect, the woman, depending on the local clinical practice, may stay in bed and wait until it is time to push, or, alternatively, may be allowed to ambulate, see relatives, drink some fluids, and have an active relationship with the partner. Nevertheless, she may feel bored or passive and may begin to worry that the labor is taking a long time, or about how the baby is tolerating the labor. The waiting is sometimes very challenging. The woman needs distraction, conversation, and reassurance that the staff is monitoring the labor and the baby.

Even though her pain is under good control, other discomforts may bother the woman, especially if she is unprepared for them. If the anesthesiologist has used a relatively high concentration of local anesthetic or a continuous infusion, she may have a heavy, numb feeling in her legs and the disappearance of the painless feeling of the uterine contractions.

Other common sensations during labor may be feeling too warm or too cold, trembling, having a window of pain in the area covered by the epidural, the so-called breakthrough pain, itching, if opioids have been used, nausea, or an uncomfortable position.

A sense of accomplishment and optimism arises when the woman reaches complete dilation: a main goal been achieved. In some hospitals at this stage, the epidural is turned down to give the woman painful sensations to guide her bearing-down efforts. This very questionable option is often intolerable for the woman, and she begs for a return of the epidural. She may feel weak or selfish for not being able to handle the pain, which is actually greater than it might have been had she not had the epidural.

In some hospitals the choice is the directed pushing, as soon as dilation is complete. In this case, if the epidural analgesia is too intense, women may feel unable to push effectively, and may find it difficult to follow directions, because they get no feedback from their efforts. They need reassurance that they will push more effectively as they continue. Sometimes, rather than being reassured, they are constantly told to push harder or longer. They feel criticized and inadequate. It seems to take a very long time and exhaustion and discouragement may arise. They should be praised for their efforts and reminded that it takes time for the baby's head to mold or rotate. Resting through a contraction or two also gives a welcome break.

A third option is to delay pushing until the baby is visible at the vaginal outlet or she feels the urge to push. This is the easiest on the mother and baby, and results in fewer instrumental deliveries, but it may lengthen the duration of the second stage [23].

When it is time to push, the partner can provide very important feedback to the woman, and give her incentive, for example by relaying the intensity readings from the contraction monitor to her as she bears down. Feedback like this shows the woman how well she is doing, and gives her a real sense of accomplishment that she might otherwise not have. As the baby's head presses on the rectum, reaches the perineum, and distends the vaginal opening, the woman may feel it pleasantly if the epidural analgesia is adequate; but on the contrary this may come as an unwelcome shock after having been comfortable if the epidural analgesia has been withheld or is inadequate at this stage.

Respecting women's autonomy means to respect her ability to choose a desired birthing position and/or to have a freestyle labor and delivery. Modern epidural analgesia may provide adequate pain relief, maintaining the painless sensation of bearing down and allowing the mother to assume any birthing position thanks to the absence of motor block and therefore is perfectly compatible with any parturient's decision. The possibility of choosing a freestyle position, especially during the second stage, may improve women's confidence so improving the sense of control and the feeling of having played a truly active role in the birth of their child [24].

24.4 Conclusion

Nowadays, almost all the typical side effects of labor epidural witnessed in the past are now no longer observed, and, very frequently, a painless but "spontaneous" vaginal delivery is now really possible.

Epidural analgesia, if performed with the new techniques and new drugs, should no longer be included in the list of barriers to childbirth humanization, but may be included in the facilitators. Anesthesiologists who want to practice humanized childbirth must be aware that if the parturient has an epidural, she still needs emotional support and assistance, since the absence of pain, usually obtained so effectively by epidural analgesia, does not mean absence of suffering.

References

1. Anonymous (1953) *Lancet* 61:453. Saturday, 14 May 1853, London
2. Kranke P, Lavand'homme P (2012) The relief of pain in labour and the role of remifentanyl. *Eur J Anaesthesiol* 29:116–120
3. Wagner M (2011) Fish can't see water: the need to humanize birth. *Int J Gynecol Obstet* 75: S25–S37
4. Misago C, Umenai T, Onuki D et al (1999) Humanised maternity care. *Lancet* 354:1391–1392
5. Page L (2000) Human resources for maternity care: the present system in Brazil, Japan, North America, Western, Europe and New Zealand. *Int J Gynecol Obstet* 75:S81–S88
6. Mitchell LM (2001) *Baby's first picture: ultrasound and the politics of fetal subjects*. University of Toronto Press, Toronto
7. Davis-Floyd R (1994) Culture and birth: the technocratic imperative. *Birth Gaz* 11:24–25
8. Hausman BL (2005) Risky business: framing childbirth in hospital settings. *J Med Humanit* 26:23–38
9. Bosch X (1998) Spanish doctors criticized for high tech births. *BMJ* 317:1460
10. Tew M (1988) *Safer childbirth? A critical history of maternity care*. Free Association Books, London, p 314
11. Leeman L, Fontaine P, King V et al (2003) The nature and management of labor pain: part II. Pharmacologic pain relief. *Am Fam Physician* 68:1115–1120
12. Behruzi R, Hatem M, Fraser W et al (2010) Facilitators and barriers in the humanization of childbirth practice in Japan. *BMC Pregnancy Childbirth* 10:25–42
13. World Health Organization (1996) *World Health Organization appropriate technology to birth and delivery*. Seminar on birth and delivery in the State of Sao Paulo. Reestablishing quality of care in birth and delivery
14. Hodnett ED, Gates S, Hoffmayr G et al (2007) Continuous support for women during childbirth. *Cochrane Database Syst Rev* 10:CD003766
15. Leslie MS, Storton S (2007) Step 1: Offers all birthing mothers unrestricted access to birth companions, labor support, professional midwifery care: the coalition for improving maternity services. *J Perinat Educ* 16:10S–19S
16. Merskey H (1976) Pain terms: a list with definitions and notes on usage. Recommended by the International Association for the Study of Pain (IASP) subcommittee on taxonomy. *Pain* 3:249–252
17. Lowe NK (2002) The nature of labor pain. *Am J Obstet Gynecol* 186:S16–S24
18. Wuitchik M, Bakal D, Lipshitz J (1990) Relationships between pain, cognitive activity and epidural analgesia during labor. *Pain* 41:125–132
19. Lally JE, Murtagh MJ, Macphail S et al (2008) More in hope than expectation: a systematic review of women's expectations and experience of pain relief in labour. *BMC Med* 6:7. doi:10.1186/17417015-6-7
20. Hodnett E (2002) Pain and women's satisfaction with the experience of childbirth: a systematic review. *Am J Obstet Gynecol* 186:5160–5172
21. Chapman L (2000) Expectant fathers and labor epidurals. *MCN* 25:133–138
22. Capogna G, Camorcia M, Stirparo S (2007) Expectant fathers' experience during labor with or without epidural analgesia. *Int J Obstet Anesth* 16:110–115

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23. Fraser W, Marcoux S, Krauss I (2000) Multi-center, randomized, controlled trial of delayed pushing for nulliparous women in the second stage of labor with continuous epidural analgesia. *Am J Obstet Gynecol* 182:1165–1172
 24. Santos OM, Siebert ER (2001) The humanization of birth experience at the University of Santa Catarina maternity hospital. *Int J Gynaecol Obstet* 75:S73–S79

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25.1 Introduction

Within surgical disciplines, obstetrics and anesthesia carry a greater risk of medicolegal liability although with improved monitoring and practice standards issued by national scientific societies such as the American College of Obstetricians and Gynecologists (ACOG) and the American Society of Anesthesiologists (ASA). Pain relief is becoming an area of concern. Analgesia refers to the relief of pain without the loss of consciousness. Modalities of analgesia during childbirth include regional analgesia, systemic opioid analgesia, continuous labor support, pudendal blocks, immersion in water during the first stage of labor, sterile water injections in the lumbosacral spine, hypnosis, and acupuncture [1]. Even though there are multiple options for labor pain management, women often experience pain during childbirth in accordance with their expectations. The ASA Closed Claims database [2] has shown that since the 1990s the proportion of claims associated with general anesthesia has progressively declined while the proportion associated with regional analgesia has steadily increased although the majority of maternal injuries are minor (e.g., headache, back pain, pain during analgesia, neuropsychological consequences). In addition, [3] it reported that postnatal depression may be more common when analgesia is not used and pain during labor has been correlated with the development of posttraumatic stress disorder. Furthermore, men are also affected by severe labor pain. A survey of first-time fathers showed that the men whose partners received an epidural felt three times as helpful and involved during

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labor and delivery and had less anxiety and stress, as compared with men whose partners did not receive an epidural [3]. All these situations may reflect unrealistic expectations and dissatisfaction with parturient care, and litigation serves the purpose not only of reparation of injury and deterrence of standard of care but also of emotional vindication. This is especially true in Italy where labor analgesia is becoming an important public health issue with its introduction free of charge in SSN as LEA (Essential Levels of Assistance) (DPCM April 23, 2008, art. 37) but, at the same time, it is not performed in all hospitals [4] due to lack of funding. The problem is clear. Labor analgesia has to be considered an important aspect of the obstetric anesthesia practice, and the provision of adequate pain relief and management has foundation in the law of negligence. The law of medical negligence emphasizes taking reasonable care in all aspects of patient management and, with respect to this particular issue, doctors (the anesthetist as well as the obstetrician, midwife, neonatologist, and labor and delivery nurses) may breach their standard of care by failing to exchange information with the parturient and also with other members of the woman's family as well as with the obstetric team; by failing to provide appropriate prenatal education and to acquire informed consent; by treating the pain inadequately or by failing to counteract adverse reaction and eventually perform acute resuscitation.

For all these reasons, in this chapter we intend to look at the above-mentioned medicolegal issues by firstly exploring the meaning of the "informed" consent and, secondarily, providing an overview of the concept of "standard of care" with particular regard to its relationship with the clinical practice guidelines.

25.2 The Informed Consent

25.2.1 Ethical Meaning and Legal Requirements of Consent

With respect to other specialties, obstetric anesthesia presents unique challenges to the process of informed consent because it involves several additional elements to the basic elements of consent. This is primarily due to the particular suffering being experienced by the parturient characterized by stress and discomfort, and often to the lack of time available for the consideration of the risks and complications consequent to the anesthesia. This circumstance has led some to question the capacity of the parturient in labor to really give consent to the anesthesia. Despite the pain and the fatigue, many surveys have shown that parturients are capable of giving consent [5, 6], if the particular circumstance does not affect the ability to provide informed consent. Other surveys [7] show the same results even after the administration of opioid analgesia.

To be a valid requirement and to have a judicial validity, the consent must be correctly formulated and validly expressed. This implies correct information about procedure, risks, benefits, and alternatives, providing the parturient with useful indications for orienting herself to self-determine, making also a responsible choice.

25.2.2 The Information Requested by Patients Before Anesthesia

It should be clear and it has been demonstrated [8] that parturients want to know about risks. Informational elements concern clearness in explaining information, that is the information about the risks of the anaesthesia, above all when the consent is requested for emergency cases, such as an urgent caesarean section.

With respect to the amount of information, as noted by Krzysztof and Kuczkowski [9], several rules have been proposed. The “one percent rule” states that the anaesthesiologist should inform the patient of complications that have an incident greater than 1 %. In situations, where there is a serious risk of death and because death represents the worst risk, the anesthesiologist may adopt the “death rule” informing the parturient of the extreme risk of dying after anesthesia. However, the anesthesiologist can choose to inform the parturient of the possible risks that a “reasonable” patient (reasonable rule) would want to be informed about. The “all-risk rule” recommends informing the parturient of all the possible risks resulting from anesthesia independently from their incidence. Finally, information about the risks of anesthesia can be delivered pursuant to the “good judgement rule” according to which the anesthesiologist should inform the parturient about the risks without frightening her.

With regard to the disclosure itself, as noted by Hoehner [10] the parturient should be advised about the procedures, the benefits, the potential risks, the complications, and alternative options, when possible, without giving excessive information during labor because this could generate more confusion and anxiety.

25.2.3 How to Obtain Consent for Obstetric Anesthesia

When possible all the information regarding labor should be given before the procedure takes place, during the antenatal period, discussing techniques, risks, complications, and the benefits of anesthesia and analgesia. This can be easier for a planned epidural anesthesia if chosen by the parturient. There are, however, circumstances, like emergencies, that require the abbreviation of consent process: when it is not possible to acquire the patient’s consent.

Understanding the contradictions and risks is fundamental to obtaining a valid consent. In this regard it is important to highlight that a Cochrane review of 20 trials involving a total of 6,534 women estimated that the relative risk of cesarean delivery with epidural analgesia during labor as compared to other methods or with no analgesia was 1.07 (95 % confidence interval, 0.93–1.23). Epidural analgesia does increase the duration of the second stage of labor by 15–30 min and may increase the rate of instrument-assisted vaginal deliveries as well as that of oxytocin administration. Equally, three randomized, controlled trials showed that early initiation of epidural analgesia (cervical dilatation, <4 cm) does not increase the rate of cesarean delivery among women with spontaneous or induced labor, as compared to early initiation of analgesia with parenteral opioids.

Another aspect is the higher risk of an increase vacuum- or forceps-assisted vaginal delivery with regional analgesia in laboring patients (relative risk [RR] = 1.42; 95 % confidence interval [CI], 1.28–1.57; 23 trials; $n = 7,935$) [11]: a meta-analysis showed that there is no reduction statistically by discontinuing the epidural analgesia late in the second stage of labor [12]. Conversely, discontinuing epidural analgesia late in the second stage resulted in inadequate pain relief for 22 % of study participants versus 6 % of participants reporting inadequate pain relief when the epidural was continued throughout the second stage of labor ($P < 0.05$) [12].

Therefore, the use of a combination of epidural and spinal analgesia reduces the need for instrumental vaginal deliveries (absolute risk reduction = 8.6 %; number needed to treat = 12), but it may increase the likelihood of a newborn needing resuscitation (absolute risk increase = 1.6 %; number needed to harm = 63) [13].

Finally, we retain its importance to stress that from the ethical and medicolegal point of view analgesia options should be explored early in the prenatal period. Encouraging patients' participation in pain management may help reduce pain and increase their satisfaction in the childbirth experience.

25.2.4 Pros and Cons to Written Consent in Labor Analgesia

As suggested in 2006 by the Association of Anaesthetists of Great Britain and Ireland, [14] in many instances, verbal consent is sufficient. However, surveys conducted by Gerancher [7] and White et al. [15] show that the written consent may reinforce what has been said verbally. This improves the knowledge about benefits, risks, and the complication of a labor anesthesia, so it is generally perceived as helpful for the parturient and her partner. This increased comfort is also due to the fact that parturients retain a copy of the consent they gave, so that they can read it again. For this reason, the consent form should be concise and clear.

A written consent is also helpful in the case when the patient decision goes against anesthetists' advice (e.g., refusal to undergo anesthesia as a result of religious obligations in an emergency situation).

25.3 The Concept of "Standard of Care"

The concept of standard of care is often discussed among physicians, and the legal definition of this term is frequently not well understood sometime by those doctors who are required by the judge, lawyer, or patient to give a technical opinion in cases of medical litigation. Its relevance derives from the fact that to establish medical negligence and successful litigation requires that three key criteria should be satisfied. The claimant must first be owed a duty of care, established whenever a patient undergoes treatment, and rarely contested. Secondly, a breach of that duty of care by a failure to provide the required standard medical care must be established. Thirdly, as a direct result of this breach of duty (causality), the claimant must have suffered physical or psychological harm, or other tangible losses.

Then, in medical litigation, a key step for the claimant is to prove that physicians failed to meet the required “standard of care.” In Italy, due to the lack of a specific law code for the physician–patient relationship, the meaning of this important concept derives mainly from the Court of Cassation (Corte di Cassazione) jurisprudence. This Court represents the last resort for both the civil and the criminal jurisdiction and has the power to correct a lower instance court’s interpretation or application of the law. Traditionally, with regard to article n. 1176 of the Italian civil code, the Court of Cassation states that “standard of care” means that any physician has the legal duty to adhere to what any other physician with the same professional preparation and training as well as scrupulous attention would have reasonably done in the similar clinical and organizational conditions. The discrepancy between the two conducts determines a negligent and/or imprudent and/or unskillful behavior with consequences for the physician in both the civil and the criminal jurisdictions.¹

Usually, the indication of what can be considered standard in a given clinical situation is set by the medical doctor required to give a technical opinion as the consultant in the court. In other words the consultant should give a balanced view of what is currently the best practice and what is perhaps appropriate for the level of skill and training of the doctor involved in the litigation also taking into consideration the concrete availability of all instruments and the organization of the place where the medical activity was performed. These physicians should know which analgesia options are available at the delivering institution, the patient’s desire for regional analgesia, the availability of continuous labor support, and the potential for complications related to specific interventions. This is because it is common for the expert’s opinion about what he would have done in the same circumstances to be directly translated into a blame or culpability judgment although this judgment should be determined by the courts. The expert opinion can make a difference in the decision as to whether a given fact was due to mishap or negligence, but it must be noted that to reflect on a particular event is very different from being analytical during the course of such an event! Expert opinion is normally supported by published articles and by personal experience.

Consequently, the strength of evidence to support an expert opinion on standard of care is fundamental. A systematic review, for example, would have a higher weighting than a case report. Few randomized controlled trials of the effects of analgesia administered during labor are performed maybe due to the fact that it would be considered unethical to randomly assign women to a placebo (no pain relief) if epidural analgesia is available. Most trials have compared the use of epidural analgesia with that of systemic narcotics [3]. There is strong evidence showing the contraindication of analgesia during labor and delivery in cases of coagulopathy (including ongoing thrombocytophlyaxis with low-molecular-weight

¹ In the Italian criminal code, the negligent injury is described as “an event that, even if it happened against the intention, occurred due to negligence, imprudence, unskillfulness or failure to comply with laws, regulations, orders and disciplines” (art. 43, Italian Criminal Code).

or unfractionated heparins) or uncorrected maternal hypovolemia. There is evidence that contraindicates labor analgesia if there is an infection at the needle-puncture site or if there are clinical signs of increased intracranial pressure because neuraxial techniques could lead to herniation if dural puncture occurred. These conditions, as well as making arrangements for emergency equipment to be immediately available to treat serious untoward reactions (hypotension, respiratory compromise, and in rare cases, seizures and cardiac arrest) at the time of placement of the nerve block, represent examples of “standard of care” for all the members of the obstetric care team (anesthetist, obstetrician, midwife) which, of course, must have adequate training or experience and must take care to communicate and exchange information in the ever changing environment of labor and delivery.

A more objective measure to determine the legal “standard of care” is represented by the identification of clinical practice guidelines (CPG) although a doctor cannot be considered responsible simply because he doesn’t follow them. CPG are in fact not an absolute requirement. They cannot guarantee any specific outcome and, in addition, require a period of time for dissemination and integration into clinical practice (so-called learning curve). In other words, they are not a “cookbook” and discretion lies at the heart of clinical judgment that needs to take into account a number of individual circumstances. Furthermore, as has already been mentioned, establishing medical responsibility also requires the satisfaction that due to the breach of a given duty of care (causality) the claimant has suffered physical or psychological harm or other tangible losses. Nevertheless, despite all these arguments that support the fact that the CPG should not constitute a de facto legal standard that is applied in all cases, in Italy future medicolegal cases may rely increasingly on clinical guidelines to determine benchmarks for acceptable clinical practice.

25.4 Clinical Practice Guidelines and the Standard of Care

A brief discussion on the use of CPG as defining the standard of care is warranted due to their recent introduction into the Italian legal system by the law 189/2012.² Literally, article 3 named “Responsibility of any health professional” states that *“Any health care professional that adheres to CPG and best practices accredited by the science community is not liable for criminal negligence. In such cases, however, it remains subject to the obligation referred to in article 2043 of the Civil Code. The judge, also in the determination of damages, shall take due account of the conduct referred to in the first period.”* At present, only two cases have been under scrutiny by the criminal court of Cassazione (Cassazione IV sezione 24 gennaio 2013 n. 11493 and Cassazione 29 gennaio 2013 n. 16237), and from both of them the role of CPGs in defining “negligence” remains not completely clear for at least

² Legge di conversione 8 novembre 2012, n. 189 e pubblicato in Gazzetta Ufficiale 10 novembre 2012, n. 263.

three key points. May all guidelines influence clinical practice and support the establishment of legal standards? What does best practice mean and how has it to integrate guidelines? Who is required to distinguish between slight or gross fault (“colpa grave o lieve”) considering that no legal definition is yet available? Extensive reviews of this issue are available in the national medicolegal and legal literature [16]. We emphasize only some aspects. Undoubtedly, clinical guidelines are statements that have been systematically developed and which aim to assist clinicians in making decisions about treatment for specific conditions. They are meant to facilitate good medical practice, and their development is a structured process by the techniques of evidence-based medicine. Among the several limitations of clinical guidelines we want to focus on the fact that they are elaborated by various authoritative bodies and that the scientific community does not usually distinguish between national or international clinical guidelines (e.g., NICE, Clearinghouse, etc.) [17, 18]. The above-mentioned law 189/2012 also requires that the conduct of any health professional has also to adhere to the “best practice” that includes the guidelines as well as all the scientific information studied at university and evidence of the literature. It is clear then that the intention of this law was to anchor the evaluation of the “standard of care” to something objective that is in conformity with customary practice endorsed by the responsible body of medical opinion. However, it is realistic that in medical litigation it should be possible for some guidelines to have greater status than others only on the basis of the expert opinion required by the court. Thus, in order to contribute to the provision of a framework for structured judicial decision-making physicians called to respond for malpractice as clinicians and consultants in the legal context have to be more prepared to justify the logical basis of their conduct/opinion in a specific case by referring to the various clinical CPGs and documenting the reason for deviating from them.

References

1. Schrock SD, Harraway-Smith C (2012) Labor analgesia. *Am Fam Physician* 85(5):447–454
2. Chadwick HS (2005) Obstetrics anesthesia. *Minerva Anestesiologica* 71:483–486
3. Hawkins JL (2010) Epidural analgesia for labor and delivery. *N Engl J Med* 362:1503–1510
4. AROI Guideline. http://www.aaroiemac.it/site/Allegati/documenti/doc_utili/doc_utili_2008/tra_taglio_cesareo_e_partoanalgesia.pdf. Accessed 28 Sept 2014
5. Pattee C, Ballantyne M, Milne B (1997) Epidural analgesia for labor and delivery: informed consent issues. *Can J Anaesth* 44:918–923
6. Knapp RM (1990) Legal view of informed consent for anesthesia during labor. *Anesthesiology* 72:211
7. Gerancher JC, Grice SC, Dewan MD, Eisenach J (2000) An evaluation of informed consent prior to epidural analgesia for labor and delivery. *Int J Obstet Anesth* 9:168–173
8. Jackson GN, Sensky T, Reide P, Yentis SM (2011) The capacity to consent to epidural analgesia in labour. *Int J Obstet Anesth* 20(3):269–270
9. Krzysztof M, Kuczkowski MD (2003) Informed consent, the parturient, and obstetric anaesthesia (Editorial). *J Clin Anesth* 15:573–574

10. Hoehner PJ (2003) Ethical aspects of informed consent in obstetric anaesthesia – new challenges and solutions. *J Clin Anaesth* 15:587–600
11. Anim-Somuah M, Smyth RM, Jones L (2011) Epidural versus non-epidural or no analgesia in labour. *Cochrane Database Syst Rev* (12):CD000331
12. Torvaldsen S, Roberts CL, Bell JC, Raynes-Greenow CH (2004) Discontinuation of epidural analgesia late in labour for reducing the adverse delivery outcomes associated with epidural analgesia. *Cochrane Database Syst Rev* (4):CD004457
13. Comparative Obstetric Mobile Epidural Trial (COMET) Study Group UK (2001) Effect of low-dose mobile versus traditional epidural techniques on mode of delivery: a randomized controlled trial. *Lancet* 358(9275):19–23
14. Association of Anaesthetists of Great Britain and Ireland (2006) Consent for anaesthesia. <http://www.aagbi.org/publications/guidelines/docs/consent06.pdf>. Accessed 28 Sept, 2014
15. White LA, Gorton P, Wee MYK, Mandal N (2003) Written information about epidural analgesia for women in labour: did it improve knowledge? *Int J Obstet Anaesth* 12:93–97
16. Fiori A, Marchetti D (2013) L'articolo 3 della Legge "Balduzzi" n. 189/2012 ed i vecchi e nuovi problemi della medicina legale (The third article of the law 189/2012. Old and new questions for the legal Medicine). *Riv It Med Leg* 2:563–573
17. Samanta A, Samanta J, Gunn M (2003) Legal consideration of clinical guidelines: will NICE make a difference? *J R Soc Med* 96:133–138
18. Fearnley RA, Bell MDD, Bodenham AR (2012) Status of national guidelines in dictating individual clinical practice and defining negligence. *BJA* 108(4):557–561