Atlas of Colonoscopy

Techniques • Diagnosis • Interventional Procedures

Helmut Messmann

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Atlas of Colonoscopy

Techniques · Diagnosis · Interventional Procedures

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Preface

Flexible colonoscopy is now nearly fifty years old. In 1957, the first attempts at constructing a flexible colonoscope were made in Japan by Matsunaga and Hirosaki.

Now, almost half a century later, colonoscopy has become a vital part of gastroenterology. Advancements in recent years—especially in chip technology—have led to previously unseen standards in image quality, which continues to gain in importance, especially in combination with new staining techniques. In addition to the enormous significance of diagnostic colonoscopy, interventional colonoscopy also plays a major role in gastroenterological endoscopy. New techniques have enabled the removal of increasingly larger polyps by means of mucosectomy, without the need for surgical intervention. Measures for achieving hemostasis, managing anastomotic leakages, and placing decompression tubes are also part of a more conservative approach using minimally invasive endoscopy, and increasingly avoiding surgical intervention.

The endoscopy team at the Augsburg Clinic in Augsburg, Germany performs more than 13 000 endoscopies per year, including a large number of interventions, providing us with a wealth of experience to draw on and the source of inspiration for writing this book. Additionally, we used only the latest equipment in creating this book—including zoom endoscopy—in order to produce pictures of superior image quality.

This book is aimed at health-care professionals who are interested in learning more about colonoscopy. However, it also of interest for the experienced gastroenterologist who is already familiar with colonoscopy, providing useful tips and tricks organized by experienced physicians in an informative and instructive manner. It is my hope that we can provide our readers with a good atlas, filled with numerous interesting findings and pictures, to support learning and further education in the area of colonoscopy.

We hope you enjoy reading this volume and look forward to receiving any comments or suggestions that may assist us in continuing to offer our colleagues a top-quality book.

Helmut Messmann

The Augsburg endoscopy team (from left to right):
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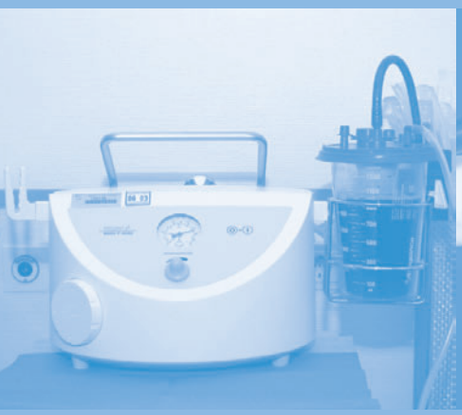



I General Information




1 General Information Regarding Examination

G. Jechart

Introduction

In the thirty years since 1971 when total colonoscopy was first described (16), significant technical advancements have been made in terms of instrument handling and imaging capability. Nevertheless, colonoscopy remains a procedure requiring manual dexterity and concentration. The experienced examiner can now successfully reach the cecum in 98% of patients and in most cases can also reach the terminal ileum. Difficulties can be posed by a mobile and elongated sigmoid colon or transverse colon as well as by postoperative intestinal fixations and other adhesions. The entire examination generally takes around 30 minutes. Rapid advancement and inspection up to the cecum is desirable, considering the discomfort to the patient, though a careful examination of all colon segments when withdrawing the instrument is essential for a thorough examination.

Proper training and experience are necessary for correct diagnosis. The diagnostic spectrum of colonoscopy encompasses not only macroscopic assessment of the condition of the mucosa, but also the possibility of collecting a targeted biopsy sample and, more recently, the use of dye spraying techniques and magnification (see Chapter 3). The instrument channel of the flexible endoscope allows for therapeutic treatment during the examination to an extent not possible with any other imaging technique. Polyps, for example, can be removed at first diagnosis and bleeding can be stopped immediately.

Thus, colonoscopy is a technically demanding examination procedure with a high clinical yield combined with the capability of therapeutic intervention.

Indications and Contraindications

Indications. An assessment of the condition of the colonic mucosa is important where there are clinical indications of colitis, i. e., abdominal pain, diarrhea, malabsorption, perianal bleeding as a result of possible intestinal ischemia, inflammation, erosions and ulcers of various geneses, polyps and tumors, diverticula, or vascular malformations. Changes in bowel habits and an increasing tendency toward constipation are cause for performing an endoscopic search for a stricture in the intestinal lumen, e. g., due to neoplasia, diverticular myochosis (thickening of the circular muscle layer), or postinflammation stricture (Tab. 1.**1**).

Thickening of the intestinal wall can be viewed using imaging techniques such as sonography (Fig. 1.**1**), computed tomography, and magnetic resonance imaging. A resulting pathological finding is an indication for colonoscopy that often can provide greater accuracy and allows taking a biopsy.

Early detection and cancer prevention. Colonoscopy is becoming increasingly important for early detection and the prevention of colorectal carcinoma in the asymptomatic general population. According to the guidelines established by the German Federal Committee of Doctors and Health Insurers (*Bundesausschuss der Ärzte und Krankenkassen*) on 5 October 2002 and based on recommendations from the German Society of Digestive and Metabolic Diseases (*Deutsche Gesellschaft für Verdauungs- und Stoffwechselkrankheiten, DGVS*), colonoscopy should be performed as a part of cancer prevention every 10 years among those aged 55 and over in the general population (14). Given the polyp–carcinoma relationship according to Vogelstein and the results of large cohort studies in the USA and Europe, there is no doubt about the effectiveness of endoscopic polyp removal in carcinoma prevention (17). With regard to these indications as well, total colonoscopy has proved itself over sigmoidoscopy and Hemoccult testing (11) (Tab. 1.**2**). Considering the current capacity for colonoscopy it would take 10 years to screen the US population following these guidelines (15).

Table 1.**1** Indications for colonoscopy

- Constipation
- Diarrhea
- Abdominal pain
- Bleeding per rectum, unexplained anemia, weight loss
- Postpolypectomy surveillance
- Prevention/aftercare colorectal carcinoma
- Pathological thickening of the colon wall detected by other imaging procedures
- Primary tumor search with metastasizing malignancy, if resulting therapeutic measures

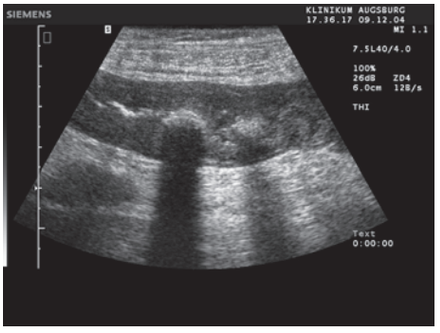


Figure 1.**1** **Thickened intestinal wall in the sigmoid colon.** Ultrasound examination of the left lower abdomen.

Contraindications. Only in a limited number of situations do the risks of colonoscopy outweigh the benefits of its diagnostic value. Contraindications include suspected intestinal perforation, imminent risk of perforation accompanying acute diverticulitis, deep ulcerous lesions, or vascular necroses (Tab. 1.**3**).

The overall condition of the patient should always be assessed to determine whether he could tolerate the physical strain of preparing for colonoscopy and endoscopy, including conscious sedation. Colonoscopy in patients with a recent myocardial infarction is associated with a higher rate of minor cardiovascular complications compared with control patients. (3)

Table 1.**2** Recommendations for cancer prevention

Population	Periodic colonoscopy for cancer prevention
General population	Once every 10 years starting at age 55
Patients with colorectal polyp	Colonoscopy check-up once every three years, if no pathological findings at first examination, then further check-ups every five years
Patients with hamartomatosis polyposis	No general surveillance recommendations
Immediate family member with colorectal carcinoma or polyp at *> 60 years of age*	Ten years earlier than the age of the index patient at which carcinoma/polyp occurred, repeat every 10 years
Immediate family member with colorectal carcinoma or polyp at *< 60 years of age*	First colonoscopy at age 40, repeat every 10 years
Immediate family member with FAP (familial adenomatous polyposis)	*Genetic carriers:* starting at age 10, annual rectosigmoidoscopy, if polyp detection then colonoscopy; after proctocolectomy annual pouchoscopy *Noncarriers:* same as general population
Immediate family member with HNPCC	Starting at age 25, annual colonoscopy
Patients with colitis ulcerosa	For pancolitis > 8 years of age or left-sided colitis > 15 years of age: complete colonoscopy with annual biopsy for two years, then once every two years
Patients with Crohn disease	No general recommendations at this time

Table 1.**3** Contraindications for colonoscopy

- Perforated intestine
- Acute diverticulitis
- Deep ulcerations
- Severe ischemic necroses
- Fulminant colitis
- Cardiopulmonary decompensation

Attention

- The physical stress of preparation for the examination and the colonoscopy itself limits its use in seriously ill patients.

Preparing for the Examination

Oral preparation. Thorough bowel cleansing is essential for a sufficient endoscopic examination of the colon. The development in 1990 of a nonabsorbable electrolyte solution (polyethylene glycol, PEG) by Fordtran was a significant improvement over earlier laxatives using sodium sulfate and modified forms are still in use today. But, due to the large quantity of liquid that must be consumed (up to 4 L) and the salty taste, these solutions are not tolerated by all patients. Their effectiveness has, however, been verified by numerous studies; data on sodium phosphate solutions (e.g., Fleet) and whether these are an improvement in terms of cleanliness and patient acceptability are less conclusive (8). Though they may appear to be a viable alternative for some patients, caution should be exercised if the patient has kidney insufficiency given the high phosphate content.

Enemas and clysmas. The use of an irrigator is recommended for patients who, due to an obstruction, cannot be prepared for examination using an oral solution. If the patient is admitted for emergency endoscopy, a quick cleansing using a clysma is a feasible option for partial colonoscopy.

Complications and Risks

Perforation, bleeding, and infection. Endoscopy of the colon entails risk of perforation, injury to blood vessels causing bleeding, and infection (Figs. 1.**2**, 1.**3**). The rate of complications can be minimized if the examiner takes precautions such as advancing the instrument only under conditions of high visibility. Sigmoidoscopy involves an average perforation rate of 1.8 per 100 000 examinations; bleeding severe enough to require a blood transfusion and perforations requiring surgical repair occur at the same rate so that the number of patients who experience a serious complication is 6.4 per 100 000 (10).

Comparing diagnostic and therapeutic colonoscopy (1, 4), statistics indicate that, with a total morbidity of 0.4%, more complications arise from therapeutic measures, such as polypectomies (1.2% vs. 0.2%) (Tab. 1.**4**).

Treatment. Not all complications require surgical intervention. Bleeding can be stopped in 92% of patients endoscopically and infections can be controlled with antibiotics. Injury to the serosa related to perforation of the intestine is painful for the patient and in most cases is surgically repaired before peritonitis occurs. In some patients, gaping wound edges can be closed with endoscopically applicable clips and further healed with a liquid diet and antibiotics (5).

Cardiopulmonary complications. The use of analgesics for colonoscopic examination increases the risk of cardiopulmonary complications, even when the utmost caution is exercised in selecting medication and dosage (cf. Chapter 4). Older and comorbid patients are especially at risk for medicamentosus hypotension, tachycardia, and respiratory failure.

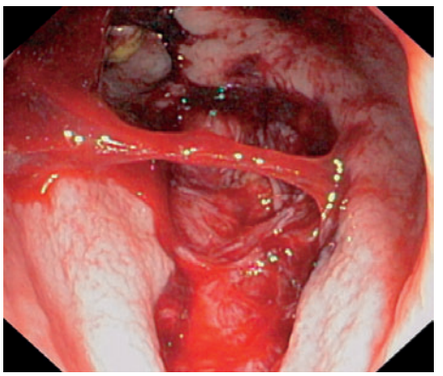


Fig. 1.2 **Intestinal perforation**

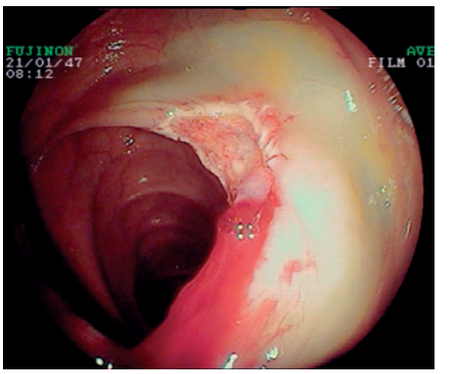


Fig. 1.3 **Intestinal bleeding following an endoscopic polypectomy.**

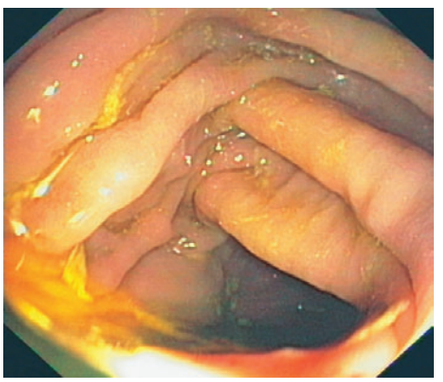


Fig. 1.4 **Intestinal lumen with low air insufflation.**

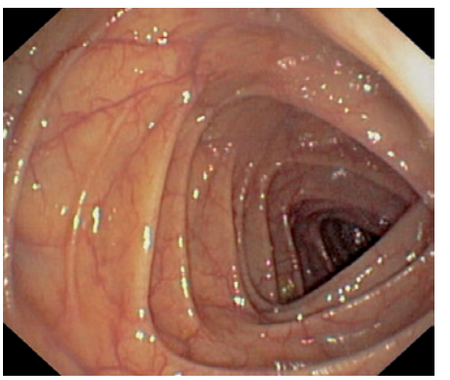


Fig. 1.5 **Intestinal lumen with high air insufflation.**

Table 1.4 Complications arising from colonoscopy (1, 4)

Total morbidity	0.4 %
for diagnostic colonoscopies	0.2 %
for therapeutic colonoscopies	1.2 %
Bleeding	0.2 % of all colonoscopies 0.3–6.1 % of therapeutic colonoscopies
Perforations	0.1 % of diagnostic colonoscopies 0.1–0.3 % of therapeutic colonoscopies
Mortality	0–0.006 % of all colonoscopies

Attention

- Cardiopulmonary complications are the leading cause of death related to colonoscopy.

General Principles

The success of the procedure is determined by the cooperation of the physician, assistant, and patient, all of whom must adjust to varying patient conditions in terms of anatomy and pain threshold, to the different equipment positions and functions, and to varying levels of training and expertise on the part of the examiner and assistants.

Insufflation of air. In order to inspect a 1.20-m-long, stool-filled hollow organ, it must be cleansed prior to the procedure and then distended using air during the examination. The distention of the intestinal lumen is vital for advancing the colonoscope under constant visualization, but it is also the source of much of the discomfort experienced by the patient through the mechanoreceptors and pain receptors. Using CO_2 instead of air may be advantageous due to its quicker absorption. Nevertheless, a good rule of thumb for all colonoscopies: use as little air as possible and as much air as necessary (Figs. 1.**4**, 1.**5**).

Mesenteric discomfort. The patient may also experience discomfort from the mechanical strain on the mesentery. This occurs when the advancement of the colonoscope is prevented, for example, by looping in the sigmoid colon. An experienced examiner can determine the position of the instrument by the movement of the endoscope in the intestine and can attempt to avoid looping or to pull back (cf. Chapter 5) before the patient experiences noticeable discomfort.

Patient care. The examiner should continue to pay close attention to the patient while concentrating on the advancement of the colonoscope on the monitor screen. The effort to produce a thorough and accurate diagnosis is almost equally important to the patient as the subjective experience of colonoscopy, which includes all aspects of the examination, including the experience of discomfort, receiving sufficient information and the maintenance of dignity.

Five basic rules of colonoscopy
1. Do not advance the endoscope without a clear view of the lumen.
2. Do not advance the endoscope if there is any resistance.
3. When in doubt, pull back.
4. Use as little air as possible and as much air as necessary.
5. Pay attention to patient's pain reaction.

Anatomy of the Colon

The endoscopist views the colon from a perspective unlike that of any other visualizing technique, viewing the inner relief of the "intestinal skeleton," which is made up of three straplike bands of longitudinal muscles (tenia coli) and numerous half-moon-shaped cross-folds (semilunar folds) which give rise to the pouchlike haustra between them (Fig. 1.**6**).

Structure of the intestinal wall. The intestinal wall can be divided microscopically into four layers, the structure of which does not vary significantly from the six macroscopic segments of the colon (rectum, sigmoid colon, descending colon, transverse colon, ascending colon, and cecum) (Figs. 1.**7**, 1.**8**). The endoscopic forceps biopsy usually takes samples limited to the mucosa. In order to collect deeper proportions of the intestinal wall (e.g., submucosa) it is practical to use a snare.

Colon segments. The division of the colon into segments is based primarily on anatomical rather than functional aspects. Only the rectum and cecum are unlike the other segments in that they function as reservoirs.

Cecum. Passage from the ileum to the cecum is restricted by the Bauhin valve that prevents the backward flow of the contents of the intestine out of the colon into the small intestine. The Bauhin valve is made up of two lips with a reinforced circular muscle layer that permit the opening of a narrow slit, the ileocecal valve, and that merge into two membrane folds at the front and back (Fig. 1.**9**).

The cecum is normally located intraperitoneally in the iliac fossa of the lower right abdomen. In the final months of pregnancy, the beginning of the large intestine grows up the inside of the right side of the abdomen. A "displaced" cecum can result if it remains at the level of the liver.

The three tenia of the cecum converge in a star shape at the end of the (vermiform) appendix that is not intubated during colonoscopy.

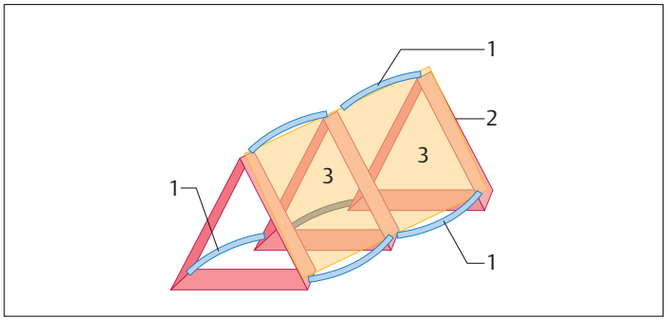


Fig. 1.**6** **Schematic structure of the colon wall.** Tenia (1), pliquae semilunares (2), and haustra (3).

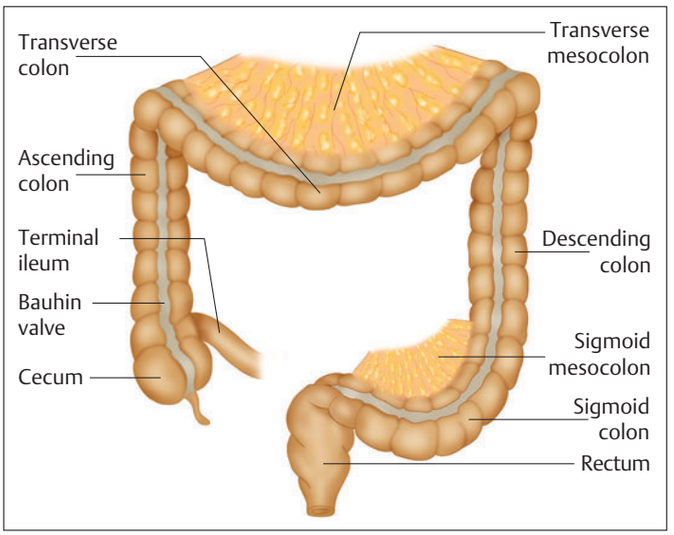


Fig. 1.**7** **Anatomy of the colon.**

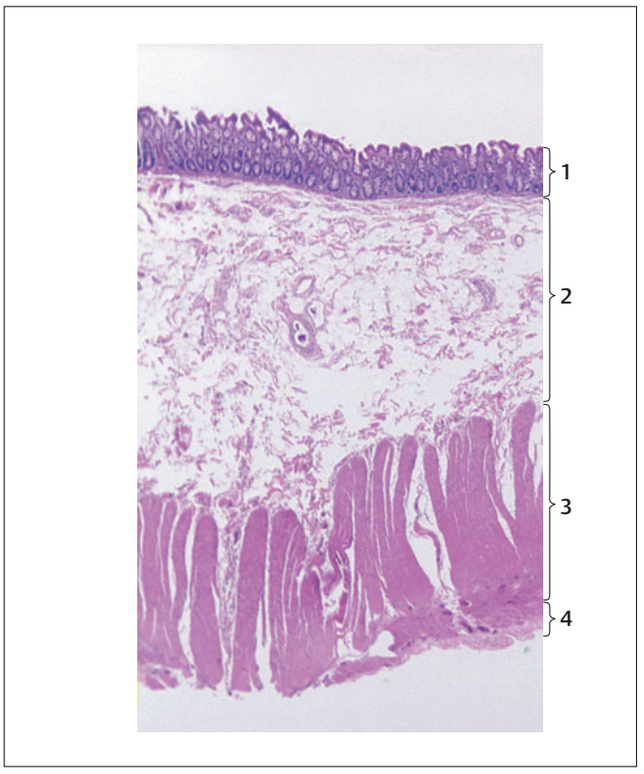


Fig. 1.**8** **Microscopic structure of the intestinal wall** (with the kind ▷ permission of Prof. Dr. H. Arnholdt, Pathological Institute, Klinikum Augsburg).
(1) Mucosa: deep, close together crypts with numerous goblet cells and enterocytes, high brush border;
(2) Submucosa: loose connective tissue layer, latticelike formation of collagen fibers, blood, and lymph vessels, vegetative plexus submucosus;
(3) Muscularis propria: strong inner circular muscle layer, outer longitudinal layer, reinforced tenia, plexus myentericus;
(4) Serosa: peritoneal covering.

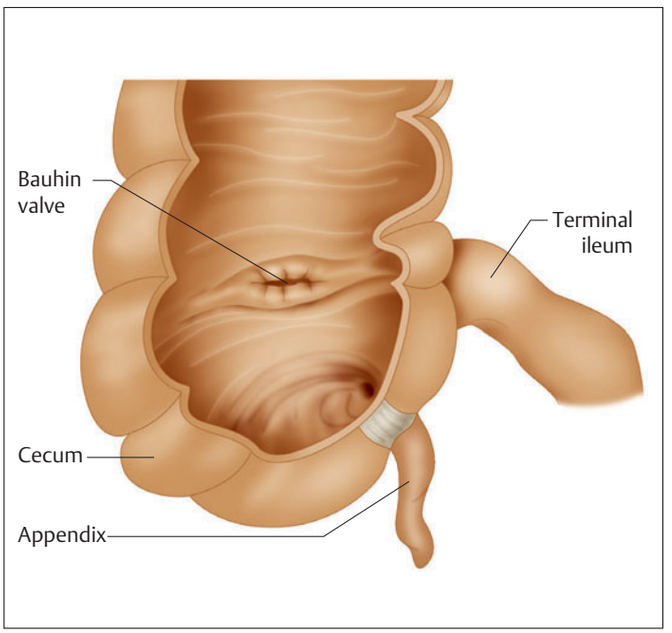


Fig. 1.**9** **Detailed view of the cecum and Bauhin valve.**

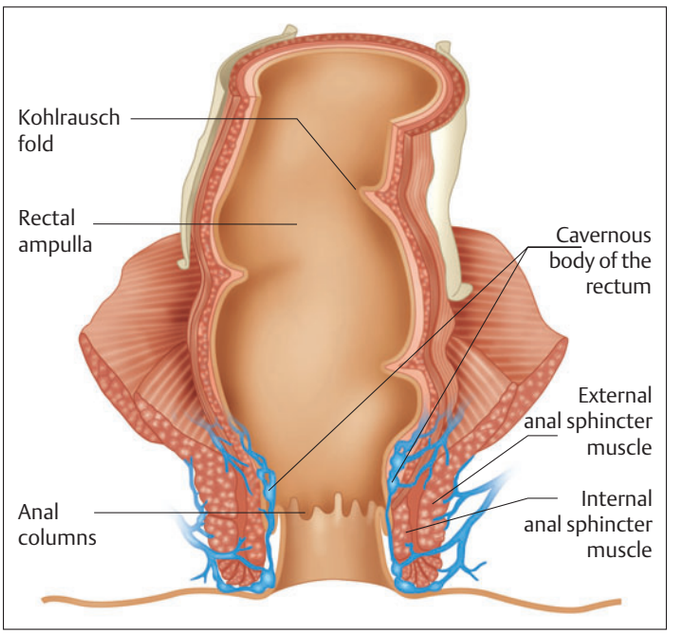


Fig. 1.**10** **Rectum and anal canal.**

Table 1.**5** Mesenterial fixation of the colon

Intestinal segment	Mesentery	Meaning for endoscopy
Sigmoid colon Transverse colon	Persistent, mobile mesentery	Makes endoscope passage difficult due to mobility in abdominal cavity
Descending colon Ascending colon Cecum	Retroperitoneal fixation of the mesentery	Endoscopic passage easier
Rectum	Primarily retroperitoneal	Good maneuverability of the endoscope

Mesentery. The mesentery is a double layer of peritoneum, which, during the embryonic phase, attaches the ascending colon and the descending colon to the back wall of the abdomen, creating a retroperitoneal fixation; the mesentery persists as a free attachment for the transverse colon and sigmoid colon so that they remain mobile (Tab. 1.**5**). Because of this, the passage of the endoscope can result in colon movements and even looping in the abdominal cavity (see Chapter 5).

Rectum and anal canal. The 15–20-cm-long rectum is closed off to the outside by the hemorrhoidal zone, where the anal columns containing arterial and vascular bundles are located. Together with the reinforced muscle layers of the internal and external anal sphincter muscles, the hemorrhoidal zone supports bowel continence. The epidermis extends 2–3 cm into the anal canal. Cranially limited by a transverse fold (the Kohlrausch fold), the rectal ampulla is a highly expandable area that functions as a reservoir (Fig. 1.**10**).

Attention

- Precise knowledge of anatomy is essential for the management of colonoscopy, correct description of pathological findings, and understanding the clinical features of intestinal diseases.

References

See Chapter 2.

2 Basic Examination Technique and Colonoscopy Workstation

G. Jechart

Learning Examination Technique

Learning colonoscopy technique requires motivation, manual dexterity, concentration, and patience on the part of the trainee. Already practiced in the technique of endoscopically examining the upper digestive tract, the beginner will learn about similarities and differences related to using a gastroscope vs. using a colonoscope.

Instrument Features

All endoscopes can be divided into three sections: the insertion tube, which is advanced in the patient, the instrument control head, where the physician can maneuver the endoscope tip and has access to the water/air supply and the instrument channel and, finally, the universal cord and plug, which connect the instrument to the supply unit (Fig. 2.**1**).

Insertion tube. The insertion tube of the video colonoscope consists of a ca. 130-cm-long tube containing optical fibers, digital wires, air and water nozzles, the instrument channel, and Bowden cables for better mobility.

Located at the tip of the endoscope are the lens and video chip, which produces the image (Fig. 2.**2**).

The last 15 cm of the insertion tube are especially flexible and can bend in all four directions, allowing better maneuvering in the gastrointestinal tract. The degree of flexion of the colonoscope is 180° up/down and 160° to the right/left. Compared with the colonoscope, the gastroscope can be moved to a greater degree upward, but to a lesser degree in all other directions (Figs. 2.**3**, 2.**4**, Tab. 2.**1**).

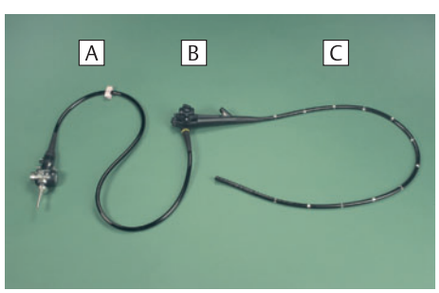


Fig. 2.**1** **Main parts of the colonoscope.** A Universal cord and plug, B Instrument control head, C Insertion tube (courtesy of Mr. Wirth, photo archive, Augsburg Clinic).

Fig. 2.**2** **Tip of colonoscope** (courtesy of Mr. Wirth, photo archive, Augsburg Clinic).

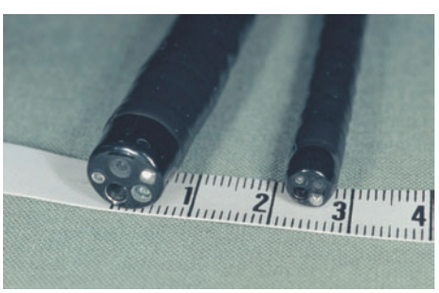
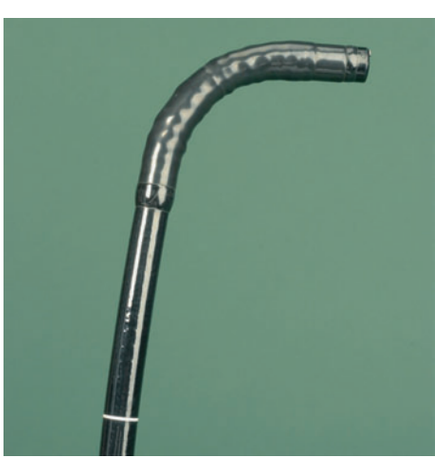


Fig. 2.**4** **Comparing the outer diameters** of the distal end of a colonoscope (left) and a gastroscope (right) (courtesy of Mr. Wirth, photo archive, Augsburg Clinic).

a



b

Fig. 2.**3** **Maximum angling downward.**
a Colonoscope.
b Gastroscope.
(Courtesy of Mr. Wirth, photo archive, Augsburg Clinic.)

Table 2.**1** Comparing standard video endoscopes (example: Olympus)

Standard video endoscopes	Colonoscope	Gastroscope
Maximum angling	Up 180° Down 180° Right/left 160°	Up 210° Down 90° Right/left 100°
Outer diameter, distal end	12.8 mm	10.2 mm
Length of insertion tube	133 cm	103 cm
Angle	140°	130°

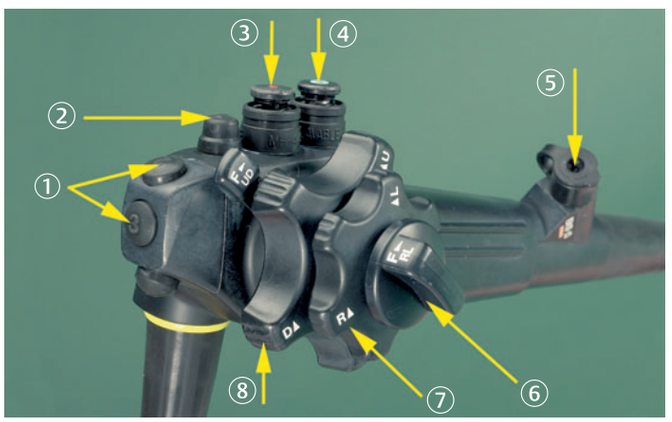


Fig. 2.**5** **Control head of a video colonoscope:**
1 Function buttons, e.g., video recorder remote control
2 Freeze button
3 Suction button
4 Air/water button
5 Instrument channel
6 Locking device
7 Angling wheel (right/left)
8 Angling wheel (up/down)
(Courtesy of Mr. Wirth, photo archive, Augsburg Clinic.)

The sigmoidoscope measures only 60 cm in total length. Because of its high degree of maneuverability, it is sometimes used in patients where the indications for examination are limited to the sigmoid colon and rectum.

Different colonoscope models can vary in length, outer diameter, and width of the instrument channel.

Instrument control head. The functions necessary for maneuvering the tip the endoscope, for suction, cleansing, and air insufflation are all located on the control head. (Fig. 2.**5**).

The opening to the instrument channel is somewhat below the air/water cylinder, but before the air/water channel merges with the suction channel. The diameter of the inside of the instrument channel is between 2.8 mm and 3.7 mm, allowing the insertion of endoscopic accessories such as biopsy forceps or polypectomy snares (Fig. 2.**6**).

Video endoscopes also have remote control buttons that, according to model, may have various functions. These buttons can generally be used for freeze frames, video recording, printing, and adjusting illumination intensity (peak and average). Newer generations are equipped with so-called big chips that allow the projection of a high-resolution screen-size image onto a video monitor. The image can be digitally enhanced using modern image processing technology (e.g., Olympus CV-160) for structure enhancement, variable by several levels during and even after the examination (see Chapter 3).

Universal cord. The universal cord connects the endoscope to the light source, air supply, water supply, suction pump, and video processor. The video processor transmits the image to the monitor screen on the video tower (Figs. 2.**7**–2.**10**).

Operating the Endoscope

Before every examination, the suction, cleansing, and air insufflation functions on the endoscope must be checked and the "white balance" set on the video processor. The physician's left hand holds the control head of the endoscope while the right hand moves the insertion tube or controls fine adjustment of the outer angling wheel on the control head (Fig. 2.**11**).

Suction and cleansing. The index finger of the left hand can be used to depress the suction button while the middle finger can either press the air/water button lightly for insufflation or more firmly to activate the washing system (Fig. 2.**12**).

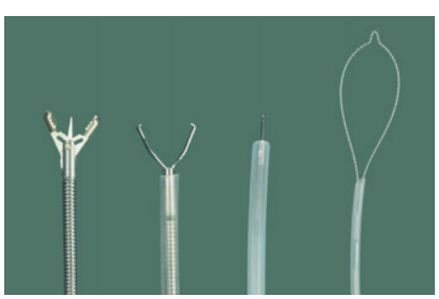


Fig. 2.**6** **Colonoscopy accessories** (from left to right): biopsy forceps, clip applicator, injection needle, and polypectomy snare (courtesy of Mr. Wirth, photo archive, Augsburg Clinic).

Fig. 2.**7** **Universal plug on an endoscope** (courtesy of Mr. Wirth, photo archive, Augsburg Clinic).

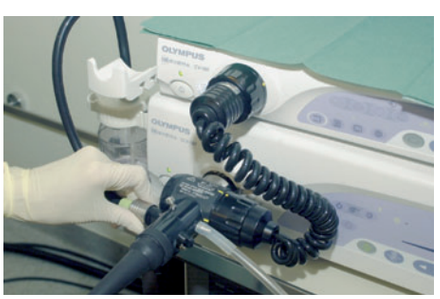


Fig. 2.**8** **Plugging the universal cord into the processing unit** (courtesy of Mr. Wirth, photo archive, Augsburg Clinic).

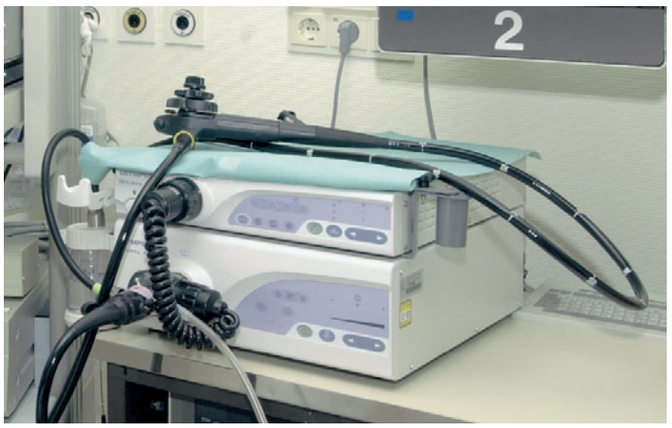


Fig. 2.**9** **Video processor (above) and light source (below)** (courtesy of Mr. Wirth, photo archive, Augsburg Clinic).

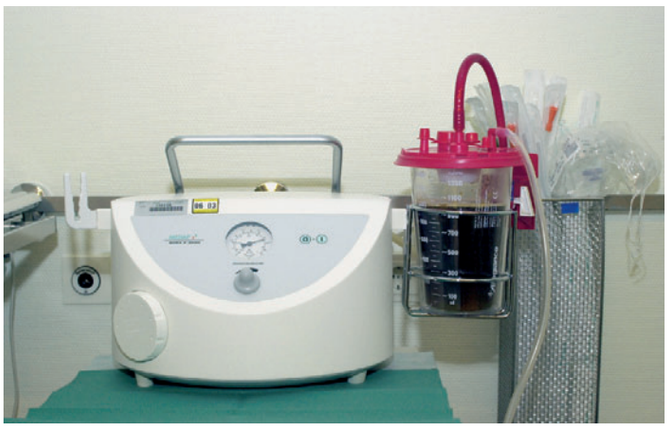
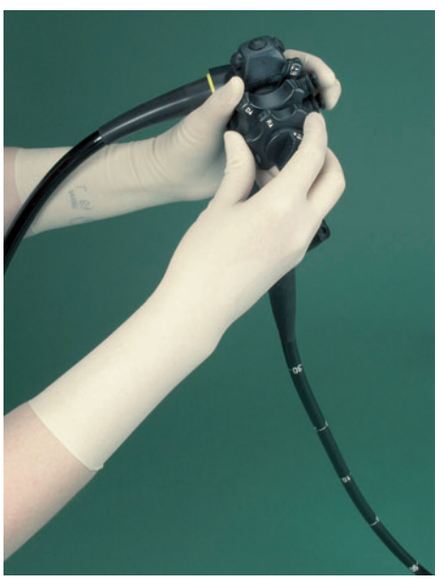


Fig. 2.**10** **Suction pump** (courtesy of Mr. Wirth, photo archive, Augsburg Clinic).

a



b

Fig. 2.**11** **Operating the instrument.**
a The examiner's right hand guides the tube or
b Fine adjustment of the endoscope tip by moving the angling wheel.
(Courtesy of Mr. Wirth, photo archive, Augsburg Clinic.)

a

b

Fig. 2.**12** **Operating the valves.**
a Suction button.
b Air/water channel.
(Courtesy of Mr. Wirth, photo archive, Augsburg Clinic.)

Flexion. Two wheels control the angling of the endoscope in different directions. Using the thumb of the left hand, the large wheel can be turned to move the tip of the endoscope up or down while the smaller wheel directs the tip of the endoscope right and left (Figs. 2.**13**, 2.**14**, Tab. 2.**2**). Each wheel has a locking device so that it can be fixed in one position, allowing, for example, the right hand to remain free to use the instrument channel (Fig. 2.**15**).

Advancing the endoscope. In an experienced and well-coordinated team, the assistant can advance the insertion tube in the colon while the physician uses both hands on the control head to steer the tip of the endoscope (Fig. 2.**16**). However, some examiners prefer to advance the shaft themselves in order to better feel the position of the instrument. In this case, the right hand is used only for fine adjustments using the angling wheels on the head of the endoscope (Fig. 2.**17**).

Fig. 2.**13** **Moving the large angling wheel.**
a Downward.
b Upward.
(Courtesy of Mr. Wirth, photo archive, Augsburg Clinic.)

Fig. 2.**14** **Moving the endoscope tip** corresponding to the maneuvering of the large angling wheel. **a** Upward (cf. Fig. 2.**13 a**). **b** Downward (cf. Fig. 2.**13 b**) (courtesy of Mr. Wirth, photo archive, Augsburg Clinic).

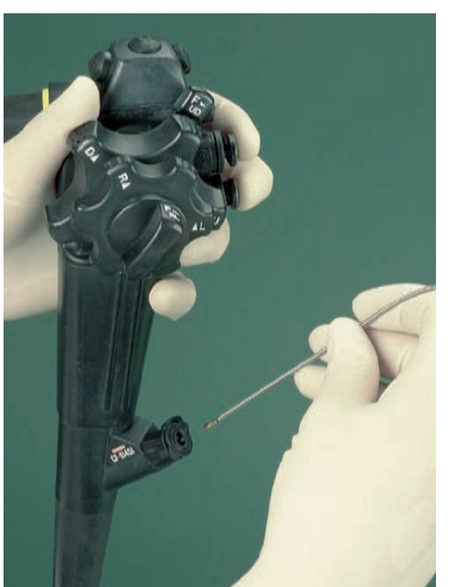


Fig. 2.**15** **Inserting biopsy forceps into the instrument channel** (courtesy of Mr. Wirth, photo archive, Augsburg Clinic).

Table 2.**2** Maneuvering the endoscope tip using the angling wheels

Rotation of the angling wheel	Movement of the endoscope tip
Large wheel: **toward** examiner	Raises the tip
Large wheel: **away** from examiner	Lowers the tip
Small wheel: **toward** examiner	Turns the tip to the left
Small wheel: **away** from examiner	Turns the tip to the right

Simulators

Maneuvering the endoscope and manual technique. The novice must first take time to study the functions of the endoscope. It is important to practice instrument handling before performing the first examination on a patient. The development of three-dimensional computer simulation can enable the trainee to practice maneuvering the endoscope and to develop the necessary manual dexterity (Figs. 2.**18**, 2.**19**).

According to one study evaluating usage of the Simbionix system, two hours of practice per day for three weeks are necessary in order for a novice to approach the level of handling expertise of an experienced endoscopist (6). Depending on the computer program, the virtual endoscopy simulator can simulate normal and pathological findings in the colon, helping to improve later detection of pathologies on actual patients.

Therapeutic interventions. Even though therapeutic interventions can now be computer simulated, experts agree that computer-simulation training of the novice endoscopist can offer only limited improvement in technique and skill; an actual examination situation is much more complex due to differences in mucosal properties and physiological factors. Thus, costly animal models are used in some courses aimed at maintaining endoscopic competency, with small groups practicing specific techniques (e.g., hemostasis) under more realistic conditions. The EASIE concept developed in Erlangen, Germany, is an animal-part simulator that integrates organs from a slaughterhouse into the model and, using perfusion, simulates in-vivo endoscopic interventions (7) (Fig. 2.**20**).

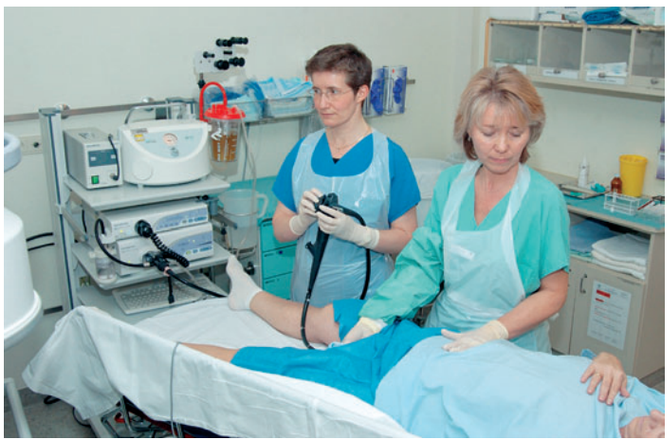


Fig. 2.**16** **The assistant advances the endoscope** (courtesy of Mr. Wirth, photo archive, Augsburg Clinic).

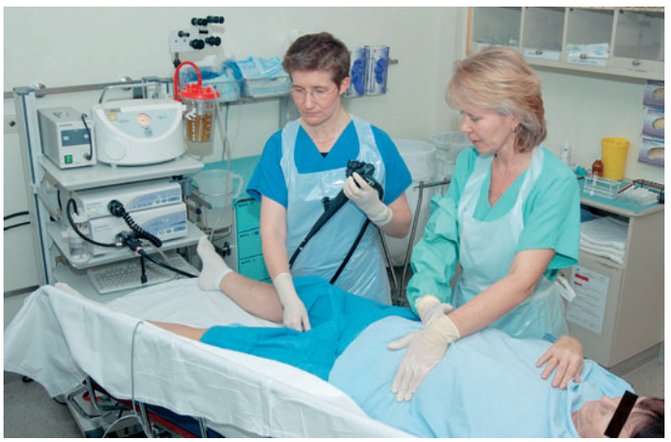


Fig. 2.**17** **The physician advances the endoscope** (courtesy of Mr. Wirth, photo archive, Augsburg Clinic).

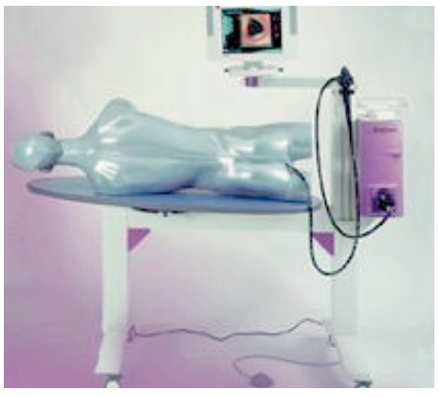
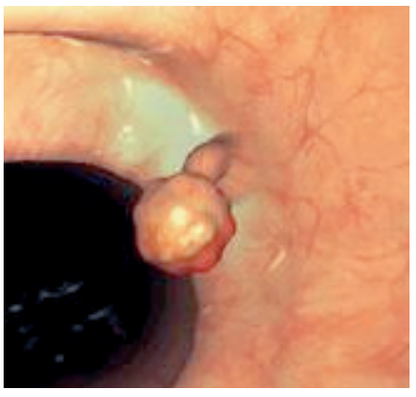


Fig. 2.**18** **Simbionix computer simulator** (courtesy of Simbionix).

a



b

Fig. 2.**19** **Virtual training** (courtesy of Simbionix).
a Manual skill training.
b Virtual colonoscopy using a simulator.

Training on a Patient

Observation. Despite modern computer-assisted learning techniques, observing while an experienced endoscopist performs colonoscopy is a key part of training for the beginner. It is important that the explanation by the endoscopist is suited to the trainee's level of training and that the handling of the instrument in technically difficult situations is described. An expert description of pathological findings enables the student endoscopist to better identify pathologies already seen in textbooks.

Withdrawing and advancing the instrument. The first practical exercise to be performed on the patient is withdrawing the instrument from the cecum to the rectum, taking care that all segments are sufficiently visualized. The next objective is to learn how to advance the instrument to the cecum. For this step, the trainer advances the shaft while the student operates the control head. Only after advancement to the cecum can safely and successfully be performed should training begin for intubating the Bauhin valve (Tab. 2.**3**).

Simple and complex interventions. Simple interventions such as performing a forceps biopsy, removing small polyps, and performing hemostasis by injection should be mastered before performing colonoscopy without supervision, in order to spare the patient an unnecessary further examination.

More complicated maneuvers, such as the removal of larger or sessile polyps, the use of clip applications, dye spraying techniques, performing mucosectomies, balloon dilations, or bougienage, are only performed after acquiring sufficient experience in basic technique, e.g., during gastroenterological training. Training centers such as CCEPDT (Competence Centers in Education, Procedure Development, and Training), for example, offer quality-controlled courses taught by skilled endoscopists experienced in teaching (7).

Table 2.**3** Steps in colonoscopy training on a patient

Steps	Objective of basic colonoscopy training
1	Observe examination procedure
2	Withdraw the instrument from the cecum to the rectum
3	Advance the instrument to the cecum under the guidance of an experienced endoscopist
4	Advance the instrument to the cecum under supervision, but without direct assistance
5	Intubation of the Bauhin valve
6	Interventions: forceps biopsy, removal of small polyps, simple hemostasis

The Colonoscopy Workstation

Layout and instrumentation in modern endoscopy units, where colonoscopy is performed under quality control, are designed according to normative standards, and also conduct regular hygiene checks.

Examination Room Set-up

The design of the colonoscopy workstation should meet not only the requirements of an ergonomic examination procedure and a patient-friendly atmosphere, but also must comply with regulations concerning ventilation and installation of electrical equipment. Keeping dust, micro-organisms and odors to a minimum is just as important for personnel as it is for the patient (Fig. 2.**21**).

Every examination room must have a hygienic area for hand washing. Direct access to a patient bathroom is also desirable.

The room where the preparations for endoscopy take place must be in the immediate vicinity of the examination room and must be able to be divided into clean and unclean zones.

Hygiene Standards for Reprocessing Equipment

Automated reprocessing. After use, the endoscope is wiped off in the examination room and placed in a container with a tenside-based (e.g., Bodedex forte) cleansing solution where it is

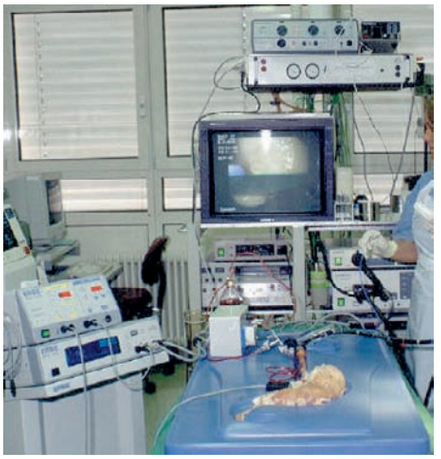


Fig. 2.**20** **EASIE model** for training in endoscopic interventions (courtesy of Dr. Maiss, University Clinic Erlangen).

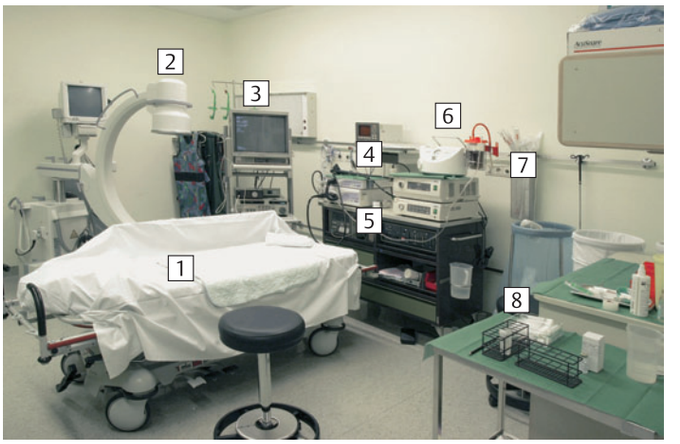


Fig. 2.**21** **Colonoscopy workstation.** Examining table (1), Radiography equipment (2), Video tower (3), Power unit, endoscope with video processor (4), Light source (5), Air supply/pump (6), Suction pump (7), Instrument table for accessories (8) (courtesy of Mr. Wirth, photo archive, Augsburg Clinic).

flushed. Aldehyde should not be used as it may cause protein fixations in the instrument channels. The endoscope is then disconnected from the power unit and brought to the storage area where it is again immersed in a cleansing solution and checked for leakages. A careful brush cleaning can reduce bacterial count by four log levels (Fig. 2.**22**). The valves should be removed and, together with the accessories, cleaned mechanically and enzymatically using an ultrasonic cleaner.

Reprocessing and disinfection procedures. Further cleaning of the endoscope can, in theory, be accomplished manually or automatically. However, automatic-washer disinfectors have distinct advantages with regard to protecting personnel from potential health hazards, as well for standardization of disinfection procedures (12).

The instrument and accessories are loaded separately into baskets or trays in the automatic-washer disinfector and the endoscope is attached to the automatic cleaning system (Fig. 2.**23**). A solution of either 2.4% glutaraldehyde or 10% succine dialdehyde is used for disinfection. The final rinse cycle is completed using sterilized water.

After thermochemical reprocessing and disinfection have been completed (ca. 40 minutes) the endoscope can be removed and allowed to dry. Air can be aspirated through all channels to speed up the drying process.

The endoscope is stored in a hanging position and without reattaching the valves to avoid recontamination resulting from residual dampness (Fig. 2.**24**).

Hygiene standards and tests. The majority of infections cited in the literature result from lacking hygiene, so it is vital that cleaning and disinfecting the instrument be conducted only by qualified personnel with up-to-date training (2). Hygiene standards exist for cleaning and disinfecting flexible endoscopes and accessories (13). Conducting quarterly tests as a quality control measure for equipment cleaning is recommended (9) (Fig. 2.**24**). This includes checking cleaning, disinfection, testing for microorganisms in all endoscope channels and lens washing systems. Detection of *Escherichia coli* or other enterobacteria or enterococci, in particular, is evidence of inadequate reprocessing. Correct endoscope reprocessing, disinfection and sterilization procedures are essential, and the health and safety of the patient, users and third parties must not be endangered.

Radiography Equipment Regulations

Radiographic screening is occasionally used to determine the position of the instrument in the patient. Commonly, a mobile radiograph is used, which is passed briefly over the patient's abdomen. The examination room must comply to specific standards, for example, must be large enough so that the axis of the radiographic path is at least 1.5 m from the nearest wall in order to avoid hazardous reflection of the ray.

Operating knowledge of radiography equipment (professional certification) is mandatory as is the wearing of appropriate protective clothing. A radiographic dosimeter must be worn on an appropriate position on the upper body, underneath the protective apron (Fig. 2.**25**).

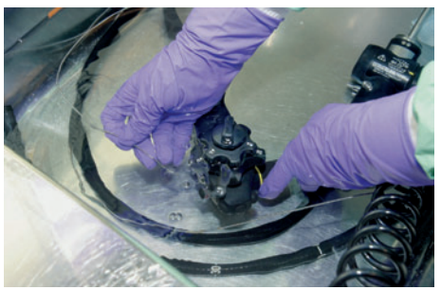


Fig. 2.**22** **Brush cleaning the endoscope after use** (courtesy of Mr. Wirth, photo archive, Augsburg Clinic).

Fig. 2.**23** **Loading the endoscope in the automatic washer–disinfector** (courtesy of Mr. Wirth, photo archive, Augsburg Clinic).

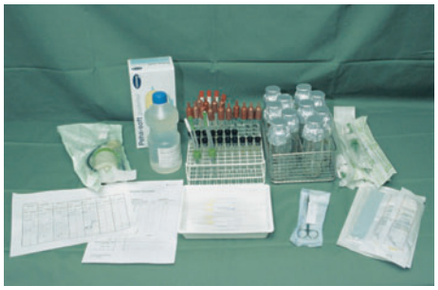


Fig. 2.**24** **Quarterly hygiene tests are essential** (courtesy of Mr. Wirth, photo archive, Augsburg Clinic).

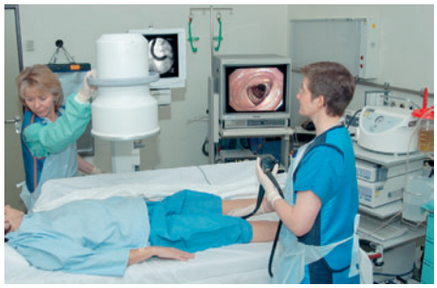


Fig. 2.**25** **Using radiography during colonoscopy** (courtesy of Mr. Wirth, photo archive, Augsburg Clinic).

Fig. 2.**26** **Storing the endoscopes in lockers** (courtesy of Mr. Wirth, photo archive, Augsburg Clinic).

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3 Modern Endoscopic Techniques

M. Bittinger

The use of video endoscopes has led to a significant increase in the quality of visualization compared with examinations performed using fiberoptic instruments. The charged-coupled device (CCD) chips used in modern video endoscopes now provide such high resolution that it is possible to detect even tiny details on the mucosal surface, which has led to significant advancements in detecting small and flat lesions, especially flat polyps. Yet, increasing resolution using video chips is only one means of increasing diagnostic yield in endoscopic procedures. Other advancements made over recent years, which will be discussed here, are:

- digital structural enhancement,
- magnifying or zoom endoscopy,
- chromoendoscopy,
- fluorescence endoscopy.

Digital Structure Enhancement

The higher resolution of modern video chips is accompanied by improved processing of the video signal by the computer to which the endoscope is attached. When the signal is transmitted to the image on the monitor, image processing (digital structure enhancement) allows surface structure details to be emphasized (Fig. 3.**1**). Extremely high resolution of detail is possible, especially when used with magnifying endoscopy and chromoendoscopy as discussed below.

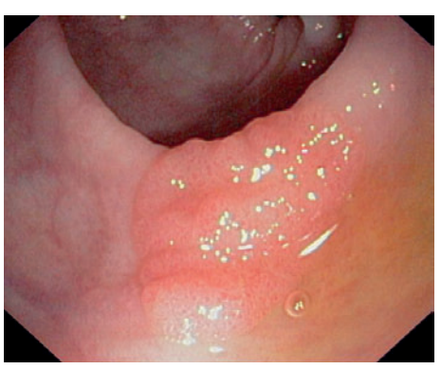
Magnifying or Zoom Endoscopy

Magnifying or zoom endoscopes enable image enlargement to a point nearly comparable to intravital microscopic examination of the mucosa.

Automatic and electronic magnification systems. Enlargement can be achieved using a built-in powered lens system, computer-supported electronic magnification to produce a digitally enlarged image, or a combination of the two. Electronic magnification is technically simpler from an instrument standpoint and does not require moveable parts, but the enlarged image often appears "grainy." The degree of detail in electronic magnification depends on the resolution offered by the CCD chip used, which limits the degree of magnification attainable. Mechanical enlargement, which uses moveable lenses, has the advantage of optical zooming (as opposed to mere digital approximation), similar to that of a microscope, with current magnification ranging from 100–150-fold. It has the disadvantage, however, of having to be integrated into the endoscope in a moveable lens system, including motorization (either manually with a linkage system or using a built-in miniature servo motor).

Discriminating detail. Extreme enlargement allows a high level of discrimination of the selected image. Yet, to achieve a sharp picture, the endoscope tip must be fixed very close to the mucosal surface being examined (focal distance). To do so, transparent caps are placed on the endoscope tip and then set on the mucosa to keep it at the correct distance from the instrument. Large vessels in the area or respiratory movements may cause movement artifacts, rendering zoom endoscopy impossible in isolated cases.

Magnification can reveal surface structure detail of even the smallest vessels (Figs. 3.**2**, 3.**3**), especially when combined with surface staining techniques (chromoendoscopy, see below).



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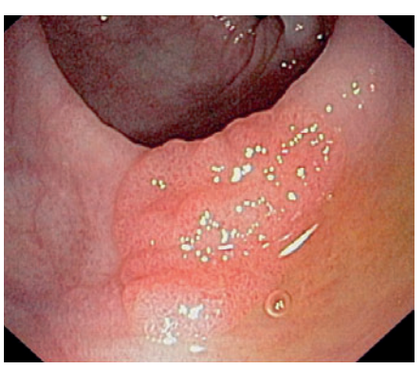


b

Fig. 3.**1** **Broadbased colonic adenoma.**
a View without digital structure enhancement.
b View with digital structure enhancement. The surface structure can be seen more clearly.

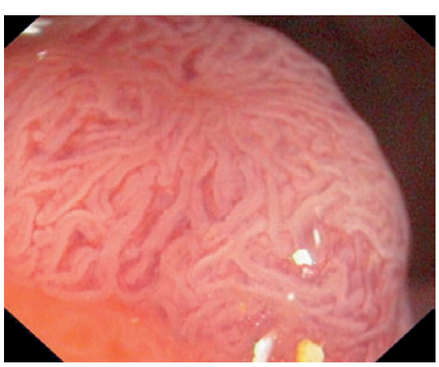


Fig. 3.**2** **Detailed surface structure of a tubulovillous adenoma in the colon.**

Table 3.**1** Overview of the most commonly used dyes and the corresponding techniques for chromoendoscopy of the colon

Dye	Type	Concentration	Side effects	Application technique
Indigo carmine	Contrast dye	0.4 % (0.1–1 %)	None	▶ No special pretreatment necessary, remove impurities with water if necessary. ▶ Apply using spraying catheter, discrimination possible immediately
Crystal violet (usually used with indigo carmine)	Absorptive dye	0.2 %	None	▶ No special pretreatment necessary, remove impurities with water if necessary. ▶ Apply using spraying catheter, discrimination possible after ca. one minute
Methylene blue	Absorptive dye	0.5–1 %	Possible green coloration of urine, DNA-damage (?)	▶ Surface pretreatment necessary: spray with ACC solution (10 %) to remove mucosal layer, ▶ Rinse after 1–2 minutes, then apply dye, discrimination possible after 2–3 minutes

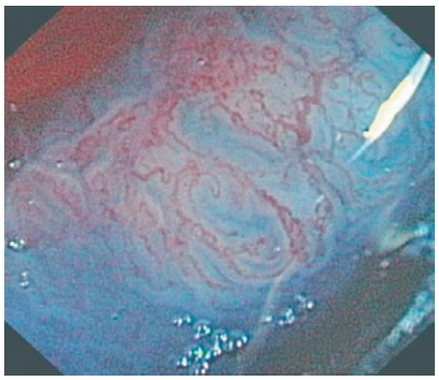


Fig. 3.**3** **Pathological vessels on the surface of a colonic carcinoma.**

Chromoendoscopy

Chromoendoscopy is a simple method that can increase the therapeutic yield of endoscopy, in particular, with regard to diagnosing dysplasia and detecting and treating flat neoplasias in the colon. Dye spraying helps distinguish fine surface details on the mucosa, allowing, on the one hand, better detection and classification of otherwise easily missed flat polypoid lesions, while, on the other hand, making it easier to distinguish between the polyp and the surrounding mucosa.

Dyes. Either absorptive or contrast dyes can be used. Contrast dyes are not absorbed by the mucosa but instead collect in tiny grooves on the mucosal surface, emphasizing surface detail. Absorptive dyes, unlike contrast dyes, are actively absorbed by the mucosa. Their use thus is also referred to as intravital staining. The dye is absorbed differently by dysplastic or malignant tissue than by healthy tissue, giving rise to differences in staining pattern, which allow for better differentiation between healthy and diseased tissue.

Examinations of the colon use mostly contrast dyes; indigo carmine is the most frequently used. Absorptive dyes (methylene blue, crystal violet) are used more often in the upper gastrointestinal tract (especially in the esophagus), though they can also be used in the colon. Contrast dyes are much easier to use than absorptive dyes as they can be sprayed using a dye-spraying tube without specially preparing the surface and there is no waiting time for absorption by the mucosa. The extra time required for staining using indigo carmine is minimal and the resulting gain in visual information impressive.

Table 3.**1** provides an overview of the three most commonly used dyes and related techniques for their use.

Pit pattern classification. Combining zoom endoscopy with surface staining enables differentiation of the mucosal surface comparable to that achieved using a microscope. This technique forms the basis of pit pattern classification of surface structures of polypoid colonic lesions, which was introduced by Kudo in 1996. The pit pattern classification is useful for further distinguishing between nonneoplastic (hyperplastic or inflammatory) and neoplastic (adenomatous or malignant) lesions. Modern high-resolution video endoscopes are generally sufficient for pit pattern classification, even without using special magnification techniques.

The pit pattern classification divides the tiny pits on the surface of polypoid lesions by size and form into five groups (pit patterns I–V). Group III is further subdivided into two subgroups (Type IIIS [S for small] and Type IIIL [L for large]). ▣3.**1** gives an overview of the classification system and corresponding characteristics. With a little practice, correct classification is straightforward and reproducible.

Type I and Type II findings correspond to nonneoplastic lesions, Types III–V are nearly always neoplastic lesions, whereby Type V lesions are highly suspicious for the presence of malignancy. Systematic studies have shown that pit pattern classification has a sensitivity of 92–98 % and a specificity of 61–95 % for distinguishing between neoplastic and nonneoplastic lesions. Thus, its routine clinical use is worthwhile.

Fluorescence Endoscopy

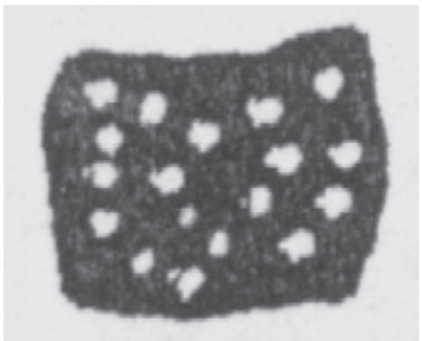
Fluorescence endoscopy is a new procedure that increases endoscopic detection of poorly visible malignancies or premalignancies. Fluorescence can be endogenous or exogenous and is commonly induced using 5-aminolevulinic acid.

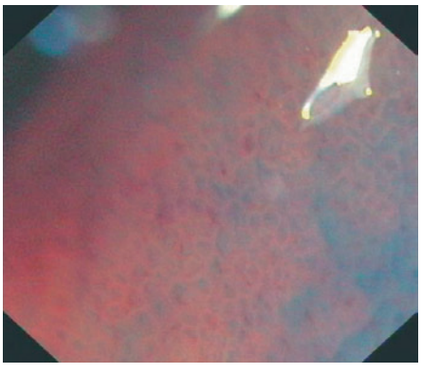
Exogenously induced fluorescence. Special sensitizers are administered exogenously (locally or systemically as an oral solution) and accumulate selectively in malignant tissue. Exposure to light of a certain wavelength then induces selective (red) fluorescence.

3.1 Overview and examples of pit pattern classification

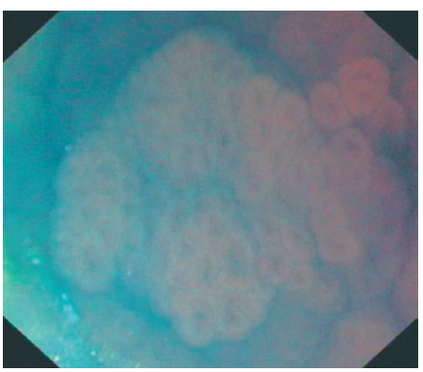
Pit pattern type and characteristics

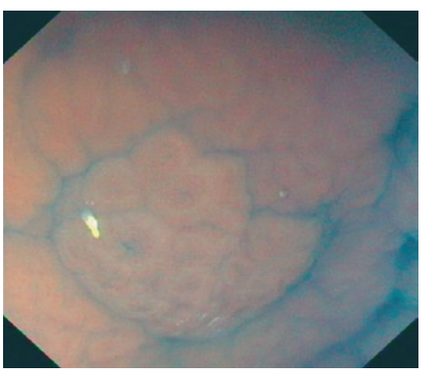
I round pits



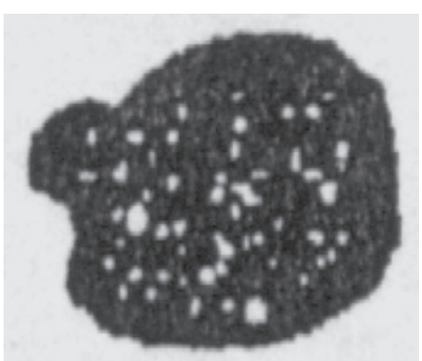


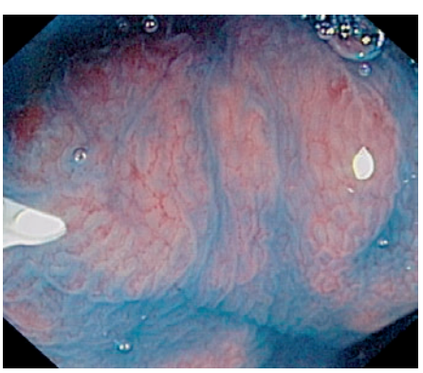
II stellar or papillary pits

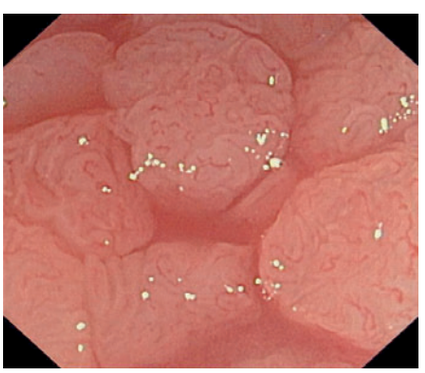




III S small tubular or round pits

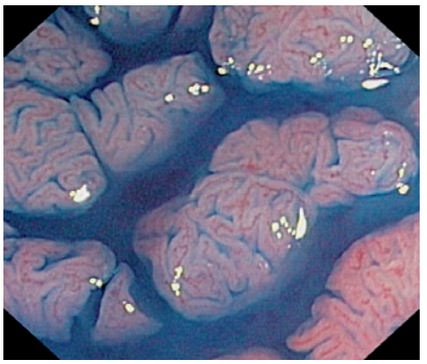


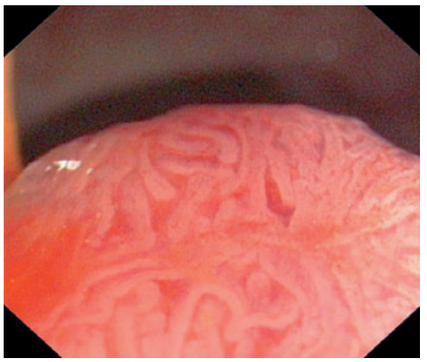




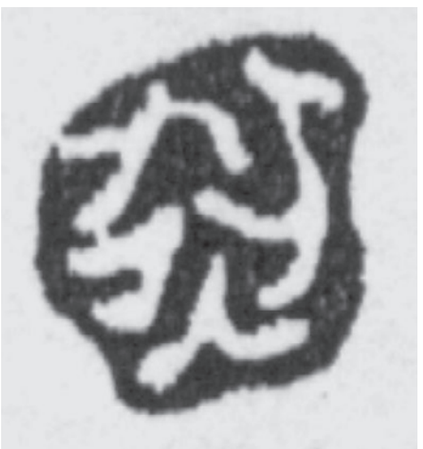
3.1 cont.

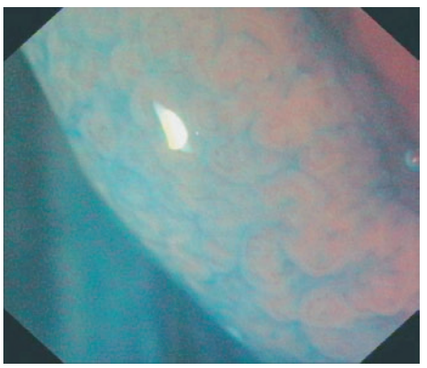
III L large tubular or round pits



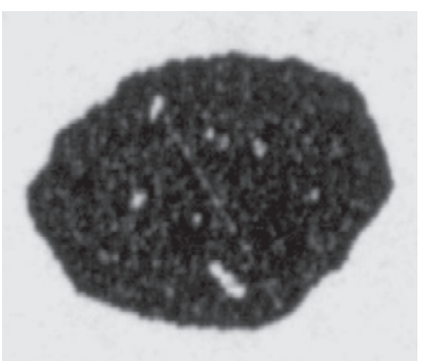


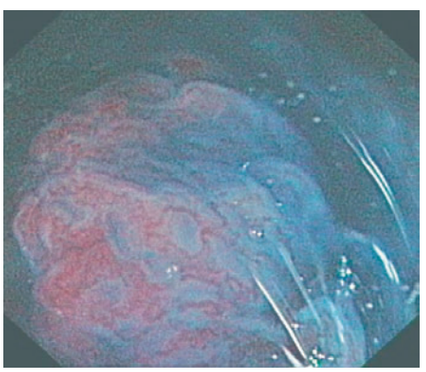
IV gyruslike or branchlike pits

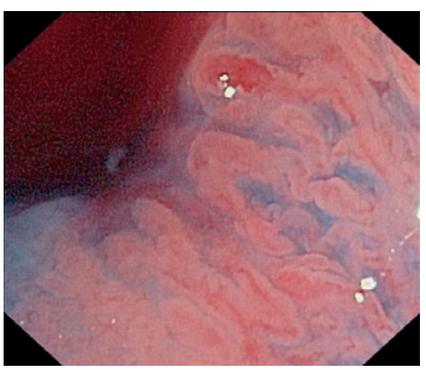


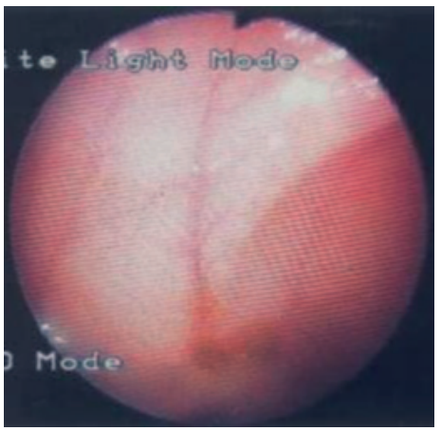


V nonstructural pits









a

b

Fig. 3.4 **Patient with ulcerative colitis.**
a Conventional white light endoscopic image with clear vascular structure.
b Using blue light one sees selective red fluorescence of a small mucosal area corresponding to the histological "flat adenoma" with dysplasia. The patient was sensitized with 20 mg/kg 5-aminolevulinic acid five hours beforehand.

Exogenously induced fluorescence following sensitization with 5-aminolevulinic acid is currently most widely used in urology. Preliminary use of this method in gastroenterology has given hope that it may lead to the development of improved early detection of carcinomas and dysplasias related to Barrett esophagus, ulcerative colitis, and other premalignant tissues. Figure 3.**4a** shows an apparently nonpathological colon from a patient who has had ulcerative colitis for several years. Figure 3.**4b** shows a selective red fluorescence of a "flat adenoma" in the same patient.

Autofluorescence. The use of autofluorescence has been most extensively studied in pulmonology research. Endogenous fluorophores (such as collagen, elastin, FAD, NADH) can be found in various concentrations and states of oxidation in malignant and nonmalignant tissue, allowing tissues to be distinguished based on their different autofluorescence. This ultimately leads to reduced green autofluorescence, so that pathologies appear red or brown.

Disadvantages. Significant disadvantages of the method are that glass-fiber endoscopes have to be used and that the administration of 5-aminolevulinic acid for exogenous fluorescence induces light sensitivity in the patient.

Prototypes of new video endoscopes are currently being evaluated, as is the use of new locally administered sensitizers that do not result in light sensitivity.

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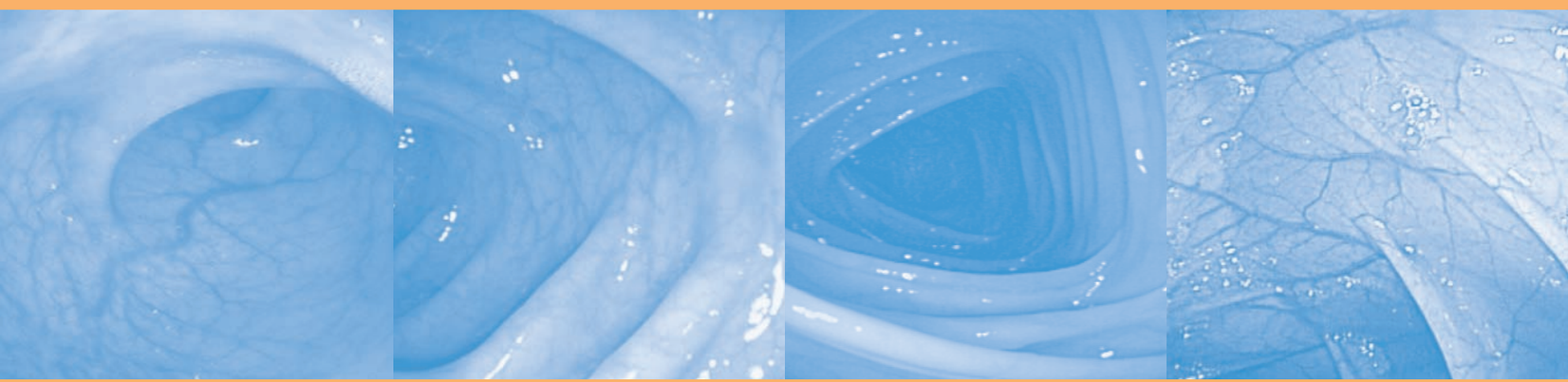
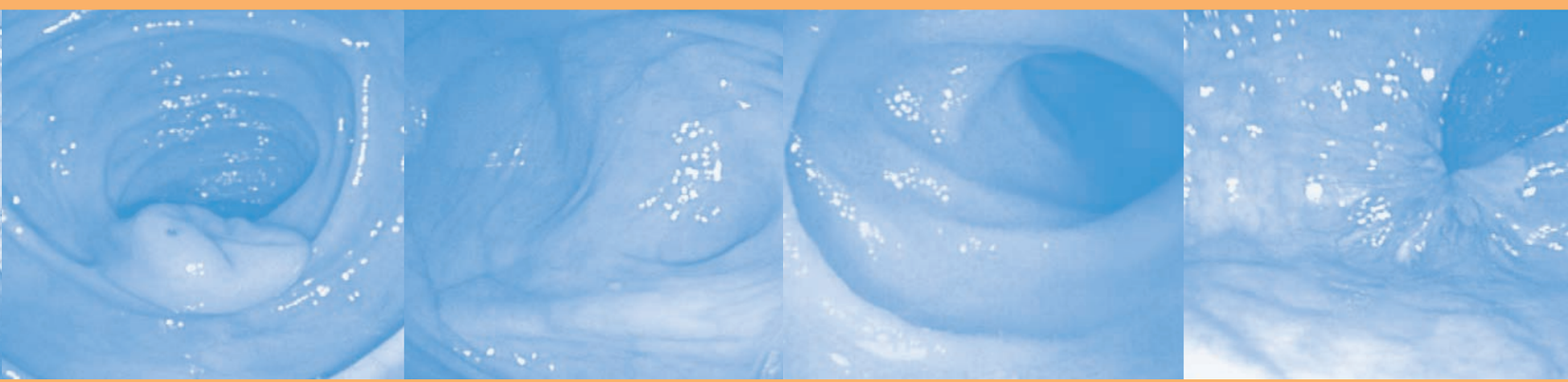


II Normal Examination Procedure and Non-pathological Findings



4 Before the Examination

A. Probst

Informing the Patient

The decision if colonoscopy is the best diagnostic or therapeutic approach should be made based on present indications (see also Chapter 1). If the indication supports the need for colonoscopy, the patient must be informed prior to examination about the necessity, procedure, and possible complications of the examination and he must sign a consent form.

Serious complications are rare and the vast majority are due to cardiopulmonary complications. Risk varies greatly from one individual to the next, depending on cardiopulmonary disease status and the use of an analgesic. In addition to more general risks, there are also potential complications specifically related to colonoscopy (foremost bleeding and perforation; see also Chapter 1). In any discussion preceding colonoscopy, the patient must also be informed of the possibility of polypectomy, which entails increased risk.

For outpatient examinations, the patient must be cautioned against performing any activities that could cause harm to him or others for a period of 24 hours following sedation (e.g., driving, operating heavy or complex machinery, signing important documents such as contracts). Alternative examination and therapeutic options should also be mentioned. Standardized written consent forms may make discussion and documentation easier, but they cannot replace an informative discussion. The patient must be given sufficient time between discussion and the actual examination to consider his decision; according to German law, the examination can be performed no sooner than one day after the discussion between patient and physician (7).

Sedation and Medication

Sedation and Analgesics

Colonoscopy can theoretically be performed without sedation, and there are no fixed rules for premedication. Nonetheless, premedication improves examination conditions for both patient and physician. This has been confirmed by results from a study by Terruzzi et al. comparing routine premedication prior to colonoscopy to "on-demand" sedation during the examination. Among patients who began the procedure without sedation, 66% requested an analgesic during the examination and a larger number of them also refused to undergo another colonoscopy in the future (22% vs. 10% in the comparison group) (10).

Benzodiazepines. The vast majority of patients visiting our endoscopic unit receive routine sedation prior to colonoscopy. "Conscious sedation" and, if possible anesthesia-induced amnesia, are desirable. Sedation is generally administered intravenously, using a benzodiazepine (Midazolam, Diazepam, Diazemuls); Midazolam (Dormicum; 0.07–0.1 mg/kg i. v.) has the advantages of a pronounced amnestic effect and a short half-life of 1.5–3 hours.

Opiates. Additional analgesics are sometimes used to assist colonoscopy; the most common is a combination of benzodiazepine and opiate (e.g., Dolantin). Dolantin (0.6–1 mg/kg) is administered intravenously. If combining substances one must be aware that the sedative effect of a benzodiazepine can be exponentially increased when used in combination with opiates, increasing the risk of respiratory depression. Flumazenil (Anexate) and Naloxon (Narcanti) are antagonists for benzodiazepines and opiates.

Propofol. Another option is the use of propofol, which rapidly induces hypnosis and has a short half-life of 2–5 minutes. However, propofol has a very narrow therapeutic index; in other words, a small change in dosage can produce either a sedative or a narcotic effect. A notable side effect is the possibility of a pronounced drop in blood pressure; patient blood pressure must therefore be monitored closely. No antagonist is available for this drug and various professional organizations strongly recommend that propofol only be used when an anesthetist is immediately available (4, 9). However, results from a study in which nurses administered propofol during colonoscopy under supervision of the endoscopist (a nonanesthesiologist with training in emergency medicine) did not report any complications (8). In our opinion, propofol should only be used when a trained physician, experienced in emergency medicine, is present alongside the examiner to monitor the patient's condition.

Cardiopulmonary complications. In 0.1–0.5% of patients premedication causes serious cardiopulmonary complications. Thus, the adequate administration of medication and monitoring of the patient during and after examination are of the utmost importance.

Other Medications/Endocarditis Prophylaxis

Spasmolytics. In addition to analgesics, antispasmodics should be available during colonoscopy to inhibit intestinal peristalsis (e.g., Butylscopolamine [Buscopan]; or Glucagon if there are contraindications).

Prevention of Endocarditis. If the patient has preexisting cardiac disease, the prevention of endocarditis and the risk of bacteremia must be considered prior to examination. The risk of bacteremia is ca. 4% for colonoscopy and ca. 2% for sigmoidoscopy; polypectomies do not significantly increase the risk for bacteremia. However, given the high risk of endocarditis among patients with heart valve replacement or a medical history of endocarditis, an antibiotic prophylaxis must always be used. For low-risk or moderate-risk patients (e.g., hereditary or acquired heart valve disease without previous endocarditis, mitral valve prolapse with mitral insufficiency), antibiotics are not strictly required for ileocolonoscopy (7). Currently, there are no standard recommendations regarding medication; however, for endoscopy of the lower gastrointestinal tract *Enterococcus faecalis*,

in particular, should be considered a potential source of infection. In our clinic we administer ampicillin and gentamycin for patients with especially high risk before the examination begins and ampicillin again six hours after examination (orally for outpatient procedures, if appropriate). If the patient has a penicillin allergy, we use vancomycin combined with gentamycin (Tab. 4.**1**). Our procedures are based on the recommendations of the American Heart Association (AHA) (2).

Allergies and Contraindications. The patient must be carefully interviewed prior to examination regarding allergies or other contraindications related to any medications. Penicillin allergy is a common example of information required for using an endocarditis prophylaxis. Possible contraindications must also be ascertained if Buscopan is to be used, and the patient should be asked specifically about these. If the patient reports any contraindications or if they are indicated (e.g., glaucoma, urinary retention, tachyarrhythmia), it cannot be used. It is also advisable to inquire about possible pregnancy prior to colonoscopy.

Table 4.**1** Endocarditis prophylaxis based on recommendations of the American Heart Association (2)

Indications
High risk for endocarditis: ▶ mechanical and bioprosthetic heart valves ▶ previous bacterial endocarditis ▶ cyanotic congenital heart disease
Antibiotic prophylaxis
▶ ampicillin 2 g i. v. and gentamycin 1.5 mg/kg i. v. (maximum 120 mg) before colonoscopy; ampicillin 1 g i. v. six hours after colonoscopy (alternative: amoxicillin 1 g orally six hours after colonoscopy) ▶ in case of penicillin allergy: vancomycin 1 g i. v. (over 1–2 hours) and gentamycin 1.5 mg/kg i. v. (max. 120 mg) by start of colonoscopy

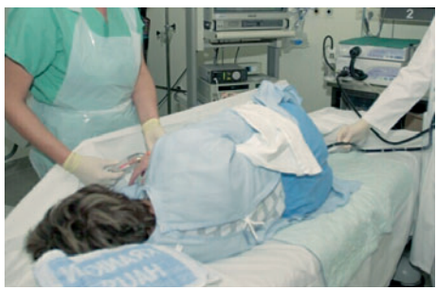
Patients with Pacemakers or Metal Implants

Pacemakers. Prior to examination, it must be known whether the patient has a pacemaker or acute pacemaker dependence. Especially if a high frequency electrical current is going to be used (e.g., for polypectomy), the pacemaker type must be known and it must have been checked no more than three months prior to examination. If a high frequency current is going to be used, ECG monitoring during colonoscopy is mandatory and the pacemaker must also be checked on the same day after intervention. When applying the neutral electrode, the distance between the pacemaker cover and the active electrode (= localization of the endoscope tip) must be > 15 cm and the distance between the active and neutral electrodes must be smaller than that between the pacemaker and the active electrode. Implanted defibrillators (ICD) must be deactivated while using a high frequency current and reactivated afterward (using a ring magnet). Otherwise, there is danger that the antitachycardia function could misinterpret the high frequency impulses and respond with inadequate shock. When inactivated, there is no protection against malignant arrhythmias; the examiner must be prepared to perform defibrillation.

Metal implants. The examiner must also be aware of the presence of any metal implants when applying the neutral electrode. Again, the distance between the neutral electrode and the active electrode (= location of the endoscope tip or polypectomy) must be smaller than that between the active electrode and any metal implant. Artificial hip joints are particularly relevant for colonoscopy.

Positioning the Patient

Prior to colonoscopy, the patient should be supine for examination of the abdomen. In addition to general clinical examination procedures, special attention must be paid to any surgical scars and hernias (inguinal, umbilical, or incisional hernias). At the beginning of the actual colonoscopy, the patient should be in the left lateral position, with his knees bent and pulled up. In this position, perianal inspection is possible, as is digital palpation and the insertion of the endoscope tip into the anus. Position may vary during the rest of the colonoscopy. Ileocolonoscopy can often be completed in the left lateral position without a problem (Fig. 4.**1 a**). In many patients, however, changing to the supine position after the colonoscopy has begun makes the rest of the procedure easier (Fig. 4.**1 b**). This position has the advantage of enabling external compression and "splinting" (especially for the sigmoid and transverse colon; see also Chapter 5) and it also makes localization of the endoscope easier. For "trouble spots," like passing the rectosigmoid junction or the hepatic flexure, as well as intubation of the ileocecal valve, repositioning the patient (from the left lateral position to supine or even the right lateral position) can also be helpful. The position for withdrawing the endoscope after reaching the terminal ileum depends on the examiner's preference, though most of the time it is done with the patient in the supine position (Fig. 4.**1 b**).



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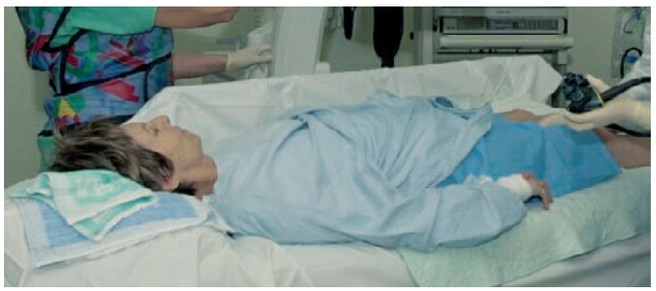


b

Fig. 4.**1** **Patient position during colonoscopy.**
a Beginning of colonoscopy: patient in left lateral position.
b After changing position: the patient is supine.

Safety of the Patient and Monitoring

Serious cardiopulmonary complications (occurring at a rate of 0.1–0.5 %) account for the overwhelming majority of complications related to endoscopy. Patients should be carefully monitored during and after colonoscopy, so that complications can be recognized and treated adequately, or, if possible prevented before they arise.

Risk assessment. Every individual patient's cardiopulmonary risk should be evaluated prior to examination. A useful reference is the classification of risk groups by the American Society of Anesthesiologists (ASA) in the ASA physical status classification system (Tab. 4.**2**) (5). Additional risk factors are cited by the German Society of Digestive and Metabolic Diseases (DGVS) (Tab. 4.**3**) (7).

Monitoring heart rate and blood pressure. The extent of monitoring required during intervention should be determined by individual risk constellation. In addition to clinical monitoring and observation of the patient, which is always foremost in importance, pulsoximetry, blood pressure monitoring, and continuous ECG monitoring can also be used. An i. v. should be available at the beginning of every colonoscopy, and blood pressure should be measured at least once. As is the case for all other endoscopic examinations, pulsoximetry for the duration of the colonoscopy is standard procedure.

ECG and oxygen saturation. ECG monitoring is recommended for at-risk patients with preexisting cardiac conditions and is mandatory for patients with pacemakers. Severe hypoxemia with a peripheral oxygen saturation below 90 % was found in 41–50 % of colonoscopies that did not use supplemental oxygen prophylactically; the main risk factor was low oxygen saturation prior to the examination (< 95 %). In one study, the rate of hypoxemia was reduced from 65 % to 17 % by prophylactically supplying 2 L of oxygen (3). Supplemental oxygen should always be administered for at-risk patients or in the case of a clinically relevant drop in oxygen saturation ($sO_2 < 90\,\%$).

Qualified assistants. A qualified assistant must be in charge of monitoring the patient, as the examiner must concentrate on performing the colonoscopy. The assistant must also be qualified to assist with CPR, if necessary. In addition to technical requirements for monitoring the patient, all endoscopy units must be equipped with emergency resuscitation, intubation, and artificial respiration equipment.

Endoscopy units also must have a recovery room where postoperative patients are monitored (especially following sedation). This room must be supervised by qualified nursing staff and can serve as a type of transition area between the examination room and in-patient area on the one hand and the discharge area of the clinic on the other.

Outpatient colonoscopy. The patient can only be discharged from outpatient colonoscopy once his vital signs have stabilized, when he can walk without assistance, and when he has minimal or no pain. The patient must be informed of the possibility of complications such as bleeding or perforation (especially after polypectomy) arising later and that he must return immediately if any of these occur (1). Patients in our outpatient endoscopy clinic receive a written information sheet (and are informed verbally during an outpatient discharge conversation). The final decision to discharge the patient is made by the examiner.

Table 4.**2** Physical status classification system of the American Society of Anesthesiologists (ASA) (5)

Class	Definition
ASA I	normal, healthy patient
ASA II	mild to moderate systemic disease without incapacitation
ASA III	severe systemic disease and limited activity
ASA IV	severe, life-threatening disease
ASA V	moribund patient, not expected to survive more than 24 hours with or without an operation

Table 4.**3** Risk groups according to the German Society of Digestive and Metabolic Diseases (DGVS) (7)

- ASA Class III–IV
- Heart insufficiency in NYHA stage III–IV
- Severe coronary heart disease
- Aortic stenosis, stage III–IV
- Severe lung disease ($pO_2 < 50$ mmHg, $pCO_2 > 50$ mmHg, or FEV1 < 1.0 L)
- Coagulation disorder (PTT < 50 %, thrombocytes < 50/nl)
- Patient's age > 70 years old

References

See Chapter 5.

5 Inserting the Endoscope and Advancing It in the Colon

A. Probst

Inspection and Palpation

Inspection. The examination begins with an inspection of the perianal region. The patient should be in the left lateral position with his knees bent and pulled up. A simple inspection can detect skin changes, scars, anal skin tags, hemorrhoids, anal fissures, anal venous thromboses, fistula, injuries, or prolapse (anal or rectal prolapse). Any findings must be noted later in the examination report. Figures 5.**1**, 5.**2** show examples of pathologies detected during inspection. The diagnostic report should include exact localization: for example, distance from the anus or a description of location as if the patient were in the dorsal recumbent position (at the 12-o'clock position ventral to the anus).

Palpation. Following inspection, a digital examination of the anal canal and distal rectum must be completed before the actual endoscopic examination begins. Attention should be paid to palpable endoluminal abnormalities (polyps, tumors, foreign objects) as well as to extraluminal appearances. Male patients can also undergo a prostate check. An assessment of sphincter tonus as well as any noticeable discomfort during the examination (inflammation, fissures) should be included in the palpation findings. Patients who have been prepared for routine endoscopy will have an empty rectal ampulla. Emergency patients are another matter, however. Especially in the event of acute gastrointestinal bleeding, in addition to endoluminal inspection, characterization of stool contents can provide important additional information (melena, fresh blood, coagulum, stool), helping to infer the source and intensity of bleeding and making the rest of the diagnostic procedure easier. Figure 5.**3** provides some examples of endoscopic pathological findings that can be detected during digital palpation.

Table 5.**1** provides a summary of possible findings from inspection and palpation prior to endoscopy.

Table 5.**1** Inspection and palpation prior to endoscopy

Inspection	Palpation
▶ Skin changes (eczema, ulcers, condyloma)?	▶ Intestinal contents (stool, blood, coagulum)?
▶ Signs of swelling (periproctic abscess)?	▶ Sphincter tonus?
▶ Injuries?	▶ Pain (inflammation, anal fissures)?
▶ Scars (surgical operations)?	▶ Endoluminal obstruction (polyps, tumors, hemorrhoids)?
▶ Anal skin tags?	▶ Impressions from an extraluminal aspect?
▶ Hemorrhoids?	▶ Prostate?
▶ Anal venous thrombosis?	▶ Stenosis (passage of finger or endoscope)?
▶ Anal fissure?	▶ Anastomosis?
▶ Fistula openings?	
▶ Anal prolapse/rectal prolapse?	

Passing the Anal Sphincter

After completing inspection and palpation and, if necessary, administering an analgesic, the actual endoscopic examination can begin. A local anaesthetic lubricating jelly, such as a lubricant containing Lidocain, should be applied liberally. The endoscope tip is then inserted in the rectum and guided digitally without visualization. The examiner should explain to the patient the steps being taken and inform the patient that he may experience the urge to evacuate his bowels. The endoscope tip is inserted in the direction indicated by preceding palpation; as a rough guide, the direction of the anal canal runs in a line between the anus and the navel. After "blindly" inserting the endoscope 4–5 cm,

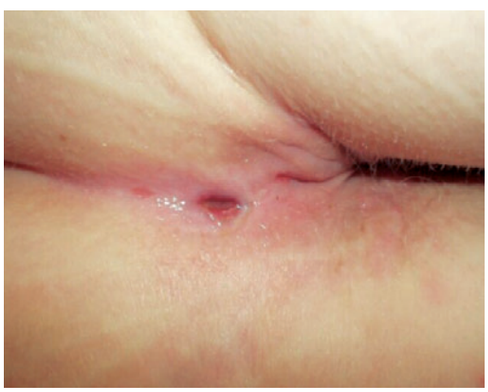
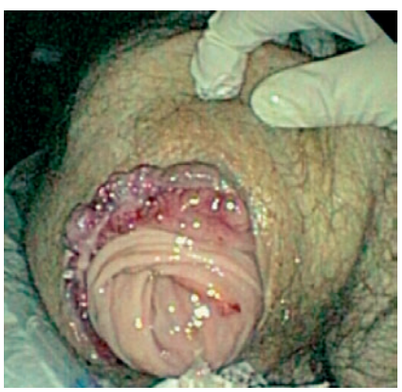


Figure 5.**1** **Examining the perianal region in the left lateral position:** patient with Crohn disease; reddened fistula opening at about the 6-o'clock position.



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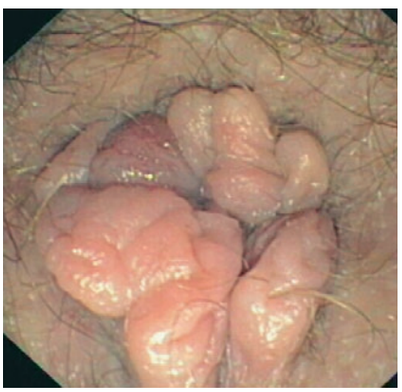

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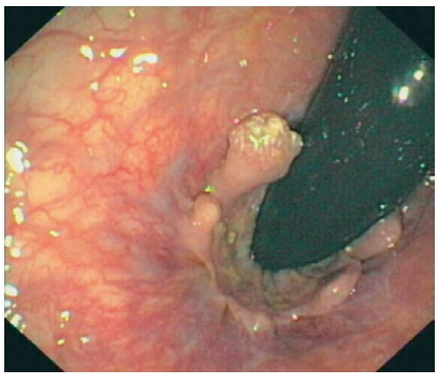
Fig. 5.**2** **Inspecting the perianal region.**
a Total rectal prolapse.
b Pronounced circular anal skin tags.



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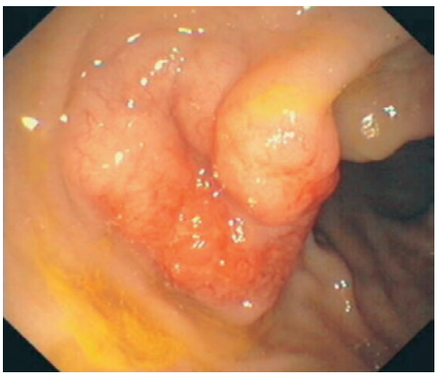

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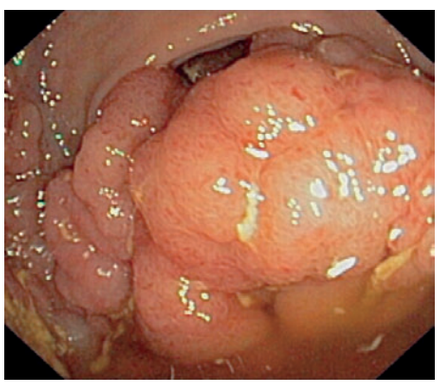

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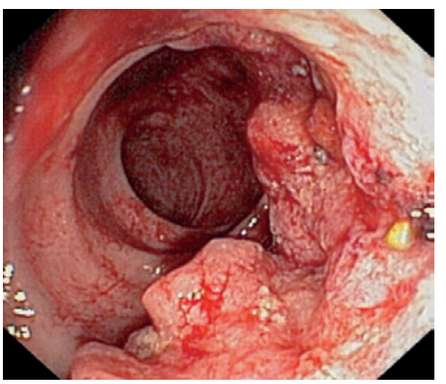


d

Fig. 5.3 **Endoluminally palpable obstructions.**

a Soft, stalklike obstructions with smooth surface in the anal canal (hypertrophied anal papilla on the dentate line, endoscope inverted in rectum).

b Sessile, submucosal obstruction with indentation in the center, 6 cm above the anus (histology: lymphoma).

c Large, endoluminal obstruction 5 cm above the anus (luminal obstruction due to polyp; histological adenoma with severe intraepithelial neoplasia).

d Hardened semicircular obstruction in distal rectum (broadbased growing carcinoma with spontaneous bleeding).

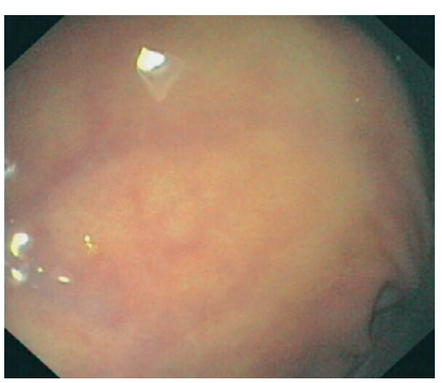


Fig. 5.4 **After "blindly" advancing the endoscope in the rectum, the instrument is withdrawn.** Direct view of the rectal wall. After withdrawal and air insufflation the lumen can be seen (lower right).

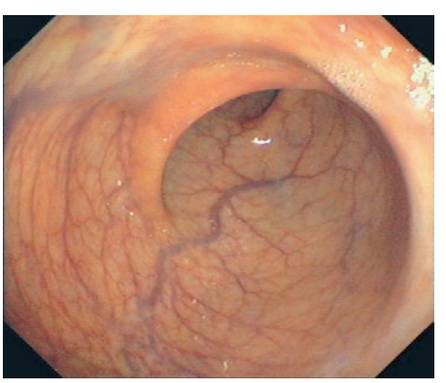


Fig. 5.5 **Centering the rectal lumen before continuing colonoscopy.**

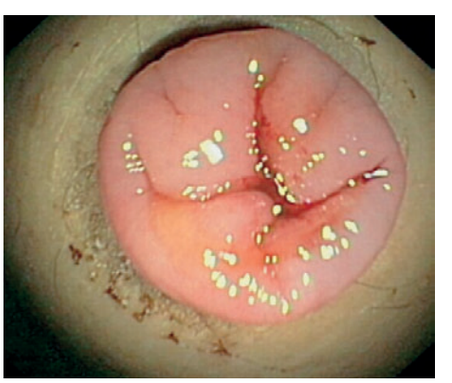


Fig. 5.6 **Colostomy** (appositional streaks of blood due to acute lower gastrointestinal bleeding).

air is insufflated and the endoscope tip is pulled back until the lumen of the distal rectum can be seen (Fig. 5.**4**). The rectal lumen is then centered in the middle of the monitor screen (Fig. 5.**5**) and the endoscope is advanced under visualization of the lumen to the rectosigmoid junction.

At this point in the examination there has not yet been sufficient inspection of the distal rectum or anal canal, which will be more closely examined on withdrawal of the endoscope later (possibly also using retroflexion of the endoscope in the rectum; see below).

Endoscope Insertion in Postoperative Patients (Colostomy/Ileostomy)

Examination options. If the patient has a colostomy/ileostomy as a result of an operation with lost intestinal continuity, endoscopy of the colon via the anus and ileum is often impossible and must be performed through the stoma (Fig. 5.**6**). Before the examination, the examiner should know the extent of the operation(s), the type of stoma, and its location. In the case of an end ileostomy, only the anastomosed small intestine can be examined endoscopically; the remaining colon, if there is any, is not reachable via the stoma. If the patient has a colostomy, the

proximal colon and terminal ileum can normally be examined without a problem. If the patient has a double-barreled ileostomy or a colostomy, the intestinal segments proximal and distal to the stoma can be examined (Fig. 5.**7**).

Inspection and palpation. Before the actual endoscopy, a thorough inspection of the area surrounding the stoma and digital palpation of the anastomosed intestinal segment should be performed. Special attention should be paid during inspection to signs of prolapse and mucosal abnormalities involving the visible intestinal mucosa as well as irregularities on the surrounding skin. Along with detecting endoluminal irregularities, the purpose of palpation is to ascertain the width of the lumen and the direction of the anastomosed intestinal segment. Knowing the width of the stoma and insertion direction is essential for inserting the endoscope and choice of instrument used is determined in part by the palpated diameter of the lumen.

Inserting and advancing the endoscope. Insertion of the instrument is eased by the examiner's finger and the use of air insufflation; it is inserted until the intestinal lumen comes into view. The lumen is then centered on the monitor screen before further advancing the endoscope. The rest of the examination ultimately depends on remaining intestine. Colonoscopy through the stoma can be made more difficult by loss of original intestine, increased postoperative mobility of the remaining intestine, or angulation as a result of postoperative adhesions.

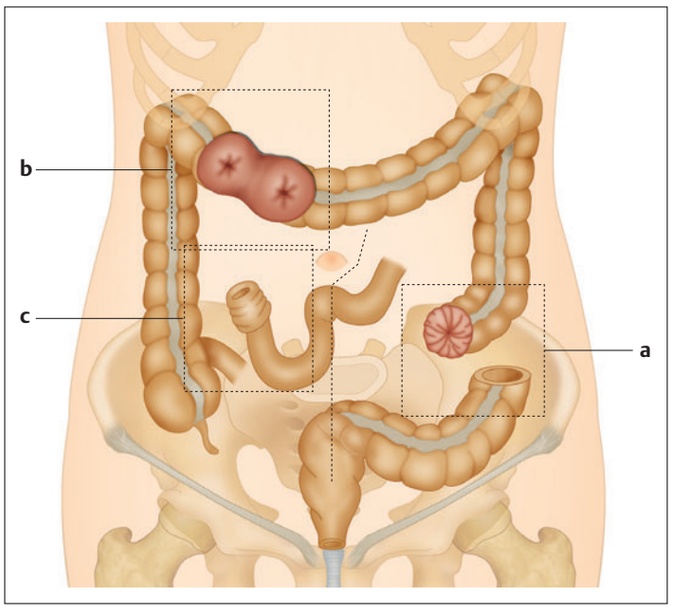


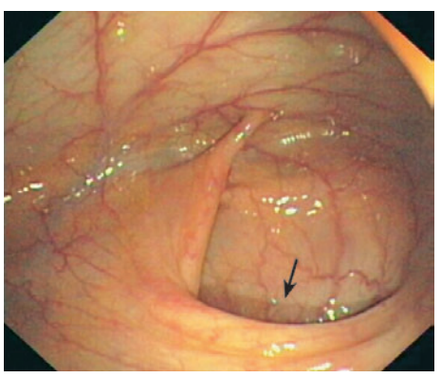
Fig. 5.**7** **Schematic illustration of various stomas.** a: end colostomy, b: double-barreled colostomy, c: end ileostomy.

Advancing the Endoscope in the Sigmoid Colon (Sigmoidoscopy)

Normal procedure. After reaching the rectosigmoid junction about ca. 16 cm proximal to the anocutaneous line, the endoscopy of the sigmoid colon begins. The sigmoid colon is situated intraperitoneally and is highly variable in length. The junction between rectum and sigmoid colon often appears as an acute bend in the lumen. The sigmoid colon can also be recognized by its prominent, circular folds. Passing the sigmoid colon with the patient lying in the left lateral position is unproblematic in simple cases where the sigmoid colon shortens itself, enabling easier passage through curves. Passing the sigmoid-descending junction is often more difficult in this position, especially for more slender patients, as the sigmoid colon is forced into the left abdomen, narrowing the angle of the junction with the descending colon. Changing position to the supine position—or, especially for slender patients, to the right lateral position—allows the sigmoid colon to fall more into the middle and right lower abdomen, thereby straightening the angle and making passage of the endoscope tip into the descending colon significantly easier.

Constant visualization of the lumen is desirable for passing the sigmoid colon. The instrument should be kept as straight as possible, without significant bowing or looping. However, individual differences in length and course of the sigmoid colon can make viewing the lumen more difficult and in some patients, looping cannot be avoided.

"Blind" advancement of the endoscope and changing patient position. If the view of the colonic lumen is obstructed or prevented by sharp angling, the examiner can attempt to ascertain luminal direction and briefly point the instrument tip without visualization in the presumed direction of the lumen, using gentle pressure to advance the endoscope in this direction. The presumed direction of the lumen is often indicated by shadowing (Fig. 5.**8**). Such maneuvers, which are performed only in exceptional cases, require experience, a light touch, and extreme concentration. The procedure must be stopped if macroscopic changes to the nearby mucosal surface (blanching, bloodless-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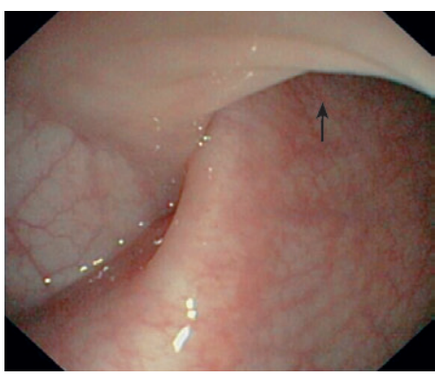

b

Fig. 5.**8 a**, **b** **Acute angling of the lumen** (example shown: sigmoid-descending junction). The direction of the lumen cannot be seen either at about the 7-o'clock position (**a**) or at the 12-o'clock position (**b**), but it can be presumed, in part due to shadowing (arrows). In exceptional situations, the endoscope tip can be very carefully advanced in the presumed direction without visualization.

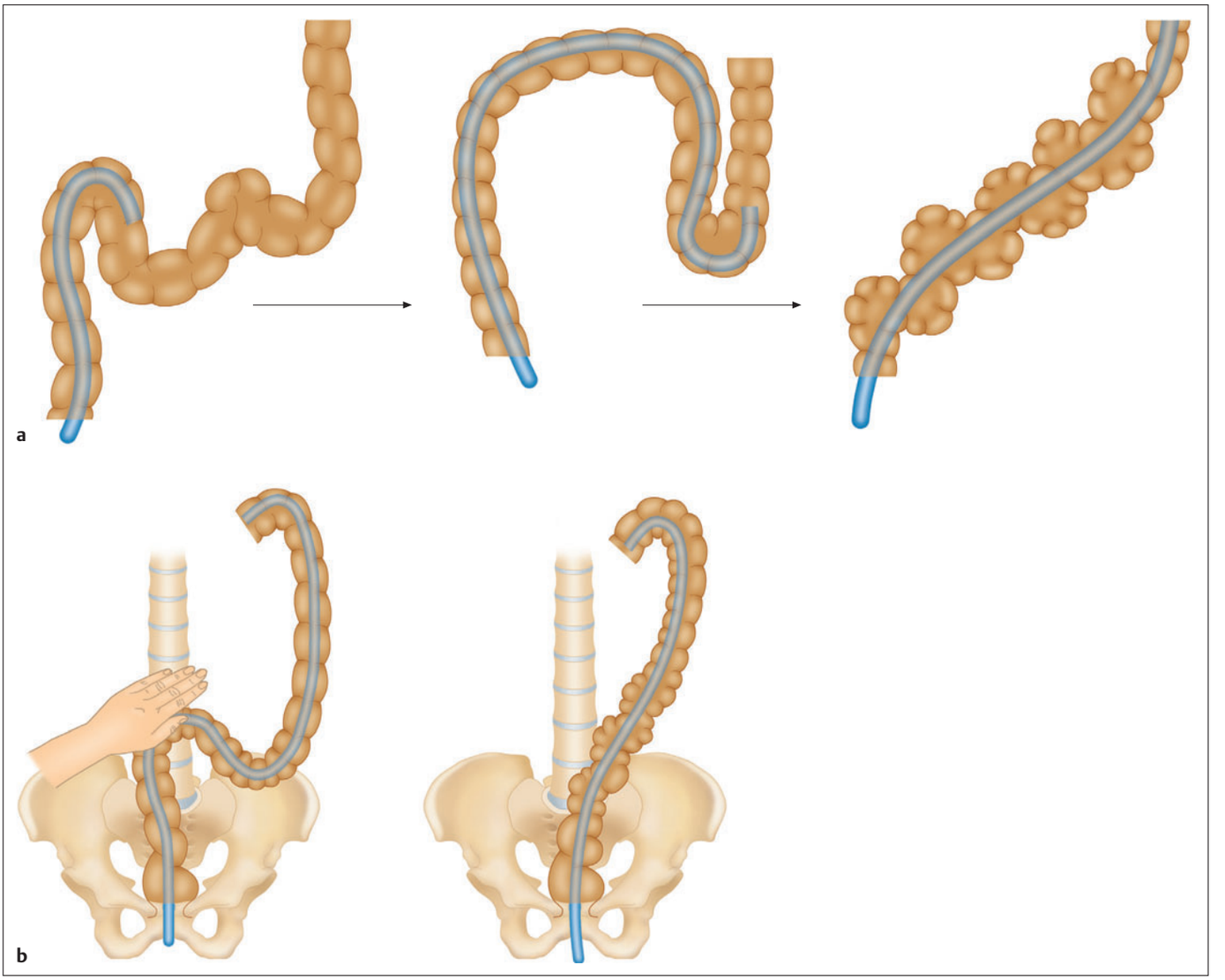


Fig. 5.**9** **Looping in the sigmoid colon.**
a Straightening the loop by withdrawing the instrument and desufflating air (or suctioning insufflated air).
b Straightening the loop using external hand pressure and withdrawing the instrument.

ness of mucosal vessels) are observed, or if there is increased resistance to advancement of the instrument and discomfort to the patient as these are signs of increased danger of perforation. Sharp kinks of the lumen can often be minimized or even eliminated by changing the position of the patient; the intraperitoneal location of the mobile sigmoid colon makes this easier. In addition to the supine position, the right lateral position can also be helpful in some situations. Changing the patient's position does not increase risk and thus must always be attempted first before resorting to "blind" advancement of the instrument.

Bowing and Looping. An additional problem in passing the flexible sigmoid colon is bowing and looping of the endoscope. Disparity between the amount of colonoscope introduced into the rectum and the amount of advancement of the tip in the lumen is a sign that a loop is forming. In extreme cases, the instrument tip no longer moves proximally in the colon when advanced or even moves "paradoxically" in the direction of the anus. Pronounced looping in the sigmoid colon can result in the entire instrument being "used up" before reaching the descending colon; it can also create discomfort for the patient and increase risk of perforation, and, ultimately, make it impossible to complete the colonoscopy.

To counteract looping, the examiner can withdraw the instrument prematurely, and, if necessary, repeatedly, to the beginning of the loop. This can straighten the already intubated colon segment and allow gradual advancement proximally. Suctioning air when withdrawing the instrument can also be helpful (Fig. 5.**9 a**).

If looping still cannot be entirely prevented or counteracted, and is impeding the continuation of the procedure, the use of external hand pressure can be helpful in fixing or "splinting" the sigmoid colon (Fig. 5.**9 b**; see below). Using external compression preventively can often counteract looping (prophylactically). The optimal localization for applying pressure

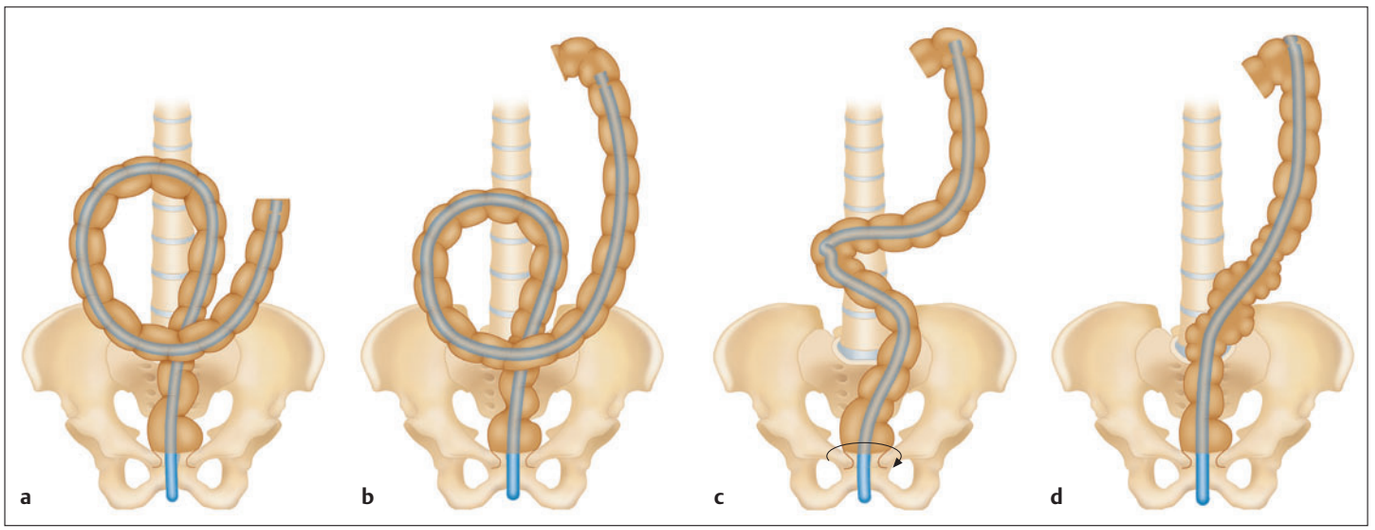


Fig. 5.**10** **Alpha loop technique.**
a Alpha loop.
b–d Straightening the loop by pulling the endoscope back and rotating the shaft clockwise.

can be found by palpation. In rare cases of pronounced or atypical looping, brief use of radiography may be necessary for orientation.

A further option for straightening the lumen and making it easier to pass the proximal sigmoid colon and the sigmoid-descending junction is the so-called alpha-loop maneuver. Rotating the endoscope 180° counterclockwise in the sigmoid colon creates a loop (similar in shape to the Greek letter alpha; Fig. 5.**10 a**) which makes further advancement easier. The loop can be straightened after reaching the descending colon or the splenic flexure (by rotating the colonoscope clockwise). The procedure is detailed schematically in Fig. 5.**10 b–d**.

Advancing Further to the Hepatic Flexure

Sigmoid-descending junction. After passing the sigmoid colon, the junction with the descending colon is reached. Sharp angling of the lumen, due to the "secondary retroperitoneal" position of the descending colon, can make it difficult to pass the sigmoid-descending junction. Unlike the flexible sigmoid colon, which is located intraperitoneally, the descending colon is fixed on the posterior abdominal wall. Looping or excess air insufflation in the sigmoid colon during preceding advancement of the endoscope can increase angling. Thus, after passing the sigmoid-descending junction, it is recommended that the sigmoid loops should be straightened by carefully withdrawing the instrument and suctioning excess air. This reduces the pull on the mesentery, which can cause discomfort to the patient, and also makes further advancement of the instrument easier.

In the case of a long and highly flexible sigmoid colon, applying external hand pressure or using the alpha maneuver can make entering the descending colon easier (Fig. 5.**10**). The actual beginning of the descending colon (corresponding to its distal endpoint) is usually evident when a longer intestinal section with a somewhat oval-shaped lumen and a relatively straight path becomes visible (Fig. 5.**11**). A visible fold in the lumen on the other side of this segment often indicates the splenic flexure.

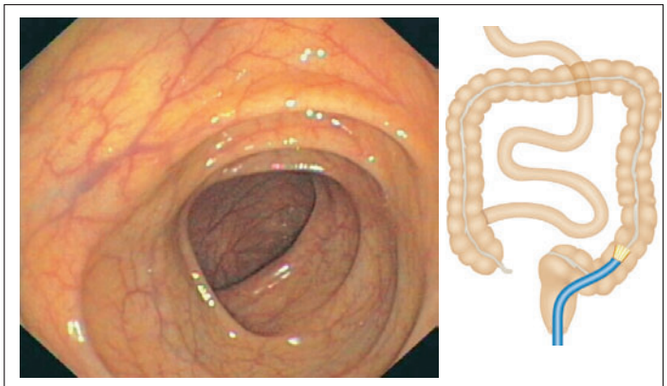


Fig. 5.**11** **View into the descending colon.** Relatively straight path, oval lumen, and evenly spaced haustrations.

Another—albeit less reliable—sign that the splenic flexure has been reached is the "bluish" coloration of the spleen visible through the colon wall (Fig. 5.**12**). After successfully passing the sigmoid-descending junction, advancing the endoscope in the descending colon is generally unproblematic. Nonetheless, splinting the sigmoid colon can still be helpful.

Splenic flexure. At the proximal end of the descending colon the splenic flexure is reached, marking the transition to the transverse colon. The transverse colon is located intrapertioneally, running across the upper abdomen to the hepatic flexure. The splenic flexure is highly variable with regard to position and degree of angling. A high flexure, which is located beneath the diaphragm, results in a larger angle (> 90°) between the descending colon and the transverse colon compared with a more caudal location (< 90°). In extreme cases, the flexure can be made of an ascending and a descending limb (Payr disease),

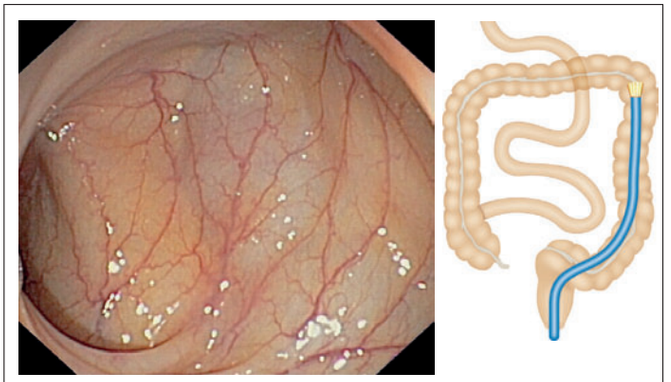


Fig. 5.**12** **Splenic flexure** with luminal impression, shimmering of spleen.

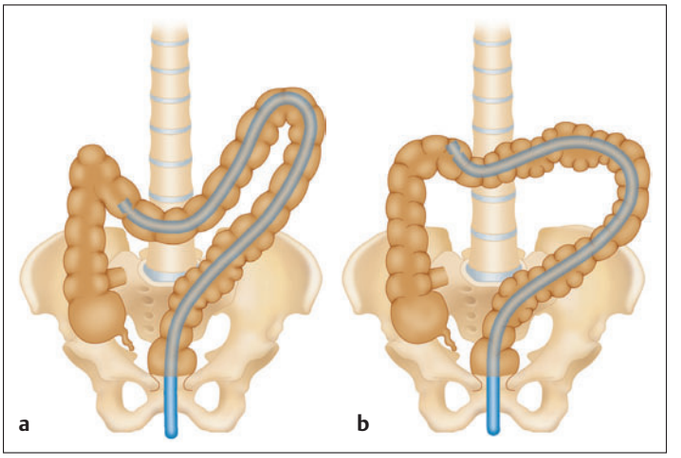


Fig. 5.**13** **Variations of the splenic flexure** with different angles between the descending colon and transverse colon.
a A "high" flexure, ca. 180°.
b "Drooping flexure."

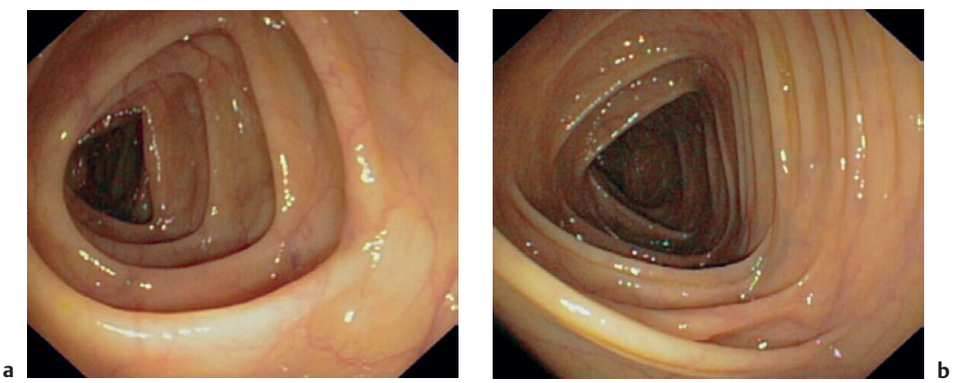


Fig. 5.**14 a**, **b** **View into the transverse colon** after passing the splenic flexure: typical triangular configuration and pronounced, evenly spaced haustration.

creating an angle of 180° (Fig. 5.**13**). Passage can be especially difficult if the splenic flexure is displaced vertically. In such cases, "pushing up" the endoscope in the more distal colon (especially the sigmoid colon) followed by withdrawing the instrument can advance the endoscope in the left side of the transverse colon. This is basically the same procedure as the alpha maneuver described above, though instead of forming a complete loop in the sigmoid colon, merely the beginning of a bend or an incomplete loop (combined with external pressure if necessary) is sufficient (cf. Fig. 5.**9 b**, Fig. 5.**10**).

Transverse colon. Recognizing that the transverse colon has been reached is usually simple, given its typical triangular-shaped lumen and strong, evenly spaced haustrations (Fig. 5.**14**). Compared with the relatively uniform, straight path of the descending colon, the position of the transverse colon is more variable due to its intraperitoneal position and fixation on a mesocolon, which may vary in length. The fixation on both retroperitoneal fixated colon flexures causes it to bend convexly and ventrally. The middle of the transverse colon, however, droops caudally. The path between splenic and hepatic flexures can vary greatly; at the one extreme, the transverse colon can be nearly horizontal, while at the other it can "droop" all the way down to the minor pelvis (Fig. 5.**15**). This results in any number of related difficulties in passage and therefore also advancing the endoscope in the hepatic flexure.

External pressure can lift a drooping midtransverse colon cranially and enable the advancement of the endoscope to continue (see below). It is also possible to push the endoscope "up" after reaching the most caudal point in the drooping transverse colon. If the instrument is then carefully withdrawn, a cranial displacement of the midtransverse colon and corresponding straightening of the transverse colon can ease passage and retrieve "used-up" endoscope length. Passage of the transverse

colon and reaching the hepatic flexure is sometimes only possible using a combination of advancing/withdrawing and external hand pressure. Optimal cooperation between examiner and assistant is essential. Pronounced angling of the lumen toward the ascending colon is a sign that the endoscope is reaching the hepatic flexure (Fig. 5.**16**).

Hepatic flexure. The fixation of the hepatic flexure and the ascending colon to the posterior abdominal wall combined with the mobility of the intraperitoneally located transverse colon can result in sharp angling at the hepatic flexure. The situation is similar to the transition described above from the intraperitoneally situated sigmoid colon to the retroperitoneally fixated descending colon; the difficulties passing the hepatic flexure are analogous. If at this point the patient is still in the left lateral position, it is strongly recommended that he should change position if problems passing the hepatic flexure are encountered; the patient should be supine or even in the right lateral position. In some cases, simply changing the position of the patient results in visualization of the previously displaced lumen of the ascending colon and can enable the examiner to overcome the flexure without a problem. If passage continues to be difficult, it is often necessary to push the endoscope up until the instrument tip is placed where the ascending colon begins. This part of colonoscopy often causes discomfort to the patient. As soon as the instrument tip is positioned in the ascending colon, it should be straightened by pulling back. This assists further advancement considerably and often the endoscope tip moves further toward the cecum as a result.

Applying external pressure can also be a significant help with the hepatic flexure. Splinting the sigmoid colon, a drooping transverse colon, or both can help straighten the endoscope, preventing repeated looping which "uses up" endoscope length while helping to reach the ascending colon successfully. If this does not work, additional external hand pressure on the right flank with the flat of the hand placed dorsally or slanted laterally to apply pressure directly to the flexure can be very helpful (see below).

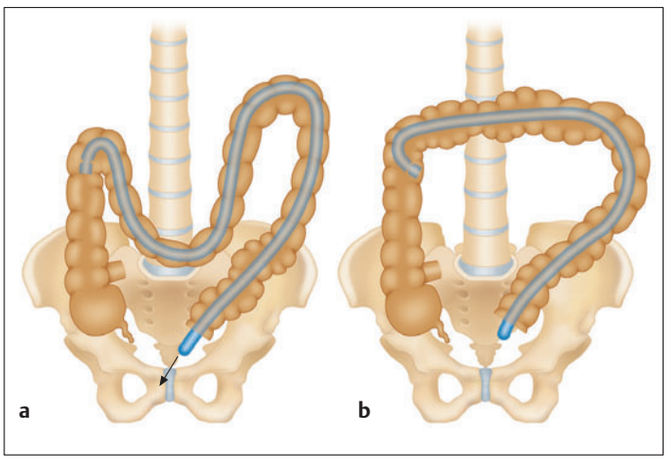


Fig. 5.**15** **Schematic illustration of various paths of the transverse colon.**
a "Drooping" transverse colon.
b Nearly horizontal transverse colon.

Proximal Colon

After passing the hepatic flexure, the view opens up to the proximal segments of the large intestine. In addition to the capacious ascending colon, the cecal pole and ileocecal valve are often visible at the end of the field of vision (Fig. 5.**17**). Often after passing the hepatic flexure, there can be a certain unnecessary advancement of the endoscope in the more distal colon segments. Thus, after positioning the endoscope tip securely in the ascending colon, it is recommended that the endoscope be carefully withdrawn and straightened. This alone can often further advance the instrument, in some cases even reaching the base of the cecum. If this does not succeed, active advancement of the endoscope is necessary. For passing the ascending colon,

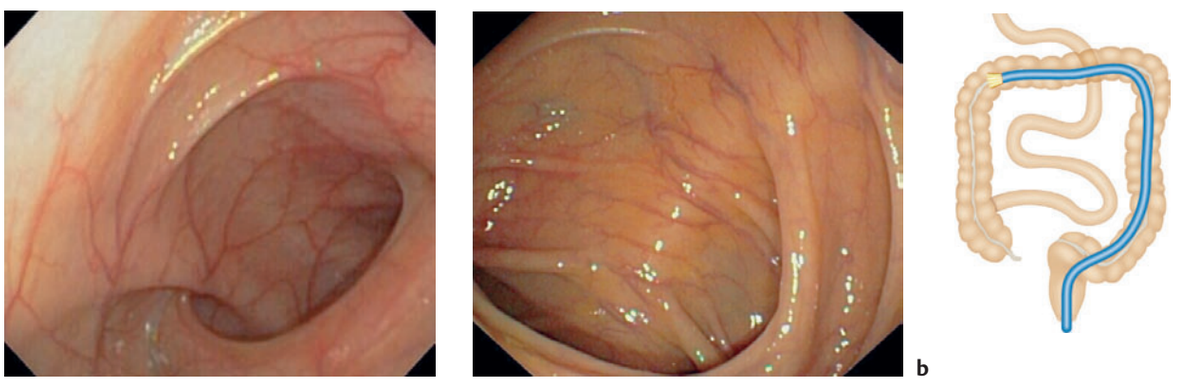


Fig. 5.**16** **Hepatic flexure from an endoscopic aspect.**
a Ascending colon toward the 5-o'clock position. **b** Ascending colon toward the 7-o'clock position.

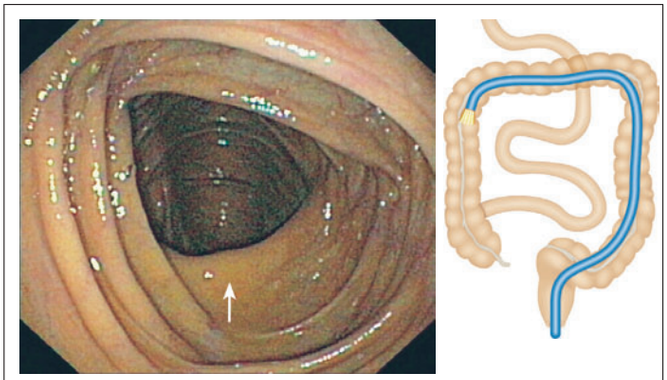


Fig. 5.**17** **View into the ascending colon** after passing the hepatic flexure. The ileocecal valve, seen as a yellowish, thickened fold, is on the lower edge of the lumen (arrow) in the distance.

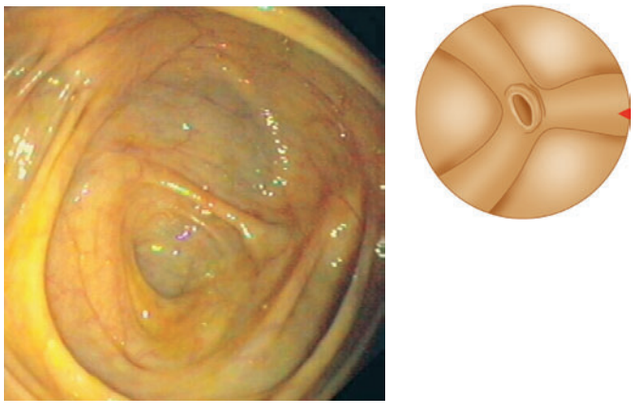


Fig. 5.**18** **View of the base of the cecum.** Convergence of the three tenia originating at about the 3-o'clock, 11-o'clock, and 6-o'clock positions; appendix orifice in the center of the image.

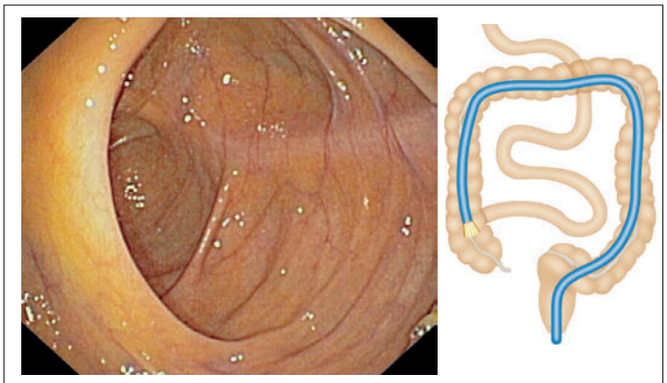


Fig. 5.**19** **View of the ileocecal valve** (thickened, yellowish semicircular fold on the left) and tilted cecum, obscured view into the cecum from this aspect.

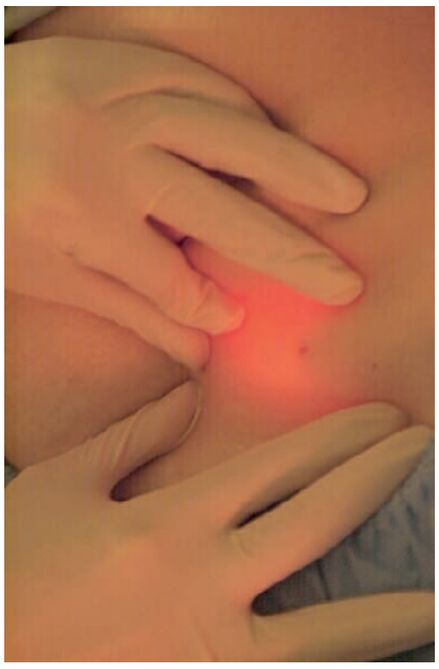
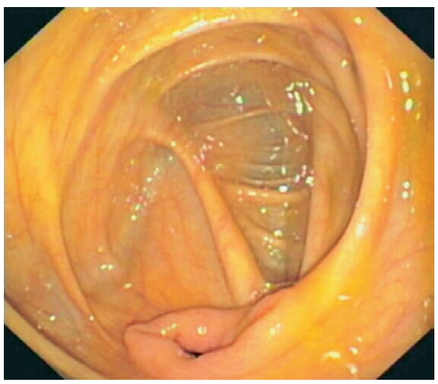


Fig. 5.**20** **Transillumination of the endoscope tip** through the abdominal wall of the lower right abdomen upon reaching the cecum.

advancing and withdrawing to straighten it, applying external hand pressure to prevent inefficient loss of instrument length in the flexible, more distal colon segments, or a combination of both procedures can be helpful. In some cases, the base of the cecum can ultimately be reached only by additionally repositioning the patient; especially for advancement in the right hemicolon, positioning the patient on his right side can make advancement easier.

Base of the cecum. Identifying the base of the cecum is usually unproblematic, given its characteristic morphology and the proximity of the ileocecal valve. The base of the cecum is characterized by a folded pattern produced by the three converging taenia; the appendix valve or invaginated appendiceal orifice is located at its center (Fig. 5.**18**). The ileocecal valve is located a few centimeters distally, and usually appears as a yellowish, thickened fold, separating the cecum from the ascending colon (Fig. 5.**19**). Position and flexibility of the cecum vary depending on its fixation to the dorsal abdominal wall. A broadly fixated cecum on the posterior wall of the abdominal cavity (as a continuation of the fixation of the ascending colon) results in a mostly immobile cecum. The range of normal anatomy encompasses all possible variations, including the complete lack of such a fixation, resulting in extreme cases in a highly mobile cecum and a possible inversion of the cecal pole or only the appendix. This explains why the base of the cecum can in some cases be completely visualized from the proximal ascending colon, but not in the case of an inverted or tilted cecum, which requires precise advancement in the cecum (Fig. 5.**19**). It is a good idea to document the images of the cecum (with or without the ileocecal valve) as a record of completion of colonoscopy. Along with the typical endoluminal morphology, reaching the cecum can often also be confirmed by visible transillumination of the endoscope tip in the lower right abdomen (Fig. 5.**20**).



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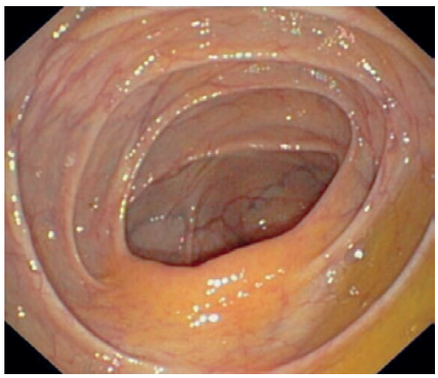
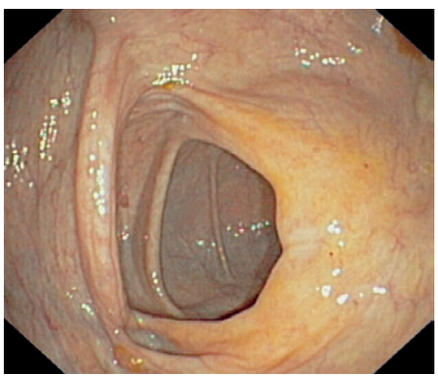


b

Fig. 5.**21** **Ileocecal valve.**
a The valve opening is easily identified from the ascending colon.
b The valve is turned toward the cecal pole and not identifiable from the ascending colon. The indentation in the center of the valve could indicate the location of the valve opening.



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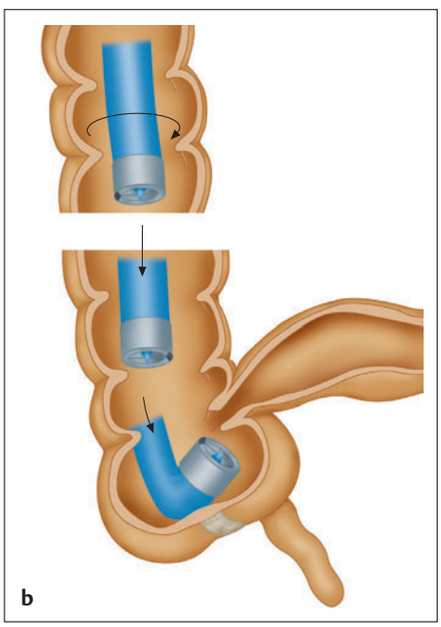

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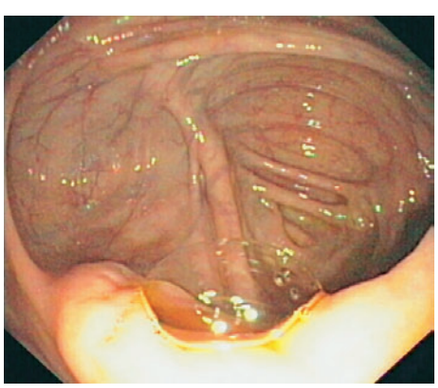
Fig. 5.**22** **Ileocecal valve.**
a The valve opening is presumably located at the indentation on the valve.
b The endoscope tip must be almost completely inverted, as illustrated here.



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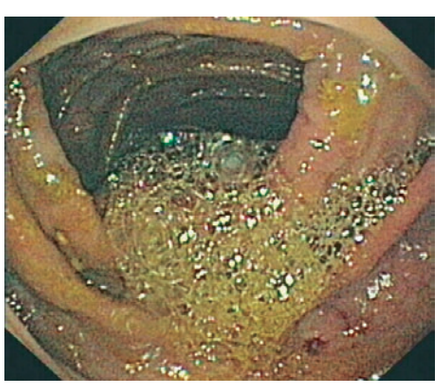


b

Fig. 5.**23 a, b** **Ileocecal valve.** Secretion of fluid from the ileum (single or multiple air bubbles) can help localize the valve opening.

Intubating the Ileocecal Valve and Terminal Ileum

The ileocecal valve is located above the base of the cecum and is usually easily seen from the proximal ascending colon, though its morphology and orientation can vary greatly. In some instances the valve opening can be clearly identified from the ascending colon, but a protruding superior valve lip and an inverted cecum can make the identification of the valve opening from the ascending colon impossible. In such cases, the ileocecal valve can often only be identified as a yellowish, thickened and slightly raised fold in the lumen (Figs. 5.**21**–5.**23**).

Depending on the individual morphology of the valve, the position of the valve opening will also vary. This can present significant difficulties for intubation. Nevertheless, valve intubation and inspection of the terminal ileum should be attempted. Intubation of the valve and inspection of the terminal ileum is an important part of the endoscopic examination for

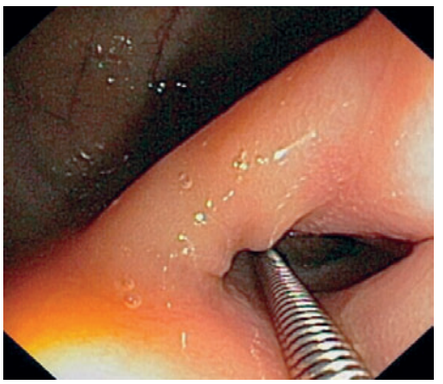


Fig. 5.**24** **Intubation of the ileocecal valve.** The biopsy forceps inserted in the valve help guide the endoscope, making valve intubation easier.

evaluating certain conditions (e.g., suspected Crohn disease, sonomorphological changes to the ileum). In emergencies involving bleeding in the lower gastrointestinal tract, inspection of the ileum helps localize the source of bleeding and often provides important information for the differentiation of ileal or colonic bleeding.

Valve intubation technique. Valve intubation technique depends on the morphology of the valve. Before intubation, the position of the valve opening must first be determined. If the valve is clearly visible, the endoscope can generally be advanced from the ascending colon into the ileum without a problem. If the valve opening is not readily identifiable, a careful inspection of the valve should first be made. Signs of the valve opening include secretion of small intestine contents (often foamy, bubbling; Fig. 5.**23**) or a visible indentation on the valve (Figs. 5.**21 b**, 5.**22**). Suctioning air out of the cecum can sometimes turn the valve opening around away from the base of the cecum and toward the ascending colon, making it visible. However, in some cases the valve opening remains difficult to identify and can only be seen from the base of the cecum. If this is the case, a complete retroflexion of the endoscope in the cecum may be required (Fig. 5.**22**).

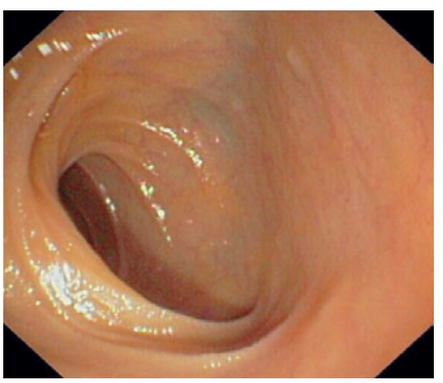
After identifying a valve opening not passable from distal, the endoscope tip is placed in the cecum and slowly and carefully withdrawn in the direction of the already identified valve opening. As soon as the valve opening can be seen, the instrument tip can be advanced again toward the terminal ileum using air insufflation (in doses). Several attempts are often necessary in difficult situations. If intubation of the valve is still not possible, in exceptional situations a closed biopsy forceps can be used to guide the endoscope through the clearly identified opening in the direction of the ileum (Fig. 5.**24**). The morphology of the small intestine mucosa makes the terminal ileum immediately recognizable. Compared with the smooth and shiny mucosa of the large intestine, the ileum mucosa has a velvety surface. Occasionally, the villi of the small intestine can be seen macroscopically; there is no haustration of the lumen. The ileum should normally be inspected until the instrument is used up, which can require > 20 cm (Fig. 5.**25**). In a small number of cases (< 5 %) intubation of the ileocecal valve remains impossible despite every possible attempt.

▣ 5.**1** illustrates the most important stages in examination procedure.

Looping and Using External Compression Techniques

Using external compression or splinting techniques can often make the examination much easier (for both the patient and examiner) and in some cases, it may be the only means of completing ileocolonoscopy. Proper use of compression technique requires close cooperation between the examiner and assistant; compression should be performed with targeted, steady pressure without using force. The localization of optimal pressure points is for the most part based on the experience of the examiner and assistant. In some cases, palpation can be used to locate the position of the endoscope or colon. With slender patients in particular, position of the instrument in the colon and incorrect advancement direction can be palpated through the abdominal wall. The use of radiographic screening or other methods (see below) can help determine position and in extreme cases may be necessary for ascertaining the exact position of the endoscope. External hand pressure can be used, in particular, for flexible colon segments, such as the sigmoid colon, transverse colon, and cecum, which are located intraperitoneally and attached to the posterior abdominal wall by a mesocolon. Retroperitoneally fixed colon segments, such as the ascending colon and descending colon, generally do not require splinting.

A number of variations are possible for using splinting to counteract undesirable bowing or looping and to help straighten the colon. However, for certain difficulties occuring frequently, there are standard techniques that will be described below.



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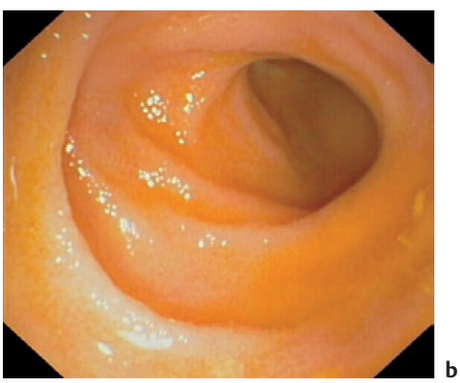

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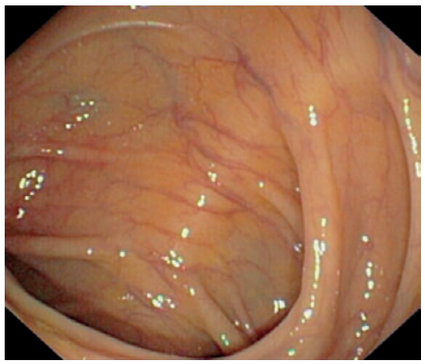
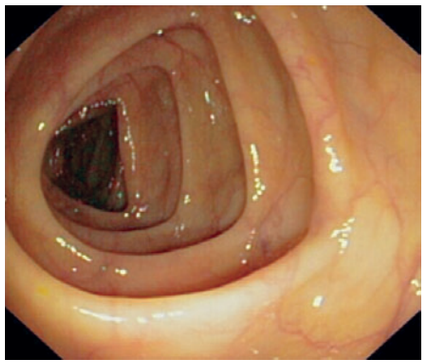
Fig. 5.**25 a**, **b Terminal ileum:** velvety mucosal surface and lacking haustrations.

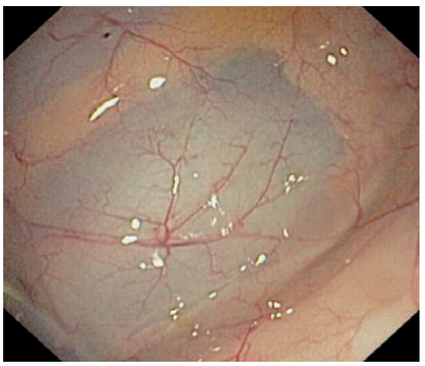
5.1 Illustrated summary of normal examination procedure



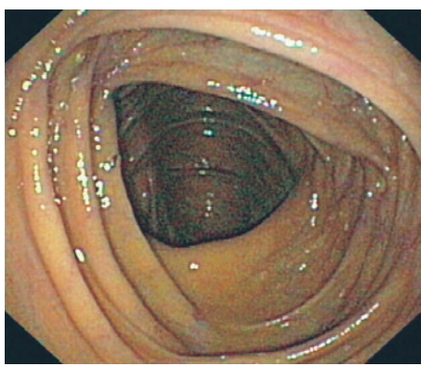
Hepatic flexure



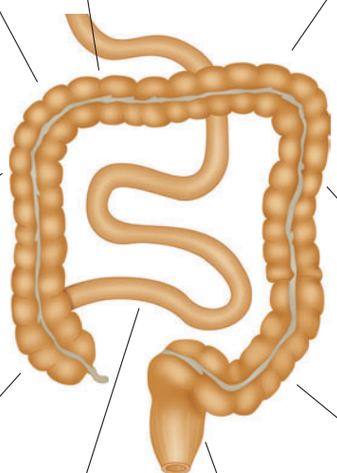
Transverse colon

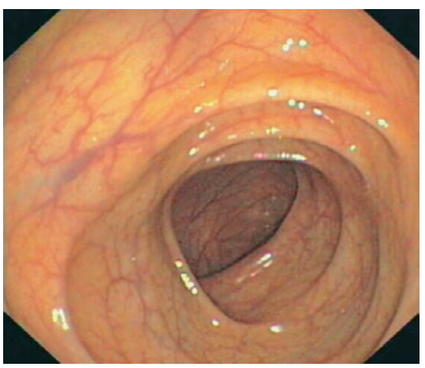


Splenic flexure

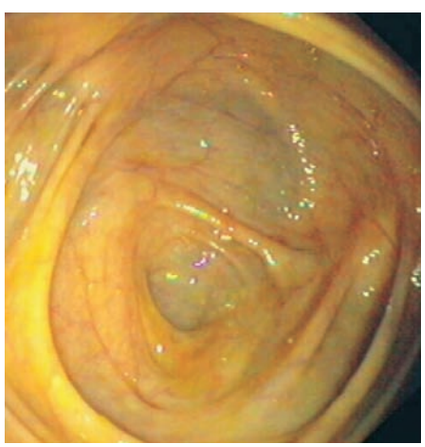


Ascending colon

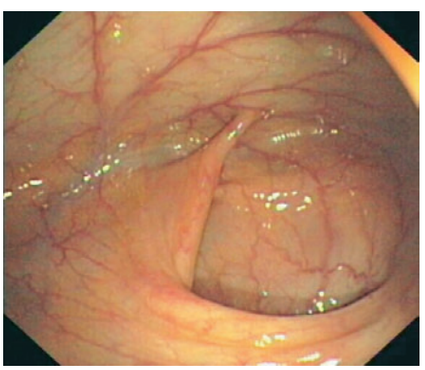




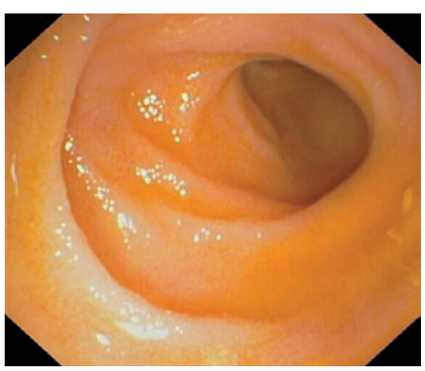
Descending colon



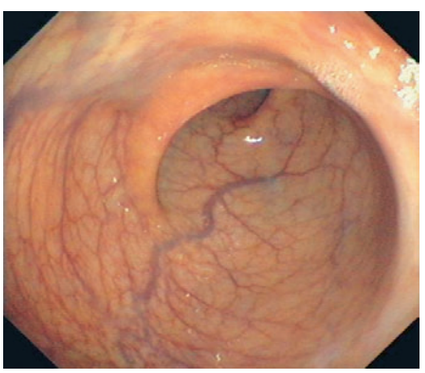
Cecum



Sigmoid colon



Ileum



Rectum

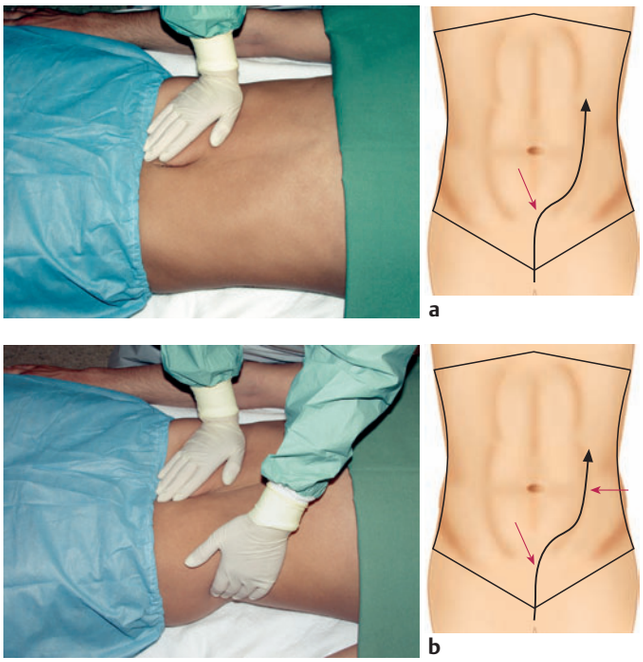


Fig. 5.**26** **Applying pressure over the sigmoid colon.**
a Simple pressure over the sigmoid colon in the supine position.
b Combined pressure over the sigmoid colon in supine position.

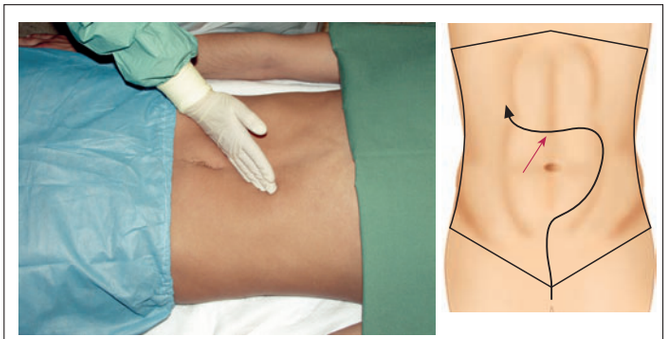


Fig. 5.**27** **Applying pressure over the transverse colon.**

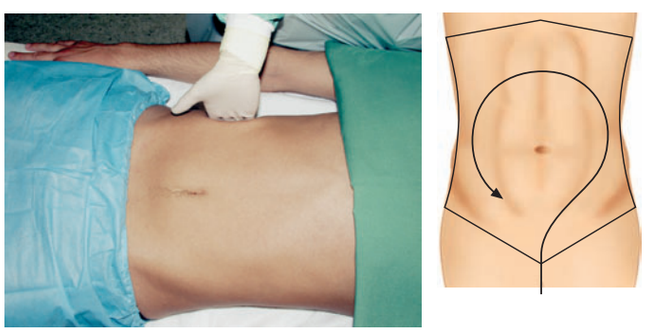


Fig. 5.**28** **Applying pressure on the right flank** for passing the hepatic flexure.

Applying Simple Pressure over the Sigmoid Colon in the Left Lateral Position

At the beginning of the colonoscopy the patient is lying in the left lateral position. After reaching the rectosigmoid junction pressure can be applied to the lower left abdomen to splint the sigmoid colon and avoid the formation of a loop. Changing position is not necessary.

Simple and Combined Pressure with Patient in the Supine Position

If applying simple pressure in the left lateral position is difficult (e.g., for adipose patients) or if pressure applied in this position is unsuccessful, it is recommended that the patient should change to the supine position. In this position, pressure on the midabdominal region can fix the flexible sigmoid colon in the lower left abdomen and counteract looping (Fig. 5.**26 a**). The effect is enhanced by additional use of the other hand on the patient's left lateral side, preventing the movement of the sigmoid colon this direction (Fig. 5.**26 b**).

Transverse Pressure

The transverse colon, which can vary in the extent to which it droops caudally, in extreme cases hanging down even into the minor pelvis, can pose difficulties for advancing the colonoscope. Along with the flexible sigmoid colon, it can be a further indication for using external hand pressure. Lifting the drooping midtransverse colon segment cranially can straighten the transverse colon and simplify advancement to the hepatic flexure (Fig. 5.**27**).

Pressure on the Right Flank

Applying external pressure to the right flank over the hepatic flexure can be helpful for passing the flexure. This can make turning the endoscope tip around easier as it comes out of the transverse colon and advances caudally in the direction of the colon (Fig. 5.**28**).

Pressure over the Cecum

In some cases, external pressure applied over the cecum can be helpful for complete inspection of the cecum (especially for a mobile cecum) and for intubation of the ileocecal valve (Fig. 5.**29**).

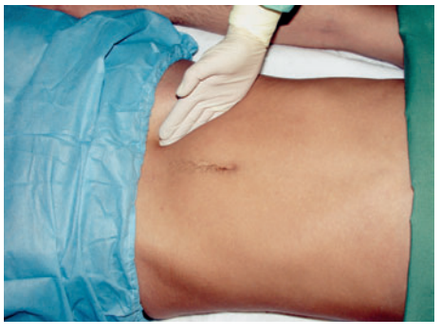
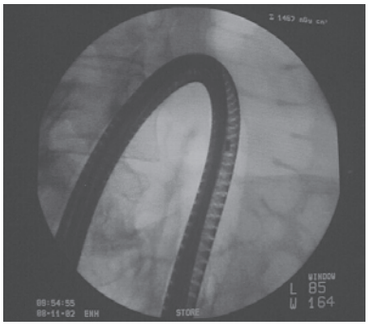


Fig. 5.**29** **Applying pressure over the cecum.**



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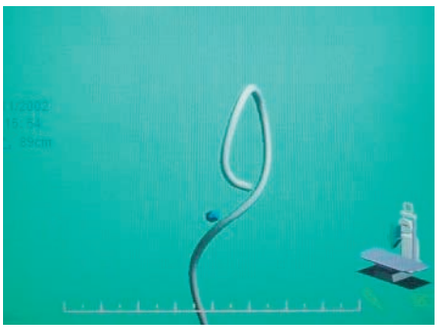

b

Fig. 5.**30** **Determining endoscope position.**
a Using radiography screening for looping in the sigmoid colon (partial image).
b Using Scope Guide (Olympus) for computerized three-dimensional positioning (same situation as in **a)**.

Localizing the Endoscope

Unlike endoscopy of the upper gastrointestinal tract, determining the position of the instrument during colonoscopy is not always easy. Identifying the rectum and rectosigmoid junction at the beginning of the procedure is unproblematic. Also, certain anatomical structures, such as the base of the cecum with the appendiceal orifice, ileocecal valve, and terminal ileum can be easily identified based on their morphology. Further indicators that can assist in localization include the configuration of the colon lumen, possible shimmering of parenchymatous organs through the colonic wall (the liver at the hepatic flexure and the spleen at the splenic flexure), and transillumination that sometimes occurs through the abdominal wall. However, given the variability of the colon in terms of length, path, and looping, it is often difficult to be entirely correct when determining the instrument's position based on the parameters mentioned and the length of endoscope introduced into the rectum. Nonetheless, knowing the exact position of the instrument, including the shape and extent of possible looping, is often vital for optimizing advancement of the instrument and for using external hand pressure. In addition, knowing the current position of the instrument is absolutely essential for exact localization of pathologies. This is vital for operation planning for removal of polyps or malignant tissue which cannot be removed endoscopically and for documenting original localization prior to endoscopic check ups (e.g., following polypectomy, mucosectomy, or therapeutic endoscopic measures to stop gastrointestinal bleeding).

Radiographic screening. A classic diagnostic procedure, using radiography is a highly reliable method of ascertaining the path and position of the endoscope in the colon. However, the drawback of roentgen ray exposure is obvious. The rate of radiographic screening for all colonoscopy procedures is reported in the literature as less than 10%; in our experience, the rate is probably lower than 1%.

Other imaging techniques. Three-dimensional computerized scope positioning, which uses magnetic impulses generated by the endoscope in the colon (e.g., Scope Guide, Olympus Optical Co., Ltd.), is one alternative to radiographic screening. The images are highly correlated with corresponding radiographs and correctly determine instrument position. Three-dimensional positioning avoids roentgen ray exposure and it also has the distinct advantage of continually recording instrument position during advancement, giving the examiner the option of responding quickly to looping by pulling back the endoscope or applying external hand pressure. Our own experience with the Scope Guide system (Olympus) has confirmed preliminary research findings (11) (Figs. 5.**30**, 5.**31**).

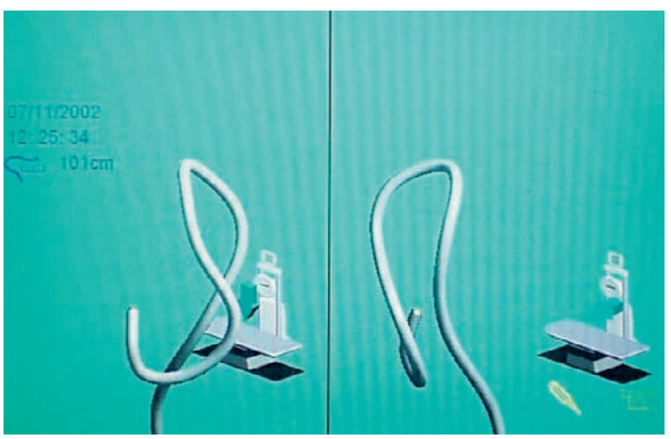

Fig. 5.**31** **Three-dimensional computerized scope positioning** (Scope Guide, Olympus): colonoscope position, split-screen display of different views.

Withdrawing the Endoscope and "Blind Spots"

Compared with advancing the scope, withdrawal after reaching the terminal ileum does not pose any technical difficulties. Close inspection of the colon is made on withdrawal, as is the collection of pathological samples and the performance of any necessary diagnostic or therapeutic interventions (biopsy, polypectomy, etc.). Slowly withdrawing the instrument and per-

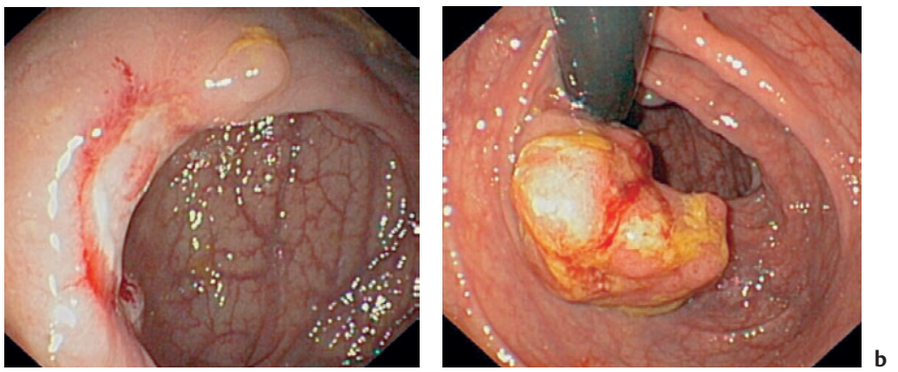

Fig. 5.**32** **Polyp in the proximal rectum. a** After endoscopic resection, a small remaining polyp piece was visible from distal next to the ulcerated resection site. Another polyp piece is likely on the other side of the fold at the lower edge of the resection site. **b** The whole polyp could only be seen by viewing (retroflexion) behind the fold. Retroflexion was performed using a gastroscope, which offers more flexibility.

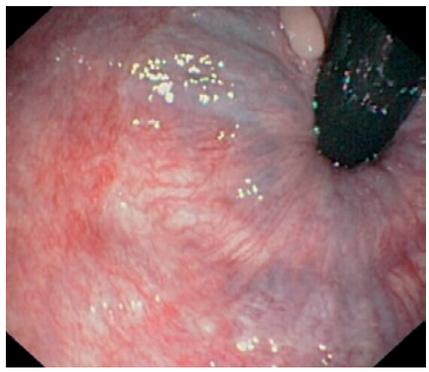

Fig. 5.**33** **Retroflexion in the rectum:** at the top right, the endoscope shaft can be seen as it comes through the anal canal. The squamous epithelium lining the anal canal can be seen circumferentially around the endoscope; the transition to the columnar epithelium of the rectal mucosa is clearly visible. A secondary finding is a small hypertrophied anal papilla directly next to the endoscope (at about the 12-o'clock position).

forming a circumferential inspection of the intestinal wall in all colon segments avoids missing smaller pathologies. While the inspection of surfaces of the folds turned toward the anus is usually unproblematic, proximal surfaces (toward the cecum) can be difficult to examine. Sufficient examination of these areas requires slow withdrawal of the endoscope and the use of adequate air insufflation. Figure 5.**32** shows a large piece of a polyp behind a fold in the proximal rectum. Despite its size, it could only be detected by slow withdrawal and careful inspection.

"Blind spots." In addition to mucosal areas throughout the colon proximal to the folds, there are also specific colon segments that should receive special attention because of their anatomical configuration. Thorough inspection of these so-called blind spots can sometimes be difficult, but is nevertheless absolutely vital. The area in the cecum underneath the ileocecal valve and the inner sides of both colon flexures can be obscured or difficult to view (see also Figs. 5.**16**, 5.**19**). Repeatedly passing the colonoscope may be necessary especially for sufficient circumferential assessment of the flexures. Additionally, administering an antispasmodic drug (e.g., Buscopan) can be helpful. Similar problems can occur in sharp curves of the winding sigmoid colon and in the rectosigmoid junction (see Fig. 5.**8**). A study by Rex et al. illustrates the problem: when colonoscopy was repeated the same day, adenomas were detected in 24% of patients where they had previously been "missed." While the majority of these were small adenomas (< 6 mm), 6% were larger than 1 cm (6). The inspection of each colon segment can only be completed when the examiner is certain that all mucosal sections have been sufficiently viewed. Withdrawal of the endoscope is the simpler part of the examination, doing so slowly and carefully is the deciding factor in the examination, diagnosis, and the overall quality of the ileocolonoscopy.

Retroflexion in the Rectum

As is the case for all other colon segments, inspection of the rectum is performed upon withdrawing the endoscope. Given the tangential view when withdrawing the instrument, the immediate supra-anal area of the distal rectum, in particular, can be insufficiently inspected if the instrument is withdrawn too rapidly. A further problem can be presented by lumen folds that do not flatten, especially if the patient cannot hold insufflated air (e.g., weak sphincter tonus). If complete inspection of the distal rectum remains doubtful, despite slowly withdrawing the instrument, sufficient insufflation, and flattening of the lumen, the examiner should perform a retroflexion of the endoscope in the rectum. After placing the endoscope tip in the proximal rectum, the tip will be deflected 180°. Visualization of the mucosa is usually lost as soon as the endoscope tip is deflected. The deflected endoscope is therefore advanced carefully with air insufflation until the distal rectum and endoscope shaft come into view. Extreme care should be used when performing this maneuver and it should be stopped if there are any signs of resistance or discomfort to the patient. Successful retroflexion can provide a direct view of the anorectal area and the dentate line. Rotating the inverted endoscope shaft can enable inspection of the entire circumference of the anorectal region (Figs. 5.**3**, 5.**33**). Retroflexion in the rectum is helpful not only for examination, but also for therapeutic measures in the distal rectum (e.g., hemostasis). In isolated cases, the anorectal region cannot be examined sufficiently using retroflexion. In such patients, a proctoscope should be used, allowing sufficient inspection of the anorectal region and the anal canal.

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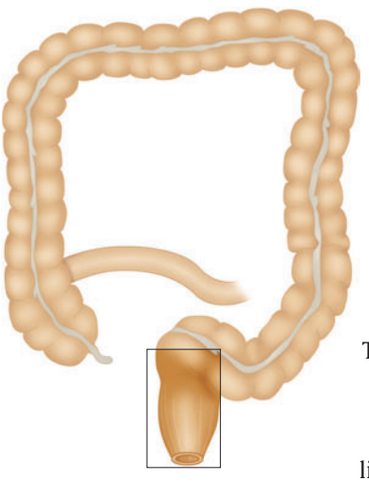
6 Normal Appearance of the Intestinal Segments

M. Bittinger

Normal Rectum



The rectum is the most distal colon segment, extending from the anal canal to the rectosigmoid junction. The dentate line marks the boundary between the anal canal and the rectum, and is characterized by a mixture of rectal mucosa (columnar epithelium) and sensitive anal skin (squamous epithelium). The fingerlike proximal ends of the squamous epithelium are somewhat thickened, forming the anal papilla. At the pouch-like distal ends of the columnar epithelium, also fingerlike in form, are the anal crypts, at the base of which are the anal glands. There is no distinct anatomical demarcation between the rectum and the sigmoid colon. As a rule, the boundary between the rectum and the sigmoid colon is considered to be at a height of 16 cm proximal to the anocutaneous line (measured using a stiff rectoscope), at which point the colon usually angles sharply toward the sigmoid colon. This demarcation is significant for treating tumors: distal to this line, rectal carcinomas in certain stages are treated differently than colon carcinomas located proximal to the line.

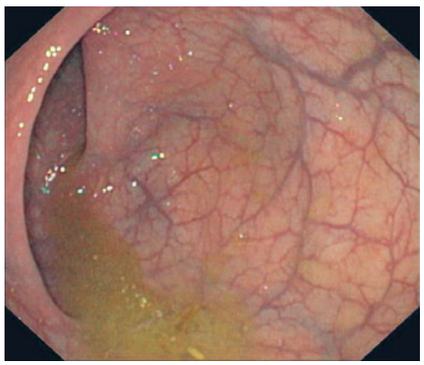
The rectal segment between these two demarcations runs fairly straight (hence "rectum") and typically has three large folds protruding into the lumen from the sides, the so-called Kohlrausch folds (Fig. 6.**1**). The distal segment of the rectum is wider than the proximal segment, forming the rectal ampulla. The proximal rectum is located intraperitoneally, while the distal segment becomes retroperitoneal in the abdominal cavity. The peritoneal reflection, about 7–8 cm from the anal margin, is deeper on the ventral side near the pouch of Douglas than on the dorsal side. This is important for transmural injuries of the rectum.

As in the rest of the colon, the rectal mucosa is smooth and reflective; the blood vessels are clearly visible and easily distinguished from the surrounding surface. As a rule, the vessels in the rectum are more prominent than in the rest of the colon. This is especially true of the venous vessels connected with the vessel branches in the region of the anal canal (hemorrhoidal plexus). The vessels range from pronounced, but still normal, submucosal veins, to pathologically widened veins (e.g., in the case of pronounced hemorrhoidal disease), to rectal varices (e.g., related to portal hypertension, ▣ 6.**1**).

Tips for examining the rectum

- When examining the proximal rectum it is important to pay attention to the areas behind the prominent rectal folds: these should be carefully inspected to avoid missing pathologies such as polyps. Sufficient air insufflation is necessary to flatten the rectal folds. This can often be difficult with patients who have a weak anal sphincter tonus and for whom the insufflation of air into the rectum may cause discomfort and the urge to evacuate the bowels. Thus, after inspecting the rectum, excess air should be suctioned.
- A common mistake toward the end of colonoscopy is withdrawing the instrument too rapidly from the distal rectum, thereby neglecting a thorough inspection of the anorectal area. Retroflexion of the instrument can enable inspection of this area and usually the dentate line (Figs. 6.**2**, 6.**5**), and can be performed on almost any patient. Chapter 5 describes the technique in detail.

6.1 Normal vascular patterns in the distal rectum



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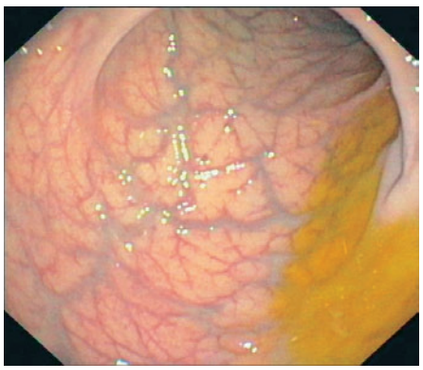
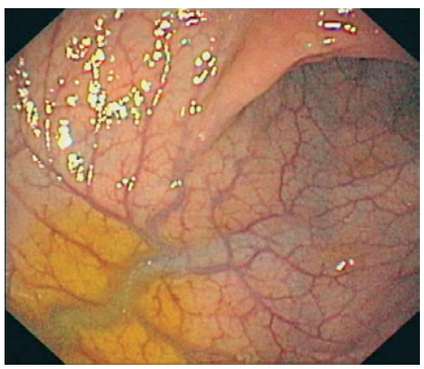
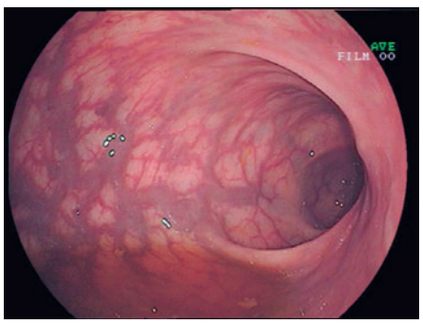


b

a Fine, but clearly visible vascular pattern contrasting sharply with smooth, reflective mucosa.
b More pronounced venous blood vessels, somewhat prominent.



c



d

c Spidery, prominent venous blood vessel with clearly visible vessel branches supplying it.
d Very prominent venous blood vessels, borderline pathology, in a patient with inner hemorrhoids.

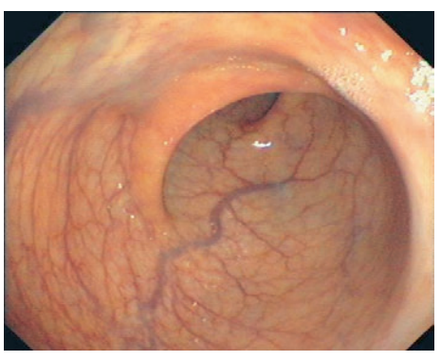


Fig. 6.1 **Rectum with crescent-shaped, infolding rectal valves.** Despite the straight path of the rectum, the folds obscure visualization. Note the distinct and pronounced vascular pattern with the typically clearly visible submucosal veins.

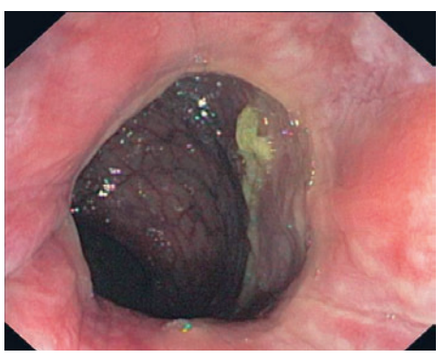


Fig. 6.2 **Anorectal area and upper anal canal, with forward-viewing instrument.** Contractions of the sphincter obscure visualization with a forward-viewing instrument, especially of the surrounding dentate line, which is barely visible.

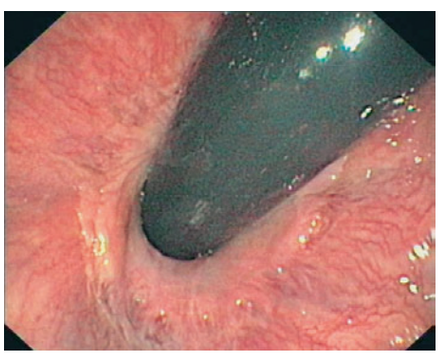


Fig. 6.3 **Anorectal area and upper anal canal viewed with a retroflexed endoscope.** Retroflexing the endoscope allows a better view of the dentate line; the transition between the pale squamous epithelium of the anal canal and the reddish columnar epithelium of the rectum is visible. The anal crypts and the slightly thickened anal papilla are also clearly visible (e.g., at about the 9-o'clock position).

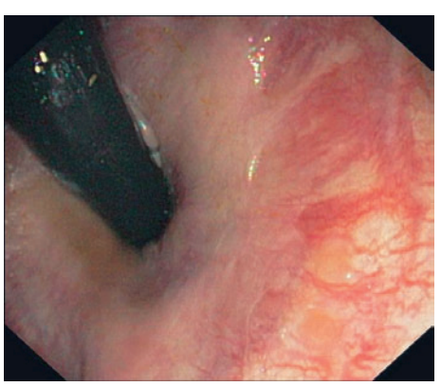


Fig. 6.4 **Close-up view of the dentate line in inversion.** The intertwining, fingerlike squamous epithelium and columnar epithelium are clearly visible. Two anal crypts can be seen at about the 2-o'clock and 3-o'clock positions and at about the 12-o'clock position (partially obscured by the instrument) a hypertrophied anal papilla.

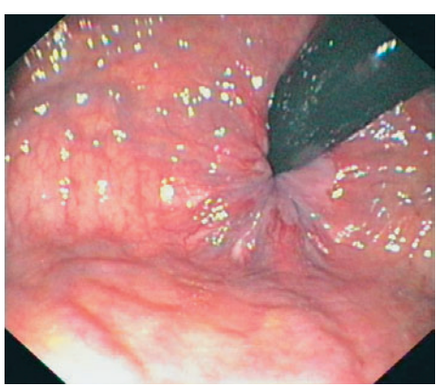
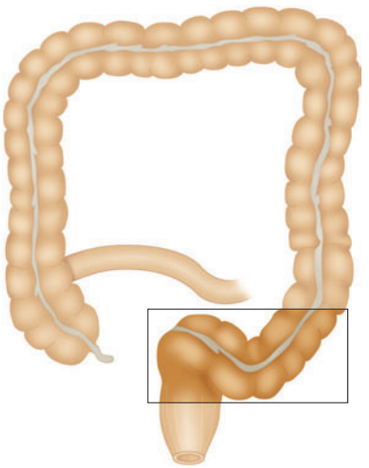


Fig. 6.5 **Normal hemorrhoidal plexus viewed in retroflexion.** The hemorrhoidal cushions, which help seal the anus, appear as bluish swellings. Vascular branches can be seen coming from the distal rectum.

Normal Sigmoid Colon



The sigmoid colon is located in the lower left abdomen between the rectum and the descending colon. Its name is derived from its S shape (sigma = the Greek letter S). The sigmoid colon is completely intraperitoneal, attached and supplied by its own mesentery (mesosigmoid) to the posterior abdominal wall. Due to its intraperitoneal position, the sigmoid colon is usually highly mobile. However, previous lower abdominal surgery, especially gynecological operations and inflammation (e.g., diverticular disease), can cause adhesions, fixing it to the abdominal wall, making passage difficult, and in rare cases even impossible. The length of the sigmoid colon can vary greatly; usually 15–30 cm long, it can be significantly longer (a so-called elongated sigmoid, not considered a pathology), which can lead to looping, and create significant problems for advancing the endoscope. As already mentioned, there is no clear anatomical demarcation at the distal end between the sigmoid colon and the rectum, though the rectosigmoid junction is usually rather sharply angled. The sigmoid-descending junction is often acutely angled, particularly in slender patients, making passage difficult. This is because the back wall of the descending colon is fixed retroperitoneally to the posterior abdominal wall, while the sigmoid colon distal to it is mobile. Advancement of the instrument thus pushes the sigmoid colon upward, causing unwanted angling.

The mucosa is shiny and smooth and the blood vessels clearly visible, though less prominent than in the rectum (Fig. 6.**6**). The lumen is round or oval-shaped (Fig. 6.**7**). The sigmoid is usually comprised of a series of curves, making visualization into the haustra more difficult (Figs. 6.**8**, 6.**9**). The sigmoid colon often is marked by strong contractions, which can also obstruct the view into the haustra. Figure 6.**10** shows an example of the previously mentioned acute angling of the sigmoid-descending junction, as can be seen, for instance, in slender patients.

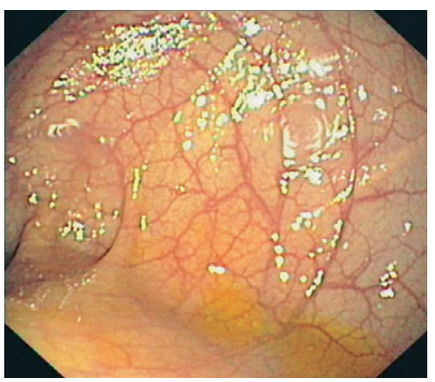
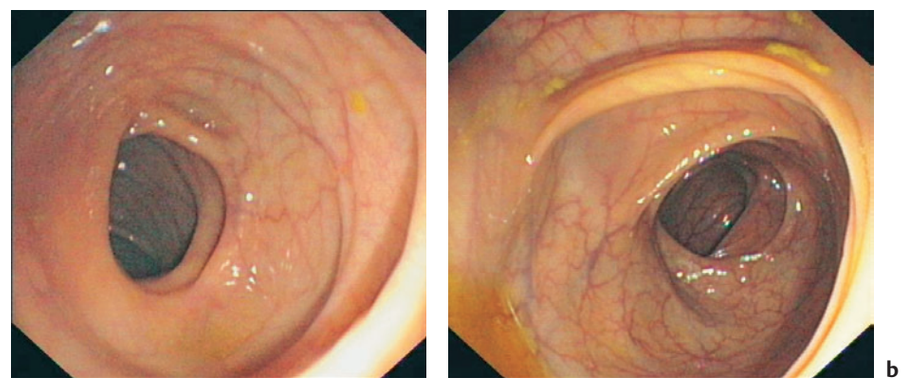


Fig. 6.**6** **Sigmoid colon.** Normal, distinct vascular pattern.



a b

Fig. 6.**7** **a, b Sigmoid colon.** Normal, round, or oval-shaped lumen.

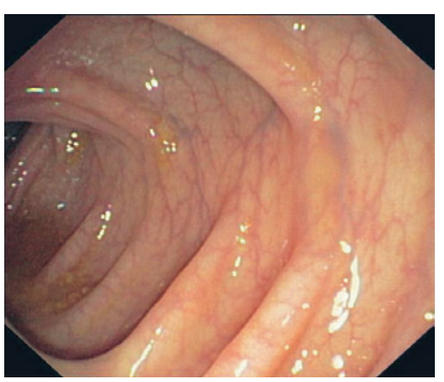


Fig. 6.**8** **Haustrations in the sigmoid colon.** The curving sigmoid colon makes viewing the inside of the curves difficult.

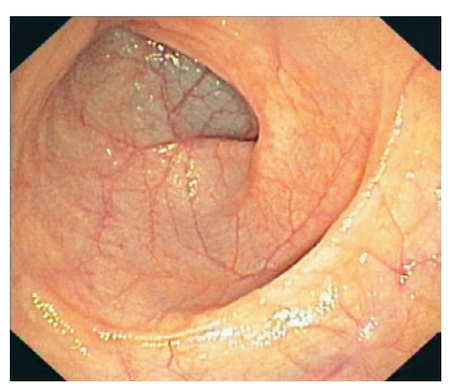


Fig. 6.**9** **Winding sigmoid**

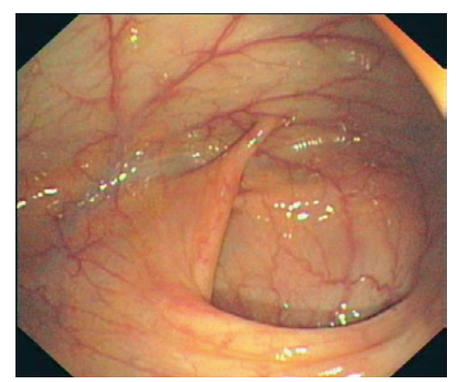


Fig. 6.**10** **Sigmoid-descending junction.** Sharp angling, folded-over appearance.

Tips for examining the sigmoid colon

- Though it can be relatively time consuming, close inspection of the haustra in the sigmoid colon when withdrawing the instrument is essential. Advancement with intermittent withdrawal, covering only a short distance, is often necessary to ensure a sufficient inspection of all areas.
- If contractions of the sigmoid colon inhibit visualization, n-butylscopolamine (note contraindications) can be given, enabling in most cases better visualization.

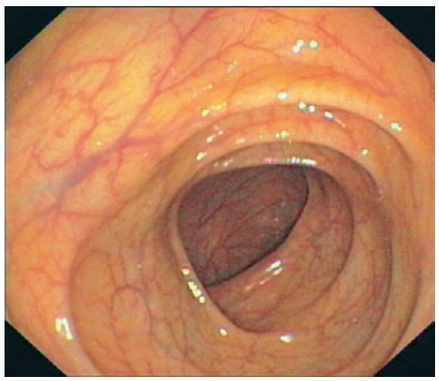
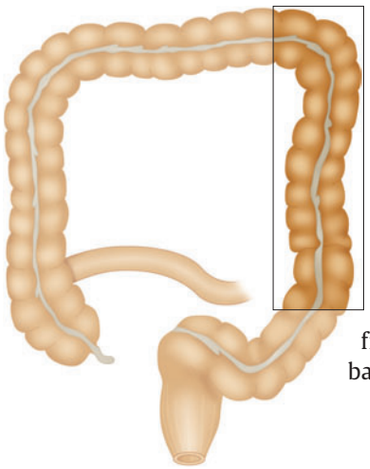


Fig. 6.**11** **Descending colon.** Normal oval or slightly triangular-shaped lumen, with normal, distinct vascular pattern.

Normal Descending Colon



The descending colon runs relatively straight along the left flank from the sigmoid-descending junction to the splenic flexure. Around 20–30 cm long, it is fixed on its posterior side to the dorsal abdominal wall; ventrally it is covered by the peritoneum lining the abdominal cavity. Due to its retroperitoneal fixation, the descending colon is barely mobile, unlike the sigmoid colon and the transverse colon.

The lumen is not as round as in the sigmoid colon, but instead is mostly triangular or oval-shaped, though the triangular shape is less pronounced here than in the transverse colon (Figs. 6.**11**–6.**14**). The haustrations are clearly visible, though usually not as pronounced as in the transverse colon. The splenic flexure, in the upper left abdomen near the spleen, separates the descending colon anatomically from the transverse colon. The spleen is often visible as a bluish coloration, shimmering through the colon wall and thus can help localize the endoscope tip (Fig. 6.**15**). The splenic flexure varies individually. In some patients, it is located high in the upper left abdomen; in others it is located deep in the abdomen, sometimes forming only a mild curve, making it difficult to localize. It can also be directly beneath the diaphragm, and in isolated cases, acutely angled, making passage of the colonoscope in the transverse colon and toward the cecum difficult.

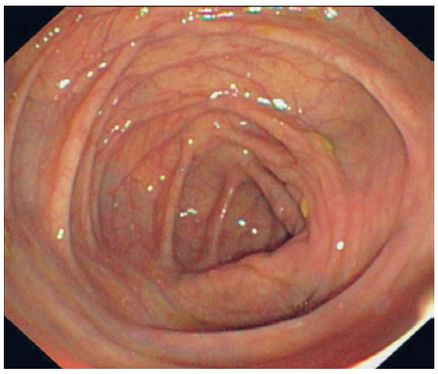


Fig. 6.**12** **Descending colon.** Normal oval or slightly triangular-shaped lumen.

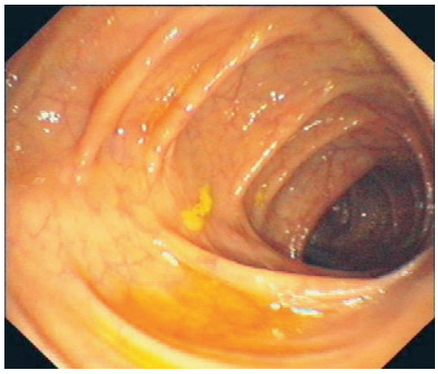


Fig. 6.**13** **Descending colon.** More pronounced triangular shape.

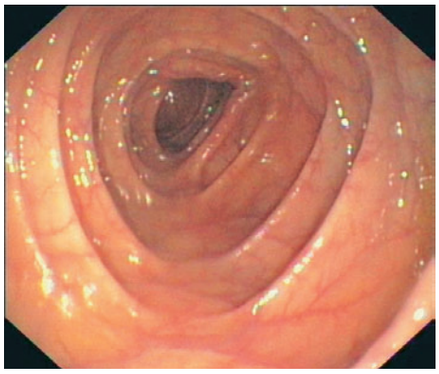
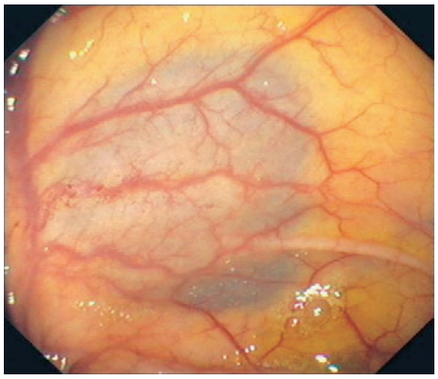
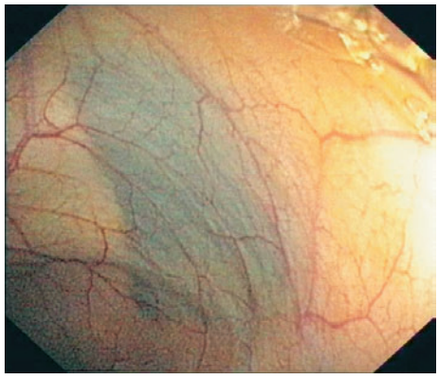


Fig. 6.**14** **Descending colon.** Clear triangular configuration, running mostly straight.



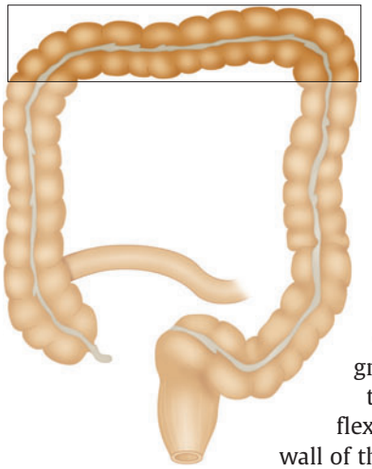
a



b

Fig. 6.**15 a**, **b** Splenic flexure. Blue coloration from the spleen shimmering through the colon wall near the splenic flexure.

Normal Transverse Colon



The transverse colon runs across the upper abdomen, connecting the descending colon and the ascending colon; the splenic and hepatic flexures at either end of this colon segment demarcate the anatomical borders. The transverse colon is entirely intraperitoneal and is supplied by its own mesocolon. On the ventral side, the greater omentum is fixed to the transverse colon. Because both flexures are fixed to the posterior wall of the abdominal cavity, while the transverse colon lies in front of other organs, its course is not straight, but instead runs in a convex arc ventrally and, to varying degrees, caudally. This can range from a mild curvature to a deep loop extending down into the pelvis. The length of the transverse colon is thus highly variable, from 30 cm to more than 50 cm. Because it lies ventrally in the abdomen, the position of the endoscope may be seen (transillumination) through the abdominal wall.

The transverse colon is normally characterized by a triangular lumen with pronounced haustrations (Figs. 6.**16**–6.**19**). The shape of a typical transverse colon is rather similar to that of a Toblerone chocolate bar. The smooth and shiny mucosa and the clear vessel pattern in the transverse colon are similar to the rest of the colon.

Because the hepatic flexure is fixed retroperitoneally, it often has a funnel-shaped winding form; just before reaching the hepatic flexure the colon curves dorsally (Fig. 6.**20**), while in the region of the hepatic flexure itself the lumen is angled caudally toward the ascending colon. The liver is often visible near the hepatic flexure as a bluish coloration, shimmering through the colon wall (Figs. 6.**21**, 6.**22**). The area of surface contact between liver and flexure is significantly larger than that of the spleen near the splenic flexure.

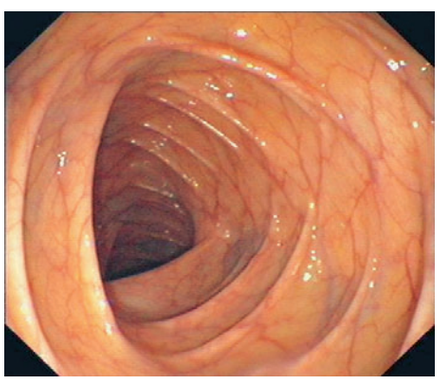


Fig. 6.**16** **Transverse colon.** Typical triangular shape.

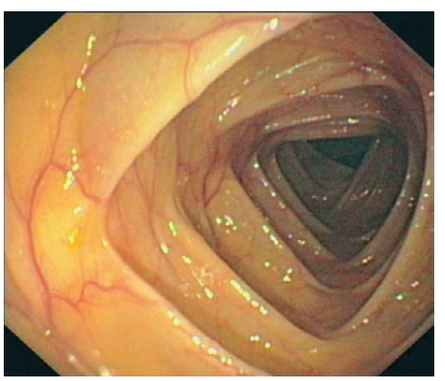


Fig. 6.**17** **Transverse colon.** Triangular lumen and strong haustrations.

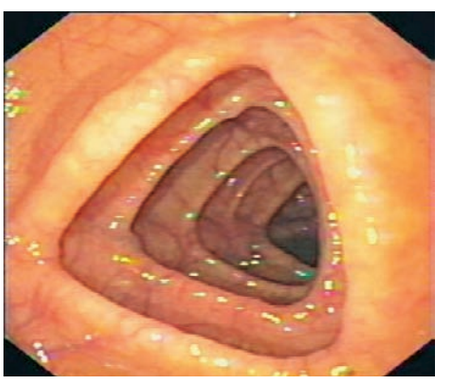


Fig. 6.**18** **Midtransverse colon** with triangular lumen.

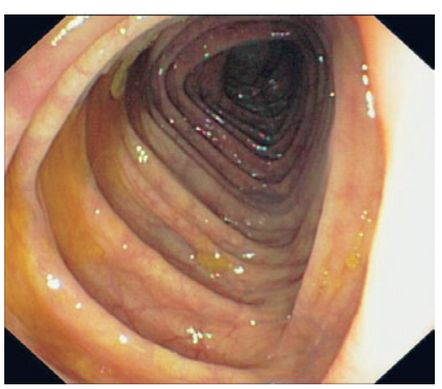


Fig. 6.**19** **Transverse colon.** Typical normal appearance, shown here with straight path.

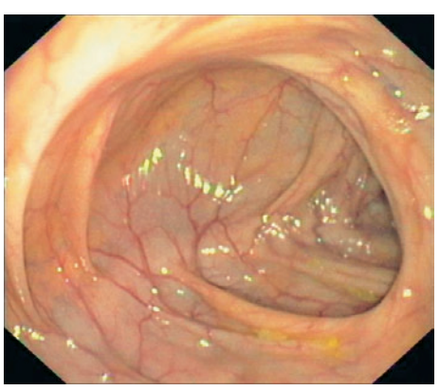


Fig. 6.**20** **Transverse colon.** Funnel-like transverse colon, angled dorsally just before the hepatic flexure. After passing this point, the instrument is angled caudally toward the ascending colon.

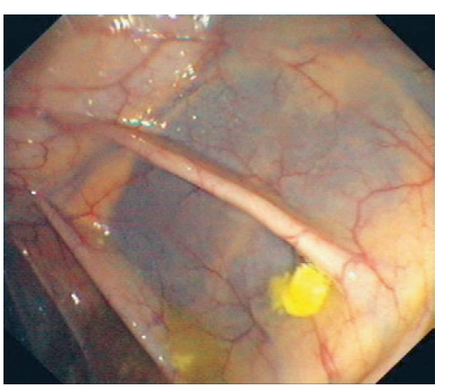


Fig. 6.**21** **Hepatic flexure** with shimmering, bluish coloration of the liver. The colon angles at this point caudally (shown here at about the 7-o'clock position).

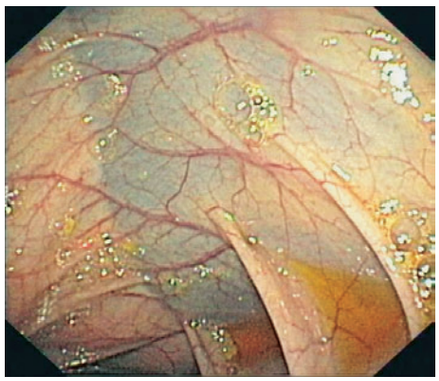


Fig. 6.**22** **Hepatic flexure.** Shimmering coloration of liver. Note the significantly larger surface area around the liver compared to the spleen at the splenic flexure (Fig. 6.**15**).

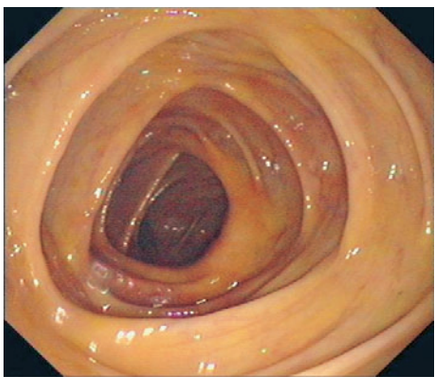
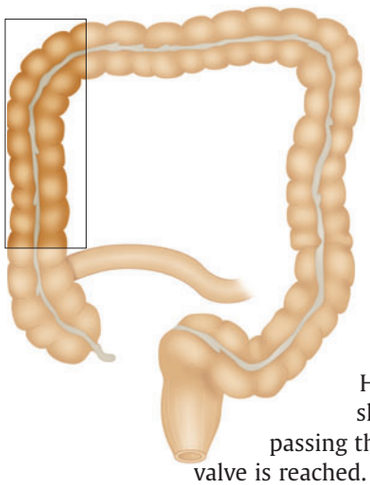


Fig. 6.**23** **Normal ascending colon** with relatively wide, triangular-shaped lumen. In the distance the yellowish Bauhin valve protruding into the lumen from the side (shown here at about the 4-o'clock position).

Normal Ascending Colon



The ascending colon runs near the right flank between the hepatic flexure and the ileocecal valve. Similar to the descending colon, the ascending colon is fixed to the dorsal abdominal wall and thus only slightly mobile, running relatively straight. The length of the ascending colon is variable, on average around 15–20 cm. However, it is occasionally very short, so that immediately after passing the hepatic flexure the ileocecal valve is reached. At the other extreme is a very long ascending colon, with the Bauhin valve located deep in the lower abdomen.

As a rule, the ascending colon has the widest lumen of any of the colon segments. The lumen is mostly triangular, similar to the transverse colon (Fig. 6.**23**), though the folds are mostly somewhat thicker and plumper than the more distal colon segments (Fig. 6.**24**). After passing the hepatic flexure, one often can already see the ileocecal valve in the distance as a yellowish arcuate fold, often with an indentation in the center (Fig. 6.**25**). Occasionally the valve is clearly prominent due to an accumulation of fat (Fig. 6.**26**). The mucosal appearance and vessel pattern in the ascending colon are the same as in the rest of the colon proximal to the rectum (Figs. 6.**27**, 6.**28**).

Tips for examining the ascending colon

- Due to the relatively thin colonic wall in the ascending colon and cecum, care must be taken to avoid perforation during therapeutic procedures such as argon plasma coagulation, laser therapy, and polyp removal. Precautionary measures for avoiding perforation, including liberally injecting flat polyps before removal with a snare, are strongly advised.

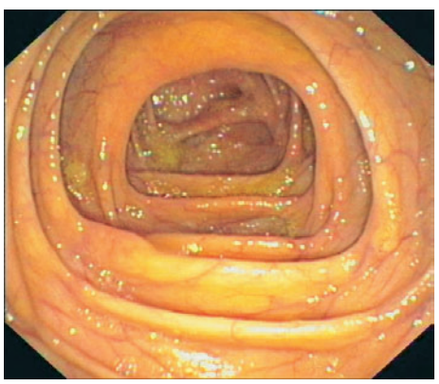


Fig. 6.**24** **Ascending colon and ileocecal valve** (yellowish, thickened fold shown here at about the 12-o'clock postion), the cecal pole can be seen in the distance. Note the relatively wide lumen in the ascending colon. The folds are somewhat thicker compared to the transverse colon.

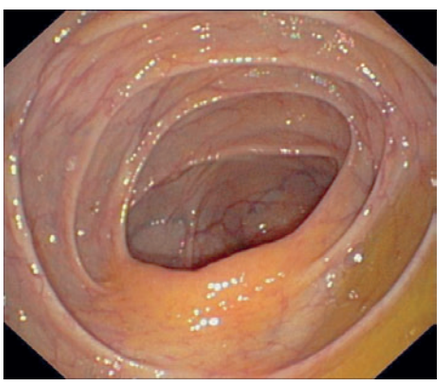


Fig. 6.**25** **Normal ascending colon** with typical aspect of the ileocecal valve (shown here at about the 6-o'clock postion) as a yellowish fold with an indentation in the center.

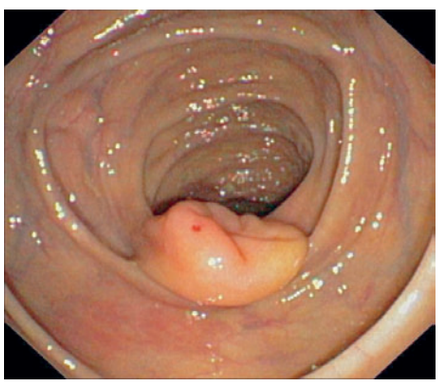


Fig. 6.**26** **Ascending colon** with lipomatous, thickened, ileocecal valve, with clearly visible opening, protruding far into the lumen. A secondary finding is the small, light red angiectasis on the valve.

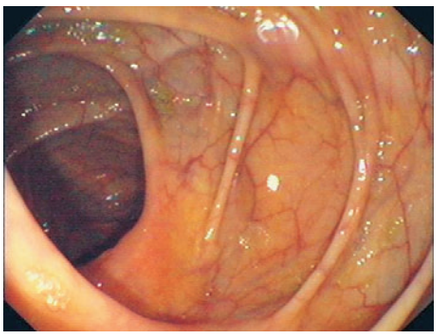


Fig. 6.**27** **Ascending colon.** Normal shiny mucosa with very distinct vascular pattern directly over the Bauhin valve, which is partially covered by a fold from the ascending colon.

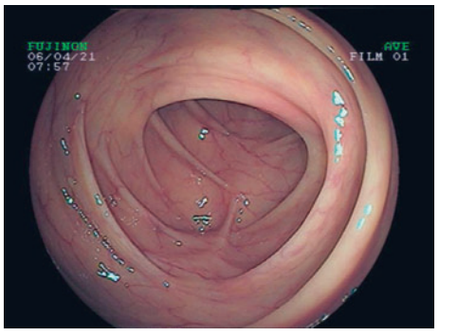


Fig. 6.**28** **Normal ascending colon** with the Bauhin valve, which appears relatively pale here (at about the 12-o'clock position). The view of the cecum is obscured by the ileocecal valve protruding into the lumen.

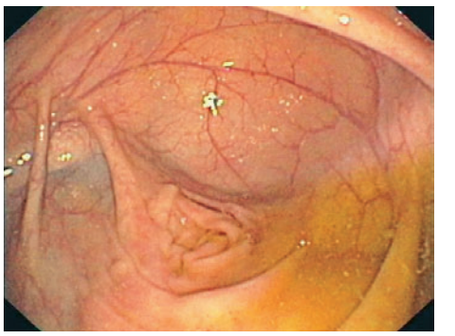
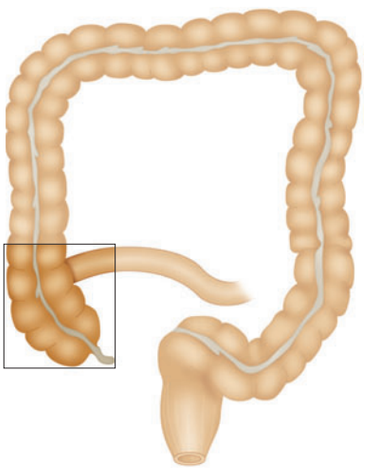


Fig. 6.**29** **Cecal pole with entrance to appendix.** The star-shaped form of the three tenia converging at the appendiceal orifice (at about the 2-o'clock, 7-o'clock, and 11-o'clock positions).

Normal Cecum and Ileocecal Valve



Cecum. The short segment of the colon between the appendix and the ileocecal valve is the cecum.

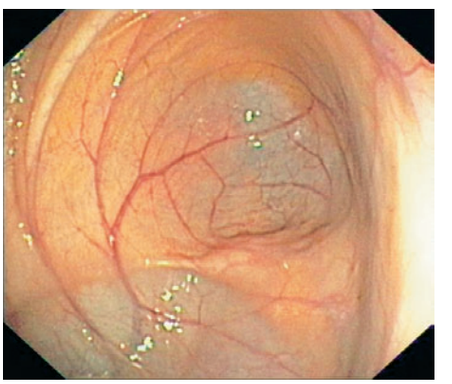


Fig. 6.**30** **Cecal pole.** One of the tenia leading to the appendiceal orifice (from about the 9-o'clock position). Note the distinct vascular pattern of the normal mucosa.

The cecum is only a few centimeters long. At its blind end, the three taenia coli converge in a star-shaped formation (Figs. 6.**29**, 6.**30**). This is where the entrance to the appendix is located, which normally can be recognized as a dotlike round or oval opening similar to a diverticulum (Figs. 6.**31**, 6.**32**). If the appendix has been removed, a raised polyplike form, the invagination of the appendiceal stump, will often be seen (Fig. 6.**33**). Like the ascending colon, the posterior side of the cecum can be retroperitoneally fixed, though it also can be completely intraperitoneal, and is then often located across from the axis of the ascending colon or inverted. This can make visualization into the cecum and advancement to the cecal pole difficult. The diameter of the cecum, like that of the ascending colon, is wider than in the other colon segments. The wall of the cecum is very thin, only a few millimeters thick.

Ileocecal valve. The ileocecal valve is the anatomical demarcation between the cecum and the ascending colon and is the point where the ileum meets the colon. The ileum enters into the wall of the colon from the side, with the sphincterlike ileocecal valve at its end, preventing backward flow of the contents of the colon into the ileum.

The ileocecal valve can have very different endoscopic appearances (▣6.**2**). It can be merely a yellowish protruding fold in the lumen (especially when the opening is toward the cecum). Or, like a papilla, it can be arcuate, protruding far into the lumen with a readily visible ileal opening. Occasionally, there is evidence of a pronounced accumulation of fat in the ileocecal valve, also known as a lipomatous valve. The valve is then an intense yellow, usually prominent and raised like a polyp. Identifying the ileocecal valve is the surest endoscopic landmark, indicating that the cecum has been reached.

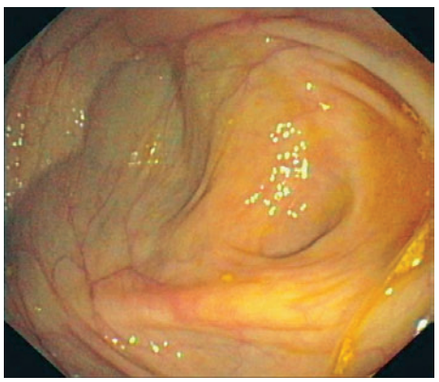


Fig. 6.31 **Close-up of appendiceal orifice,** round configuration shown here. Again two of three converging tenia are visible.

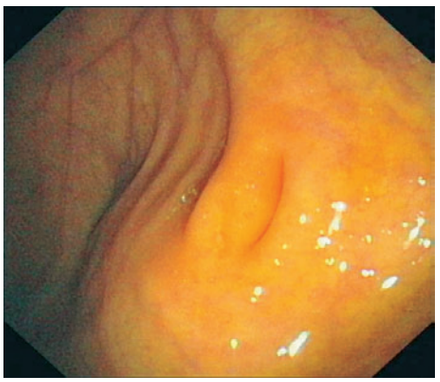


Fig. 6.32 **Close-up of appendiceal orifice,** oval configuration shown here. The very lightly spotted appearance around the orifice is caused by a small lymph follicle, often found here.

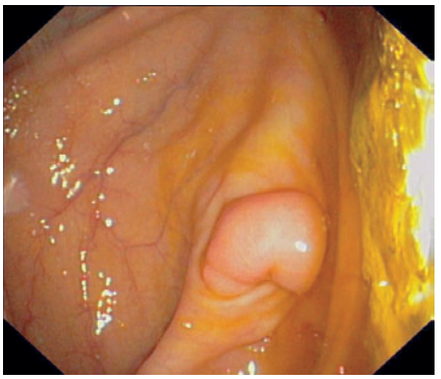
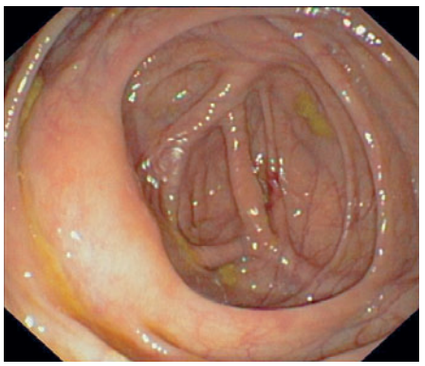
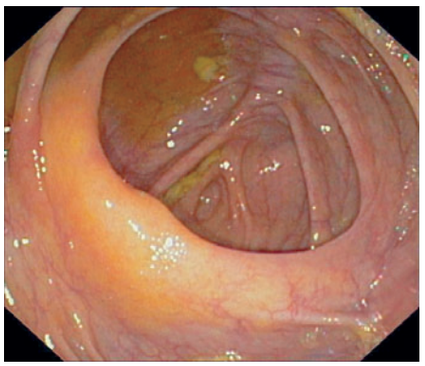


Fig. 6.33 **Invaginated appendiceal stump from previous appendectomy.** The worm-like appearance and smooth surface are typical, helping distinguish it from an adenoma. If there is any uncertainty, take only an excisional biopsy; resection of the appendiceal stump using a snare involves a high risk of perforation.

Various endoscopic appearances of the ileocecal valve



a



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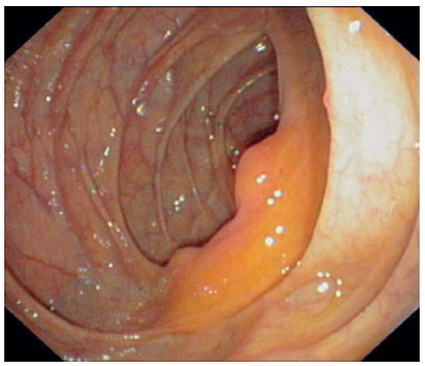
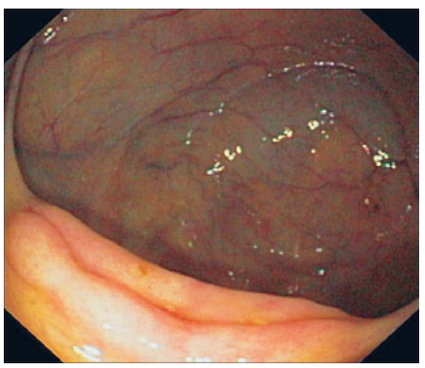
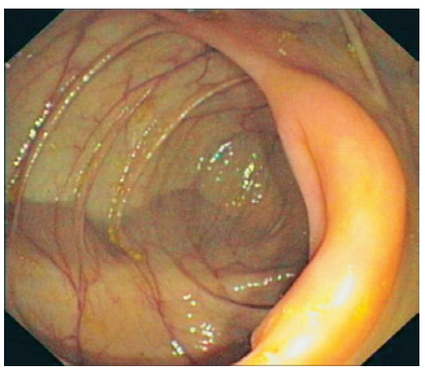


c

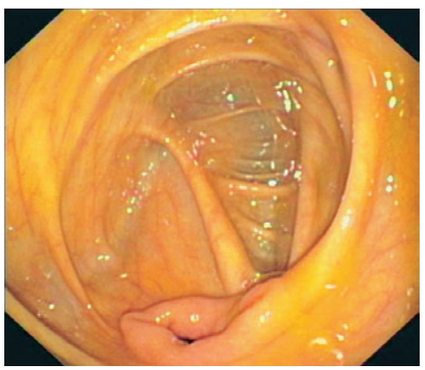
a–c Valve with the ileal opening pointing toward the cecum, usually appearing as a thickened, yellowish fold, protruding into the lumen. A more or less pronounced indentation is often visible at the orifice (**b**, **c**). Intubation of this type of overhanging valve is often difficult, due to lacking visualization.



d



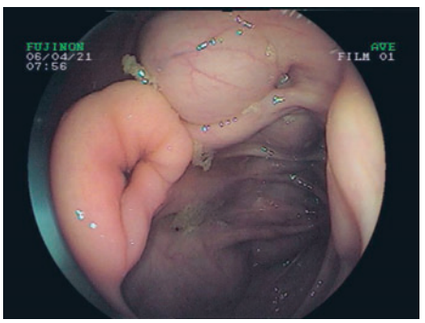
e



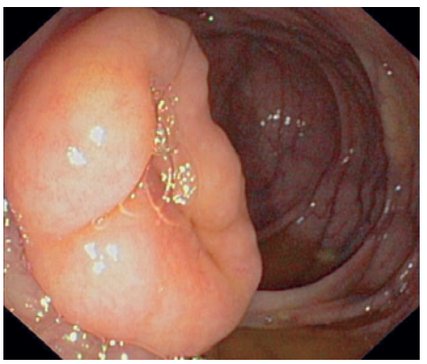
f

d–f Labial form of the valve with a clearly oval-shaped orifice. The lips are sometimes slightly open (**f**). Intubation of this type of valve is usually unproblematic.

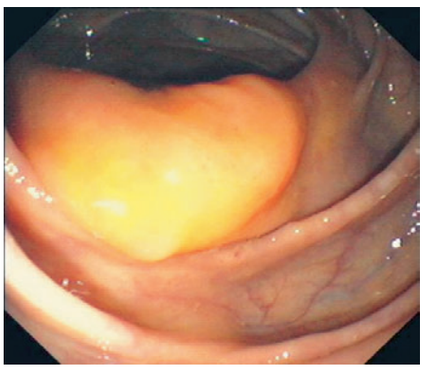
■ **6.2 cont.**



g



h



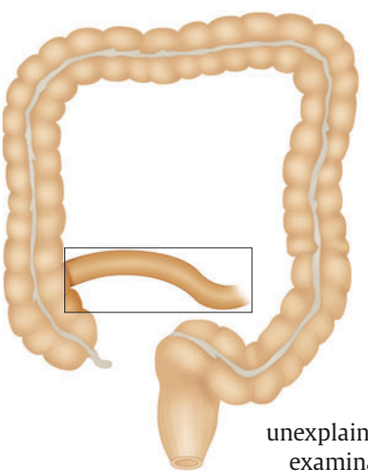
i

g–i More papillary type of valve, strongly projecting far into the lumen and usually exhibiting radial folds on the valve opening (**h**). The prominence is sometimes caused by a pronounced accumulation of fat (lipomatous valve), recognizable by the intense yellow color (**i**).

Tips for examining the cecum and ileocecal valve

- It is important that the cecum be thoroughly inspected, in order to avoid missing pathologies. The area of the cecum directly behind the Bauhin valve is especially difficult to examine in an inverted or mobile cecum. In some patients, the examiner can attempt to deflect the tip of the endoscope to the point of retroflexion, thereby allowing backward viewing of the ileocecal valve. However, this maneuver should be performed with the utmost caution because of risk of perforating the thin cecal wall. Overdistension of the cecum, which also increases risk of perforation, should likewise be avoided. This maneuver should only be performed by experienced examiners.
- It should be remembered that a raised polyplike form on the cecal pole might be an invaginated appendiceal stump from an earlier appendectomy (Fig. 6.**33**). This is an important consideration, as removal with a snare (if mistaken for an adenoma) can have adverse consequences, i. e., perforation.
- Identification of the ileal opening at the Bauhin valve can be made difficult by an overhanging valve lip or an inverted cecum. It is, however, extremely helpful for intubation of the terminal ileum to know beforehand where the valve orifice is located in relation to the valve. In addition, repeated attempts at intubation usually cause surface lesions and these can sometimes be difficult to correctly identify, e.g., they can be confused with angiectasias.

Normal Terminal Ileum



There is no consensus on whether inspecting the terminal ileum is a necessary part of every total colonoscopy. Intubation is often difficult and requires an experienced examiner. However, there are some patients in which pathologies are present only in the terminal ileum (e.g., Crohn disease). And, for certain indications (diarrhea, suspected chronic inflammatory bowel disease, unexplained gastrointestinal bleeding), examination of the terminal ileum is essential. At our center we therefore aim to perform an intubation of the Bauhin valve on every patient, in part given the benefit of practical experience gained by the examiner in performing this technique.

The terminal ileum is reached after intubating the ileocecal valve. As a rule, no more than 10–15 cm of the terminal ileum can usually be viewed. The caliber of the terminal ileum is significantly thinner than that of the colon. The transverse folds typical of the small proximal intestine are less pronounced in the terminal ileum or lacking completely (Figs. 6.**34**, 6.**35**). The mucosa is distinctly different from the colonic mucosa. Instead of a smooth and reflective surface, it is velvety and granular, this is caused by the shaggy relief of the ileum villi, which are usually visible close up (Fig. 6.**36**). Blood vessels are generally fine and thin and are not as easily distinguishable, or in much detail, as in the colon (Fig. 6.**37**). Especially in younger patients, aggregated lymphatic follicles (Peyer plaques) are often visible as small, raised polyplike forms; pronounced forms are also referred to lymphatic hyperplasia, which, usually, is not a pathology (Figs. 6.**38**, 6.**39**).

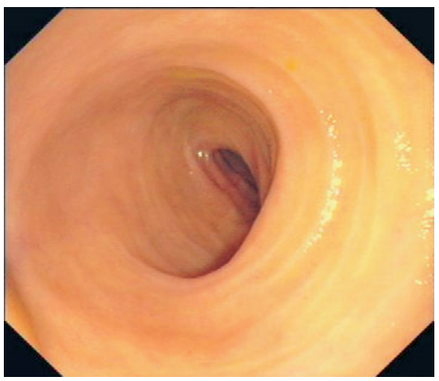


Fig. 6.**34** **Normal terminal ileum.** Transverse folds are barely visible and the mucosa has a velvety, matt surface.

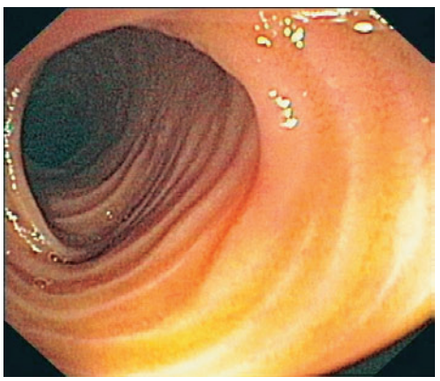


Fig. 6.**35** **Normal terminal ileum with somewhat more pronounced transverse folds.** Here, too, the fine-grained and velvety mucosal surface is clearly visible.

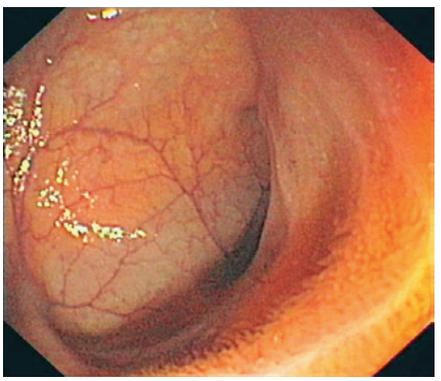


Fig. 6.**36** **Ileum mucosa** viewed from the side close-up, showing the visible surface relief of the villi.

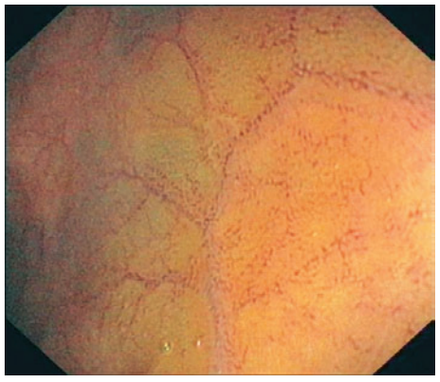


Fig. 6.**37** **Close-up view of the ileum mucosa.** Velvety surface structure, created by the villi. The blood vessels underneath are not as easily distinguishable as in the colon.

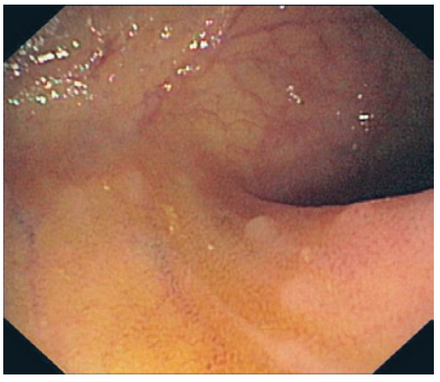


Fig. 6.**38** **Ileum mucosa** with individual lymph follicles, visible as raised lentil-like forms.

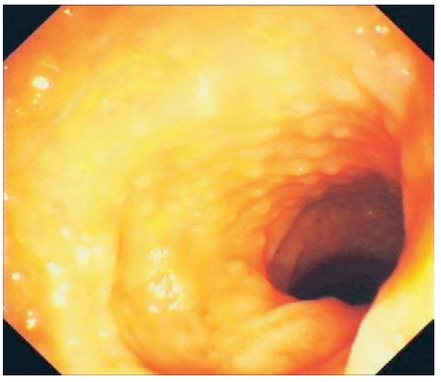


Fig. 6.**39** **Lymphatic hyperplasia in the terminal ileum** with small, multiple lymph follicles.

Tips for examining the terminal ileum

- Forcible air insufflation in the terminal ileum can cause discomfort to the patient. Air should be insufflated sparingly and suctioned intermittently, which can also serve as a check of the elasticity of the ileum.
- Intensive contractions of the ileum and its sometimes foamy contents (due to reabsorption of bile acids) can hinder examination. If these conditions prevent sufficient examination, n-butylscopolamine (note contraindications) may be injected for the former, and for the latter, an over-the-counter antigas drug (e.g., simethicone) can be given to increase visualization.

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7 Normal Postoperative Appearances

M. Bittinger

Restoring Intestinal Continuity

Following surgical resection of the colon—whether due to benign or malignant disease—the continence of elimination of feces must be restored. If continuity restores the natural pathway, i. e., so that evacuation occurs transanally, an anastomosis is necessary between resected segments. Continuity can be restored using end-to-end, end-to-side, or side-to-side anastomoses (Fig. 7.**1**).

Ileocolic anastomosis. An ileocolic anastomosis is an anastomosis between the ileum and the colon (e.g., after a right hemicolectomy). This is generally an end-to-end (Fig. 7.**2**) or end-to-side anastomosis (Fig. 7.**3**). One can usually see a slight difference between the velvety, small intestinal mucosa and the smooth, shiny colonic mucosa. The transition is usually visible as a clear, smooth border and is only seldom slightly polypoid in appearance (Figs. 7.**2**, 7.**3**).

Colocolic anastomosis. A colocolic anastomosis is constructed between two colon segments (e.g., following resection of the sigmoid colon) and is usually an end-to-end anastomosis. A normal colocolic anastomosis appears as a smooth, whitish curvilinear scar that does not noticeably narrow the lumen (Fig. 7.**4**). Sometimes larger venous vessels can be seen near the anastomosis, but not intersecting it; occasionally suture remnants or metal staples (from the use of automatic sutures) can be seen on the anastomosis (Fig. 7.**5**).

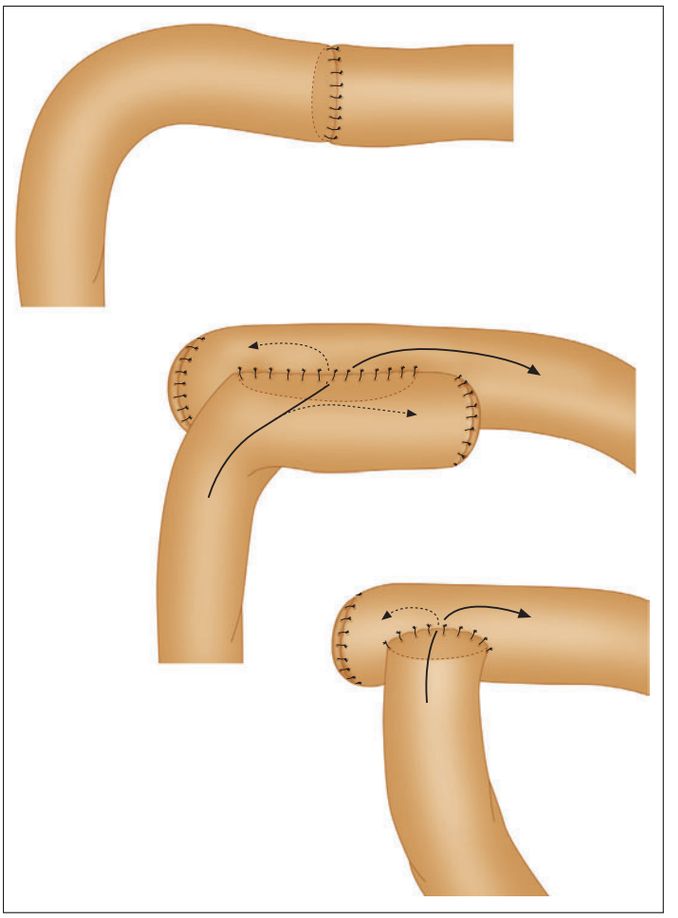


Abb. 7.**1** **Schematic illustration of options for restoring continuity following intestinal resection:** end-to-end anastomosis (top), side-to-side anastomosis (middle), end-to-side anastomosis (bottom).

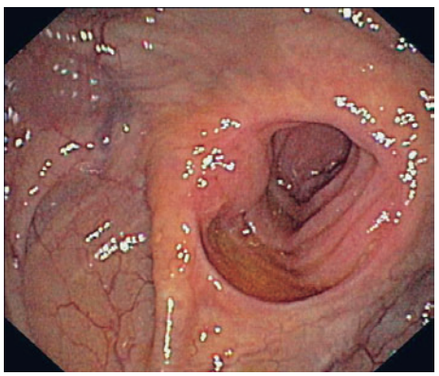


Abb. 7.2 **Ileocolic end-to-end anastomosis.** The ileum folds and velvety mucosa of the neoterminal ileum can be seen; the colon has a smooth mucosal surface and distinct vascular pattern. The anastomosis appears as a smooth ring.

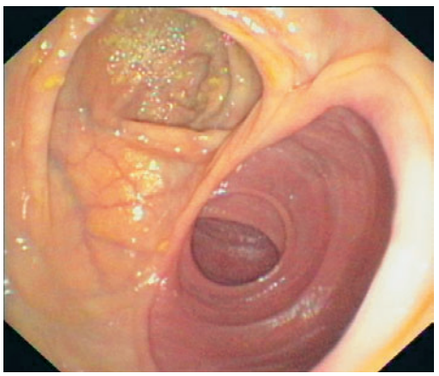


Abb. 7.3 **Ileocolic end-to-side anastomosis.** Neoterminal ileum with typical ileal mucosa at lower right; blind end of anastomosed colon at upper left. Note the difference between the vascular patterns in the ileum and colon.

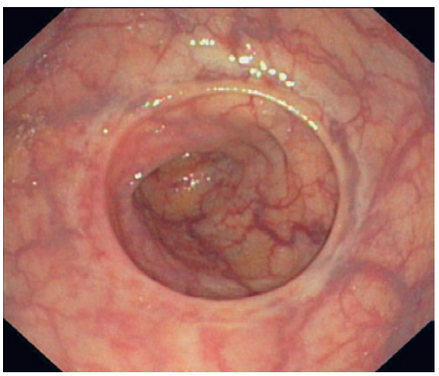


Abb. 7.4 **Colocolic end-to-end anastomosis,** visible as a smooth, whitish, curvilinear scar. Note on either side of the anastomosis ring the prominent vessels that do not intersect it.

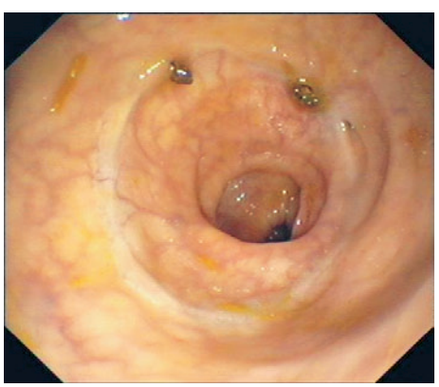


Abb. 7.5 **Colocolic end-to-end anastomosis,** shown here as a flat, white ring. Two metal staples with feces are visible at about the 1-o'clock and 11-o'clock positions; at about the 2-o'clock position is the blind end of a small diverticularlike indentation.

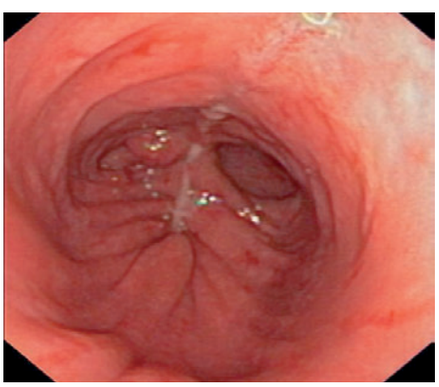


Abb. 7.6 **Ileoanal pouch (J pouch).** On the right is the opening of the neoterminal ileum, and on the left is the blind end of the ileum loop where the ileum was folded back on itself. Perpendicular to the ileum folds, the partly fibrin-covered (i. e., not recent) incision is visible between the two loops with a staple. The pouch's wide lumen functions as a reservoir for stool.

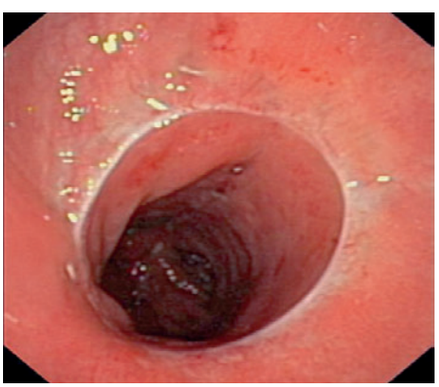


Abb. 7.7 **Anastomosis between pouch and anal canal** directly above the sphincter. In the distance, near the 12-o'clock position, a longitudinal seam is visible on the pouch; the anastomosis appears as a white ring.

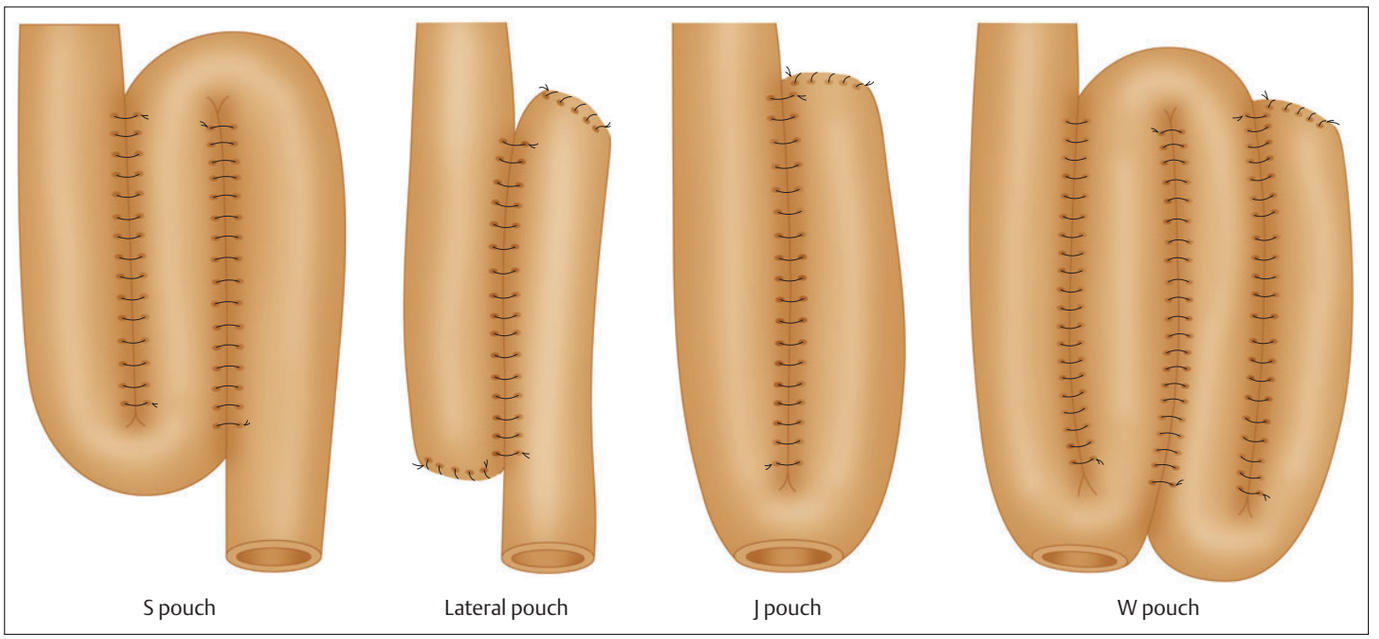
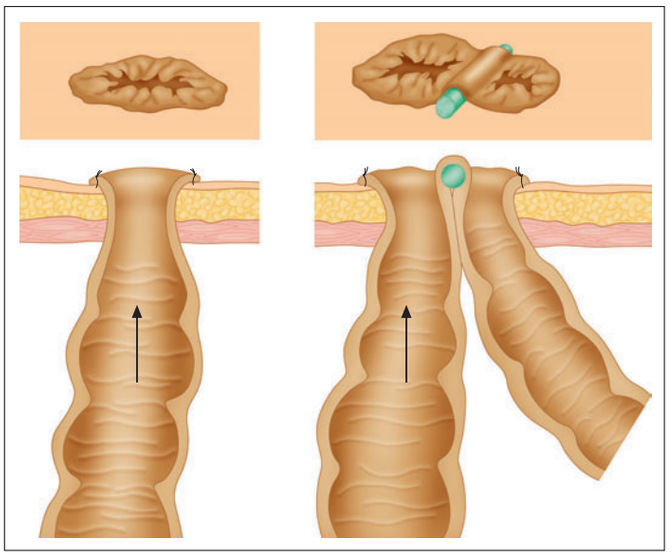


Abb. 7.**8** **Ileoanal pouches.** Schematic illustration of the four most commonly used types.

Ileoanal pouch. The ileoanal pouch is a special form of restoring continuity following proctocolectomy. This procedure is generally used for severe ulcerative colitis and ulcerative colitis with dysplasia. Unlike ileostomy, the patient retains anal continence. The ileoanal pouch is usually formed as a so-called J pouch: two ca. 15-cm-long ileum loops are placed next to each other, joined to form a reservoir, and anastomosed to the anal canal (Figs. 7.**6**, 7.**7**). The ileoanal pouch functions as a reservoir for stool, thus avoiding too frequent elimination. Other pouches are also commonly used, though significantly less often than the J pouch, which is technically the easiest to construct (Fig. 7.**8**).

Stomas

If restoration of continuity is not possible permanently (e.g., following rectal excision due to low rectal carcinoma) or temporarily (e.g., to protect a rectal anastomosis), a stoma is necessary. An ileostomy is a connection between the ileum and an opening in the skin (stoma); a colostomy refers to passage of waste through the colon to an opening in the skin. Stomas can be either endstomas or double-barreled stomas that have two openings, allowing the remaining intestinal segment distal to the stoma a means of draining (Fig. 7.**9**). A normal stoma is characterized by a sufficiently wide intestinal opening; there is no significant protruding (prolapse), and there are no signs of



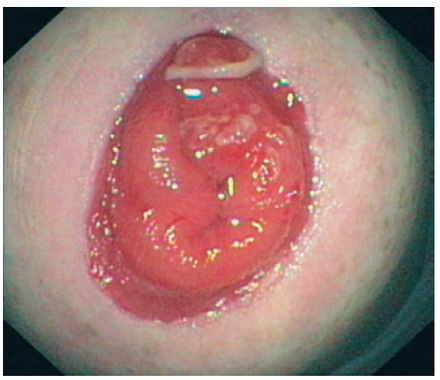


Abb. 7.**10** **Normal smooth colostomy with normal, wide opening.** The oblong, yellowish structure at about the 12-o'clock position and the whitish deposits on the mucosa next to it are feces.

◁ Abb. 7.**9** **Colostomy.** Schematic illustration of an end colostomy (left) and a double-barreled colostomy (right).

irritation to the mucosa or surrounding skin (Fig. 7.**10**). If the opening of the stoma is not wide enough to permit passage of the small finger, stenosis is indicated and must be treated. As the contents of the small intestine are much more aggressive than those of the large intestine, an ileostomy requires more care than a colostomy and skin irritation occurs much more frequently.

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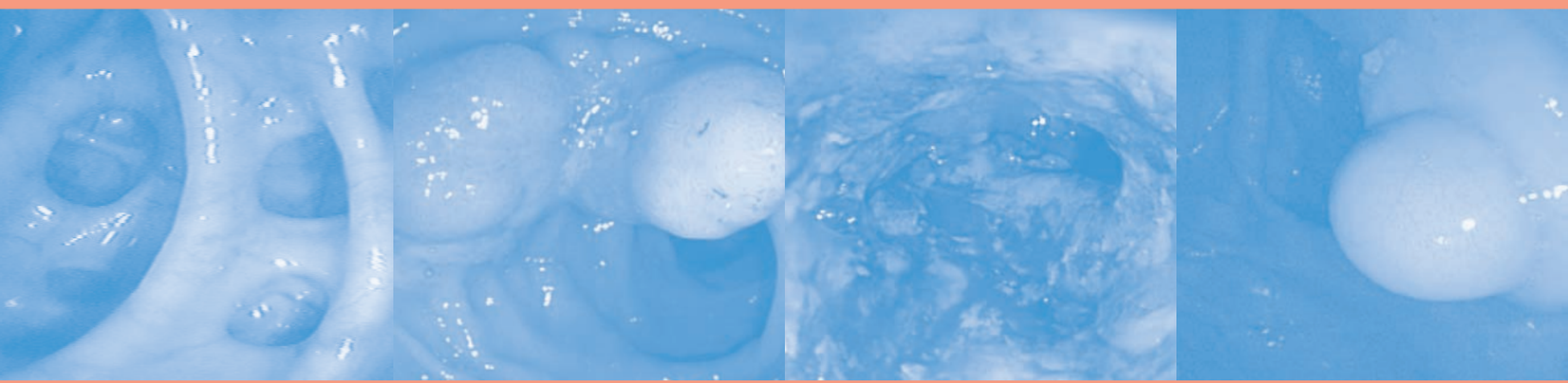
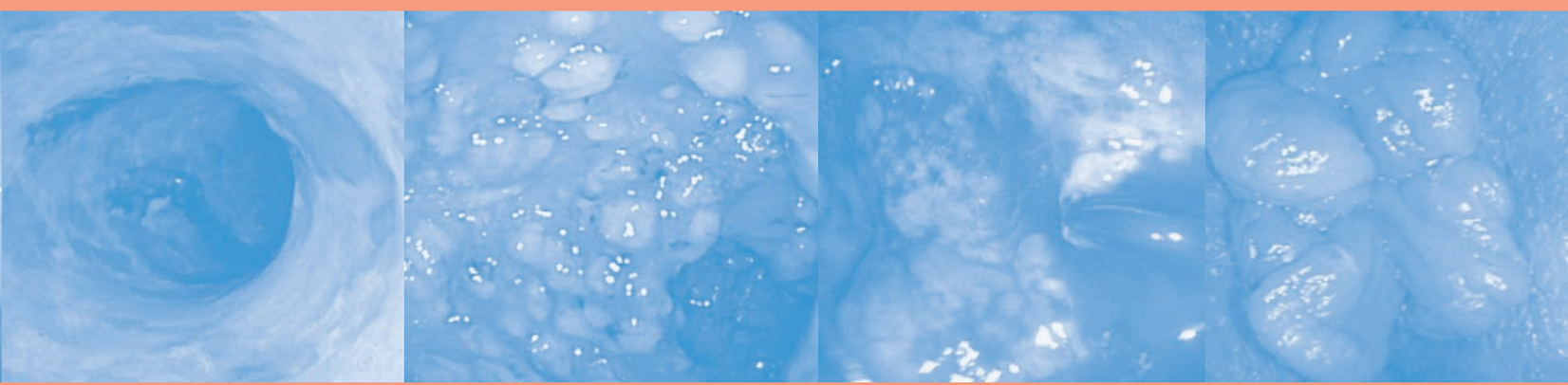


III Pathological Findings



8 Diverticulosis and Diverticulitis

M. Bittinger

Definitions

Colonic diverticula are fingerlike outpouchings protruding outward from the intestinal lumen.

True and pseudodiverticula. A distinction is made between true diverticula and pseudodiverticula. The seldom-occurring true diverticula are present at birth and usually only appear in the right hemicolon. Pseudodiverticula, which occur much more frequently, are acquired. They can be found anywhere in the colon, though they tend to appear in the left hemicolon. True diverticula are characterized by herniation of the entire colon wall, including the muscle layer. Pseudodiverticula are herniations of only mucosa and submucosa, through defects in the muscular coat. Thus, the walls of pseudodiverticula are thinner and less stable than those of true diverticula. While true diverticula usually only occur one at a time, pseudodiverticula normally occur in multiple. True diverticula and pseudodiverticula are not endoscopically distinguishable.

A further distinction can be made with regard to pseudodiverticula, which can be further divided into incomplete and complete diverticula. Incomplete diverticula include only mucosal and submucosal layers and remain intramural, located in the muscle layer, while complete diverticula protrude beyond the circular muscle layer.

Inflammation. The presence of colonic diverticula without any complications is referred to as (colonic) diverticulosis. Diverticulitis occurs if one or more diverticula become inflamed (generally symptomatic).

Diverticulosis

Clinical Picture

Epidemiology. Colonic diverticulosis is a modern-day disease, first described by Cruveilhier in 1894. Since then, its prevalence has been increasing in industrialized countries, while remaining constant in developing nations. Mean frequency (combined age groups) in western industrialized nations is 20–40%; in Southeast Asia and Africa it is only 2–4%. Disease frequency is clearly associated with age: while diverticulosis seldom occurs among those under age 30, its prevalence among those over age 80 is 50–65%. Predisposition to diverticulosis has not been shown unequivocally to vary between men and women. However, in younger age groups, the disease appears to be slightly more common among men and in older age groups it appears to be more common among women.

Symptoms. Most individuals affected by diverticulosis remain asymptomatic over their lifetime; a minority of patients, 20–30%, exhibit clinically relevant complications (diverticulitis, stenosis, perforation, or bleeding). Nonspecific symptoms that may be related to diverticulosis include cramplike lower abdominal pain (usually located in the lower left abdomen), a feeling of fullness, bloating, and irregular bowel habits. Differentiation from diverticulitis is possible if these nonspecific symptoms are not accompanied by signs of inflammation. In some cases, it is impossible to differentiate between diverticulosis and irritable bowel syndrome and it is doubtful whether the symptoms are related to diverticulosis.

Etiology. Etiology and pathogenesis of diverticulosis are attributed to the low amount of fiber in the western diet, which leads to lower stool volume in the colon. This eventually causes a decrease in lumen diameter, which in turn increases wall tension of the colon according to the Laplace law. The sigmoid colon, in particular, can be affected, due to its intense contractions and high proportion of segmentation. Stool is condensed, thereby reducing stool volume in the sigmoid, and the width of the lumen is narrower than in the right hemicolon.

Diagnosis

Role of endoscopy. Diverticulosis is most often diagnosed using endoscopy or radiology with a contrast enema (Fig. 8.**1**). More recently, computed tomography has also been used. Diagnosis using sonography is also possible under good echo conditions. Especially in cases of less pronounced diverticulosis, diagnosis sometimes occurs incidentally during endoscopy or radiology of the colon or abdomen.

Colonoscopy is usually indicated as a diagnostic tool for known or suspected diverticulosis (more often for potential differential diagnoses than for main diagnosis), though it also can be used for treatment (Tab. 8.**1**). Endoscopic surveillance is not indicated for uncomplicated diverticulosis, not least due to risk of potential complications during endoscopy, but also given the lacking treatment corollary. Signs of acute diverticulitis are a contraindication for colonoscopy due to increased risk of perforation.

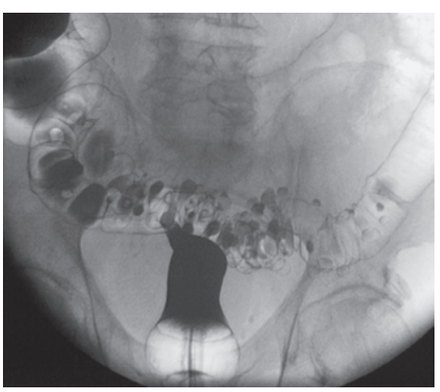


Fig. 8.**1** **Sigmoid diverticulosis, visible on radiographs with contrast enema.**

On the one hand, lacking stool volume and resulting increased wall tension (see above) lead to hypertrophy of the circular muscle layer, which can be seen endoscopically as a thickening of the circular muscle folds with an accordionlike shortening of the lumen (also referred to as myochosis) (Fig. 8.**2**). In cases of pronounced hypertrophy, the diverticula in the haustra between the folds are barely visible (Fig. 8.**3**). On the other hand, pseudodiverticula protrude through defects in the circular muscle layer. These defects, and thus the diverticula, are typically found at the points where blood vessels penetrate the muscle layer supplying the inner layers of the colonic wall (Figs. 8.**4**, 8.**5**). Decreasing elasticity of the colon wall with increasing age contributes to the formation of diverticula.

The causes mentioned above explain the more frequent appearance of diverticulosis in the left hemicolon (i. e., descending colon and especially sigmoid colon, Figs. 8.**6**–8.**9**). However, diverticulosis can affect the entire colon and occasionally there are cases where unexplained diverticulosis affects only the right hemicolon (Figs. 8.**10**, 8.**11**). For reasons that are unclear, diverticulosis of the right hemicolon more frequently involves complications (especially bleeding).

Table 8.**1** Indications for colonoscopy with known or suspected diverticular disease

Diagnostic indications
▶ symptoms of obstruction (alternating constipation and diarrhea, recent onset of constipation)
▶ unexplained abdominal pain (if accompanied by signs of inflammation, first conduct radiological or sonographic examination for signs of diverticulitis)
▶ prior hematochezia
▶ unclear radiological findings (e.g., to determine malignant/benign nature of stenoses, exclude neoplasia for hypertrophied folds or stool particles)
▶ preoperative to determine which colonic segments are affected by diverticula
Treatment indications
▶ hematochezia
▶ endoscopic treatment of postdiverticular stenoses (if surgical intervention has been declined or is considered to be risky due to accompanying disease)

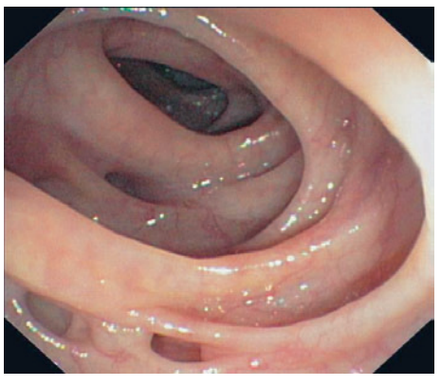


Fig. 8.**2** **Accordionlike lumen narrowing (myochosis)** and thickened folds caused by hypertrophied circular muscles, diverticulosis in the sigmoid colon.

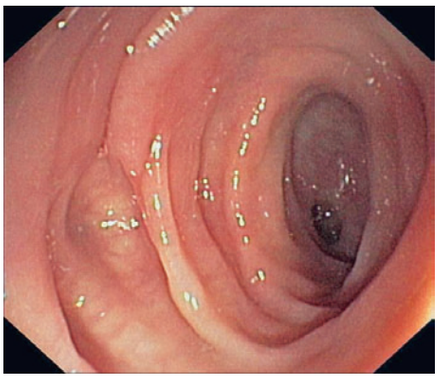


Fig. 8.**3** **Significant narrowing of the lumen (myochosis).** Visualization into the haustra harboring the diverticula is made more difficult.

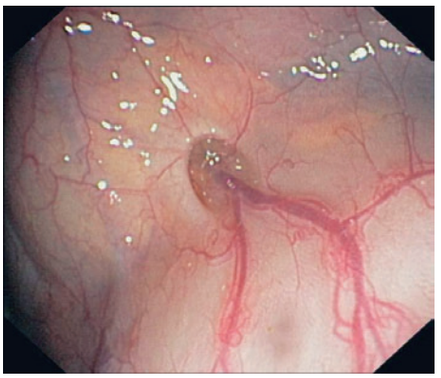


Fig. 8.**4** **Proximity of a diverticulum to a blood vessel,** which is penetrating the circular muscle layer, causing a weakness in the colon wall.

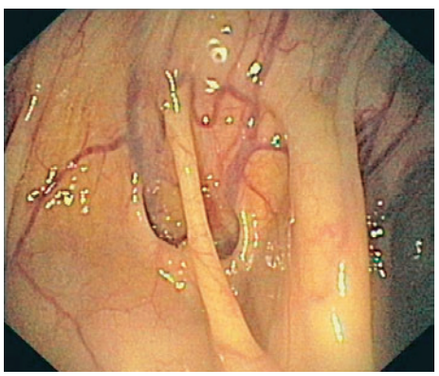


Fig. 8.**5** **Close proximity of diverticulum and blood vessels.** Slanted angle of entry of vessels into the wall and slanted path of diverticulum are clearly visible.

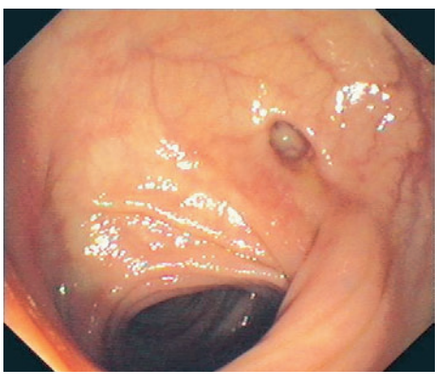


Fig. 8.**6** **Mild case of diverticulosis in sigmoid colon.** Small, single diverticulum, soft folds, no significant myochosis.

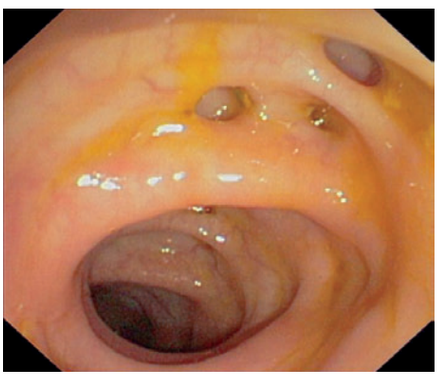


Fig. 8.**7** **Moderate case of sigmoid diverticulosis.** Several small diverticula, folds already somewhat thickened, no clinically relevant narrowing of the lumen yet.

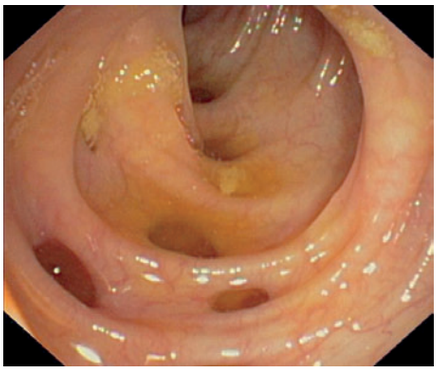


Fig. 8.**8** **More severe case of sigmoid diverticulosis.** Numerous diverticula, some already giant, folds are noticeably thickened, myochosis.

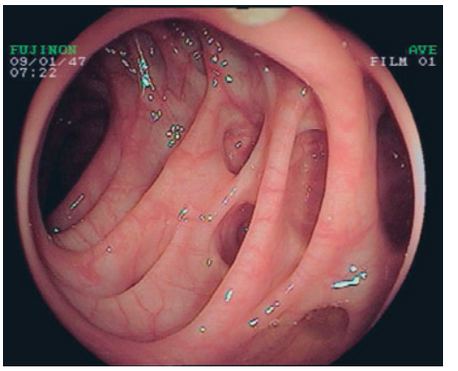


Fig. 8.**9** **Pronounced diverticulosis in the sigmoid.** Many large or very large diverticula, lumen is severely narrowed, more difficult view into haustra.

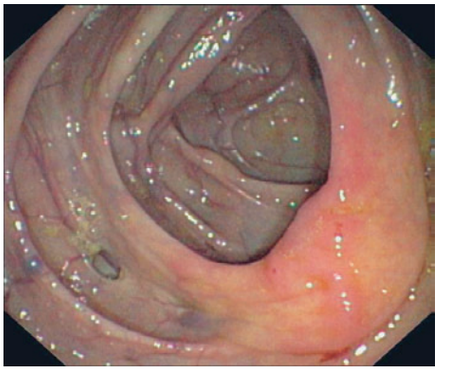


Fig. 8.**10** **Diverticulosis of the ascending colon.** Clearly recognizable ileocecal valve at right. Proximity of diverticulum to a blood vessel suggests a pseudodiverticulum.

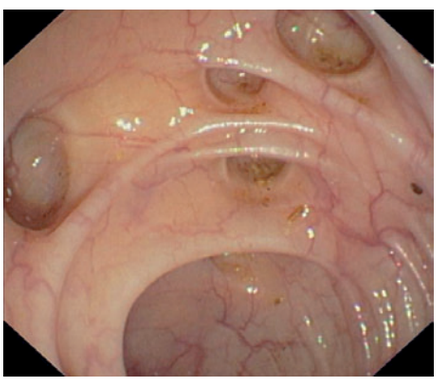


Fig. 8.**11** **Diverticulosis at the hepatic flexure.** The diverticula are still partly filled with feces.

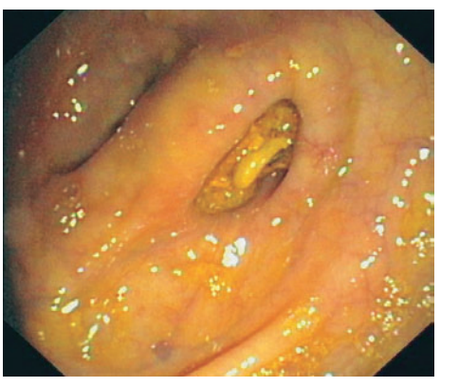


Fig. 8.**12** **Diverticulum with impacted feces.** Impacted feces impede bowel cleansing in patients with diverticulosis.

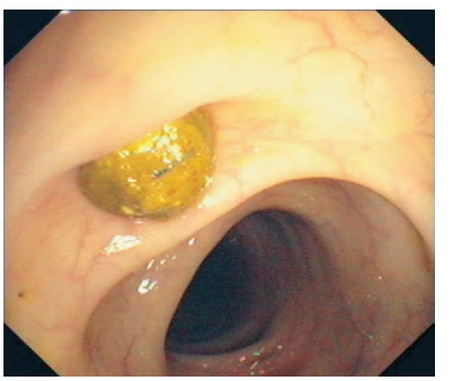


Fig. 8.**13** **Severely impacted and hardened stool (fecalith) in a diverticulum.**

Diverticulosis presents with high variability (▣ 8.**1**), ranging from solitary, barely detectable diverticula to multiple giant diverticula involving nearly the entire colon. The changes to the folds and the degree of compression and deformation of the lumen (myochosis) also varies greatly.

Endoscopic diagnostic criteria

- localization,
- size and number,
- condition of diverticula and affected colon segment.

Possible characteristics based on these criteria are listed in detail in Tab. 8.**2**.

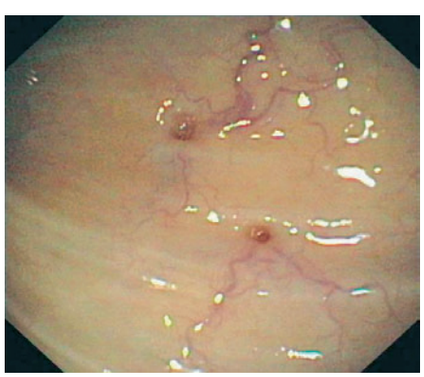
Examination procedure for diverticulosis

- Clinically relevant diverticulosis can present problems for colonoscopy. Narrowing and deformation of the lumen as well as thickening of the folds from muscle hypertrophy make identifying the colon lumen, for advancement of the instrument and visualization into the haustra, difficult. Thorough bowel cleansing is also difficult because the impacted stool is hard to mobilize out of the diverticula (Figs. 8.**12**, 8.**13**). During endoscopy, remaining stool particles can emerge from the diverticula and additionally obstruct visualization. Endoscopic examination must be performed cautiously on patients with diverticulosis. Mistaking a diverticulum for the lumen and advancing the endoscope into it can cause perforation. The endoscope must never be advanced "blindly," i. e., without clearly identifying the lumen. Some important characteristics for discerning between diverticula and intestinal lumen are listed in the following:

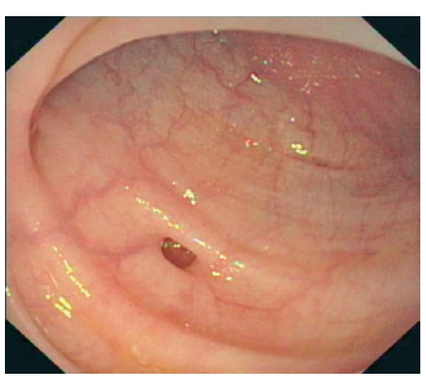
Table 8.**2** Endoscopic criteria of diverticular disease

Criteria	Description of findings
Localization of diverticulum	Include all affected colon segments, in the left hemicolon preferably with additional distance in cm (e.g., "distal sigmoid colon, 20–30 cm from the anus")
Number of diverticula	Rough quantitative classification, e.g.: ▶ solitary ▶ isolated (up to about 10) ▶ a few (10–20) ▶ multiple (> 20)
Size of diverticulum (▣ 8.1)	Rough quantitative classification, e.g.: ▶ barely visible (1–2 mm) ▶ small (around 3 mm) ▶ medium (around 5 mm) ▶ large (7–10 mm) ▶ very large (larger than 10 mm)
Diverticulum condition	▶ Contents, e.g., impacted stool (Fig. 8.**12**), fecaliths (Fig 8.**13**) ▶ Signs of inflammation: redness, swelling, secretion (e.g., pus) ▶ Signs of bleeding: erosion of diverticulum or diverticulum neck, adherent clot, blood vessel destruction
Condition of affected colon segment	▶ Muscle hypertrophy (myochosis) ▶ Stenosis, if present, list: – passability – approx. luminal width – stenosis length – probable genesis (inflammation, scarring, neoplastic) ▶ Possible external fixation (e.g., following prior diverticulitis)
Additional findings	Additional diagnoses independent of diverticula (e.g., polyps)

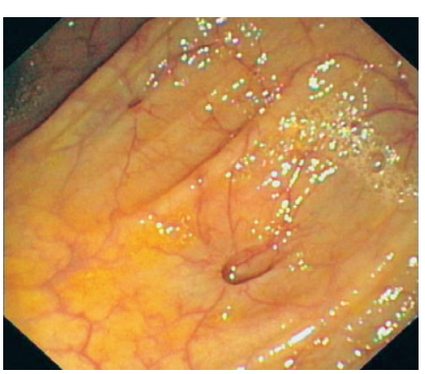
▣ 8.1 Various shapes and sizes diverticula



a

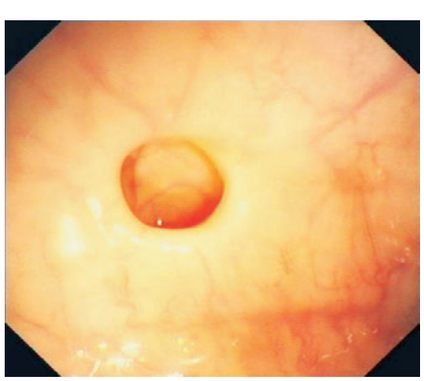


b

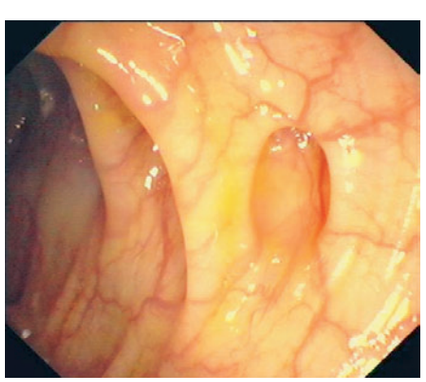


c

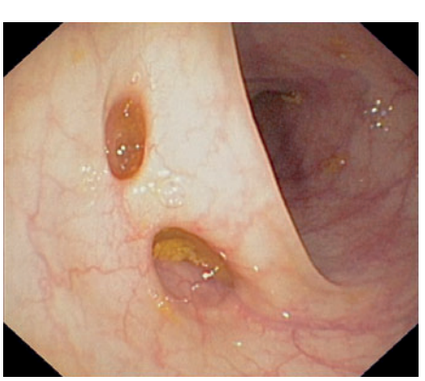
a–c very small and small diverticula (1–3 mm).



d



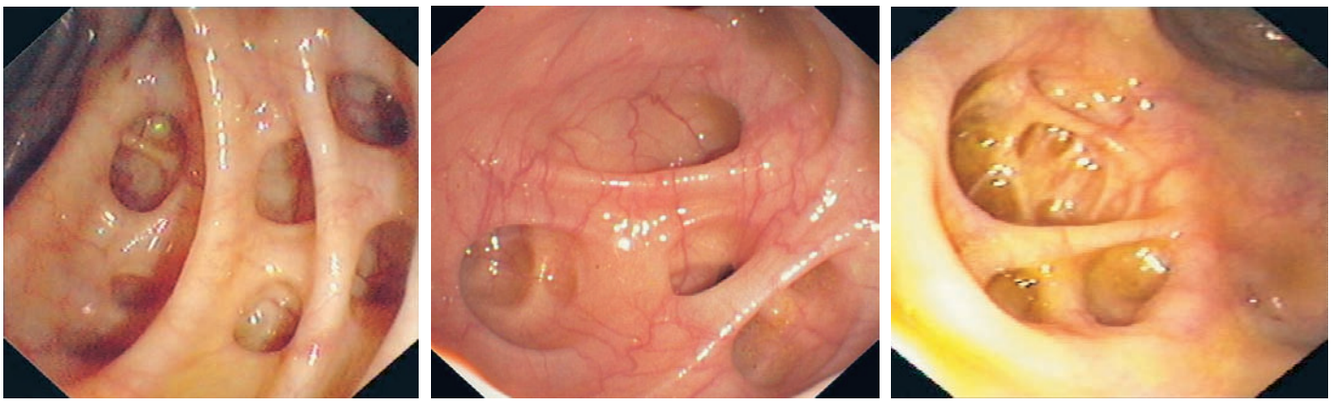
e



f

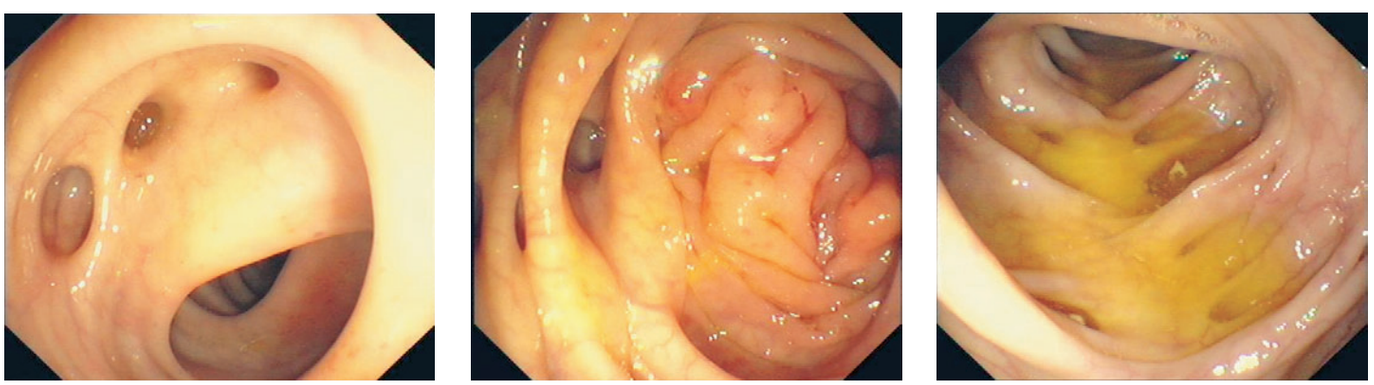
d–f medium-sized diverticula (around 5 mm).

8.1 cont.



g **h** **i**

g–i Large (about 7 mm) and very large diverticula (> 10 mm).
Proximity to blood vessels is readily visible for most diverticula. Large diverticula (**g–i**) sometimes have bronchial-like branching at the base of the diverticulum, as can be clearly seen in **i** with a ca. 12 mm giant diverticulum in the sigmoid colon. Such large diverticula always pose the risk of being confused with colonic lumen when advancing the instrument.



a **b** **c**

Fig. 8.**14** **Differentiating between diverticula and intestinal lumen.**
a Diverticular openings are round or oval, while intestinal lumen appears slitlike or half-moon shaped.
b Diverticula are located between haustral folds (left half of image); the folds converge in the luminal direction (right half of image).
c Diverticular openings are perpendicular to the longitudinal axis of the colon.

- Diverticular openings are, as a rule, round or oval, while the intestinal lumen between transverse folds of the colon is more slitlike or half-moon shaped (Fig. 8.**14 a**).
- Diverticular openings are located in the haustra, that is, between the transverse folds; the transverse folds often converge in the direction of the colon lumen (Fig. 8.**14 b**).
- Diverticular openings are perpendicular to the longitudinal axis of the colon. Looking into a diverticulum, the actual lumen is off to the side (Fig. 8.**14 c**).

If there is any uncertainty, withdraw and advance the colonoscope somewhat to perform a closer inspection before advancing it again. Changing the patient's position, or, in the case of pronounced muscle spasms, administering an antispasmodic (e.g., n-butylscopolamine; note contraindications), can be helpful.

▶ Unlike many other pathological findings, diverticula are generally better detected and inspected when advancing the instrument than when withdrawing it. Diverticula are located in the haustra, and, as more proximal colon segments tend to be shortened when the instrument is advanced, the viewing angle into the haustra is more advantageous on advancing than on withdrawal.

▶ Avoid forced air insufflation in patients with diverticulosis. Tension of the bowel wall can make insufflation painful; also, the thin diverticular walls (lacking muscle layer) make risk of perforation higher than among healthy patients. Biopsy of the diverticulum should also be avoided, given the thinness of the diverticulum wall.

▶ Occasionally a diverticulum is invaginated, protruding inward into the lumen. This is referred to as an "inverted diverticulum" (Fig. 8.**15**). Inverted diverticula can be confused with polyps, a dangerous mistake. Removal with a snare necessarily causes perforation and related adverse consequences. Unlike adenomas, inverted diverticula usually have a smooth surface and are the same color as the surrounding surface (Figs. 8.**15**, 8.**16**).

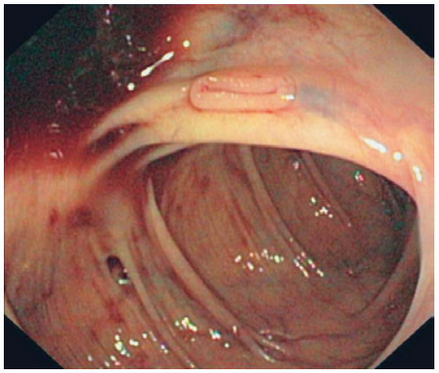


Fig. 8.**15** **Partially inverted diverticulum.** Note the smooth surface of the diverticulum and same color as surrounding area, characteristics differentiating a diverticulum from an adenoma.

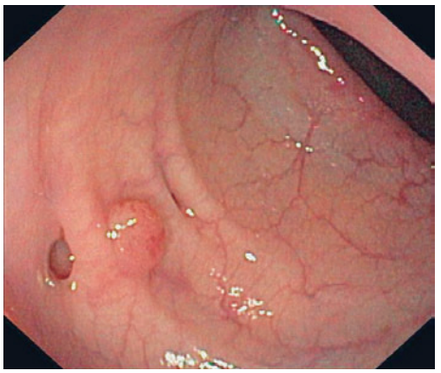


Fig. 8.**16** **Adenoma next to two small diverticula.** Fragmented reflection of light is a sign of irregular, rough surface. Appearance very different from inverted diverticulum (Fig. 8.**15**).

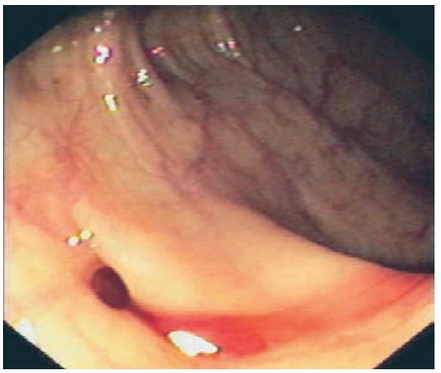


Fig. 8.**17** **Active hemorrhagic oozing from a sigmoid diverticulum** resulting from erosion of a vessel adjacent to the diverticulum.

Complications

As already mentioned, the majority of patients with diverticulosis (usually 20–30%) will not become symptomatic until complications arise. The two most common complications are, first, diverticular bleeding (Fig. 8.**17**), which frequently occurs with diverticulosis of the right hemicolon and simultaneous use of nonsteroidal anti-inflammatory drugs (NSAID) and affects some 10% of individuals with diverticulosis, and second, diverticulitis, which, including resulting complications (stenosis, abscess, perforation, and fistula formation) affects about 15–20% of individuals with diverticulosis and will be discussed in more detail in later sections.

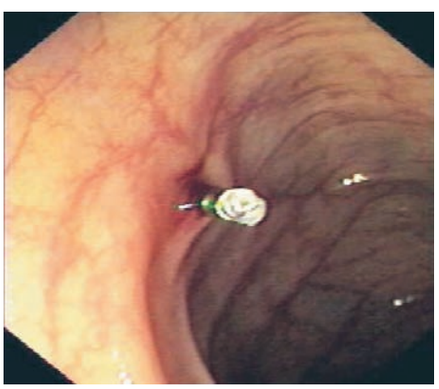


Fig. 8.**18** **Hemostasis by application of metal clip.**

Differential Diagnosis

Differential diagnosis becomes more difficult with the onset of clinically relevant complications arising from diverticular disease, which occur with diverticulitis attack or afterward. Cases involving stenosis present the problem of distinguishing between inflammatory and malignant stenosis, which can be difficult, especially using radiology. In most cases, differentiation can only be accomplished by means of endoscopy and targeted biopsy sampling. However, sufficient evaluation often requires passing the stenosis. If high-grade stenosis renders passage with a standard colonoscope impossible, a pediatric instrument or a gastroscope should be used. Distinguishing diverticulitis from an inflammation due to another reason (e.g., Crohn disease, infectious colitis, or ischemic colitis) in a colon segment with diverticula can be problematic. In isolated cases all available information must be gathered based on family history, clinical picture, endoscopy, histology, microbiology, and laboratory tests, in order to enable differential diagnosis.

Treatment

An increase in dietary fiber is sufficient for treating uncomplicated diverticulosis and no further specific therapy is necessary. Endoscopic intervention may be considered for acute diverticular bleeding or in isolated cases for strictures related to scarring following a prior diverticulitis attack.

Diverticular bleeding. If a bleeding diverticulum is identified during emergency colonoscopy (which is seldom possible given limited visualization), it is often possible to stop bleeding endoscopically. Local injection of diluted epinephrine solution (1:10 000 to 1:100 000), application of fibrin glue or metal clips (Fig. 8.**18**), or electrocoagulation can be used. Recurrent bleeding is an indication for resection of the affected colon segment.

Stenoses. Symptomatic diverticular stenoses, caused by scarring are also an indication for surgical intervention. They can, however, occasionally be treated by balloon dilation if surgical treatment has been declined or if it appears less than optimal due to accompanying disease. In such cases, the patient must be

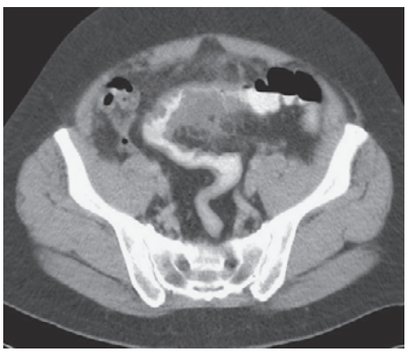


Fig. 8.**19** **Active diverticulitis in the sigmoid on a computed tomography scan using contrast agent administered rectally.** In the center of the image a highly inflammatory thickening of the sigmoid wall with inflammatory surrounding reaction and severely narrowed lumen only visible as a thin streak of contrast agent.

informed, however, that dilation is a difficult procedure due to the diverticula and that complications can occur (especially perforation).

Diverticulitis

Clinical Picture

Epidemiology. Diverticulitis is an inflammation of one or more diverticula, and is the most commonly occurring complication of diverticulosis, affecting some 20–25 % of individuals suffering from diverticulosis at least once in their lifetime. Around two-thirds of these experience only one episode of diverticulitis. Around one-third experience a relapse after the condition has been treated and has healed completely; the vast majority (up to 90 %) has a relapse within five years after the first attack.

Pathogenesis. Diverticulitis is caused by impacted stool in a diverticulum that damages the diverticular mucosa through compression and erosion and then quickly spreads via bacterial infection to the surrounding tissue (peridiverticulitis). The thin wall of the diverticulum is susceptible to microperforations, exacerbating the spread of the inflammation to the surrounding area. Surrounding tissues (fatty tissue and omentum) cover the microperforations, separating them from the abdominal cavity and free perforation in the abdominal cavity is rare, especially in early stages of diverticulitis. Inflammation usually is not limited to a single diverticulum, but affects a variously long colon segment. If inflammation spreads further to the surrounding area, abscesses and fistulas can develop in nearby organs. Fistula formation usually involves the bladder and vagina. The healing process causes, on the one hand, scarring and fibrozing, and thus adhesions and fixations between the affected colon segment and the surrounding area. On the other hand, scarring can also cause (sometimes high-grade) stenosis of the colon lumen.

Symptoms. The main symptoms of diverticulitis are pain in the lower left abdomen, fever, and positive laboratory tests for signs of inflammation (leukocytosis, increased CRP levels, and/or BSG rate). In a typical case, symptoms resemble those of appendicitis, except for their localization on the left side of the abdomen (hence: "left-sided appendicitis"). It goes without saying that in (rarely occurring) cases of diverticulitis of the right hemicolon (especially in the cecum or ascending colon), it is often impossible to differentiate clinically between diverticulitis and appendicitis. Other possible clinical symptoms include changes in bowel habits (usually constipation and even partial intestinal obstruction, occasionally diarrhea), nausea, vomiting, and dysuria and, in rare cases, light anal bleeding. Tenderness near the affected colon segment and localized guarding are usually evident during clinical exam. Resistance can also commonly be palpated in this area. It should be noted that, among patients who are immunosuppressed and older patients, diverticulitis could occur almost without the presence of symptoms.

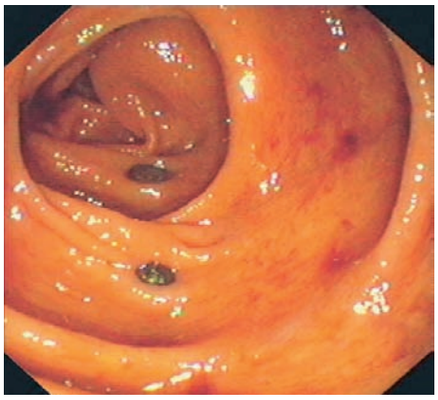
Diagnosis

Role of endoscopy. As already mentioned, signs of acute diverticulitis are a contraindication for colonoscopy, due to elevated risk of perforation. Computed tomography and sonography can be useful for diagnosing acute diverticulitis (Fig. 8.**19**), in particular, for documenting the extent of inflammation to neighboring areas and thus the appearance of clinically relevant complications such as abscess formation. These procedures therefore are preferable to contrast enemas using water-soluble contrast agents (gastrografin), which have been used in the past. Contrast enemas are, however, still used, especially in smaller units without ready access to computed tomography scans. Radiological criteria for diagnosing diverticulitis are irregularities in the contour of the diverticulum, with pointed, spiky outer contours, and narrowing of the affected colon segment, as well as evidence of contrast agent extravasation to the surrounding area. After the attack has abated, the indications for colonoscopy are those previously mentioned for colonoscopy in the case of diverticulosis (see Tab. 8.**1**).

If endoscopy must nevertheless be performed on a patient with acute diverticulitis because of unclear findings or atypical symptoms, the findings described in Tab. 8.**3** may be present, depending on the severity of disease.

Early endoscopic signs of acute diverticulitis include edematous swelling of the transverse folds and narrowing of the lumen in the affected colon segment due to swelling and the usually contemporaneous spastic muscle contractions. Reddening of the mucosa is often evident and it can be spotted or, in more severe cases, patchy; vascular pattern is often obscured and no longer sharply demarcated (Fig. 8.**20**).

In severe forms of diverticulitis, inflammation spreads beyond the immediate area around the diverticula openings (Fig. 8.**21**) to the mucosa in between (Figs. 8.**22**, 8.**23**). Occasionally, an inflamed diverticulum will have purulent secretion (Figs. 8.**24**, 8.**25**) and in extreme cases, formation of an intramural abscess may occur with intense reddening and narrowing of the lumen. Only seldom (usually an incidental finding) is inflammation limited to only one diverticulum whereby reddening and erosion at the neck of the diverticulum are limited to the diverticulum and do not affect the surrounding area (Fig. 8.**26**). In such cases, the patient may be clinically asymptomatic.



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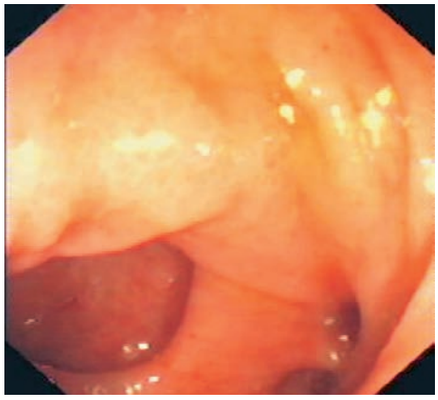

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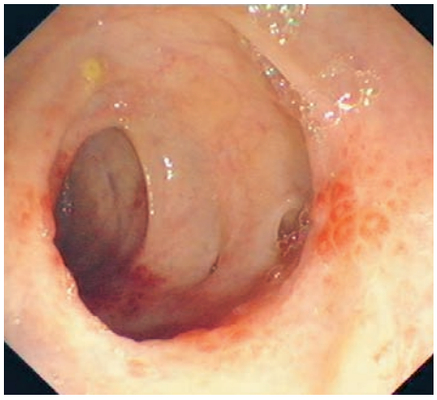

c

Fig. 8.**20** **Early phase of diverticulitis.**
a The folds are edematous (especially in the background), the mucosa has reddened, patchy areas. Stool-filled diverticulum.
b Edematous folds, moderate narrowing of the lumen due to edema and spastic muscle contraction.
c Patchy areas of reddened mucosa, relatively mild edema. Vessel pattern is partly obscured, but still discernible.

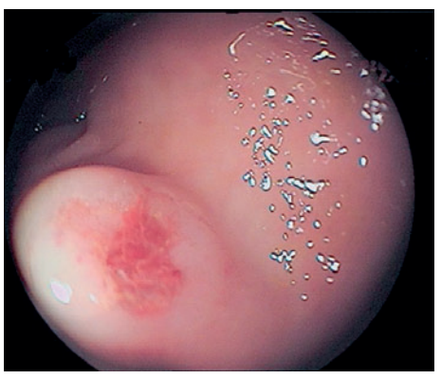

Fig. 8.**21** **Close-up view of diverticulitis.** Ringlike edema around diverticulum base. The opening is severely reddened; streaked, purulent secretion beginning to form at the base and the diverticulum opening is completely closed due to swelling.

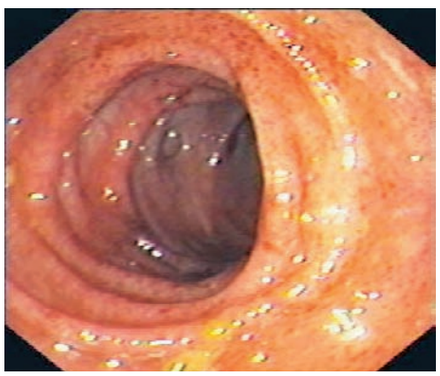

Fig. 8.**22** **Moderate case of diverticulitis.** Spread of inflammation in a longer colon segment. The mucosa is significantly reddened, vessel pattern obscured, edematous folds. A diverticular opening is visible in the distance.

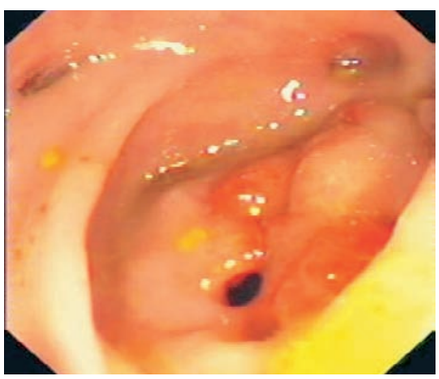

Fig. 8.**23** **Severe diverticulitis.** Inflamed, high-grade stenosis of the lumen. Note the severe edematous changes, partly raised and polyplike reddened folds at the edge of the rest of the lumen.

Examination procedure for diverticulitis

- Proceed very carefully if acute diverticulitis is detected during colonoscopy. In particular, avoid overly insufflating the colon with air given the risk of perforation.
- The instrument must be advanced carefully if there is narrowing of the lumen. If there is any uncertainty, the examination should be stopped and postponed until the diverticulitis abates.

Table 8.**3** Endoscopic signs of diverticulitis

Early signs (Fig. 8.**20**)	▶ edematous swelling of folds ▶ narrowing of lumen ▶ spastic muscle contractions ▶ obscured vessel pattern ▶ mucosal erythema
More severe inflammation (Figs. 8.**21**–8.**23**)	▶ severe swelling of folds ▶ increased narrowing of lumen ▶ patchy reddening of mucosa ▶ loss of vessel pattern
Severe inflammation (Figs. 8.**24**, 8.**25**)	▶ purulent effusion ▶ intramural abscess formation ▶ high-grade stricture ▶ substantial signs of inflammation of mucosa (intense reddening and swelling, vulnerability)

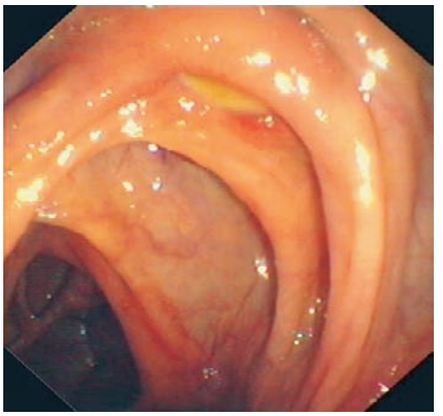

Fig. 8.**24** **Severe diverticulitis** (shown here in the ascending colon) with purulent secretion from the diverticulum (at about the 12-o'clock position). Visible reddening on the edge of the diverticulum.

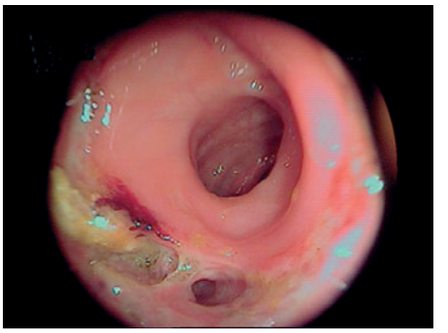

Fig. 8.**25** **Substantial purulent secretion** from a sigmoid diverticulum, severe diverticulitis. The diverticular neck is eroded and bloody, erosive changes extending from the diverticular neck toward the right and into the haustra.

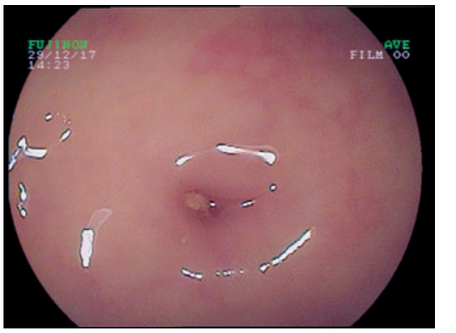

Fig. 8.**26** **Incidental finding: localized diverticulitis** in an asymptomatic patient. Stool particles visible in the diverticulum. The diverticular neck is reddened and edematous. The area surrounding the diverticulum does not exhibit any significant inflammatory changes.

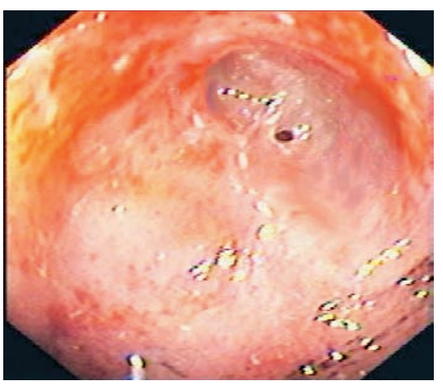

Fig. 8.**27** **Central dotlike fistula opening** in a diverticulum with inflammatory changes; a complication of diverticulitis.

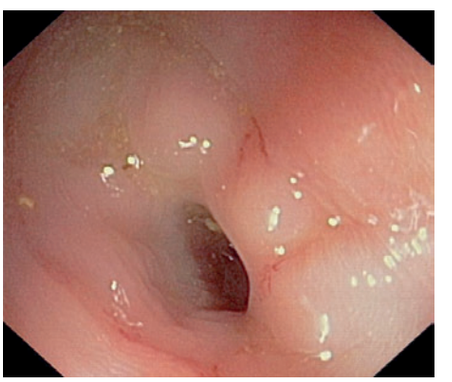

Fig. 8.**28** **Post-diverticulitis stenosis in the sigmoid colon, no longer passable.** The surrounding folds are thickened and converge in the direction of the stenosis.

Complications

Around 20 % of patients with acute diverticulitis develop clinically relevant complications. These include abscess formation, perforation, and fistulas (Fig. 8.**27**), generalization of inflammation in terms of sepsis, bleeding, and development of a stenosis related to scarring and no longer capable of regression (Fig. 8.**28**). As a rule, these complications require surgical repair. Two exceptions are, first, minor and generally self-limiting bleeding, and, second, in isolated cases, stenoses due to scarring for which treatment can be attempted using balloon dilation via endoscopy.

Differential Diagnosis

Differential diagnosis includes distinguishing diverticulitis from an acute attack of chronic inflammatory bowel disease (ulcerative colitis or Crohn disease), acute colitis of another genesis (ischemic colitis, infectious colitis), as well as colonic malignancy, the latter especially in relation to the development of permanent stenosis. In patients with seldom-occurring diverticulitis of the right hemicolon, distinguishing diverticulitis from appendicitis is difficult and in rare cases impossible. Differential diagnoses also include extraintestinal disease: primarily gynecological disorders (inflammatory or tumorous diseases of the adnexa and ovaries, endometriosis, extrauterine pregnancy), and also urological disorders (ureter stones, bladder tumors).

Treatment

Treatment of uncomplicated diverticulitis is conservative: temporary liquid diet, antibiotics (usually a combination of broad spectrum penicillin or a broad spectrum cephalosporin with metronidazole, alternatively ciprofloxacin orally for mild diverticulitis), and analgesics. There are no endoscopic treatment options.

Surgical intervention is indicated for complicated diverticulitis and recurrent diverticulitis. Surgical intervention should also be considered at the first occurrence of uncomplicated diverticulitis in patients who are immunosuppressed, in patients less than 50 years of age, and for diverticulitis of the right hemicolon.

Only in isolated cases does endoscopy play a role in complicated diverticulitis, i. e., for seldom-occurring clinically relevant bleeding and stenosis resulting from scarring, for which surgical treatment is not possible.

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9 Polyps and Polyposis Syndromes

T. Eberl

Polyps

Definition

The term "polyp" describes a mass of tissue protruding into the lumen of the bowel, without implying pathological relevance. Polyps can be stalked, round, or sessile and can vary in size. They can occur as solitary or multiple polyps. Polypoid lesions are the most common pathological finding of colonoscopy. Polyps can only be classified by histological evaluation.

Pathologically and anatomically, polyps of epithelial tissue are distinguished from those of mesodermal tissue and there are also a few other special forms. Table 9.**1** shows the WHO classification of colorectal polyps. According to WHO guidelines, epithelial polyps are divided into neoplastic and nonneoplastic (tumorlike) polyps, whereby neoplastic polyps make up some 70% of all colorectal polyps (3).

Table 9.**1** WHO classification of colorectal polyps

Neoplastic polyps	Nonneoplastic (tumorlike) polyps
▶ adenoma	▶ Peutz–Jeghers polyp
▶ polypoid carcinoma	▶ juvenile polyp
▶ carcinoid tumor	▶ hyperplastic polyp
▶ nonepithelial tumors (lipoma, leiomyoma, hemangioma, lymphangioma, etc.)	▶ benign lymphoid polyp ▶ inflammatory polyp

Table 9.**2** Distribution of colorectal polyps (Erlangen Registry of Colorectal Polyps, 1978–1993) (3)

Bowel segment	Percentage of colorectal polyps
Cecum	4%
Ascending colon	10%
Hepatic flexure	3%
Transverse colon	9%
Splenic flexure	2%
Descending colon	8%
Sigmoid colon	30%
Rectum	34%

Table 9.**3** Relationship between histology and transformation potential of various epithelial polyps (3, 5)

Histology	Percentage of all polyps	Malignant transformation at diagnosis up to
Tubular	75%	4.8%
Tubulovillous	15%	19.0%
Villous	10%	38.4%

Clinical Picture and Clinical Significance

Epidemiology. The prevalence of colorectal polyps varies greatly in different regions of the world, though it tends to be higher in western industrialized nations with high standards of living. Colon polyps are, on the whole, very common and their frequency increases with age. Some 70% of all colon polyps are adenomas, and 65% of these are located between the rectum and splenic flexure. Between 30–50% of patients with adenomas have several adenomas simultaneously; the most significant factor determining the presence of an adenoma is being older than 60 years of age. Table 9.**2** shows the approximate distribution of colorectal polyps by intestinal segment (3).

Symptoms. Most polyps do not cause any symptoms. Possible symptoms include anal bleeding (unexplained iron deficiency anemia) and signs of obstruction accompanied by abdominal pain (larger polyps). Villous adenomas have a special status related to their clinical picture as they can secrete a watery potassium-rich secretion, occasionally up to several liters per day. The patient thus suffers from watery diarrhea and mucous discharge; laboratory tests may detect hypokalemia.

Adenoma–dysplasia–carcinoma sequence. Adenomas deserve special attention as they make up 70% of all colon polyps. An adenoma is a benign neoplastic epithelial tumor in which cells arise from glandular epithelium with varying degrees of dysplasia and varying potential for malignant transformation. Current opinion holds that every adenoma has the potential to become a carcinoma (adenoma–dysplasia–carcinoma sequence). The "National Polyp Study" and the Erlangen Registry of Colorectal Polyps have identified adenoma size and polyp histology as independent risk factors in the development of a carcinoma (3, 5). The rate of malignancy increases with increasing size: at 2 cm and larger, 12% of adenomas have high-grade dysplasia and 33% of adenomas contain carcinomatous components. The relationship between histology and transformation of various epithelial polyps is shown in Tab. 9.**3**.

Atypical cells can be found in 10% of all adenomas, whereby initial proliferation is limited to a small area of the polyp. Later, the malignant cells spread, infiltrating the polyp stalk, and, finally, the intestinal wall.

Diagnosis

Diagnosis of colon polyps
- family history,
- clinical examination (rectal digital examination),
- occult blood test,
- **total colonoscopy**,
- (double) contrast enema,
- virtual colonoscopy (?).

Family history and clinical findings form the basis for diagnosis. Family history is especially important for hereditary forms of polyposis syndrome and where there is a family history of colorectal adenomas and carcinomas. Digital rectal palpation makes an important contribution to clinical findings, but it only detects only around 5–10% of polyps. Occult blood tests are not suitable as a primary screening tool for adenomas, as their sensitivity for adenomas is only 30%. Occult blood tests are negative for 93% of colon adenomas up to 1 cm in size and for 77% of adenomas 1–2 cm in size, whereby 7% of polyps of this size are malignant. Twenty-five percent of polyps larger than 2 cm escape detection and of these up to 45% are already transforming malignantly.

Role of endoscopy. Endoscopy, i. e., total colonoscopy, is essential and it is the main diagnostic tool. Endoscopy is the most informative method of diagnosing colorectal polyps. Endoscopic biopsy—or ectomy—of polyps initially has a purely diagnostic role. Not until the results of histological evaluation are available can it be decided whether endoscopy is the definitive treatment method or whether other treatments (removal of remaining polyp pieces, surgical intervention) are required. Early-stage polyps can be detected and removed endoscopically, thereby reducing the incidence of carcinoma.

Complementary procedures. Barium enemas can be used complementarily, e.g., for strictures, endoscopically impassable segments, or in cases where endoscopy is not possible. Contrast enemas should only be administered using double-contrast technique; false negative findings occur in 2–22% of patients using this technique, depending on size.

The role of virtual colonoscopy in diagnosing colon polyps remains unclear. Virtual colonoscopy can, however, hardly replace conventional colonoscopy as a screening tool: sensitivity for adenomas < 5 mm is around 30–50%; for adenomas 6–9 mm, sensitivity is at the most 80% and for adenomas > 1 cm ca. 90% (2). In more recent studies, results of virtual colonoscopy are even less impressive: the rate of detection of polyps > 1 cm is reported at 70% and among polyps 5–9 mm at only 40–60% (4). Thus, for polyps detected by virtual colonoscopy, conventional colonoscopy is absolutely essential for ectomy of the polyp; currently 20% of detections are false positives (2).

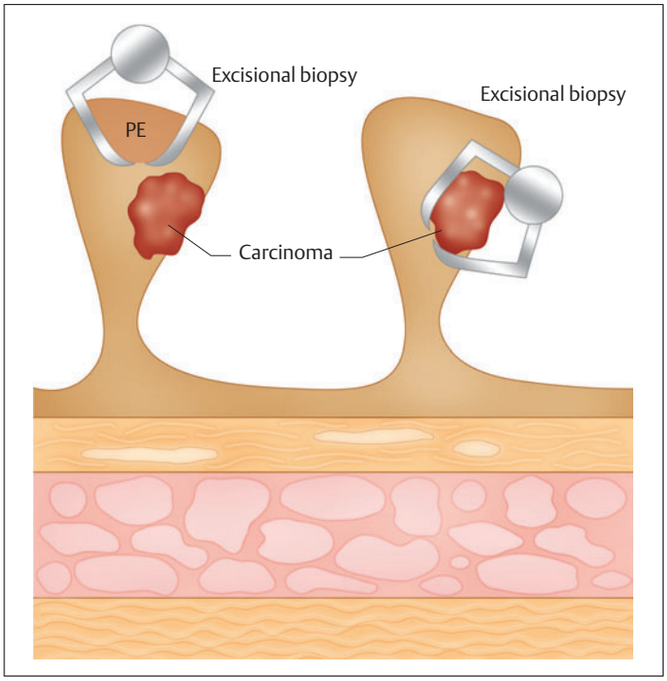
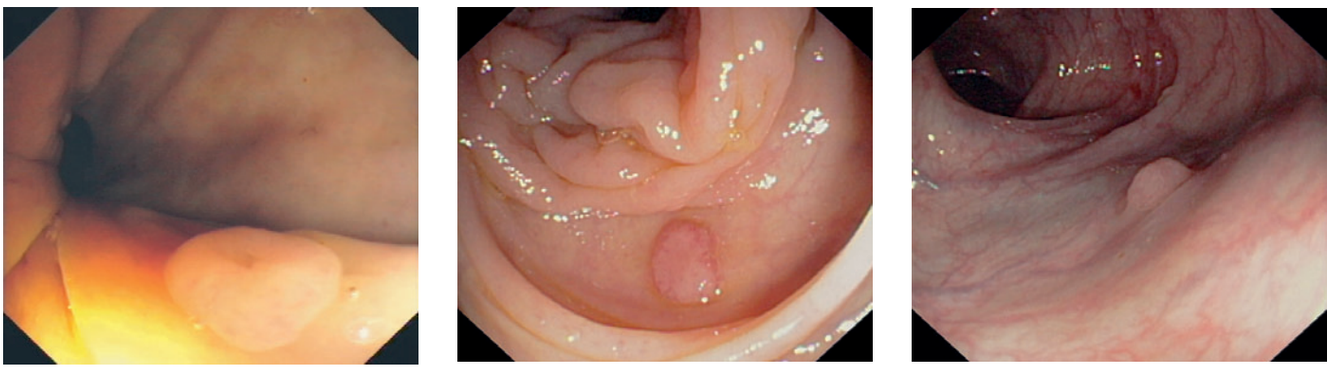


Fig. 9.1 **Problem of forceps biopsy for polyps with and without carcinomatous components.**

Examination procedures for colon polyps
- Total colonoscopy, i. e., inspection of the entire colon to the cecum, is especially vital for colon polyps. It is the main diagnostic tool and in most patients the primary treatment procedure as well.
- Rectosigmoidoscopy alone detects ca. 63% of all colorectal polyps. If an adenoma is detected, total colonoscopy is necessary to exclude the presence of further adenomas.
- Macroscopic diagnosis of a polyp necessitates its histological evaluation. With the exception of nonneoplastic polyps, which are histologically uniform, a forceps biopsy of an adenoma does not offer a reliable classification. For specific classification of adenomas, histological evaluation of the entire polyp is necessary. The polyp can be removed by endoscopic polypectomy or by surgical means.
- Very small polyps (up to 5 mm) can be removed with forceps. Total ectomy is mandatory for adenomas larger than 5 mm.

Follow-up studies on colon polyps (using double-contrast techniques) are available from "precolonoscopy" times. Of polyps larger than 1 cm, 37% grew in size during an observation period of nine years; cumulative carcinoma risk after five, 10, and 20 years was 4%, 14%, and 35% respectively (10). Based on these data, total polypectomy is indicated for all colon polyps larger than 5 mm (10), since differentiation between adenomas and adenocarcinomas is not possible based on biopsy alone (Fig. 9.1). Histological evaluation is essential for every resected polyp and must include histological classification, degree of in-

9.1 Diminutive polyps



a **b** **c**

a–c Small adenomas, less than 5 mm, covered by apparently normal mucosa, flat, sessile.

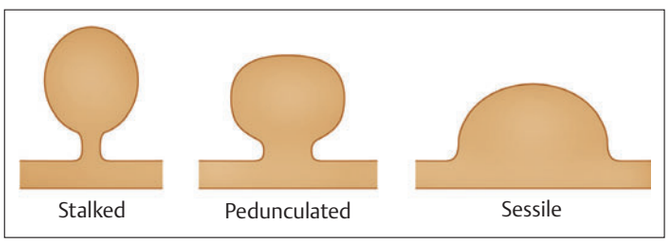


Fig. 9.**2** **Macroscopic growth forms of colorectal adenomas.**

traepithelial neoplasia and report of excisional biopsy. Chapter 18 goes into more detail concerning polypectomy technique.

Prospective colonoscopy studies have shown that if polyps smaller than 5 mm without adenoma components (hyperplastic polyps) are detected in the rectum or sigmoid colon, the prevalence of metachronous adenomas in proximal colon segments is 24–34% (1). Thus, distal hyperplastic polyps can be considered index lesions for more proximal metachronous adenomas.

Epithelial Polyps

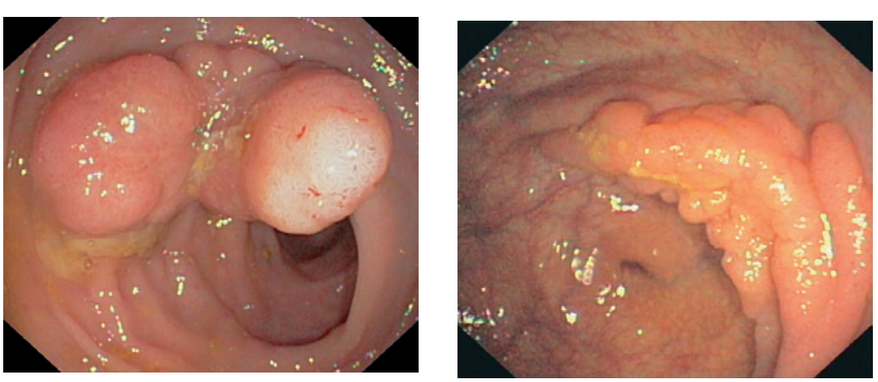
Adenomas. Typical appearance of colon adenomas varies. Differentiating between an adenoma and a hyperplastic polyp is only possible using special endoscopic techniques, in particular, chromoendoscopy and "pit pattern classification" (see Chapter 3).

The smallest adenoma can measure less than 5 mm, is covered by normal-appearing mucosa, and is flat and sessile, located on the mucosal surface, and occasionally appearing reddish in color. Polyps < 5 mm are referred to as "diminutive polyps" (▣ 9.**1**). Histologically, they are characterized by closely situated, branchlike tubuli, surrounded by lamina propria. Larger adenomas are characterized as sessile or stalked: sessile adenomas have a broad base, while stalked adenomas have a type of stalk of varying length (Fig. 9.**2**).

Macroscopically visible endoscopic criteria for adenomas

- Sessile (▣ 9.**2**), stalked (▣ 9.**3**),
- Surface: normal or mildly erythematous (▣ 9.**2 c**),
- Contour: smooth (▣ 9.**2 d**, **e**), lobed (▣ 9.**2 f–h**).

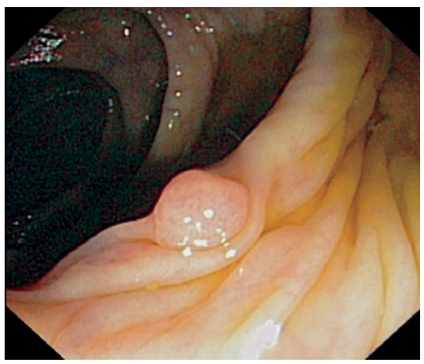
9.2 Sessile polyps



a **b**

a, b Broadbased, sessile polyps on mucosal surface.

9.2 cont.



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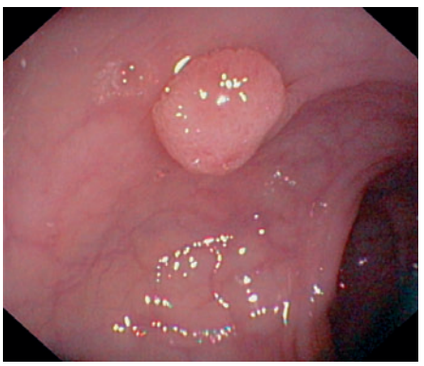

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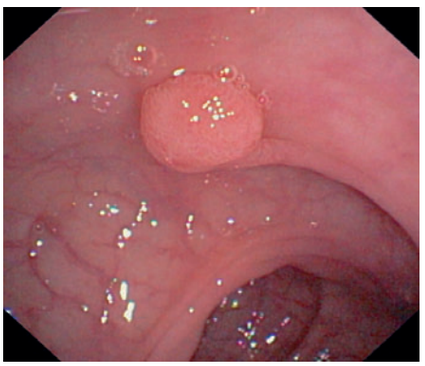
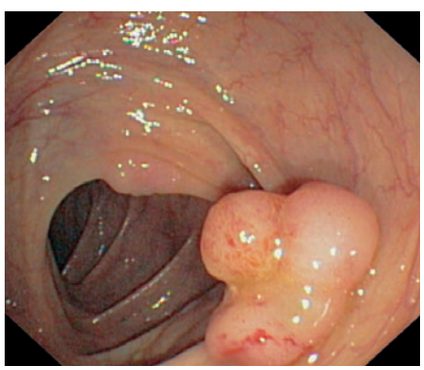


e

c–e Pedunculated polyps with mildly erythematous surface (**c**) and smooth contours (**d**, **e**).



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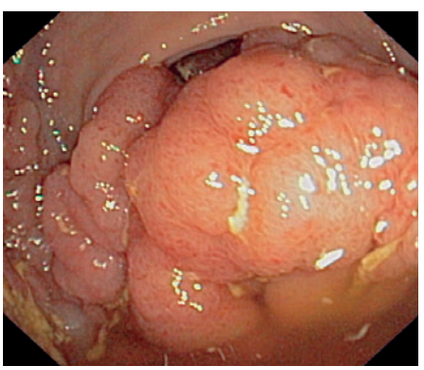

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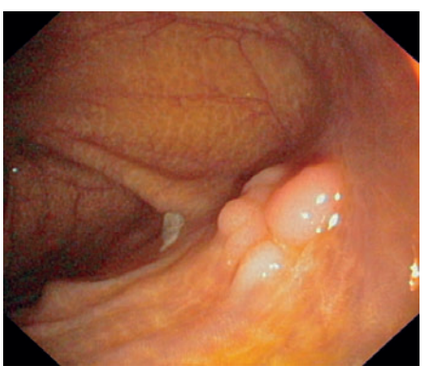
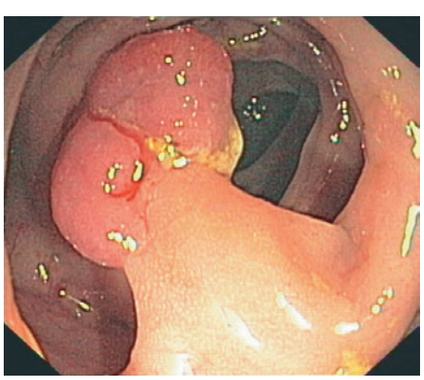


h

f–h Sessile polyp with lobular surface.

9.3 Stalked polyps



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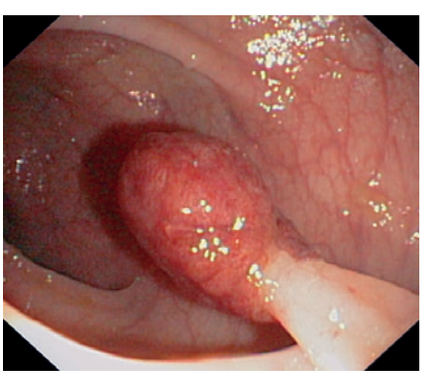

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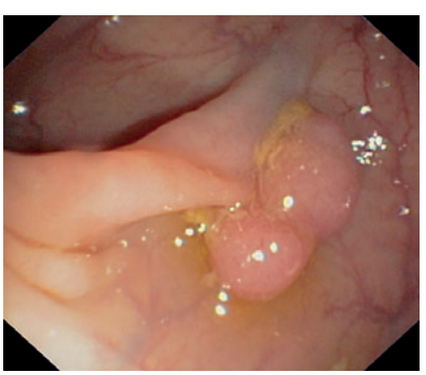

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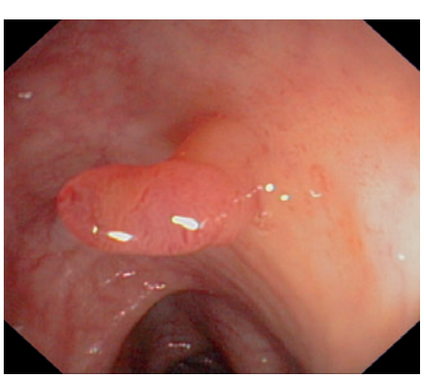
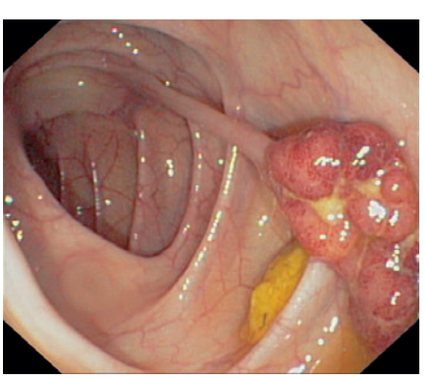


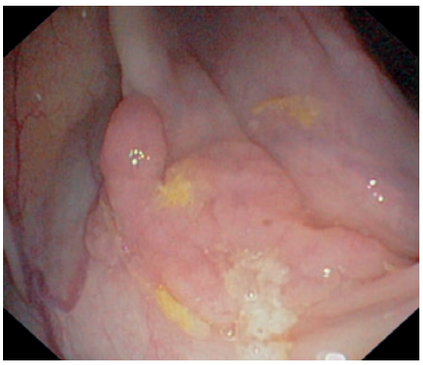
d



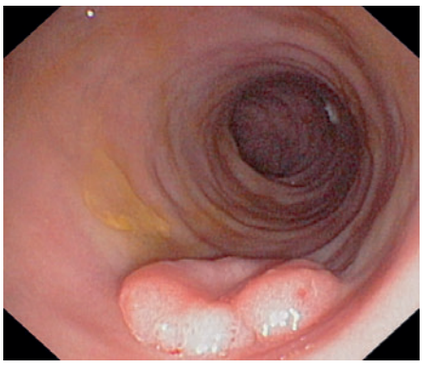
e

a–e Stalked polyps with stalks of varying lengths and thicknesses.

9.4 Flat adenomas



a



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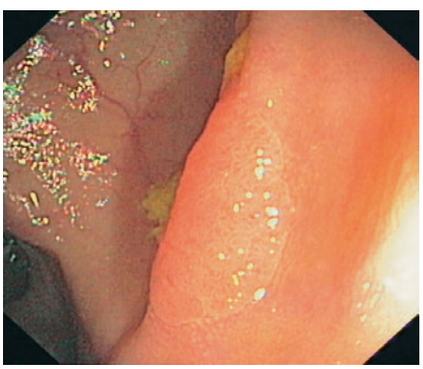

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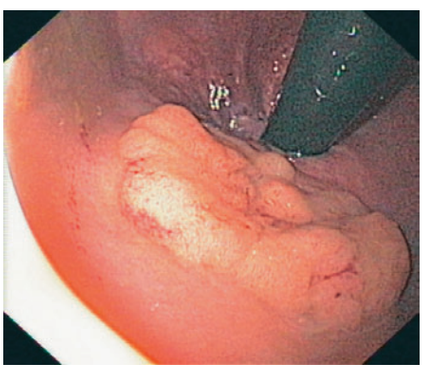
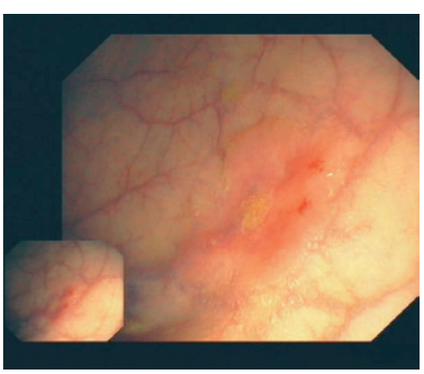


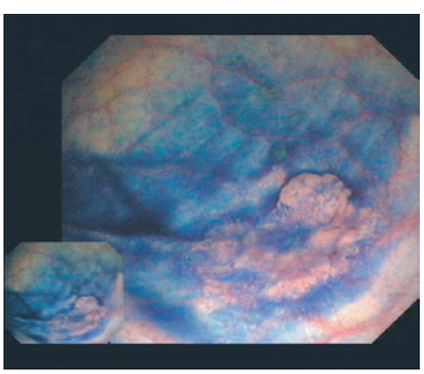
d

a–d Flat adenomas with only slightly elevated surface and a shallow depression in the center.

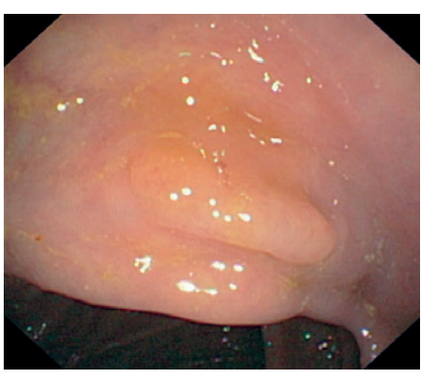
9.5 Polyps before and after staining



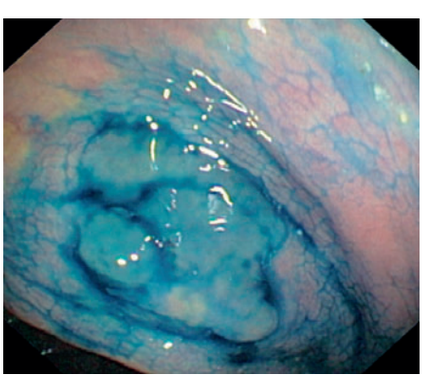
a



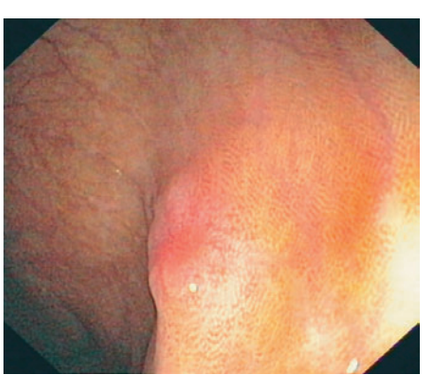
b



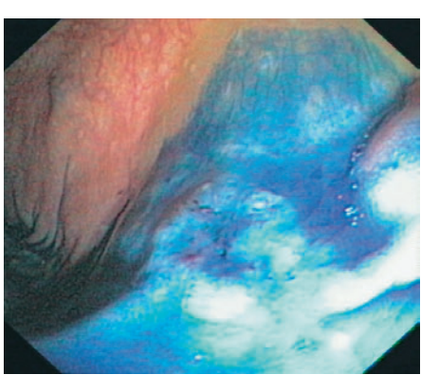
c



d



e



f

a–f The exact extent of flat adenomas can be better ascertained using staining (chromoendoscopy).

Table 9.**4** Endoscopic characteristics of histologically different adenomas (9)

Endoscopic characteristics	Tubular adenoma (▣ 9.6)	Tubulovillous adenoma (▣ 9.7)	Villous adenoma (▣ 9.8)
Shape	Sessile or stalked	Sessile or stalked (thicker stalk than tubular adenoma), medium sized	Sessile, often > 3 cm
Surface	Normal to slightly erythematous	Erythematous	Pale yellow, irregular
Contour	Smooth to lobular	Nodular, lobular	Nodular, fingerlike processes, diffuse and tubular growth

Continually improving image resolution and special endoscopic techniques have made it possible to detect even very flat adenomas which are only slightly elevated and sometimes have a shallow depression in the center. These are called "flat adenomas" (▣ 9.**4**). Chromoendoscopy can help better ascertain exact extent of invasion and borders (▣ 9.**5**). Flat adenomas can be indicators of dysplasia or even malignant transformation. (Figs. 9.**3**, 9.**4**).

In one prospective study of a large patient sample, 36 % of all adenomas found during routine colonoscopy were "flat adenomas," 4 % of the smaller "flat adenomas" (< 10 mm diameter) and 29 % of the large "flat adenomas" (≥10 mm diameter) were found to contain severe dysplasia or an adenocarcinoma (7).

Histologically, adenomas are divided into two main types: villous and tubular. Villous adenomas often have a diameter of more than 3 cm, are sessile and often malignantly transforming. The tubulovillous adenoma consists of tubular and villous adenoma components (Fig. 9.**5**). Endoscopic characteristics of the histologically different adenomas are shown in Tab. 9.**4** and ▣ 9.**6**–9.**8**.

Cancerous adenomas. Adenomas are neoplastic and can become cancerous. Some polypoid structures are made up of carcinoma tissue and contain few or no adenoma components.

Surface changes are especially characteristic of malignant transformation: deformation, ulcerated polyp head, granular or friable surface, or bleeding (▣ 9.**9**). In addition, the polyp usually appears to have a rather firm consistency (▣ 9.**9e**). If the adenoma can be depressed with a biopsy forceps into the intestinal wall, it is unlikely that deeper intestinal wall layers have been invaded.

Histological evaluation of a cancerous polyp determines further treatment. Endosonographic staging can assist in determining depth of infiltration of the bowel wall and thus can help determine treatment method.

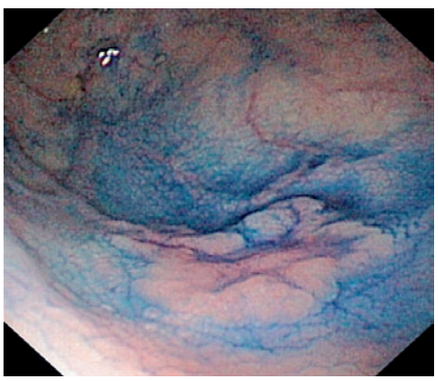


Fig. 9.**3** **Flat adenoma with depression in center,** stained with indigo carmine. Histologically severe dysplasia in transition to an adenocarcinoma.

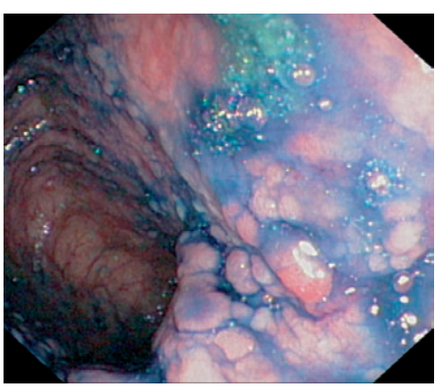


Fig. 9.**4** **Flat, villous adenoma,** presenting as multiple polyps (chromoendoscopy with indigo carmine). Histologically malignant transformation of the irregular surface at top edge of picture.

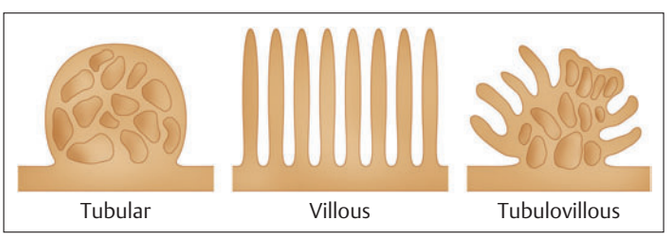
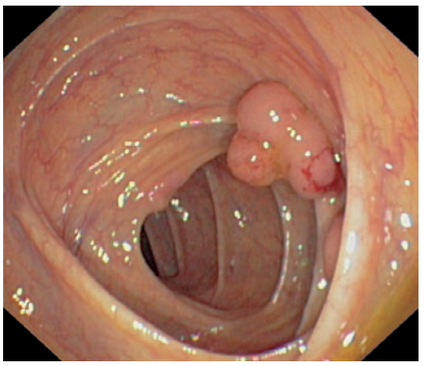
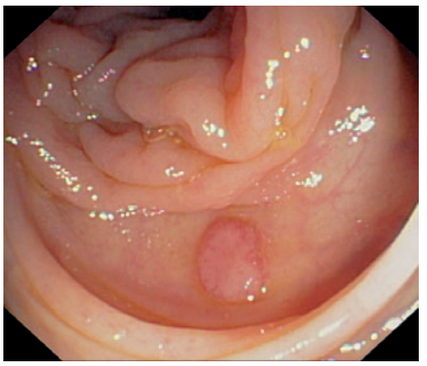


Fig. 9.**5** **Histological types of adenomas.**

9.6 Tubular adenomas

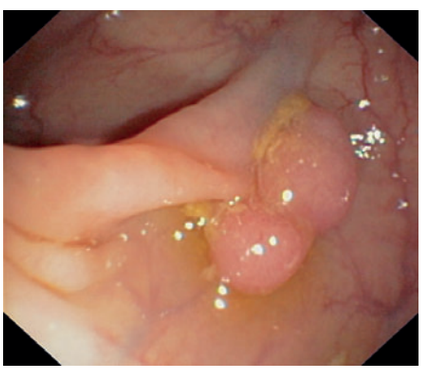


a

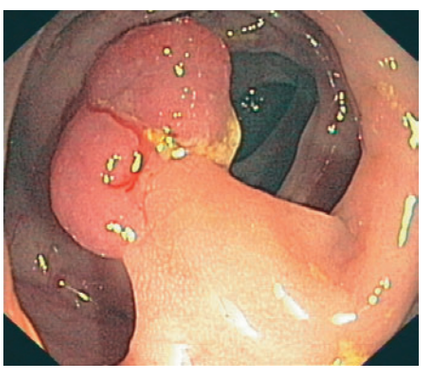


b

a, b Small, sessile tubular adenomas, normal to slightly erythematous surface, lobular contour.



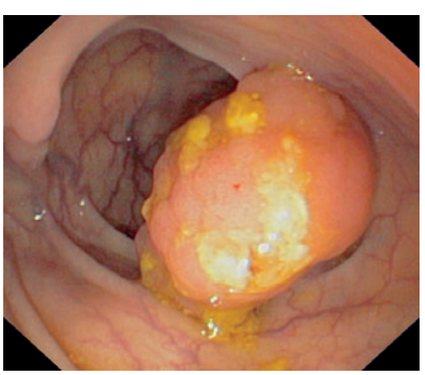
c



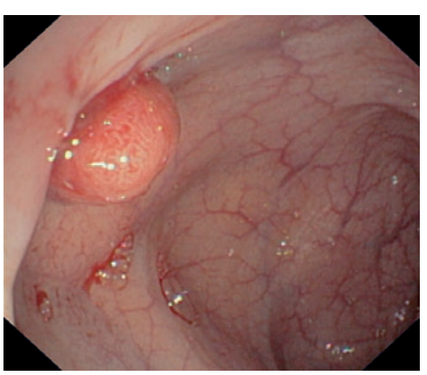
d

c Stalked tubular adenoma with lobular contour.
d Stalked tubular adenoma with smooth surface.

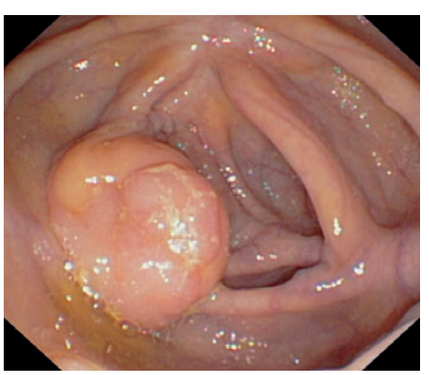
9.7 Tubulovillous adenomas



a

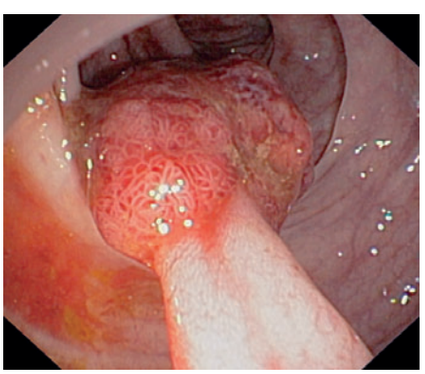


b

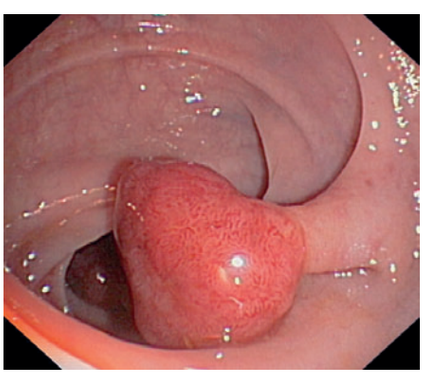


c

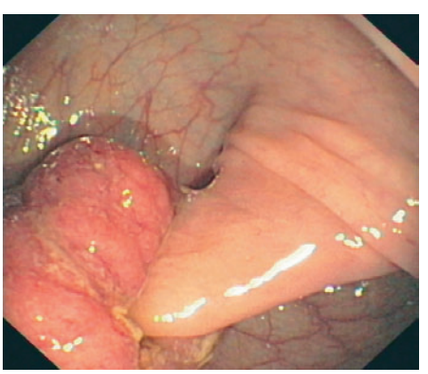
a–c Sessile, tubulovillous adenomas with erythematous surface and lobed contour.



d



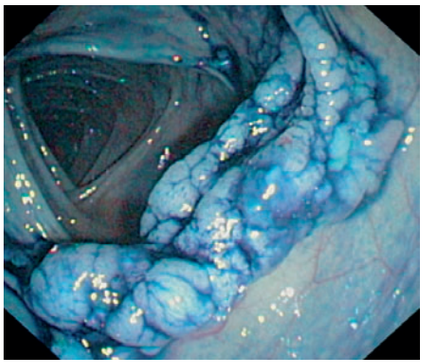
e



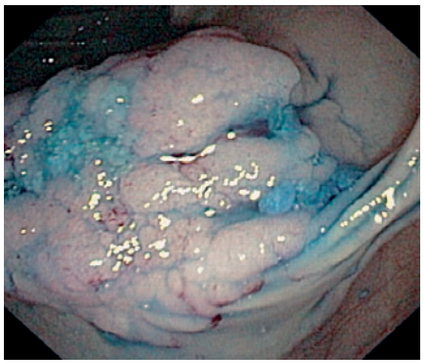
f

d–f Stalked, tubulovillous adenoma; with erythematous surface (**d**, **e**), thicker stalk than tubular adenomas (**f**).

9.7 cont.



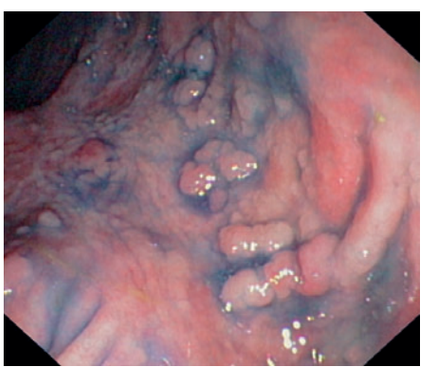
g



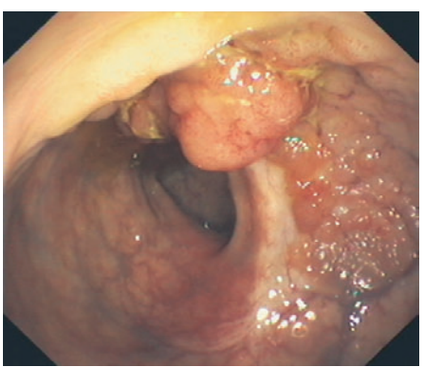
h

g, h Sessile, tubulovillous adenomas, broad-based, in a haustrum, better visualization with chromoendoscopy.

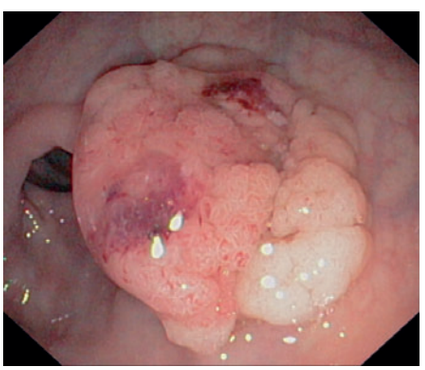
9.8 Villous adenomas



a

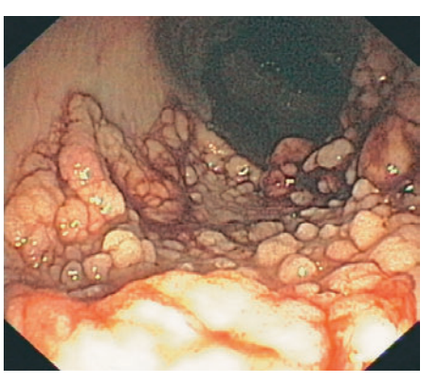


b

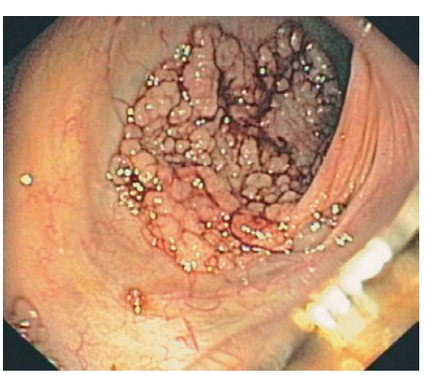


c

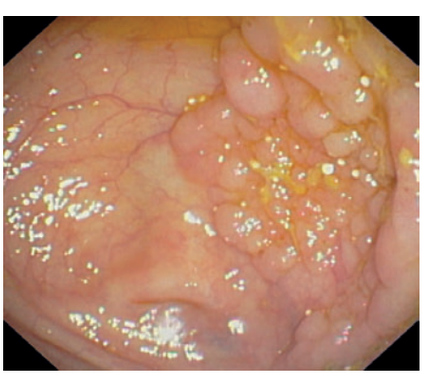
a–e Sessile, villous adenomas, broad-based, partly presenting as multiple polyps. Characteristic pale yellow and irregular surface, as well as nodular contour with fingerlike processes.



d

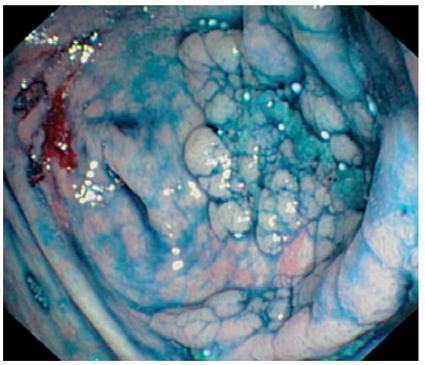


e

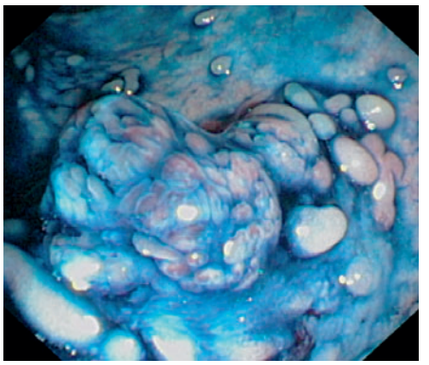


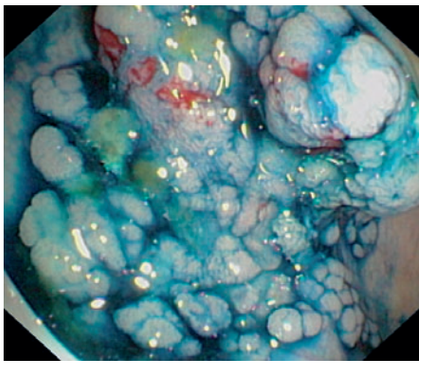
f Wide villous adenoma on the cecal pole adjacent to appendiceal orifice (lower edge of picture).

9.8 cont.



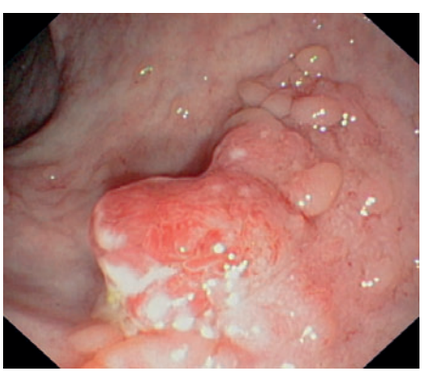
g Better visualization of this villous adenoma on the cecal pole **(f)** using chromoendoscopy.



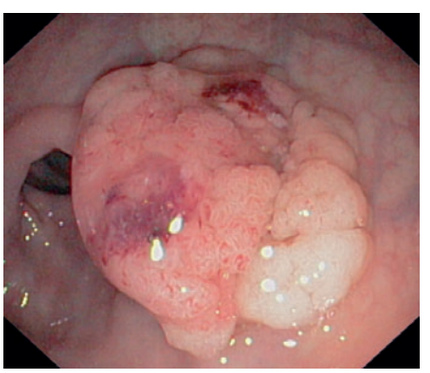


h, i Two villous adenomas with nodular contours, fingerlike processes, and tubular growth. Stained with indigo carmine dye.

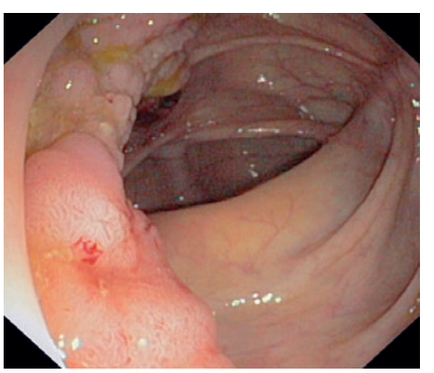
9.9 Malignant polyps



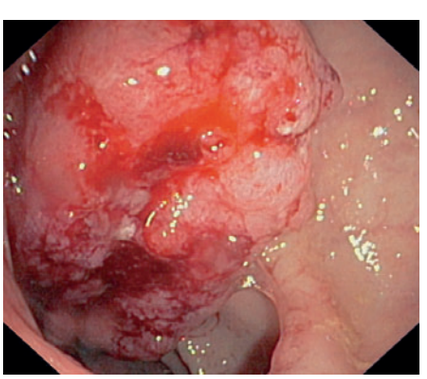
a Cancerous tubulovillous adenoma with deformed surface and ulceration at the polyp base (lower left edge of image).



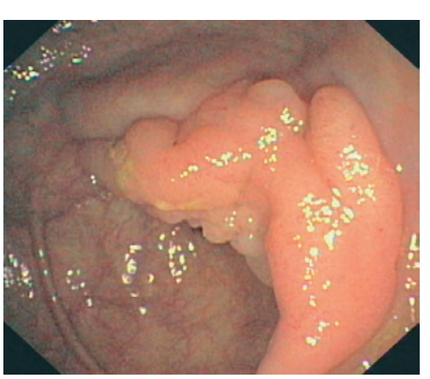
b Villous adenoma with friable surface, some spontaneous bleeding. Malignant transformation on surface.



c Wide tubulovillous adenoma, malignant transformation with surface changes: deformation, granular and friable surface, depression in center and beginning ulceration (upper left edge of image).

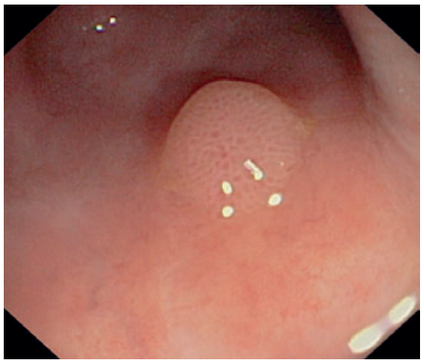


d Malignantly transforming tubulovillous adenoma with granular, friable surface, pronounced vulnerability, and bleeding tendency.

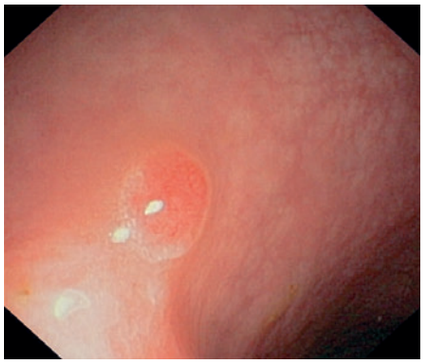


e Malignantly transforming tubulovillous adenoma, surface depression in the center and firm consistency.

9.10 Nonneoplastic polyps: hyperplastic polyps, juvenile polyps, Peutz-Jeghers polyps

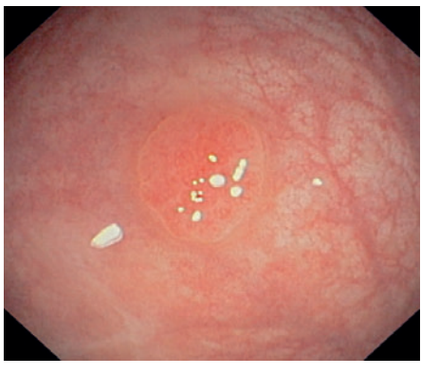


a

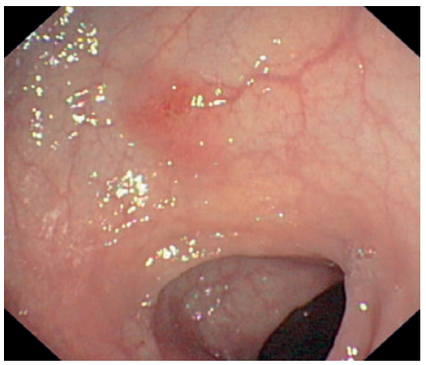


b

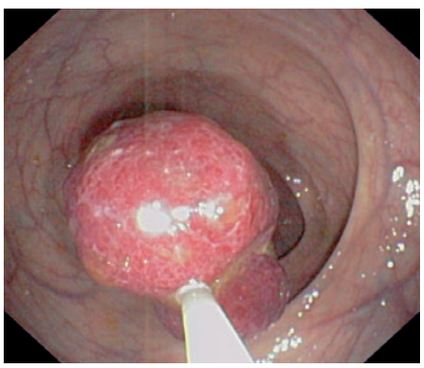
a–d Small hyperplastic polyps, sessile, 5–10 mm. Pale or same color as surrounding mucosa.



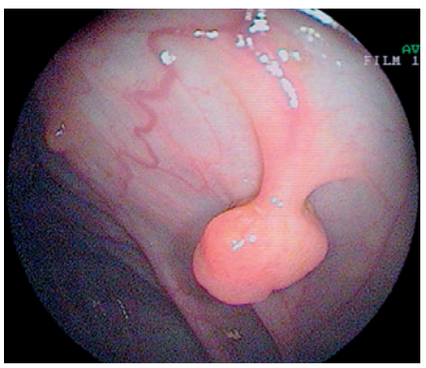
c



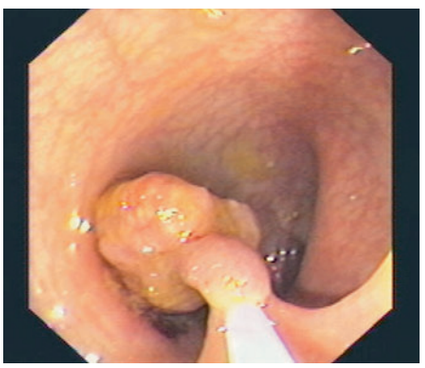
d



e Juvenile polyp, ca. 2 cm in size. Luminous red surface, friable, partly eroded.



f Juvenile polyp, stalked, nodular surface, similar to tubular adenoma.



g Stalked Peutz–Jeghers polyp with lobular surface.

Hyperplastic polyps. Hyperplastic polyps are sessile polyps with a diameter of 5–10 mm. Special endoscopic techniques (chromoendoscopy and "pit pattern classification") must be used to distinguish hyperplastic polyps from adenomas macroscopically.

Hyperplastic polyps, often called metaplastic polyps, are pale or the same color as the surrounding mucosa (▣ 9.**10 a–d**). They usually appear as solitary polyps, but in 5–10 % of patients several polyps appear in one colon segment. Larger hyperplastic polyps can be stalked and may be confused with small tubular adenomas.

Histological evaluation is also necessary for hyperplastic polyps. This requires a forceps biopsy or ectomy with histological analysis of the specimen. Histological examination can potentially be avoided in the future, but only if diagnosis based on pit pattern classification is certain and is performed by an examiner experienced in chromoendoscopy and diagnosis.

Juvenile polyps. Juvenile polyps appear in children and adolescents as solitary hamartomatous polyps.

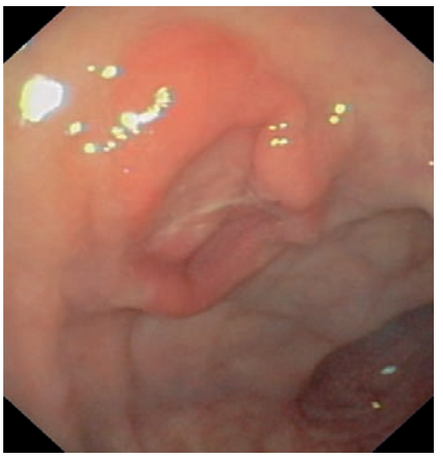


Fig. 9.6 **Heterotropic gastric mucosa in rectum** with polypoid margin, depression in center.

Viewed endoscopically, juvenile polyps are up to 3 cm large, with luminous red, friable, eroded, and/or ulcerated surfaces. The surface is occasionally nodular, similar to a tubular adenoma (▣ 9.**10 e**, **f**).

Histologically, these are cystically dilated glands with mucinous material (mucous retention cysts). They are surrounded by increased connective tissue and blood vessels and there is also an increase in inflammatory cells. Though juvenile polyps are not neoplastic per se, there is nonetheless malignant potential if adenomas or mixed juvenile adenomatous polyps appear synchronously (6).

Peutz–Jeghers polyps. Polyps occurring in Peutz–Jeghers syndrome are hamartomas with a characteristic histological appearance of tree branchlike muscularis mucosae.

Peutz–Jeghers polyps can vary in size (up to a few centimeters), and can be stalked or sessile, irregular or lobular (▣ 9.**10 g**). The polyp surface can show evidence of infarction. Further details and images can be found in the following section "Polyposis Syndromes."

Inflammatory polyps. Inflammatory polyps are caused by prior colitis attack. They are unspecific and made up of highly inflammatory, focal ulcerations of the epithelium with granulation tissue. More details and images are presented in Chapter 12.

Nonepithelial Polyps

Nonepithelial polyps include submucosal tumors, carcinoid tumors, and also more rare findings such as pneumatosis cystoides intestinalis or misplaced endometriosal tissue. More information is given in Chapter 11 "Submucosal Tumors" and Chapter 17 "Rare Diseases and Disorders."

Heterotropic gastric mucosa in the rectum is a rare finding. Viewed macroscopically, the polypoid lesion appears to have a raised margin and smooth surface, with a depression in the center (Fig. 9.**6**).

Surveillance

An interdisciplinary consensus panel has made recommendations for endoscopic diagnosis and treatment of polyps that are set forth in the guidelines of the German Society of Digestive and Metabolic Diseases (8). Polyps larger than 5 mm should be removed by polypectomy, while polyps smaller than 5 mm should be resected using forceps. There is no need for special endoscopic follow-up examination after resection of nonneoplastic polyps. Following complete resection of adenomas, surveillance colonoscopy may be required after three years, depending on degree of dysplasia. If no pathologies are found during the first check-up, subsequent check-ups should be performed every five years. Following resection of an adenoma with carcinomatous components (pT1), later check-ups depend on risk classification (low risk/high risk). In the case of partial resection of neoplastic lesions, complete endoscopic or surgical removal of the localized finding should take place as soon as possible. For more information on polypectomy, refer to Chapter 18, "Polypectomy and Mucosectomy."

Polyposis Syndromes

Definition

All colorectal polyps that can occur as solitary or multiple polyps can also occur as a polyposis syndrome, with, by definition, more than 100 polyps. A distinction is made between neoplastic and nonneoplastic polyps in polyposis syndromes. Table 9.**5** lists neoplastic and nonneoplastic polyposis syndromes.

Clinical Picture and Clinical Significance

Though only neoplastic polyposis tends to transform malignantly, there is evidence that nonneoplastic polyposis types such as hamartomas (juvenile polyposis, Peutz–Jeghers polyps) can transform malignantly in isolated cases. In order to classify a polyposis syndrome correctly, it is necessary to completely remove several (generally 5–10) polyps and evaluate them histologically.

Familial adenomatous polyposis. Familial adenomatous polyposis (FAP), or familial polyposis coli (FPC), is a hereditary autosomal dominant disease, necessarily precancerous, and by definition characterized by the appearance of more than 100 adenomas. After symptoms appear up to 70 % of patients develop a carcinoma, occurring three decades earlier than in the normal population. Gardner syndrome is associated with the related formation of osteomas of the skull, maxilla, and/or mandible, skin lipomas and fibromas, desmoids, and multiple epidermal cysts. Turcot syndrome denotes an association between colonic adenomatous polyposis and tumors of the central nervous system.

Nonneoplastic polyposis syndrome. There are also a number of nonneoplastic polyposis syndromes involving the intestinal tract, e.g., juvenile polyps, Peutz–Jeghers syndrome, and lymphoid hyperplasia. Polyps occurring in juvenile polyposis are hamartomas and are similar to solitary juvenile polyps (see above). Around one-third of juvenile polyposis patients have increased frequency of familial occurrence and carcinoma development

Table 9.5 Neoplastic and nonneoplastic polyposis syndromes

Neoplastic polyposis syndromes	Nonneoplastic polyposis syndromes
▶ Familial adenomatous polyposis	▶ Peutz–Jeghers syndrome
▶ Gardner syndrome	▶ Juvenile polyps
▶ Turcot syndrome	▶ Cronkhite–Canada syndrome
	▶ Cowden syndrome
	▶ Inflammatory polyposis
	▶ Benign lymphoid polyposis
	▶ Hyperplastic polyposis
	▶ Pneumatosis cystoides intestinalis

has been observed in 10% of patients, even though juvenile polyps are nonneoplastic.

Patients with Peutz–Jeghers syndrome have polyps in the entire gastrointestinal tract, mainly in the small intestine. A clinical sign of the syndrome is frecklelike skin pigmentation around the mouth as well as on the mucosa of the lips and mouth. The polyps can be large and can cause significant symptoms like obstruction, bleeding, and intussusception. Though these hamartomatous polyps are not precancerous lesions, carcinomas have been found in the stomach, small intestine, and ovaries.

Cowden syndrome is a hereditary autosomal dominant polyposis syndrome that also is marked by hamartomas of the gastrointestinal tract and other organs. Colorectal carcinomas have not yet been found to be associated with this very rare syndrome.

Cronkhite–Canada syndrome is a nonfamilial disorder that appears after age 40 as a generalized polyposis syndrome of the entire gastrointestinal tract involving cystic degeneration of the mucosa. It is clinically manifested as extremely watery diarrhea, occasionally mixed with blood and mucous, nail atrophy, and alopecia. Prognosis is poor as there is no available causal treatment.

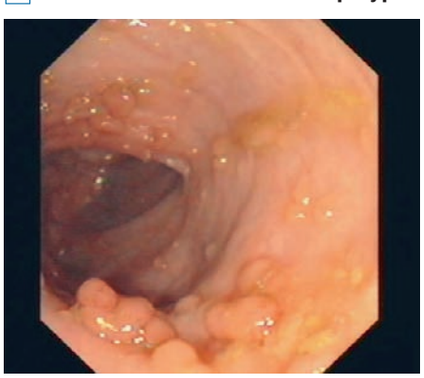
Endoscopic Diagnosis

Familial Adenomatous Polyposis (FAP)

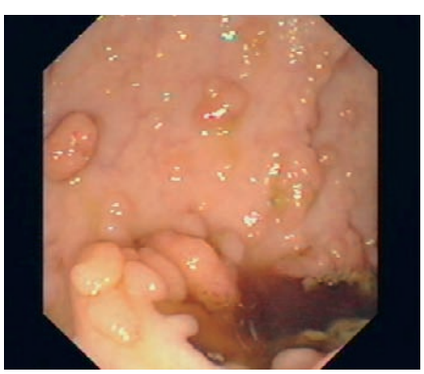
FAP is characterized endoscopically by multiple polyps, by definition more than 100 adenomas, which cover the colon mucosa in wide swathes. The presence of fewer than 100 polyps is referred to as an attenuated form of FAP. Generally, adenomas in FAP are 1–3 mm in size and are smooth with a regular border, presenting a normally colored mucosa (▣ 9.**11 a**, **b**). Another variation consists of large sessile and stalked polyps (▣ 9.**11 c**, **d**). Less common is the appearance of larger polyps (4–8 mm) in the entire colon, with normal mucosa in between them. In 70% of patients, one or multiple carcinomas are detected after the appearance of symptoms (▣ 9.**11 e**).

Multiple tiny polyps throughout the colon, occurring with Gardner syndrome, are shown in ▣ 9.**12**.

▣ **9.11 Familial adenomatous polyposis**

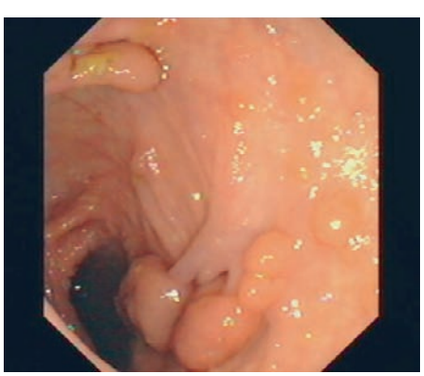


a



b

a, b Smooth, regular border, sessile adenomas, FAP, 1–3 mm large.



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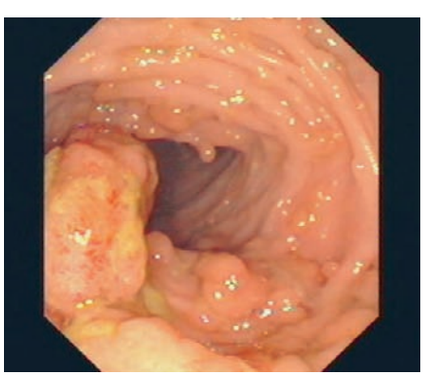
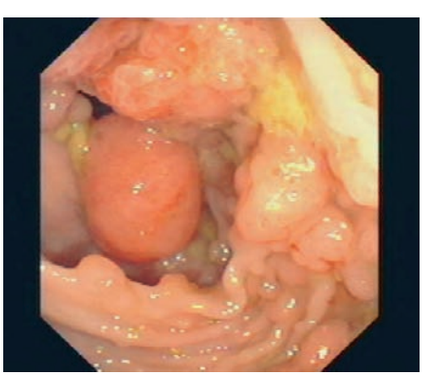


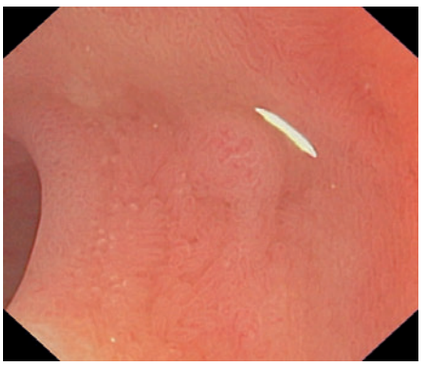
d

c, d Large, sessile, and stalked adenomas, FAP.



e Rectal carcinoma, FAP, with multiple adenomas (upper edge of image).

9.12 Gardner syndrome



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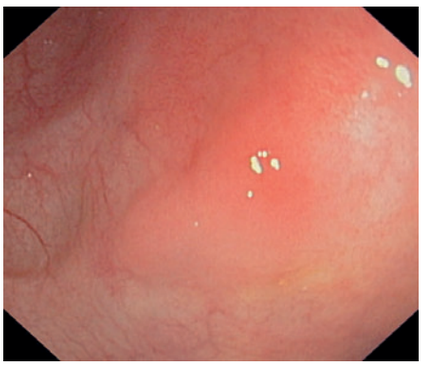

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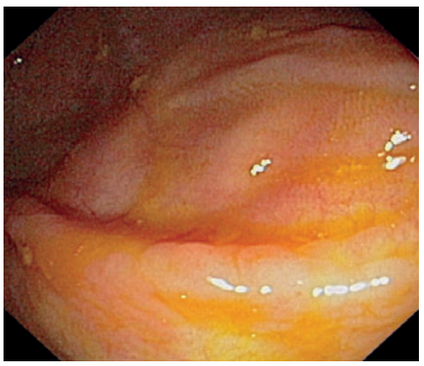
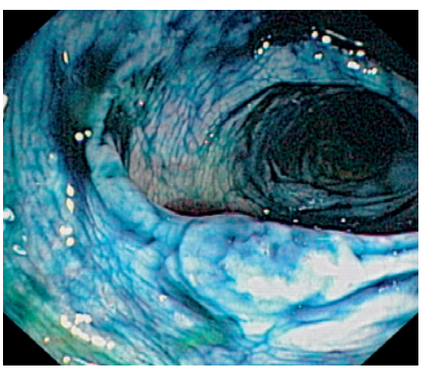


c

a–c Multiple diminutive polyps, Gardner syndrome.



d Improved detection of diminutive polyps using chromoendoscopy, Gardner syndrome.

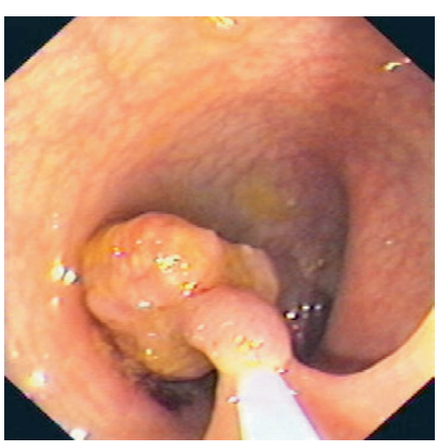


Fig. 9.7 **Stalked polyp, Peutz–Jeghers syndrome,** lobular with infarction.

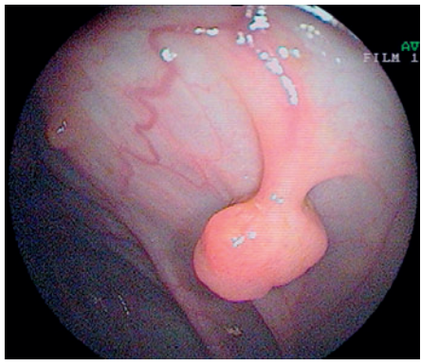
Given the risk of malignant transformation, treatment consists of a colectomy–proctomucosectomy with ileoanal anastomosis and pouch. Genetic counseling and evaluation, and, if indicated, screening tests, should be conducted on affected individuals during childhood.

Peutz–Jeghers Syndrome

Polyps related to Peutz–Jeghers syndrome are hamartomatous with a characteristic histological appearance of tree branchlike muscularis mucosae. They can vary in size (up to a few centimeters), be stalked or sessile, irregular, or lobular. The surface may show signs of infarction (Fig. 9.**7**).

If possible, polyps should be removed when symptoms manifest themselves. Asymptomatic polyps should also be endoscopically resected, even though they generally do not transform malignantly. Surgical intervention is only justified in the case of bleeding or stenosis.

9.13 Juvenile polyposis



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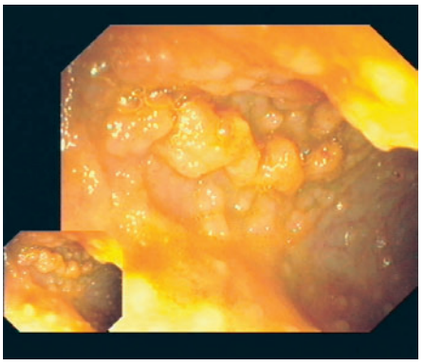
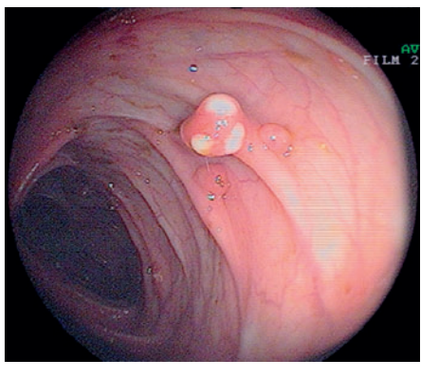


b

a Smooth surface of a juvenile polyp with known polyposis syndrome.
b Diminutive polyps in the terminal ileum, juvenile polyposis.



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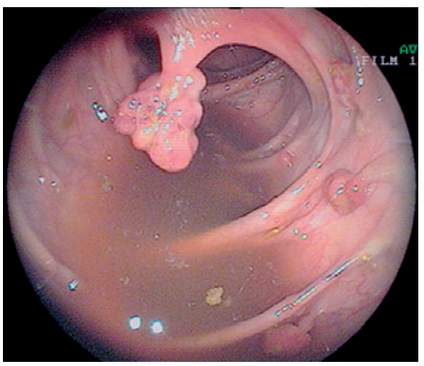

d

c, d Juvenile polyposis with stalked polyps and tiny polyps.

Juvenile Polyposis

Polyps related to juvenile polyposis can occur in the entire digestive tract, or only in the colon where they mainly appear in the rectum. Their diameter measures 3–30 mm and they resemble solitary juvenile polyps. Given their smooth surface, juvenile polyps cannot be distinguished from adenomas macroscopically (▣ 9.**13 a–c**); they also appear as diminutive polyps (▣ 9.**13 d**).

Adenomas appearing at the same time as juvenile polyposis are considered precancerous and must be removed. Subtotal colectomy with ileorectal anastomosis is recommended for multiple polyps with pronounced clinical signs.

Differential Diagnosis

Occasionally, it is difficult to differentiate FAP from inflammatory or lymphoid polyposis.

Inflammatory pseudopolyps related to ulcerative colitis resemble those of FAP. However, in FAP, the mucosa between polyps is unremarkable and vascular pattern remains visible, while in ulcerative colitis, the mucosa between polyps shows signs of changes corresponding to disease activity.

An infrequent diagnosis is lymphoid polyposis or lymphoid hyperplasia of the colon, caused by invasion of the mucosa of the rectum and colon by either a conglomeration of lymph follicles or diffuse lymphoid hyperplasia. Lymphoid polyposis manifests itself as 1–2 mm large, slightly elevated, yellow–white polyps (▣ 9.**14**).

Hyperplastic polyposis occurs as more than 100 hyperplastic polyps, which—similar to solitary polyps—are sessile and seldom larger than 5–10 mm maximum. Hyperplastic polyposis does not exhibit any malignant tendency and there is no need for regular follow-up examinations.

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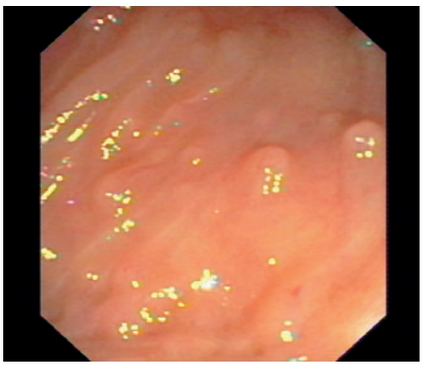
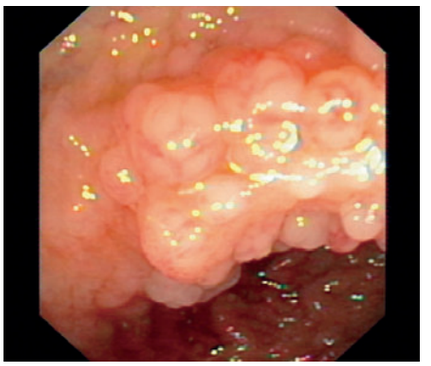
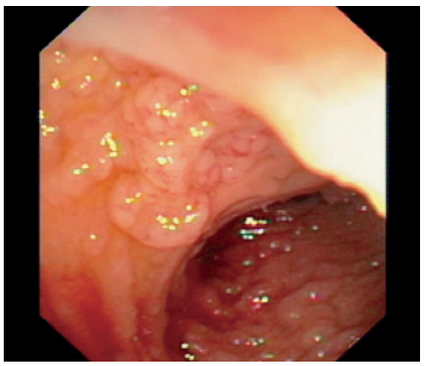
9.14 Lymphoid polyposis







a 1–2 mm large, slightly elevated, yellow-white polyp, lymphoid polyposis.

b

c

b, c Lymphoid polyposis in the terminal ileum with a conglomeration of small, yellow-white polyps.

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10 Malignant Tumors

T. Eberl

Definition and Classification

Most colorectal carcinomas are adenocarcinomas, which developed from colorectal adenomas (adenoma–carcinoma sequence). Less than 2% of malignancies are nonepithelial malignancies (lymphomas) or colon metastases of another primary tumor. Adenocarcinomas account for the vast majority of carcinomas in the colon (85%) and rectum (90%); mucinous adenocarcinomas account for only ca. 13% of carcinomas in the colon and ca. 8% of carcinomas in the rectum. Table 10.**1** gives an overview of the histological classification of primary colorectal tumors.

The degree of differentiation of adenocarcinomas is described by a grading system of the UICC, whereby the degree of differentiation or malignancy is usually divided into four grades:

- G1 = highly differentiated
- G2 = moderately differentiated
- G3 = poorly differentiated
- G4 = undifferentiated.

Signet-ring cell carcinomas are classified as G3; undifferentiated, small-cell carcinomas as G4. The TNM staging system (UICC 1993) is used especially for staging colorectal carcinomas in terms of extent of the local tumor, spread to lymph nodes, and potential distant spread (metastasis).

Table 10.**1** Histological classification of primary colorectal tumors according to WHO

Epithelial tumors	Nonepithelial tumors
▶ Adenocarcinoma	▶ GIST tumors, leiomyosarcoma
▶ Mucinous adenocarcinoma	▶ Hematopoietic and lymphatic neoplasias
▶ Signet-ring carcinoma	▶ Unclassified tumors
▶ Squamous epithelial carcinoma	
▶ Adenosquamous carcinoma	
▶ Undifferentiated carcinoma	
▶ Colloid carcinoma	
▶ Unclassified carcinoma	

Clinical Picture and Clinical Significance

Epidemiology. Incidence of colorectal carcinoma is generally high in western industrialized nations. In Germany, the rate among men is 41 per 100000 and among women it is 52 per 100000. For both men and women colorectal carcinomas are the third most common carcinoma. Frequency of disease rises steadily with increasing age, the maximum being around 75 years of age. Epidemiological studies have shown that in addition to genetic causes, there is also an association between environmental factors and the development of colorectal carcinomas. A diet rich in animal fat and low in fiber promotes the development of colorectal carcinomas. Further risk factors include chronic inflammatory bowel diseases (especially ulcerative colitis), colon adenomas, family history, prior ureterosigmoidostomy, and hereditary syndromes (familial adenomatous polyposes, hereditary nonpolyposis colorectal cancer).

Symptoms. About 60% of all colorectal carcinomas are located in the colon and around 40% in the rectum. Of those located in the colon, most (60%) are found the sigmoid colon, followed by the ascending colon. In 4–6% of patients, two or more synchronous carcinomas are found in the colon and rectum (1). The main symptom of colorectal carcinoma is the presence of blood and mucous in stool, though symptoms of right-sided colon carcinomas initially manifest themselves indirectly as iron-deficiency anemia resulting from chronic occult blood loss. Changes in bowel habits, unintended weight loss, tenesmus, and diarrhea are further typical symptoms that may indicate colorectal carcinoma. Around 10% of all colorectal carcinomas are diagnosed in emergencies related to an obstruction or perforation (1).

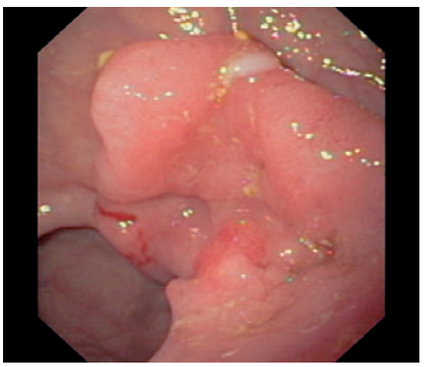
Treatment strategy. Treatment strategies for colorectal carcinoma depend on the pathological tumor stage. The primary treatment for all colorectal carcinomas is surgical resection, which can also be indicated for already metastasizing disease, for example, in order to prevent bowel obstruction (ileus) and stenosis. Additional adjuvant and neoadjuvant therapies include chemotherapy and radiation according to tumor stage; for rectal carcinomas, further treatment is determined by preoperative endosonographic T-staging (TNM staging). For metastasizing disease, chemotherapy may be a palliative treatment option.

Diagnosis

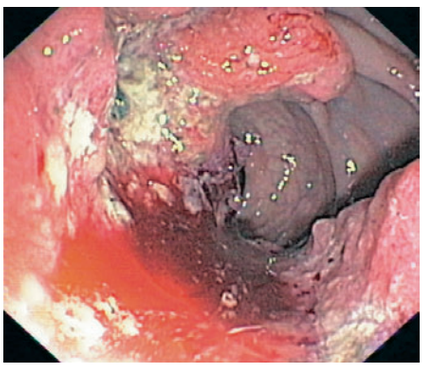
Diagnosis of colorectal carcinoma

- family history,
- clinical examination (rectal palpation),
- laboratory parameters (CEA),
- **total colonoscopy (with biopsy sampling)**,
- double-contrast enema examination of the colon, if necessary (if complete endoscopy is not possible),
- virtual colonoscopy (?),
- sonography of liver and abdomen,
- if necessary, abdominal computed tomography (CT)/ magnetic resonance imaging (MRI) of liver,
- thoracic radiography,
- endosonography, MRI with endorectal coil (rectal carcinoma).

10.1 Macroscopic growth form of colorectal carcinoma: ulcerative tumor



a



b

a Rectal carcinoma. Bowl-shaped ulceration, gray–yellow hue.
b Ulcerated colon carcinoma with inflammatory margin and petechiae.

Diagnosis is based on family history and clinical findings. Family history is especially important with regard to hereditary polyposis syndromes and for patients with a history of colorectal adenomas and carcinomas in the family. Digital rectal examination is especially important for clinical findings, as 40% of all colorectal carcinomas are located in the rectum.

Role of endoscopy. Endoscopy, i.e., total colonoscopy, is the main and essential diagnostic measure for clarification, especially of colorectal carcinoma. On the one hand, it has the potential for providing histological confirmation of diagnosis preoperatively. This is generally successful in total colonoscopy, which enables suspicious mucosal areas to be biopsied under visualization. On the other hand, endoscopy of the entire colon is absolutely indicated if carcinoma is suspected. This is necessitated by the frequent simultaneous appearance of adenomas and carcinomas: in 4–6% of patients, an additional or multiple carcinomas occur in the colon and rectum; additional colon polyps occur in 25% of patients (1).

Complementary procedures. Double-contrast barium examination of the entire large bowel has the advantage of tumor localization, but the disadvantage of lacking possibility for histological evaluation. Double-contrast examination should be performed if stenosis caused by the tumor prevents inspection of the entire bowel. The role of virtual colonoscopy in diagnosing colorectal carcinomas remains unclear, as was discussed in more detail in Chapter 9 "Polyps and Polyposis Syndromes."

The use of a stiff rectoscope in detecting rectal carcinomas should also be mentioned. Ascertaining the height of the rectal tumor and thus choice of surgical procedure is more reliable and better reproducible with a stiff rectoscope. Endosonography provides the most reliable evaluation of the depth of invasion of a rectal carcinoma and proximity to the sphincter muscle. MRI with an endorectal coil can also be useful.

Examination procedure for colorectal carcinomas

- Total colonoscopy (inspection of entire colon to the cecum) is particularly vital for colorectal carcinoma and is the primary diagnostic procedure.

Epithelial Tumors

Adenocarcinomas. Adenocarcinomas appear in the entire colon and rectal area, most frequently in the rectum and sigmoid. Four typical appearances can be distinguished.

Colorectal carcinomas: macroscopic growth forms (5)

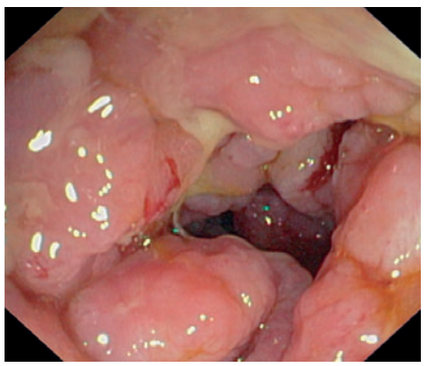
- ulcerative tumor, depressed (▣ 10.**1**),
- sessile, polypoid tumor, nodular, or lobular (▣ 10.**2**),
- annular tumor, stenosis (▣ 10.**3**),
- flat elevated tumor (▣ 10.**4**).

Ulcerative tumor. Ulcerative tumors typically have an irregular surface, which can be sometimes deeply depressed; color can range from gray–red to yellow (ulcerated) (▣ 10.**1**). The surrounding mucosa is swollen, sometimes with bright red inflammatory changes and is often a source of gastrointestinal bleeding (▣ 10.**1 b**). The consistency of the tumor as tested with a forceps or when taking a tissue sample is firm, or even rock hard and friable.

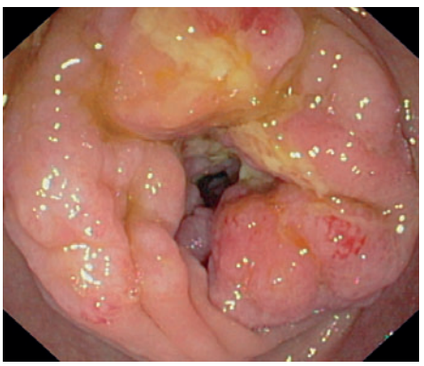
Polypoid tumor. Another growth form of colorectal carcinoma is the polypoid tumor. These tumors are not depressed and can vary in size (▣ 10.**2**). They have a nodular or lobular surface, which may have focal erosions or ulcerations (▣ 10.**2 c–e**). Tumors that are firmly situated in the intestinal wall signal infiltration of deeper wall layers.

Annular tumor. The third most common growth form is the ringlike annular, ulcerated lesion, which affects the colon wall circumferentially and is often already causing stenosis by the time of diagnosis, rendering instrument passage impossible (▣ 10.**3**). Patients mostly belong to the some 10% of individuals with colorectal carcinomas who are diagnosed in emergencies related to obstruction. Tumor infiltration of the pericolic fatty tissue leads to stricture or stenosis and thus compression and narrowing of the lumen. Differentiating between a diverticular stricture and a malignant stenosis

10.2 Macroscopic growth form of colorectal carcinoma: polypoid tumor

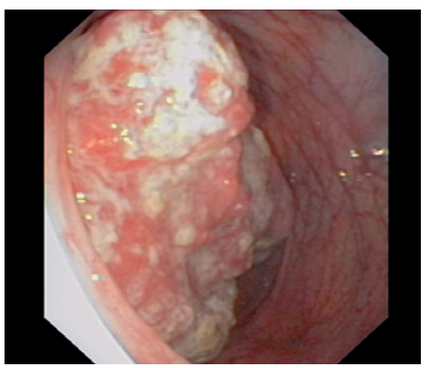


a



b

a, b Sessile, polypoid carcinoma, nodular or lobular.



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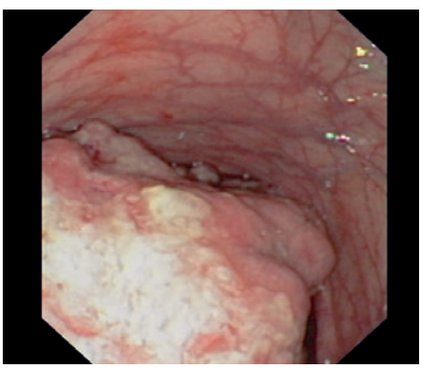

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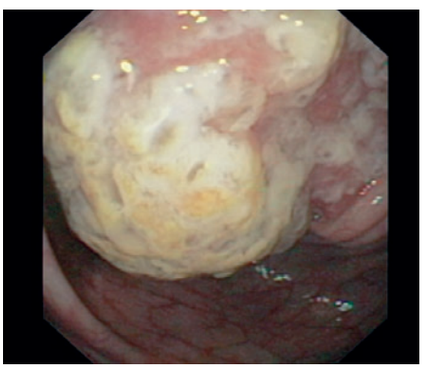
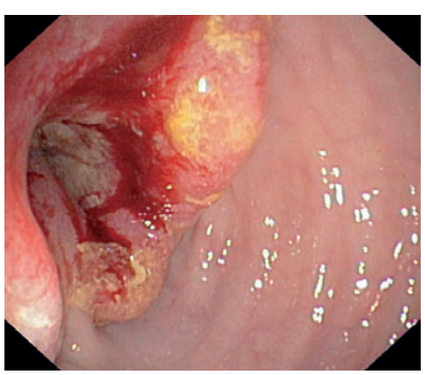


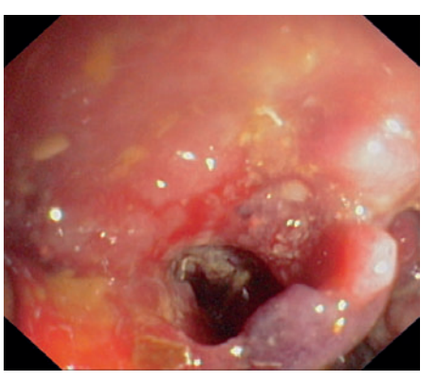
e

c–e Polypoid carcinoma with focal erosions or ulcerated surface.

10.3 Macroscopic growth form of colorectal carcinoma: annular tumor

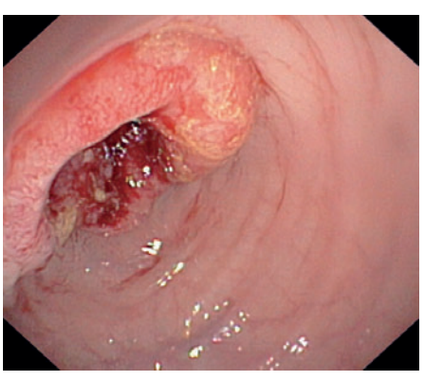


a

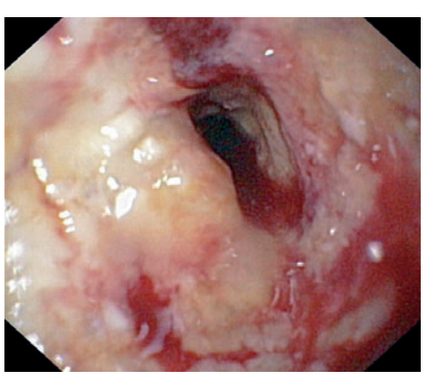


b

a, b Circular ulcerated rectal carcinoma, stenosing.



c Annular, ringlike growth pattern of a colon carcinoma, stenosing.



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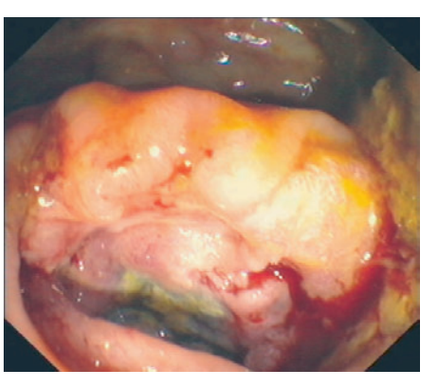
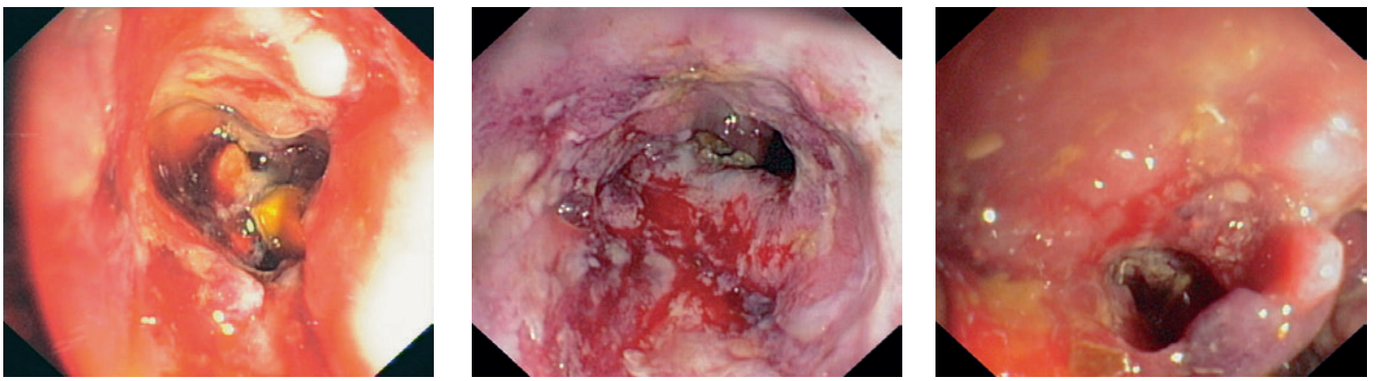


e

d, e Stenosis caused by carcinoma, ulceration in center.



a b c

Fig. 10.**1 a–c** **Carcinoma: stenosis** with irregular surface, swollen mucosa, vulnerable or spontaneously bleeding mucosa.

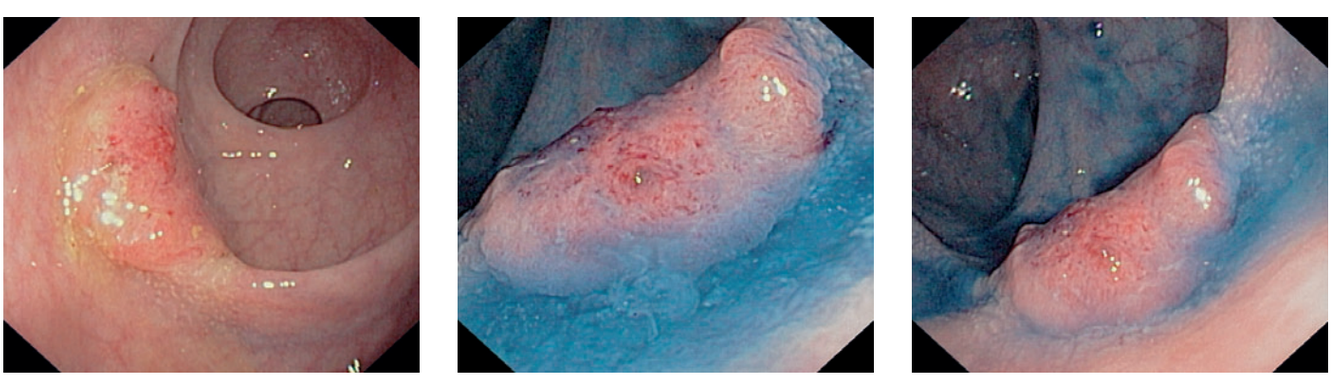
can be difficult. Malignant stenoses generally have an irregular surface, the mucosa is swollen and destroyed, vulnerable, or bleeding spontaneously (Fig. 10.**1**). Inflammatory stenoses have an edematous mucosa, a glassy, livid surface, and are without significant irregularities.

If endoscope passage is not possible, a smaller-caliber instrument should be used to attempt passage and to effectively assess the stricture. To do so, the instrument should be withdrawn slowly and multiple biopsy samples should be taken. If passage is not possible even with a smaller instrument, multiple biopsies should be taken from the distal part of the stricture that can be visualized. Further information about the malignant/benign nature of the stenosis must be then ascertained using a computed tomography scan and contrast enema.

Flat tumor: A more rare form of colorectal carcinoma is flat prominent or slightly elevated tumors with a depression in the center and/or ulceration. The tumor margins are either at the level of the mucosa or raised a few millimeters (▣ 10.**4 a**). These changes can be easily missed and are diagnosed better using chromoendoscopy or an instrument capable of producing a high resolution image (▣ 10.**4 b–e**, see also Chapter 3).

This type of tumor develops from a flat adenoma that, according to the literature, comprises 8.5–11% of all adenomas (2). This subgroup of adenomas can have a high incidence of high-grade intraepithelial neoplasia, depending on size. In a recent prospective study of a large patient sample, 36% of all adenomas found during routine colonoscopy were "flat adenomas." Four

▣ **10.4 Macroscopic growth form of colorectal carcinoma: flat tumor**

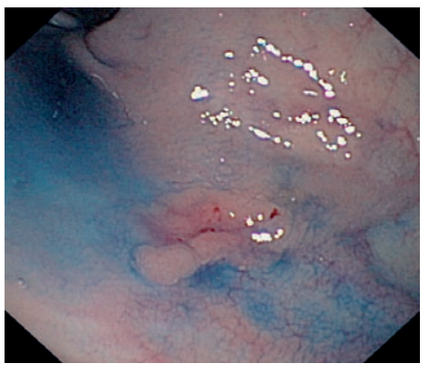


b c

a Flat elevated tumor with irregular, bumpy surface; tumor margins raised a few millimeters above the mucosa.

b, c Tumor margins of same tumor emphasized using chromoendoscopy.

10.4 cont.



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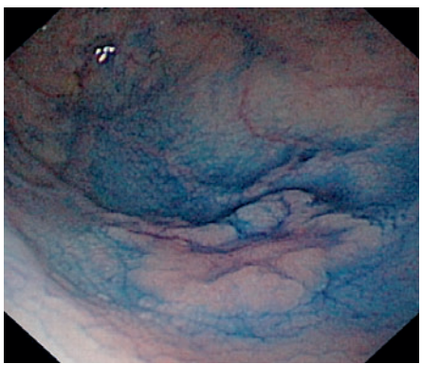
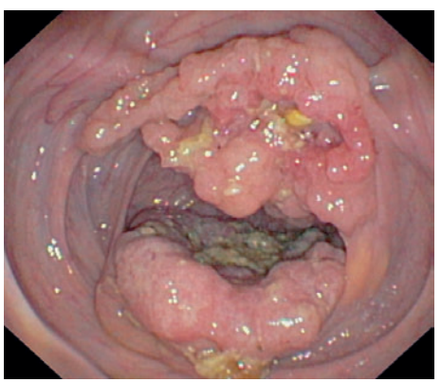
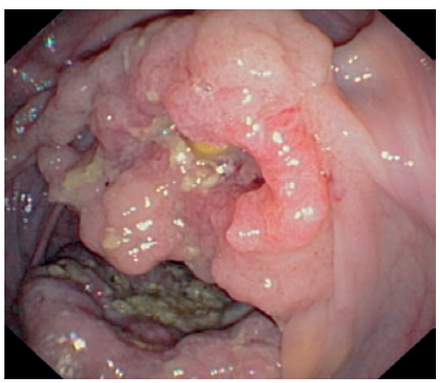


e

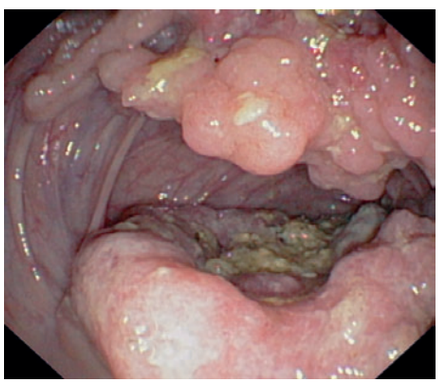
d, e Flat elevated carcinoma with depression in center, better visualization with chromoendoscopy.



a



b



c

Fig. 10.**2 a–c** **Double carcinoma in cecum,** originating at two bowlshaped growing tubulovillous adenomas. The tumor edges are not touching. The ileocecal valve is involved in tumorous process.

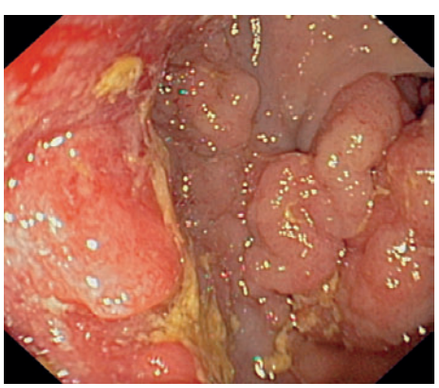


Fig. 10.**3** **Synchronous colon polyps, colorectal carcinoma.** On the left edge of the image, a carcinoma is recognizable and on the right edge a larger polyp, partly obstructing the lumen.

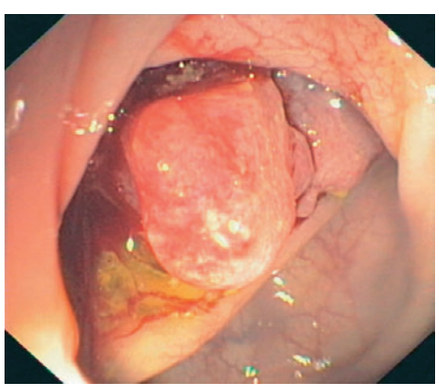
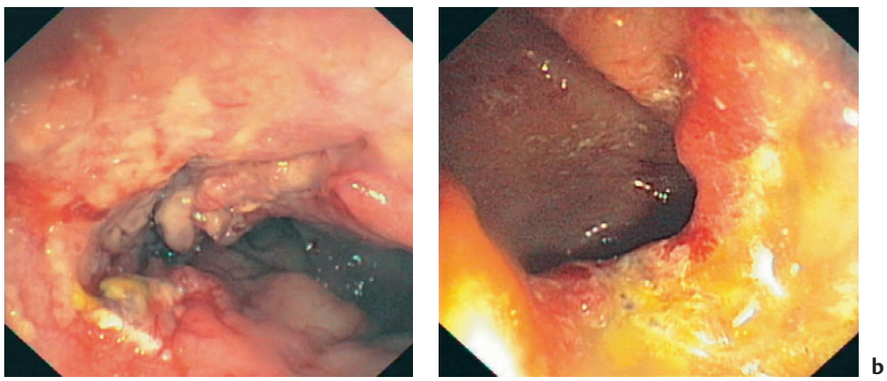


Fig. 10.**4** **Synchronous polyp, colorectal carcinoma.**

percent of the smaller "flat adenomas" (< 10 mm diameter) and 29% of the large "flat adenomas" (≥10 mm diameter) were found to contain severe dysplasia or an adenocarcinoma (3). The fact that flat adenomas also have a lower incidence than polypoid lesions of K-*ras* mutations supports the hypothesis that malignant progression of flat adenomas to carcinoma does not necessarily have to include a polypoid phase. Flat adenomas perhaps are an early phase in the development of a de-novo colon carcinoma (2).

An additional carcinoma or multiple carcinomas are found in the colon and rectum in 4.6% of patients (Fig. 10.**2**); additional colon polyps occur in 25% of patients (Figs. 10.**3**, 10.**4**). Often, large villous adenomas can be confused with carcinomas because of their irregular and friable surfaces. In such cases especially, a forceps should be used to test consistency (hard vs. soft) and whether the tumor can be depressed slightly into the intestinal wall. Further information about potential malignancy can be ascertained with multiple biopsies or an ectomy of the



a b

Fig. 10.5 a, b Ulcerating anal carcinoma with infiltration of distal rectum.

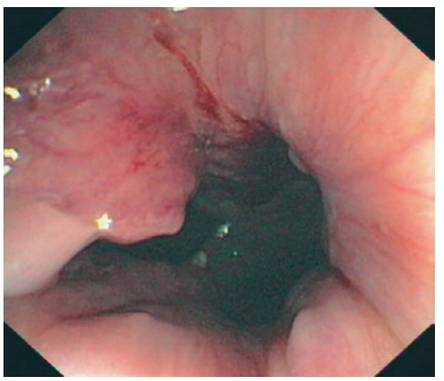
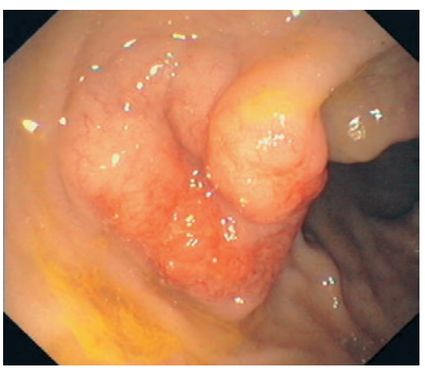
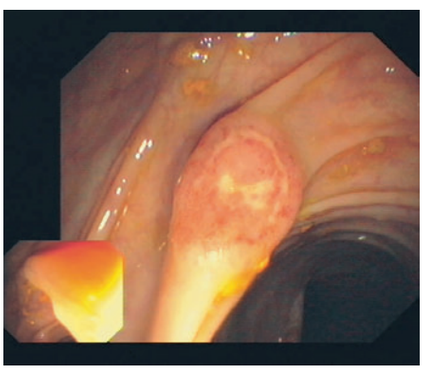


Fig. 10.6 **Squamous cell carcinoma,** originating in the anal canal (at the 10–12-o'clock position in dorsal recumbent position).

▣ 10.5 Primary colonic lymphoma

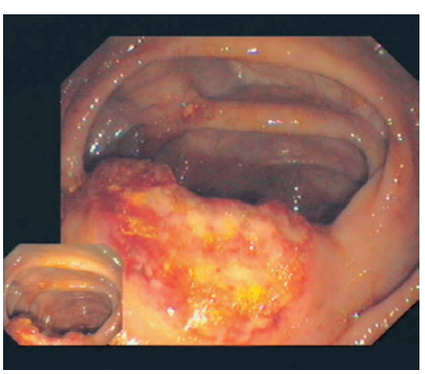


a

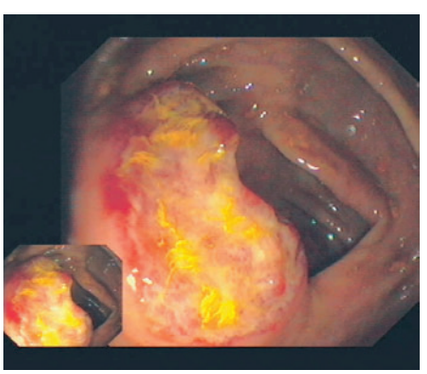


b

a, b Primary lymphoma in the colon. Polypoid tumor with firm, indurated surface.



c



d

c, d Lymphoma involving ileocecal valve with firm, indurated, partly ulcerated surface. Intermittent bleeding due to friable surface.

polypoid tumor—if technically possible—and histological analysis.

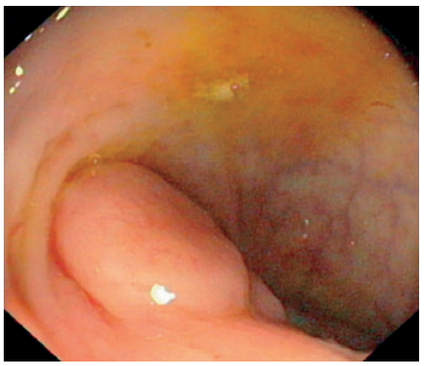
Squamous cell carcinomas. Squamous cell carcinomas are anal carcinomas that have infiltrated the distal rectum (Figs. 10.**5**, 10.**6**). A cloacal carcinoma originating in the anal transitional epithelium can also involve the distal rectum. Differentiation between a distal rectal carcinoma with infiltration of the anal canal and a squamous cell carcinoma with infiltration of the distal rectum is not always possible macroscopically.

Malignant Nonepithelial Tumors

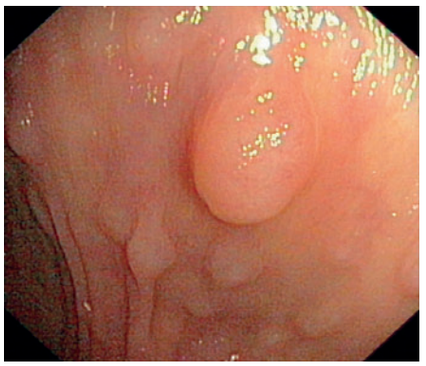
Malignant lymphoma. With regard to colonic lymphoma, a distinction must be made between primary colonic lymphoma vs. a secondary, generalized lymphoma, that is, dissemination arising from primary lymph node invasion.

Primary colonic lymphoma is very rare and presents endoscopically as a polypoid tumor with a firm and indurated surface (▣ 10.**5**). Given the size of the tumor, the lumen can become obstructed; the friable surface can also lead to tumor bleeding.

10.6 Lymphoma invasion of colon with generalized lymphoma



a Lymphoma involving rectum with exophytic growth related to generalized lymphoma.



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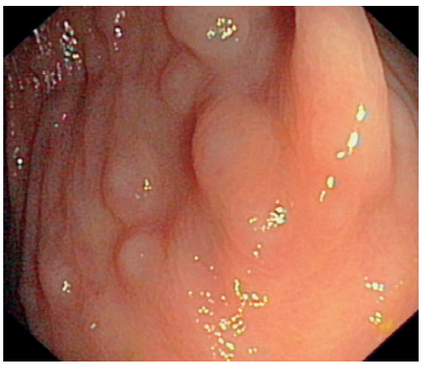
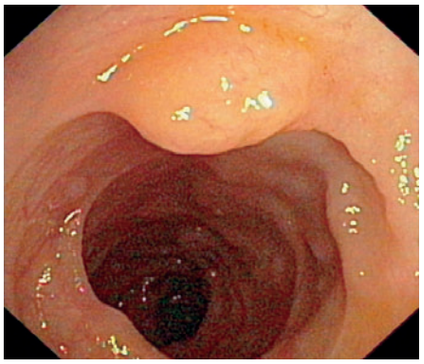


c

b, c Generalized lymphoma with invasion of the cecum. Typical appearance with multiple, small, slightly raised or flat elevated polypoid lesions.



d Generalized lymphoma, invasion of the ileum with flat elevated polypoid lesions.

Generalized lymphoma tends to occur in the left side of the colon and in the rectum. Viewed endoscopically, the affected mucosa appears friable, indurated, and erythematous; exophytic tumor growth is very rare. Another endoscopic appearance is characterized by multiple small, slightly raised, or flat elevated, polypoid lesions (▣ 10.**6**).

Gastrointestinal Stromal Tumors (GIST tumors). In most cases, these are malignant leiomyomas, often accompanied by tumor bleeding.

GIST tumors appear endoscopically as polypoid tumors with an irregular, eroded and partially ulcerated surface (Fig. 10.**7**). Macroscopic differentiation from polypoid carcinomas is not possible.

Colon metastases. Metastases of another primary tumor can infiltrate the colon directly, from an adjacent organ or via implantation metastasis in the colon wall.

If the colon is infiltrated by a malignancy from an adjacent organ, the bowel wall will show signs of edema that can cause stenosis of the lumen. In advanced stages, the tumor may project into the lumen, appearing as an exophytic tumor, which in some cases cannot at first be clearly differentiated from a colorectal carcinoma.

Metastases in the colon wall occur first within the wall and then spread into the lumen. These are also exophytic tumors, often with surface ulcerations, as these tumors grow more rapidly than their blood supply (Fig. 10.**8**)

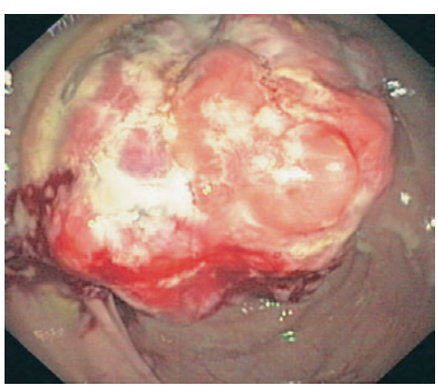


Fig. 10.**7** **Malignant gastrointestinal stromal tumor (GIST tumor) in the colon** with polypoid growth. Macroscopic differentiation from polypoid carcinoma impossible.

Surveillance

Guidelines for aftercare, as well as follow-up surveillance after operative treatment for colorectal carcinoma, were established

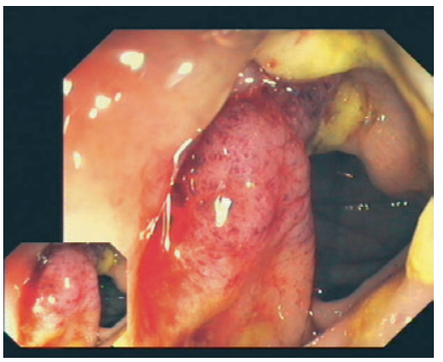


Fig. 10.8 **Colon wall, metastasis of gastric carcinoma** (adenocarcinoma) at hepatic flexure. Exophytic tumor with ulcerations cannot be clearly differentiated from a colorectal carcinoma macroscopically.

by the German Society of Digestive and Metabolic Diseases based on a meeting of a consensus group on prevention, diagnosis, aftercare, and drug treatment of colorectal carcinomas (4).

Colon carcinoma. Colonoscopy surveillance should be performed after 24 and 60 months for colon carcinomas in UICC stages I–III. Following endoscopic resection of a malignant (low-risk) colon polyp (T1 N0 M0; G1–2), another endoscopic check-up must be performed after six months.

Rectal carcinoma. Surveillance colonoscopy is indicated for rectal carcinomas in UICC stages I–III at 24 and 60 months. After local excision, rectoscopy or sigmoidoscopy must be performed at six, 12, and 18 months (4).

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11 Submucosal Tumors

T. Eberl

Definition and Classification

As the term indicates, submucosal tumors are characterized by growth exclusively in the submucosa. Submucosal tumors occur much less frequently in the colon than in the upper gastrointestinal tract.

Submucosal tumors are primarily carcinoid and nonepithelial tumors. Based on the WHO classification and nomenclature of colorectal tumors, submucosal tumors can be classified as "nonepithelial tumors" and "carcinoid tumors" (Tab. 11.**1**).

Table 11.**1** Classification of submucosal tumors

Nonepithelial tumors
▶ Benign tumors
– leiomyomas, leiomyoblastomas
– lipoma, lipomatosis
– vascular tumors: hemangioma, lymphangioma
▶ Malignant tumors
– leiomyosarcoma
– other tumors
Carcinoid tumors

Clinical Picture and Clinical Significance

Submucosal tumors often remain asymptomatic, detected incidentally during endoscopic examination or radiology of the large bowel. Clinical manifestation is rare. Erosion of the mucosa can cause gastrointestinal bleeding and larger tumors may occlude the lumen; occlusion or tumor invagination may appear as an ileus (obstruction).

Diagnosis

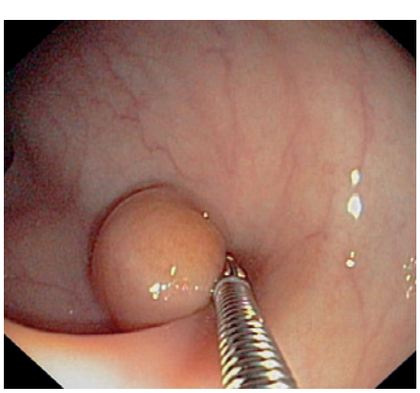
Nonepithelial Tumors

Lipomas. Lipomas are the most frequently occurring submucosal tumors of the colon and rectum, comprising ca. 65% of all gastrointestinal lipomas. They are predominantly found in the right hemicolon and multiple tumors occur in 20% of patients. Incidence rates are 0.2–0.8% based on autopsy reports (3).

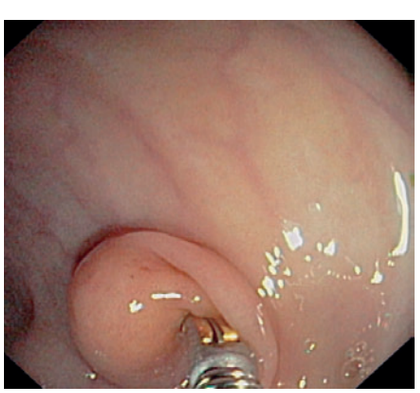
Lipomas appear as solitary or multiple submucosal lesions. They are rounded and elevated, 1–3 cm in size. Other endoscopic features include yellowish hue, smooth and translucent surface, and soft consistency (▣ 11.**1 a–e**). Lipomas are normally sessile; stalked lipomas are quite unusual (▣ 11.**1 f**, **g**). Their consistency can be tested with the instrument tip or biopsy forceps: if the lesion indents easily, this is referred to as a "pillow sign" (▣ 11.**1**) (5).

Excess adipose tissue occasionally accumulated at the Bauhin valve may make it appear to be a lipoma, though it is merely lipomatous. It is vital to recognize the difference in order to avoid resection of a lipomatous valve (Fig. 11.**2**). Endoscopic biopsy is only necessary if there is doubt about the identity of the lesion. Biopsy is indicated if the surface is irregular and/or eroded.

Repeated biopsies of a single lipoma may expose sensitive, yellow, adipose tissue underneath (Fig. 11.**3**). Snare polypectomy is only seldom necessary, and should be avoided, unless warranted by symptoms. Polypectomy is difficult, even using a high level of power, as adipose tissue is a poor conductor of electricity. Bleeding and perforation may result (4).



a



b

Fig. 11.**1 a**, **b** Testing lipoma consistency with a biopsy forceps: "pillow sign."

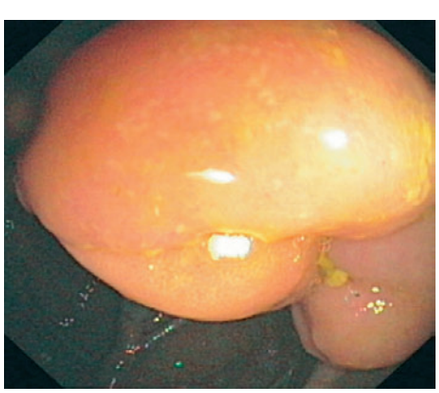
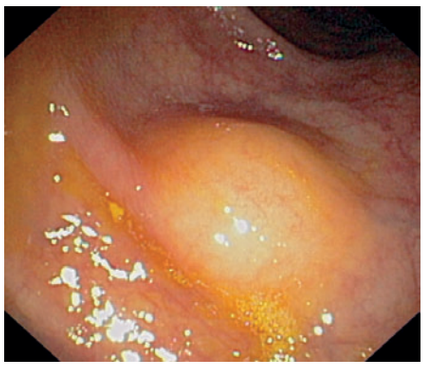
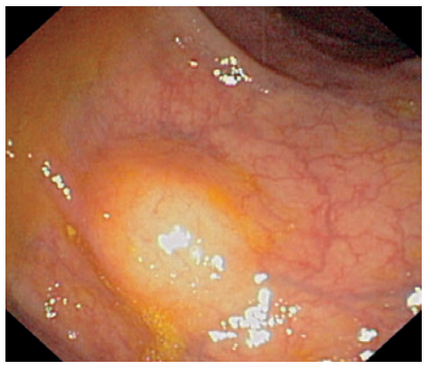


Fig. 11.**2** **Lipomatous ileocecal valve** with massive fat accumulation.

11.1 Sessile and stalked lipomas



a



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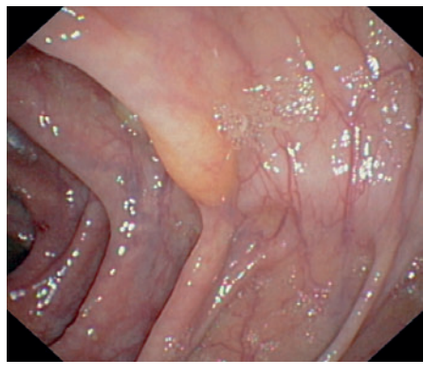
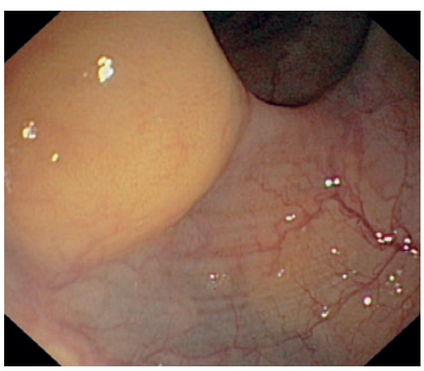


c



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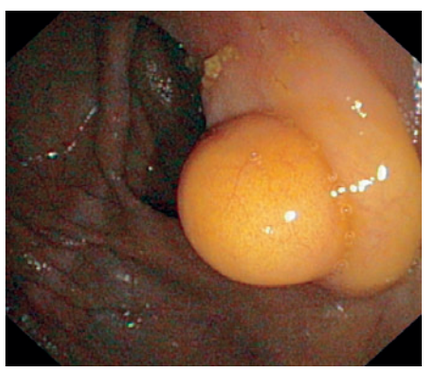
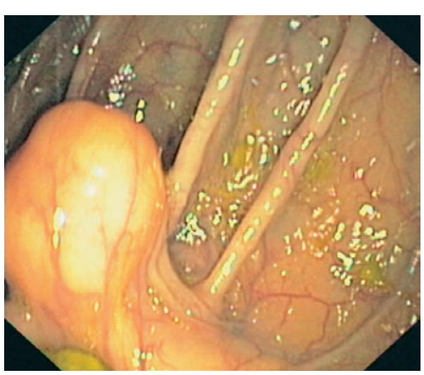
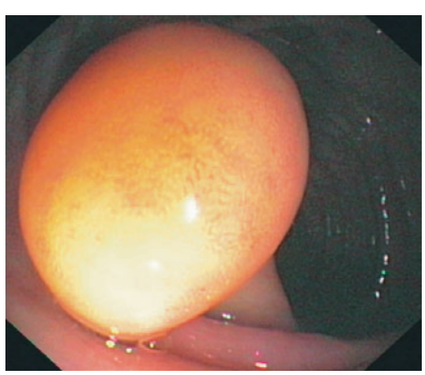


e

a–e Sessile lipomas presenting as submucosal, rounded elevated forms 1–3 cm in size, yellowish color. Their surface s smooth and translucent, soft consistency.

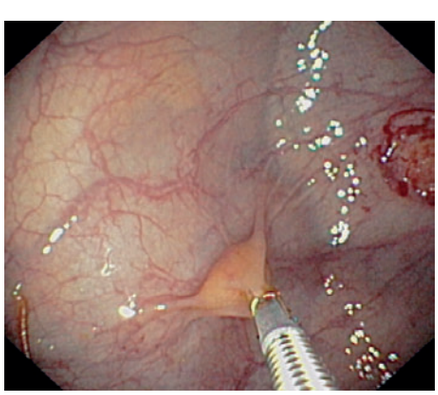


f

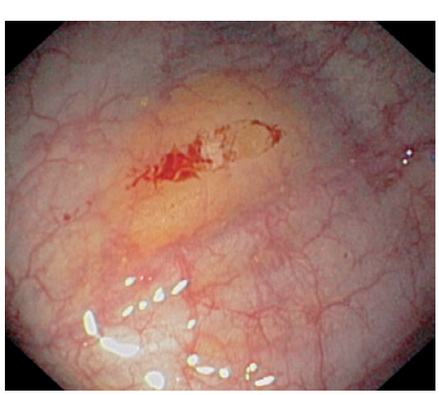


g

f, g Stalked lipomas, round with smooth margins, yellowish color.

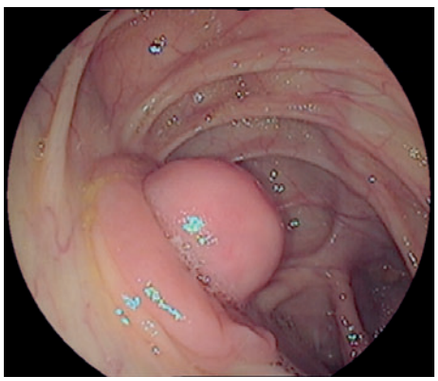


a

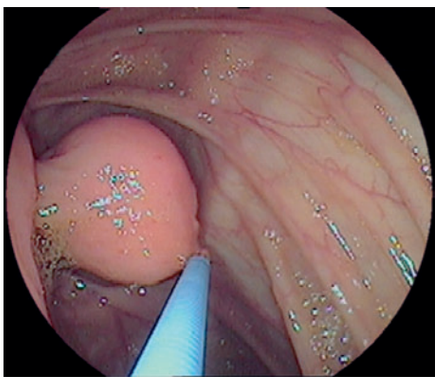


b

Fig. 11.3 **a**, **b** Buttonhole biopsy at same location on lipoma surface, yellow adipose tissue underneath.



a



b

Fig. 11.4 a, b Leiomyoma in the terminal ileum, ca. 2 cm large, protruding out of the ileocecal valve. The surface is smooth and covered with reddened, stretched mucosa.

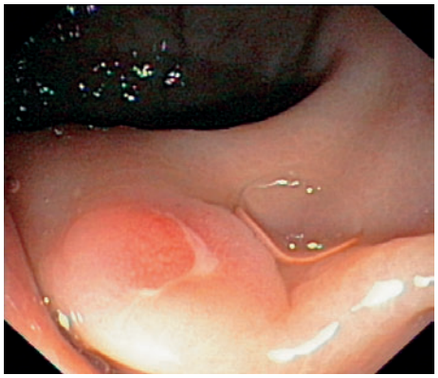


Fig. 11.5 **Carcinoid in the rectum** with eroded surface.

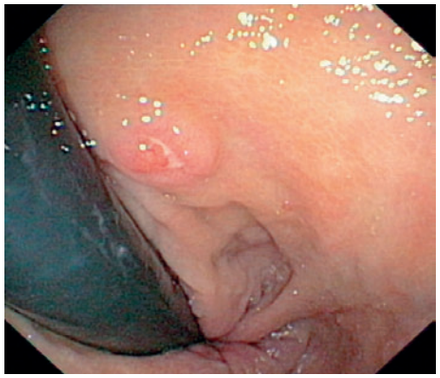


Fig. 11.6 **Rectal carcinoid** with eroded surface (retroflexed view).

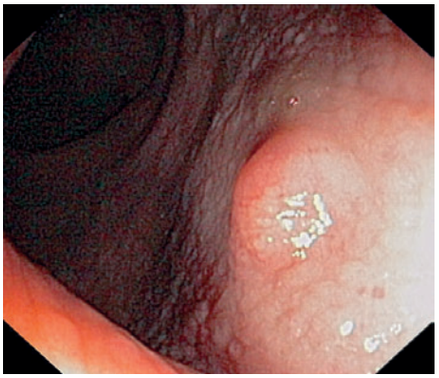


Fig. 11.7 **Carcinoid,** sessile with smooth margin and vascularized surface.

Leiomyomas. Leiomyomas are submucosal tumors of the colon.

Leiomyomas are 2–4 cm large, smooth, sessile, and covered with reddish, stretched mucosa (Fig. 11.**4**). Histological diagnosis usually yields little; diagnosis must be assured with endosonography. A typical endosonographic finding is an isolated spindle-shaped thickening of the muscularis propria. The tumor is smooth and has a well-defined margin.

Leiomyosarcomas. Histological differentiation of leiomyosarcomas and benign leiomyomas is often difficult or even impossible.

Leiomyosarcomas present as either large, polypoid tumors or as nodular tumors, growing outward toward the serosa. Their surface can be irregular or eroded.

Rare submucosal tumors. In addition to the tumors already described, there are also a few other, rarely occurring submucosal tumors, such as lymphangiomas.

Lymphangiomas present as smooth, roundish, polypoid, soft, and yielding tumors. It is generally not possible to diagnose lymphangiomas or differentiate them from other submucosal tumors macroscopically. Their location deep in the mucosa makes them difficult to reach with a biopsy forceps.

Other rare findings include pneumatosis cystoides intestinalis and misplaced endometrial tissue. Further details are provided in Chapter 17, “Rare Diseases and Disorders.”

Carcinoids

Carcinoids appear more often in the rectum (Figs. 11.**5**, 11.**6**) and terminal ileum and less often in the colon or appendix.

Carcinoids present as pale yellow, sessile tumors with a smooth margin and a shiny, vascularized surface (Figs. 11.**7**, 11.**8**). They are up to 2 cm large and usually broadbased; consistency when tested with a biopsy forceps is rather firm.

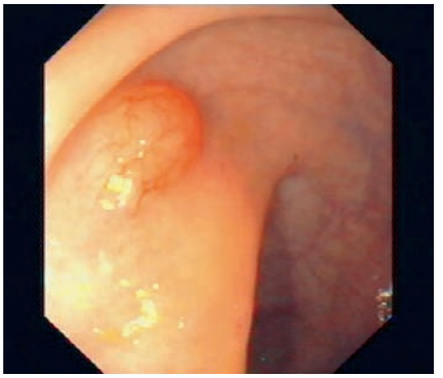


Fig. 11.**8** **Sessile carcinoid,** pale yellow hue, and vascularized surface.

Treatment

Treatment of submucosal tumors generally includes complete removal of the lesion, whereby the method depends on size and localization. For larger lesions, only surgical resection can ensure excisional biopsy. Smaller submucosal tumors can be removed endoscopically with a snare.

Lipomas and leiomyomas. Lipomas, if asymptomatic, do not have malignant potential and do not need to be removed. Leiomyomas should be removed in the midterm, especially if surveillance shows evidence of growth.

Carcinoids. There are clear treatment guidelines for carcinomas (2). Rectal carcinomas less than 1 cm in size can be removed completely (like benign tumors) using endoscopic polypectomy. Follow-up surveillance is not necessary for carcinomas of this size, as they do not present a risk of metastatization. There is no consensus in the literature on procedures for rectal carcinomas 1–2 cm in size (2). If there is no infiltration or if risks associated with surgical intervention are high, endoscopic ectomy may be considered. If the muscle layer has been infiltrated, surgical resection is absolutely essential and must be followed by endoscopic biopsy and surveillance. Surgical resection is mandatory for carcinoids > 2 cm and must be performed according to oncological criteria, in particular taking into account the proximity of regional lymph nodes and the potential for lymph node metastasis.

Appendiceal carcinoids appearing before age 16 can be removed by appendectomy. Under the age of 16, they are still generally classified as benign lesions. After age 16, right-sided hemicolectomy is indicated because of the danger of lymph node metastasis, especially for tumors larger than 2 cm (1, 5).

Table 11.**2** provides an overview of treatment options for submucosal tumors (2, 4, 5).

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Table 11.**2** Treatment options for submucosal tumors

Submucosal tumor	Endoscopic treatment	Surgical treatment
Lipoma	▶ Small lipomas (< 3 cm): snare polypectomy (if symptomatic)	▶ Large lipomas (> 3 cm); excision (if symptomatic)
Leiomyoma	▶ < 2 cm: snare polypectomy	▶ > 2 cm: extirpation
Leiomyosarcoma	▶ No endoscopic treatment	▶ Resection
Carcinoid	▶ < 1 cm: snare polypectomy ▶ 1–2 cm: snare polypectomy (if high operative risk and no deep infiltration)	▶ 1–2 cm: resection (if deep infiltration) ▶ ≥ 2 cm: oncological resection with removal of regional lymph nodes

12 Colitis—Inflammatory Bowel Diseases and Other Forms of Colitis

R. Scheubel

The following abbreviations are used in this chapter:

CD	=	Crohn disease
DALM	=	dysplasia-associated lesion or mass
DD	=	differential diagnosis
DGVS	=	German Society of Digestive and Metabolic Diseases (*Deutsche Gesellschaft für Verdauungs- und Stoffwechselkrankheiten*)
HGIN	=	high-grade intraepithelial neoplasia
IBD	=	Inflammatory Bowel Disease
LGIN	=	low-grade intraepithelial neoplasia
NSAID	=	nonsteroidal anti-inflammatory drugs
PSC	=	primary sclerosing cholangitis
UC	=	ulcerative colitis

Definition

Colitis. The intestinal mucosa has a limited number of possible reactions to microbial, chemical, or immunological irritants: edema, erythema, erosion, ulcer, necrosis, stricture, and scarring. Various diseases differ in terms of typical characteristics, intensity, and particularly distribution of these reactions, enabling differential diagnosis. Isolated changes are, on the contrary, unspecific. NB: Previous systemic or topical treatments can influence or mask characteristic changes.

Clinical Significance of Chronic Inflammatory Bowel Disease (IBD)

Ulcerative colitis and Crohn disease occur all over the world, but they appear more frequently in western industrialized countries. In North America, an estimated 1.3 million people are affected; in Europe, 1.9 million; and in Germany, 300 000. Pathophysiologically, chronic IBD manifests as a deregulated immune response of the intestinal mucosa to microbes or other environmental irritants in individuals who are genetically predisposed to increased susceptibility. In Europe there is a typical north–south gradient in incidence and prevalence. The rate of Crohn disease in Germany is near the European average, at 5.2 patients per 100 000. The first manifestation usually occurs in earlier decades, though there is a second peak later in life (especially for ulcerative colitis; less so for Crohn disease). The two diseases can be clearly distinguished in terms of immunopathogenesis and clinical appearance. Only 1 % of 5–10 % of attacks remains unclear and is classified as indeterminate colitis. Serological markers (antibodies such as ANCA and ASCA) can be useful for differential diagnosis.

Clinical manifestation. Ulcerative colitis and Crohn disease have a number of symptoms in common: diarrhea, abdominal pain and peranal bleeding. Ulcerative colitis usually occurs in episodes with intermittent periods of remission. Crohn disease may also occur in episodes, but there is an active, chronic form as well. In addition, Crohn disease can be divided into the perforating, fistulizing type, the active, chronic inflammatory type, and the fibrostenotic type. Progression and prognosis may eventually be predictable using genetic markers, but this is not yet possible.

Treatment. Anti-inflammatory drugs can be used to effectively treat the mucosal surface. Corticosteroids and immunosuppressants are the first-line therapy for severe cases. An anti-TNF antibody is sometimes used to treat Crohn disease. Nonetheless, more than 70 % of individuals with Crohn disease have to be operated on during their lifetime and many have signs of recurrence of the disease afterward. Drug therapy usually corresponds to increasing level of severity (step-up approach). A more recent approach involves beginning therapy with a combination of stronger drugs and then reducing their use (top-down approach), but this approach requires further evaluation. Remission-maintaining drugs are indicated for chronic active CD, steroid-refractory CD, and usually the perforating type. However, determining which drugs maintain remission is difficult, not least because endoscopically visible signs of activity do not correlate well with disease activity and progression (5). Ulcerative colitis refractory to treatment is frequently treated with surgical intervention, i. e., usually proctocolectomy. Proctocolectomy is often followed by inflammation in the ileal reservoir, so-called pouchitis.

Diagnosis

Diagnosis of chronic IBD is based on patient medical history and clinical findings. Endoscopy, i. e., ileocolonoscopy with biopsies, is essential and is the main diagnostic procedure. Radiography, ultrasound, and increasingly magnetic resonance imaging (MRI), as well as bacteriological and serological investigation, are also used.

Endoscopy. Colonoscopy is essential for diagnosis, differential diagnosis, and, in isolated cases, assessing disease activity. Differential diagnosis is primarily based on macroscopic findings. Though histological evaluation can also be useful for classification, the pathologist cannot function as a “referee” who makes the final decision in the case of inconclusive endoscopic findings. Endoscopy plays less of a role in monitoring treatment and determining prognosis. In isolated cases, endoscopy can be used therapeutically (CD) and it also plays an important role in carcinoma prevention.

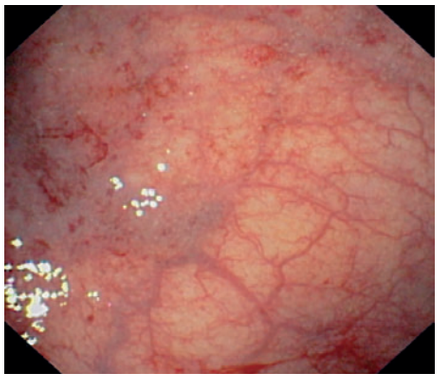


Fig. 12.1 **Ulcerative proctitis.** Sharp demarcation between inflamed mucosa (left) and normal mucosa in upper rectum.

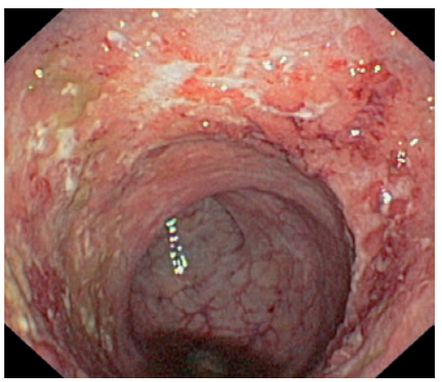


Fig. 12.2 **Ulcerative proctitis.** Florid attack in rectum with abrupt transition to normal sigmoid colon above.

Inflammatory Bowel Disease—Indications for Ileocolonoscopy:

- clarification of longstanding diarrhea (> 4 weeks),
- differentiation of inflammatory changes,
- surveillance for confirming chronic course and thus diagnosis,
- evaluation of disease activity (more for UC than CD),
- determine extent of inflammation related to grave changes in disease course,
- preoperative staging, i. e., evaluation of current attack prior to planned resection,
- routine surveillance of longstanding UC for diagnosing dysplasias (intraepithelial neoplasias) for carcinoma prevention (less clear for CD),
- clarification of recently appearing symptoms (partial obstruction, bleeding),
- therapy: stricture dilation (CD),
- future prospects: diagnosis by means of tissue biopsy for microbiological detection using gene chips

Aim of endoscopy
- diagnosis,
- differential diagnosis,
- evaluation of disease activity (?),
- treatment monitoring (?),
- evaluation of prognosis (?),
- therapy,
- cancer prevention.

Basic colonoscopic inflammatory appearances: swelling, erythema, mucoid or pus secretion, and mild to severe epithelial destruction, ranging from an erosive defect to deep ulceration.

Ulcerative Colitis

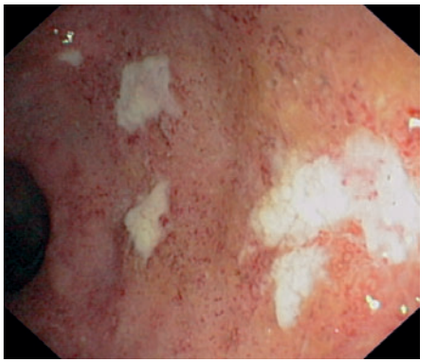
In most cases, ulcerative colitis presents with characteristic endoscopic appearances. Progression corresponds to the length and severity of episodes. Disease course is seldom of a chronic, smoldering nature.

Spread of inflammation. Active UC inflammation of the mucosa is continuous and symmetrical. It can affect the entire colon or only part of it and can occur as proctitis or proctosigmoiditis, spreading into the upper sigmoid colon; left-sided colitis can spread to the splenic flexure or become pancolitis. The rectal mucosa is typically involved. Total colonoscopy can provide exact information about the extent of inflammation. The border between affected and healthy mucosa is usually clearly demarcated (Figs. 12.**1**, 12.**2**). However, discontinuous manifestations of UC have also been cited, e.g., in the form of "proctitis" with "cecitis," so that atypical manifestations must also be considered (1, 2). ▣ 12.**4 b** from our own clinical files shows an example of UC involving the cecum, with remission of the right side and a florid attack involving the left side. Fig. 12.**3** gives an overview of the extent of colonic involvement in ulcerative colitis.

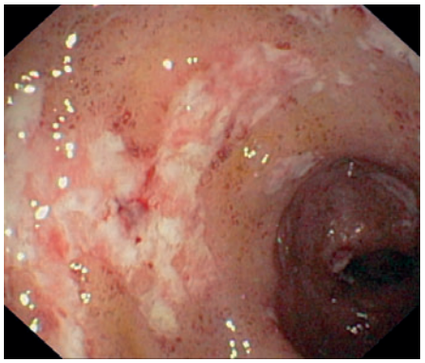
Endoscopic criteria of ulcerative colitis
- increased mucous discharge: early warning sign,
- reflection of light is fragmented, granular, lost vessel pattern: likely a sign of remission, also chronic course (Fig. 12.**1**),
- erythema and increased vulnerability: chronic, mild course,
- marked vulnerability, profuse bleeding after passage of colonoscope: sign of increased disease activity (▣ 12.**2 f**),
- widespread mucopurulent secretion with visible erythema and loss of vessel pattern: active phase (▣ 12.**1 a**, **b**),
- linear, longitudinal ulceration: active phase (▣ 12.**2 a**).

Early signs or signs of recurrence of inflammation include masses of mucous appearing on the mucosal surface as a creamy, whitish coating (▣ 12.**1 e**). With low-grade inflammation, vascular pattern can be distorted, weakened, or lost (due to edema and inflammation). This is partly caused by loss of transparency of the mucosa. Erythema is usually diffuse and is evidence of an active phase, especially when accompanied by granularity and fragmentation of reflected light (Fig. 12.**1**). Mucopurulent secretion is also evidence of increased disease activity (▣ 12.**1 a**, **b**). Figure 12.**1** shows various appearances of fibrinous exudations as well as edematous and granulated mucosa in an acute episode.

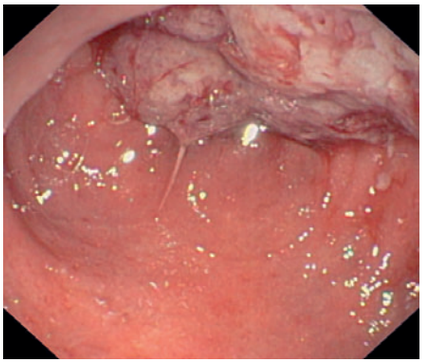
12.1 Ulcerative colitis: fibrinous exudates, edematous and granulated mucosa in an acute episode



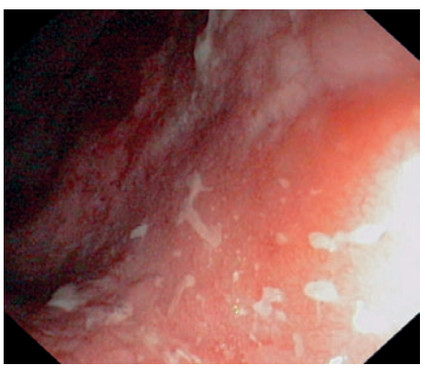
a Recent fibrinous exudates, appearing as a whitish covering on an already edematous mucosa (sigmoid colon).



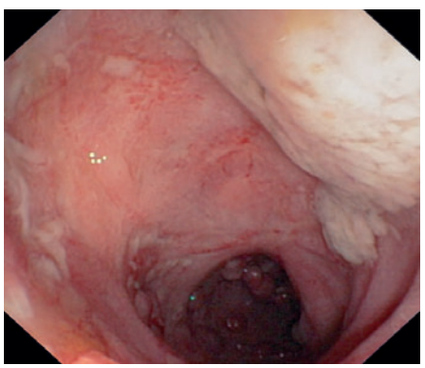
b Fibrinous material on an edematous and granulated mucosa (rectum).



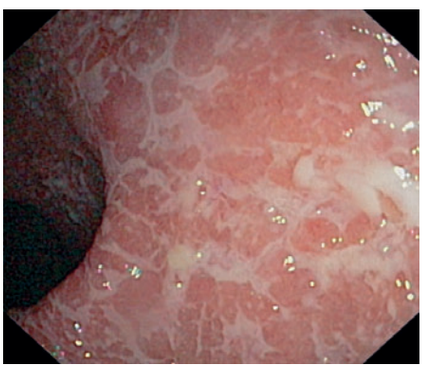
c Masses of fibrin and blood (sigmoid colon).



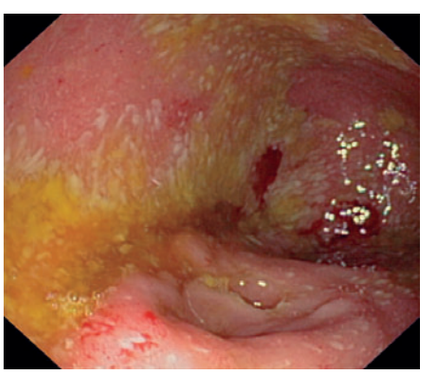
d Ulcerative proctitis: fibrinous exudates.



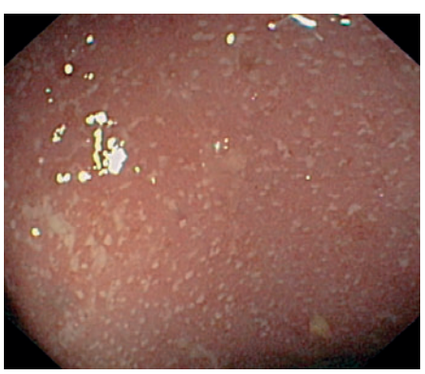
e Pancolitis, chronic ulcerations, also recent attack with fibrinous material in the ascending colon.



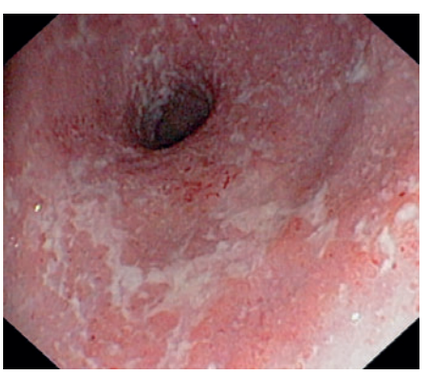
f Florid inflammation in the sigmoid colon with typical fresh fibrinous coverings, already weblike, precursor of ulceration.



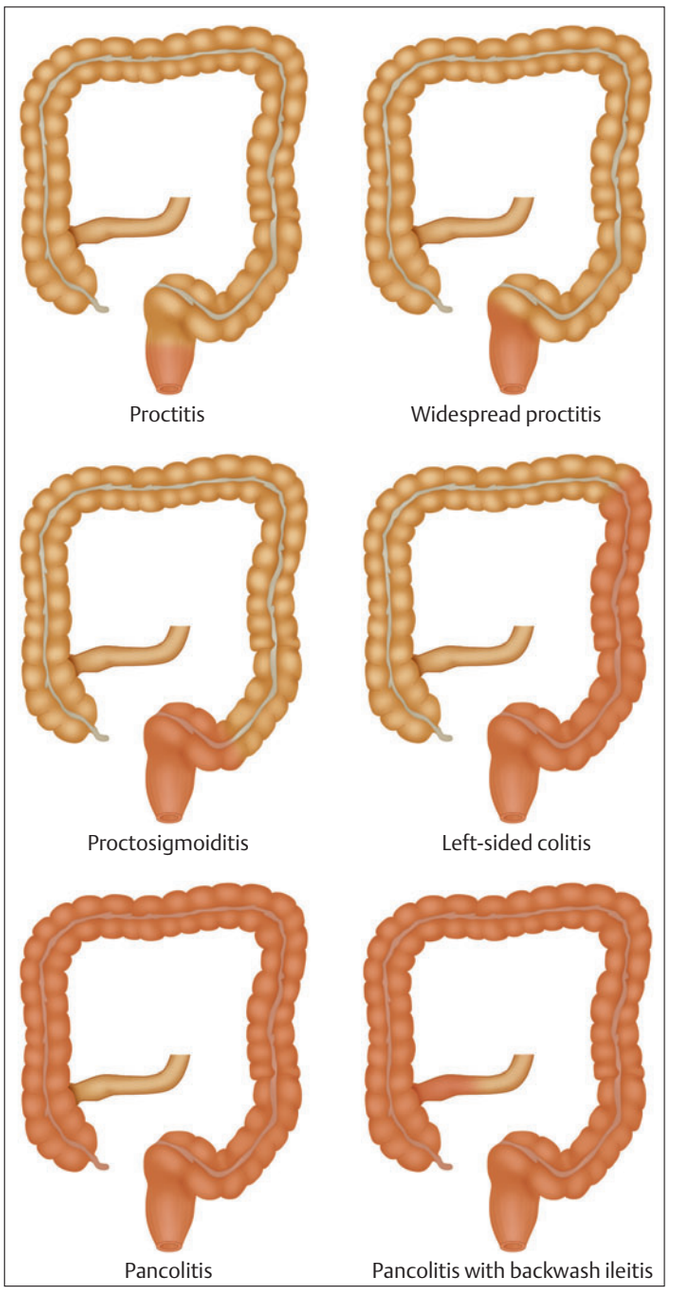
g Recent attack of pancolitis, fibrinous exudates in the cecum. The ileocecal valve and small intestinal mucosa are not involved and are not inflamed.



h Ulcerative proctitis: tiny dot-shaped fibrinous exudates (rectum).



i Florid proctitis, Löfberg (grade 2) with confluent weblike fibrinous coatings, also increased vulnerability.



Surface necroses or deep, epithelial necroses are a result of widespread epithelial destruction and abscessed crypts. Single or multiple ulcerations can be related to active UC and can be larger or smaller in size (▣ 12.**2a**). Regardless of the various shapes—linear, serpiginous ("snakelike"), circular, or oval—all of these ulcers share the common characteristic of typically being surrounded by a reddened and vulnerable mucosa (▣ 12.**2**). Petechiae are another sign of disease activity (Figs. 12.**2c**, 12.**4**). Ulcerations and mucosal destruction can sometimes cover a wide surface area, so that only islets of "normal" mucosa remain (Fig. 12.**5**). (Differential diagnosis: such extreme patchiness related to ulcerations does not appear with Crohn disease). Renewed spread of florid inflammation may initially leave individual mucosal islets with normal, intact vessel pattern (Fig. 12.**6**).

Löfberg score. Löfberg (1994) has proposed an endoscopic classification system of severity ("score") of UC. This classification is applied more often in studies than in everyday use (▣ 12.**3**).

Pseudopolyps. Pseudopolyps are knobby, bulging areas of mucosa resulting from necrosis. They occur as multiple polyps and can be several centimeters long. They often have a soft, whitish surface (Figs. 12.**7**–12.**9**), though sometimes they can be tinged with blood. Pseudopolyps can also occur as bridging folds.

Chronic course. Longstanding inflammation can lead to mucosal atrophy and loss of haustra, which gives the colonic lumen a tubelike appearance (▣ 12.**4a**) (analogous to a "stiff tube" in radiological imaging). Figure 12.**4** shows chronic forms of UC, some with atrophied mucosa.

◁ Fig. 12.**3** **Varying extent of colonic involvement in ulcerative colitis.**

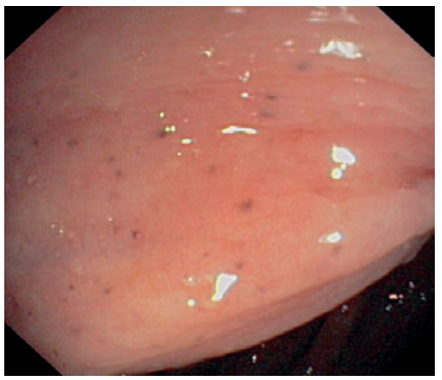


Fig. 12.**4** **Minimal inflammation,** already abating, traces of hemosiderin resulting from petechial bleeding (sigmoid colon).

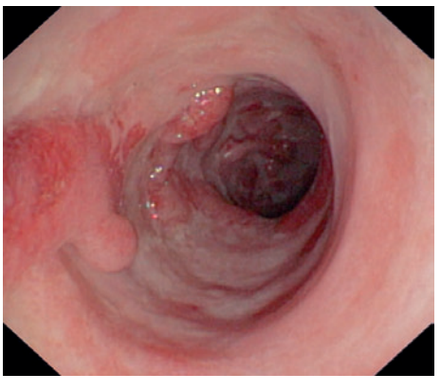


Fig. 12.**5** **Nearly circular mucosal necrosis** in chronic UC, with only pseudopolypoid, regenerated mucosa remaining (sigmoid colon).

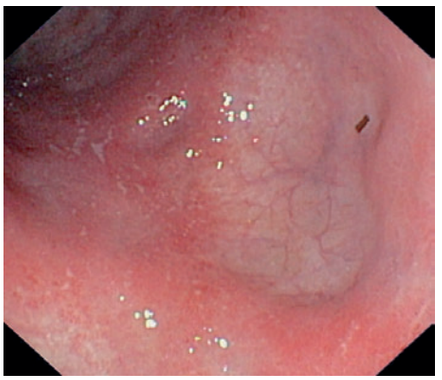
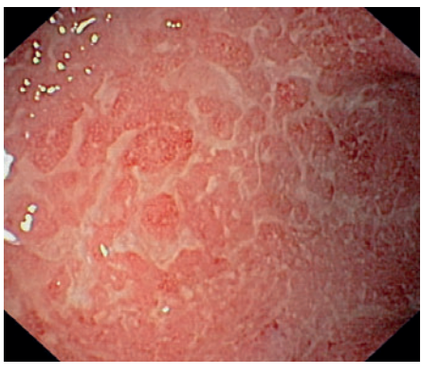
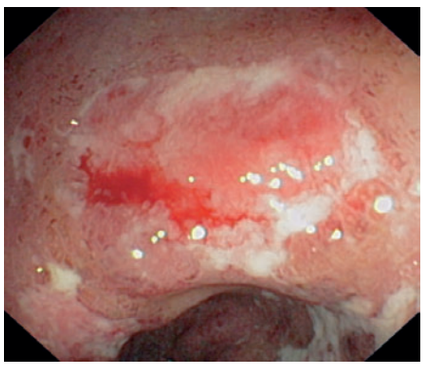


Fig. 12.**6** **Florid sigmoiditis in UC.** Inflammation spreading to proximal colon in recent onset of recurrent attack, delayed onset in specific areas (right side of image) vessel pattern is remaining, only slightly altered by prior attack.

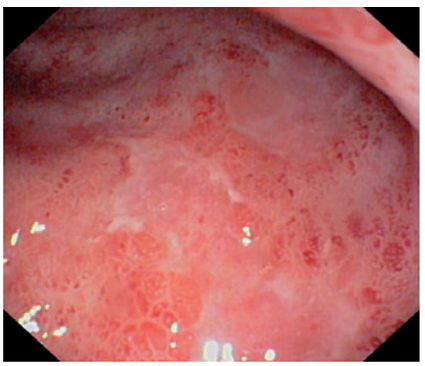
12.2 Ulcerations in ulcerative colitis



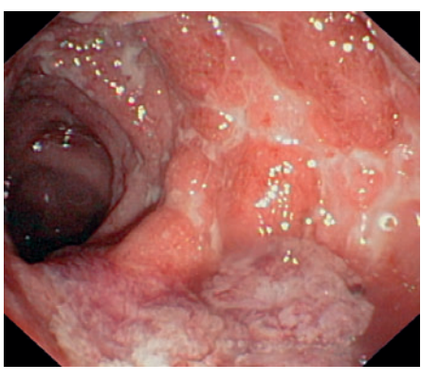
a Weblike confluent ulcerations in an acute episode (rectum).



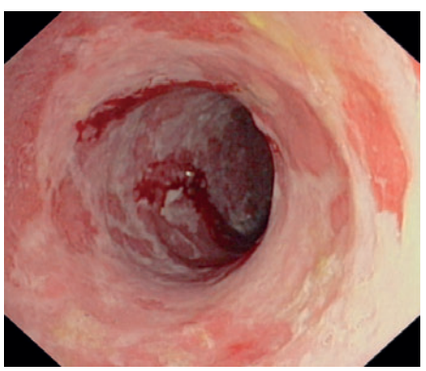
b Recent attack: flat, recent ulcer, fibrin in the background (sigmoid colon).



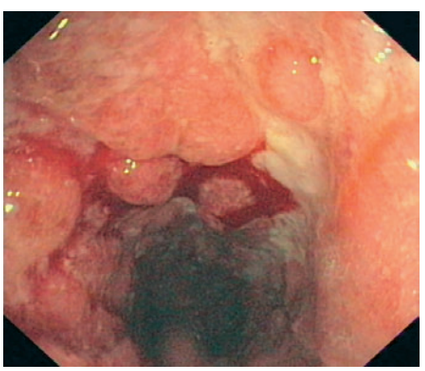
c Flat ulceration and mucosal petechiae in a treated attack (sigmoid colon).



d Confluent ulcers and fibrinous masses in an acute attack (lower sigmoid colon).

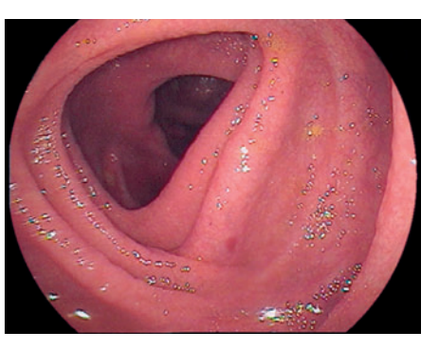


e Significant, almost circular mucosal necroses and flat ulcerations related to active chronic UC with persistent attack.

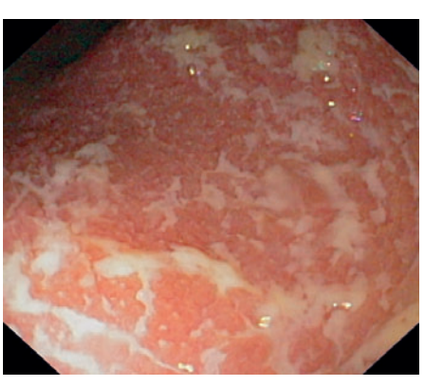


f Deep ulcerations can appear with a high degree of activity; vulnerability is overall greatly increased. Distinguishing UC from other forms of colitis is more difficult here; main criteria for UC are continuous inflammatory process and mucosal erythema.

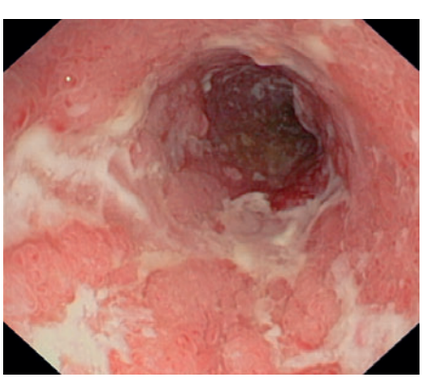
12.3 Löfberg Classification of UC



Löfberg score, grade 1
diffuse granularity, edema, fragmented light reflection, loss of normal vascular pattern.



Löfberg score, grade 2
all criteria of grade 1 + hyperemia, vulnerability, fibrinous exudates.



Löfberg score, grade 3
all criteria of grades 1 and 2 + ulcerations.

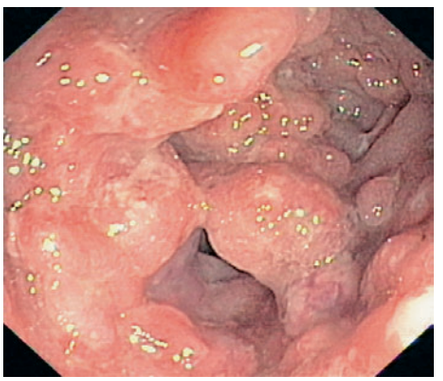


Fig. 12.7 **Pronounced development of pseudopolyps** with signs of florid inflammation on their surfaces; causing moderate stenosis.

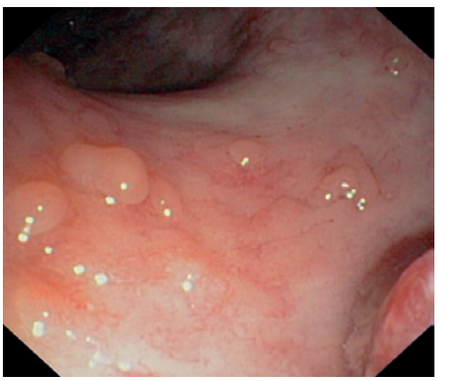


Fig. 12.8 **Pale tiny pseudopolyp,** smooth surface, thus appearing like remaining local mucosa or partially regenerated mucosa.

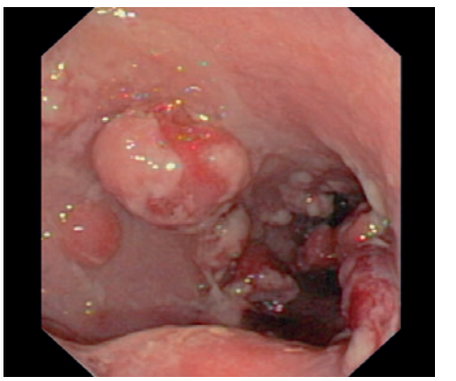
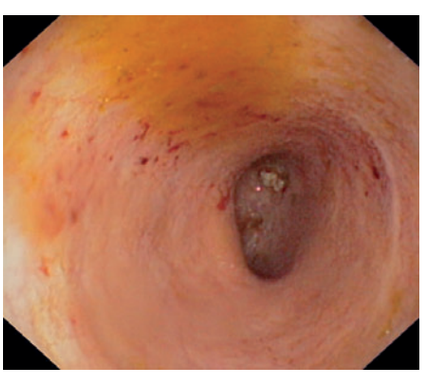
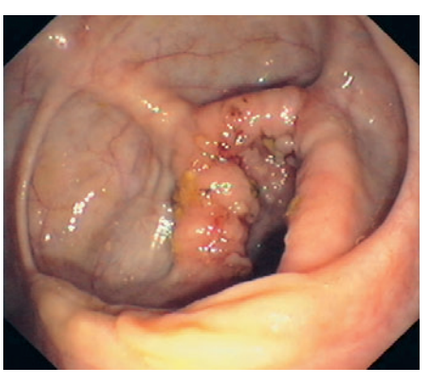


Fig. 12.9 **Pseudopolyps with typical whitish surface, similar to a sugar glaze**, which usually does not cover the polyp completely.

12.4 Clinical course of ulcerative colitis: chronic form



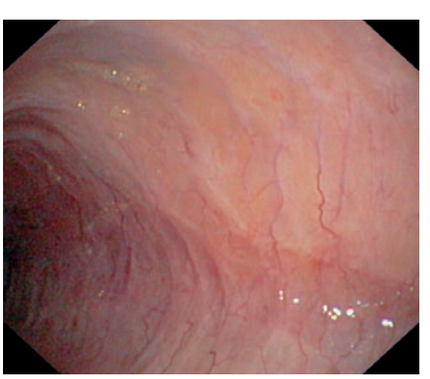
a



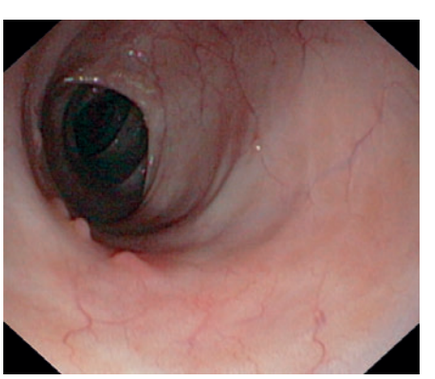
b

a Pancolitis, longer duration, atrophied mucosa, and loss of haustra in ascending colon.

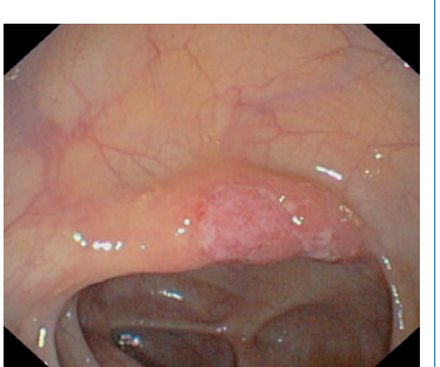
b Longstanding ulcerative pancolitis, view into the ascending colon and cecum with apparently atrophied mucosa of the ascending colon, but florid changes in cecum (cecitis).



c



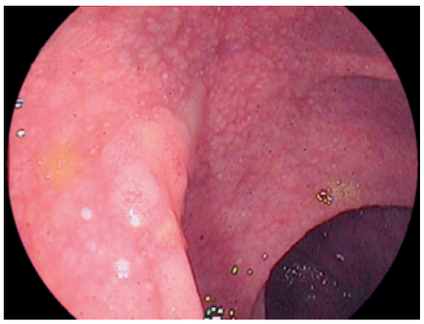
d



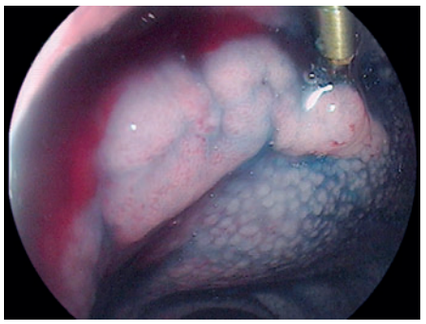
e

c–e Inactive stage. **c** Atrophied mucosa, whitish with solitary, regenerated vessel. Destroyed vessel pattern is always a sign of prior attack and after several episodes it will never return to its normal condition. **d** Another inactive area. The mucosa is flat and hardly rises during biopsy. A few elevated mucosal segments in between, so-called pseudopolyps. **e** Even in inactive intestinal segments, patchy inflammation focals can appear; in this case it is a circumscribed area which shows all typical characteristics of a recently inflamed area.

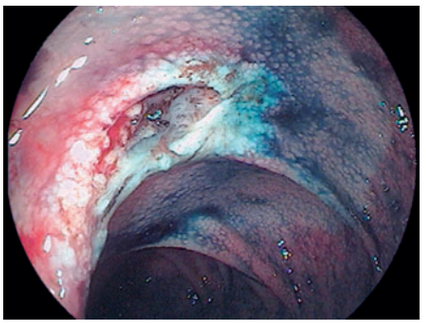
12.5 DALM (Dysplasia-associated lesion or mass) in ulcerative colitis



a

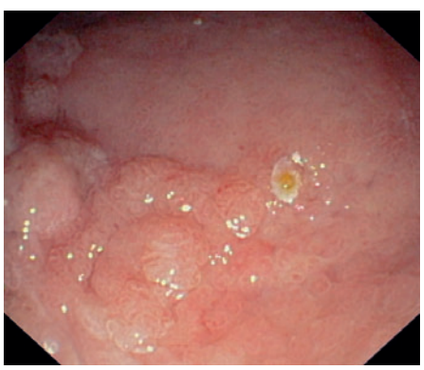


b

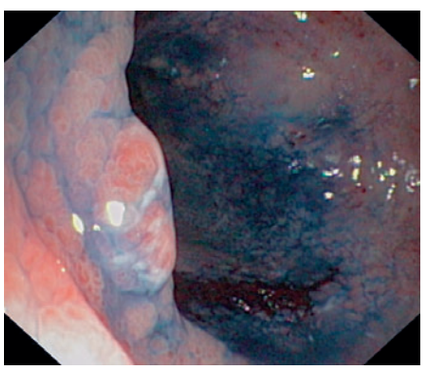


c

a–c DALM resection at the hepatic flexure. **a** Flat polypoid lesion in the hepatic flexure, atypical appearance for an adenoma: DALM, histologically LGIN. **b** After indigo carmine staining and injection: DALM at the hepatic flexure. **c** Previous endoscopic resection of the DALM lesion (histologically: no further intraepithelial neoplasias in the surrounding area).

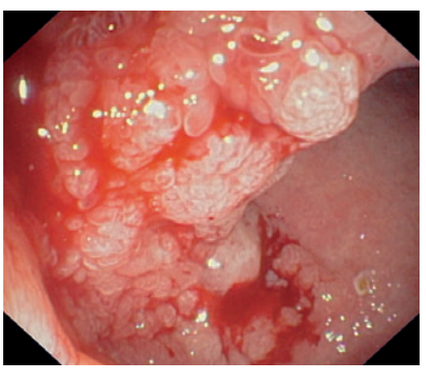


d



e

d, e DALM in the sigmoid colon. **d** Raised polypoid structure in the sigmoid colon (DALM). The patient also had backwash ileitis and PSC. **e** The somewhat displaced lesion can be better visualized against the surrounding granulated mucosa with chromoendoscopy (indigo carmine). Histologically HGIN, partly carcinoma in situ. Afterward, the patient underwent proctocolectomy, which did not reveal an invasive carcinoma.



f DALM in rectum, tinged with blood.

DALM. Polyps can occur as pseudopolyps, as adenomalike polyps, or as other types that, if they show signs of dysplasia or intraepithelial neoplasia, are called DALM (dysplasia associated lesion or mass) (▣ 12.**5**). Adenomalike polyps, and sometimes even DALM, can be removed by polypectomy, which can also be the definitive treatment (▣ 12.**5 c**).

However, polypectomy can only be the definitive therapy for DALM when the presence of dysplasia (intraepithelial neoplasias) in the flat mucosa has been investigated and ruled out using multiple biopsies. This sometimes requires a very large number of excisional biopsies. It takes 33 excisional biopsies to find dysplasia with 90% certainty and 56 excisional biopsies per examination for 95% certainty (4). Recent studies have shown that targeted biopsies following methylene blue staining could increase the rate of detection of dysplastic epithelium in flat mucosa (3) (▣ 12.**5 e**).

Note
Current DGVS guidelines recommend: after evaluation by two pathologists, colectomy is recommended for high grade dysplasia (intraepithelial neoplasias) and has to be discussed for low grade dysplasia.

Terminal ileitis ("backwash ileitis")
Pancolitis can involve the appendix (Fig. 12.**10**) and the ileocecal valve, causing a rigid and dilated opening. The last segment of the distal terminal ileum is then often involved in backwash ileitis (Figs. 12.**11**–12.**13**), usually involving diffuse swelling and reddening of the ileal mucosa (Fig. 12.**13**). Ulcerations do not occur very often and changes seldom extend more than a maximum of 12–15 cm into the ileum.

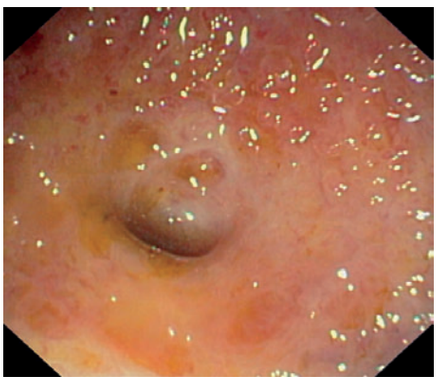


Fig. 12.**10** **Pancolitis in UC.** The appendix is also involved and the appendiceal opening shows typical ulceration.

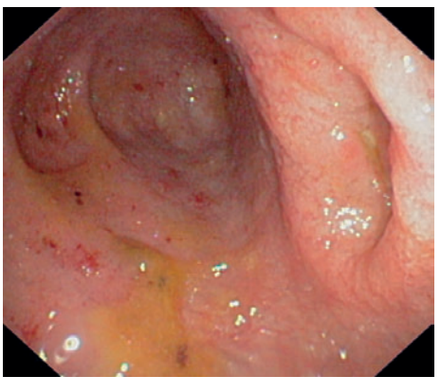


Fig. 12.**11** **Chronic pancolitis in UC.** Ileocecal valve involvement and backwash ileitis. The same patient also has PSC.

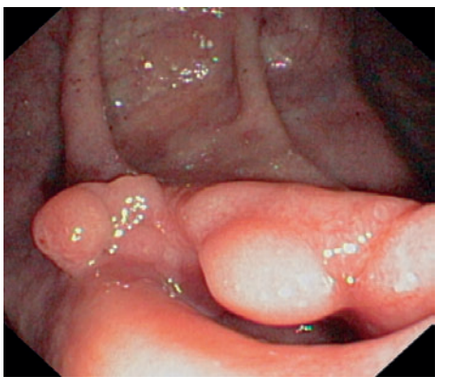


Fig. 12.**12** **Pancolitis in UC.** Ileocecal valve involved in pronounced backwash ileitis and showing polypoid changes.

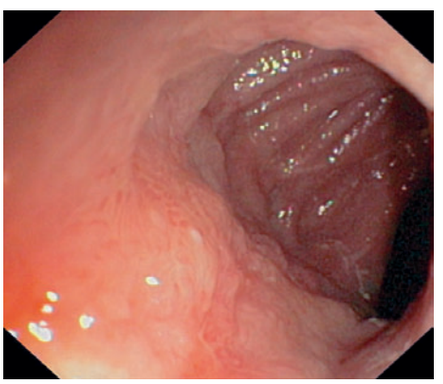


Fig. 12.**13** **Pronounced backwash ileitis.** Edema, granularity, and formation of a small aphthous ulcer in the terminal ileum.

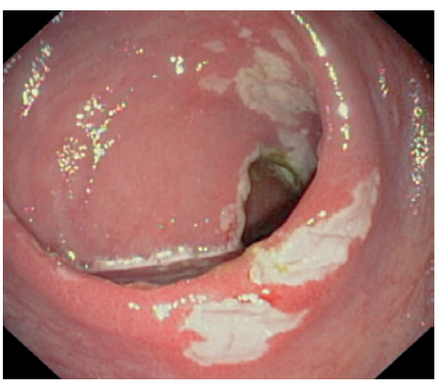


Fig. 12.**14** **Ileoanal pouch inflammation (pouchitis).** Acute inflammatory changes similar to UC with somewhat patchy fibrinous plaques and significant erythema.

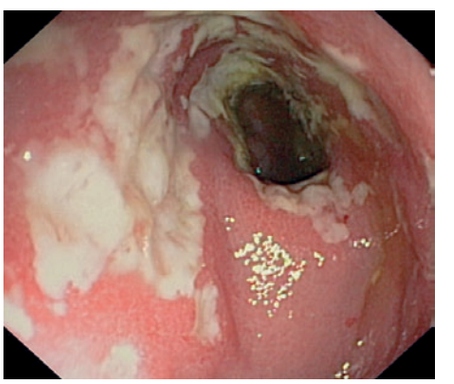


Fig. 12.**15** **Pouchitis (different segment).** The changes here are more pronounced (simultaneous radiology of this region due to carcinoma in the anal transitional zone).

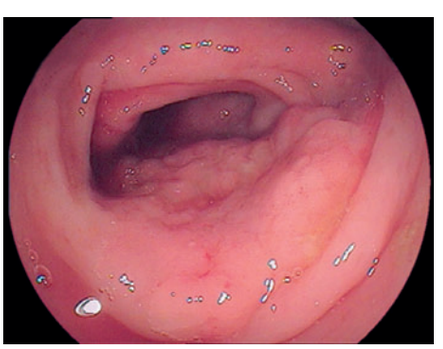


Fig. 12.**16** **Pancolitis in UC, colitis carcinoma, hepatic flexure.** Broad-based polyp with (tubulo)villous surface, appearing somewhat irregular, but not typical for a carcinoma. Stage pT3 carcinoma detected at colectomy.

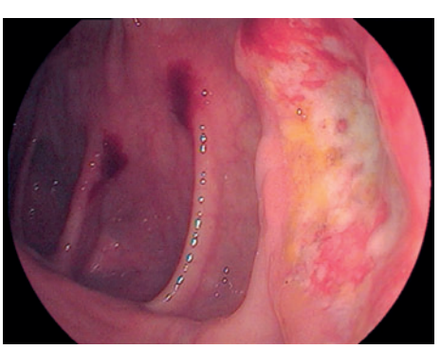


Fig. 12.**17** **Pancolitis in UC, colitis carcinoma in the ascending colon.** An apparently previously polypoid structure, now showing widespread ulceration and crater formation. Dysplasias were described at this localization six months previously. Now pT3, N1 carcinoma with three positive lymph nodes.

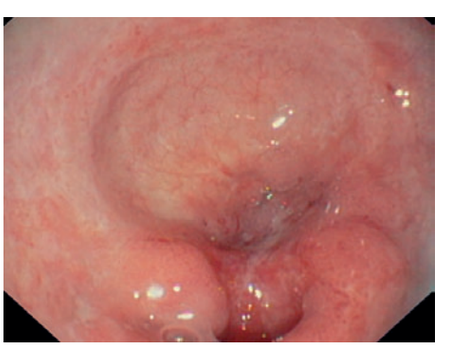


Fig. 12.**18** **Pancolitis related to UC, colitis carcinoma in the cecum,** initiated at the edge of the ileocecal valve.

Pouchitis

Pouchitis is an inflammation in the ileal reservoir following proctocolectomy and anastomosis of an ileoanal pouch to the anus. Clinical diagnosis must be confirmed by both endoscopy *and* histology (7).

The most common endoscopic criteria of acute pouchitis are erythema, edema, vulnerability, petechiae, granularity, contact bleeding, fibrinous discharge, erosions, and small ulcerations, similar to UC. Not all patients exhibit these macroscopic signs of extent of inflammation; histological evaluation can reveal a more severe degree of disease activity than is endoscopically visible. Thus, a biopsy is absolutely essential. Among other things, it can be used to classify activity according to number of crypt abscesses (Figs. 12.**14**, 12.**15**).

Toxic Megacolon

Toxic megacolon is a contraindication for colonoscopy due to high risk of perforation.

Carcinoma in Ulcerative Colitis

Carcinoma can appear in various forms, ranging from a vast polypoid area with an irregular surface to a typical, depressed tumor form. (Figs. 12.**16**–12.**18**).

Crohn Disease

Crohn disease is a chronic inflammation that commonly involves the terminal ileum and various segments of the colon. Colon involvement is present in two-thirds of patients.

Early Crohn disease (preceding aphthous ulceration) presents reddened patches with distorted vascular pattern. Aphthous erosions are considered early, relatively specific signs of Crohn disease. They are flat and usually less than 5 mm in diameter, with a characteristically narrow, reddened margin and a yellowish or grayish center, i.e., a hyperemic ring surrounding a fibrinous necrosis (▣ 12.**6**). They present in otherwise normal appearing mucosa at some distance to severe lesions.

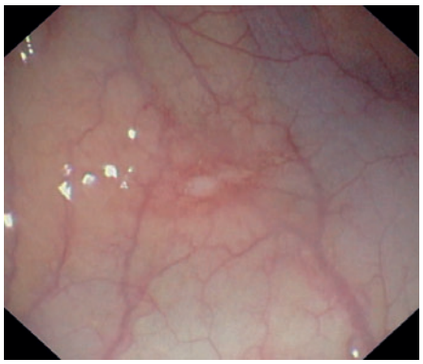
Normal mucosal segments are interspersed with abnormal segments. Interspersion of normal and affected segments is also referred to as a "skip lesion" (Figs. 12.**19**, 12.**20**). Ulcerations can be larger, but are still surrounded by normal mucosa (▣ 12.**17 a**). They can range from flat to depressed and some are winding and snakelike (serpiginous) (▣ 12.**7 b**), though they are often parallel to one another longitudinally (▣ 12.**7 c**, **e**, **f**).

A "cobblestone" appearance is typical, but not specific, arising from the intersection of longitudinal and transverse ulcerations and fissures (Figs. 12.**21**, 12.**22**). In general, cobblestoning affects shorter segments.

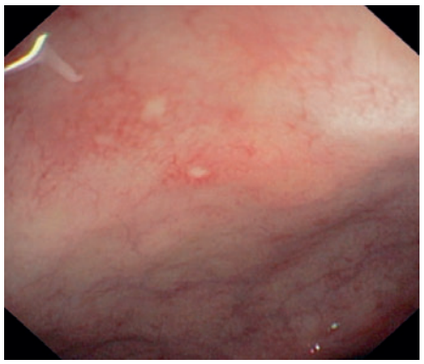
Pseudopolyps occur less frequently than with UC (Figs. 12.**23**–12.**25**). Strictures are common. They can have a purely scarred appearance, but they often also appear with ulcerations arising from underlying transmural inflammation. Scarring is the result of prior inflammatory episodes (Fig. 12.**26**).

Fistula openings are often visible and then are usually surrounded by edema and erythema. Endoscopy provides the option of introducing contrast agents into the lumen or through a stricture, e.g., using a probe inserted through the colonoscope in order to visualize fistulous tracts by means of fluoroscopy.

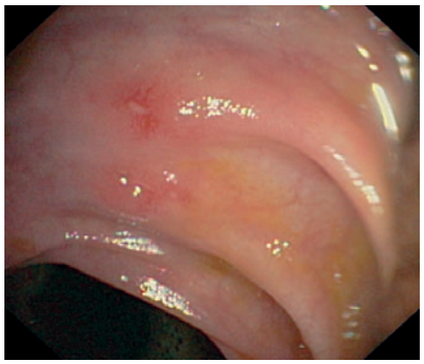
▣ **12.6 Aphthous erosions in early phase of Crohn disease**



a Typical aphthous erosions in an otherwise normal surrounding (cecum).

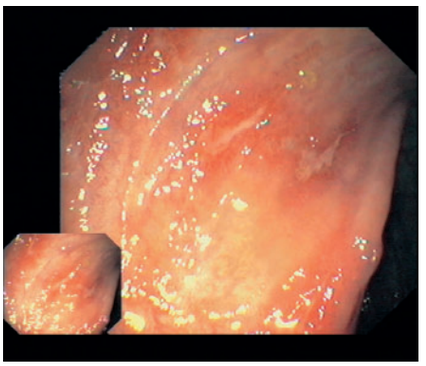


b Aphthous erosion in ascending colon.

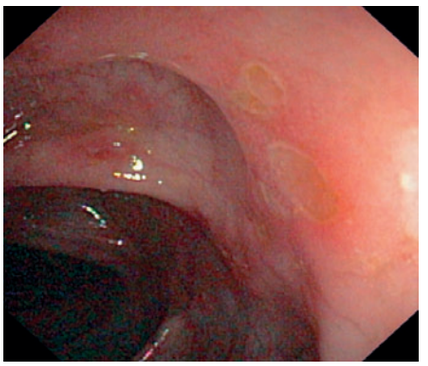


c Aphthae in the sigmoid colon. Other lesions were also nearby.

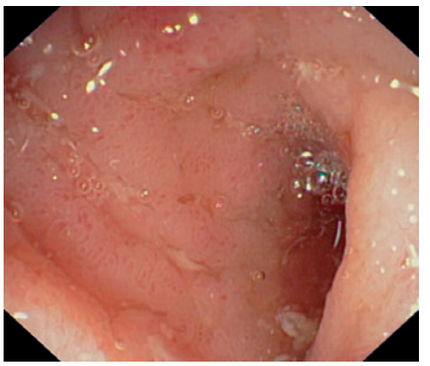
12.6 cont.



d Larger erosions (ascending colon).

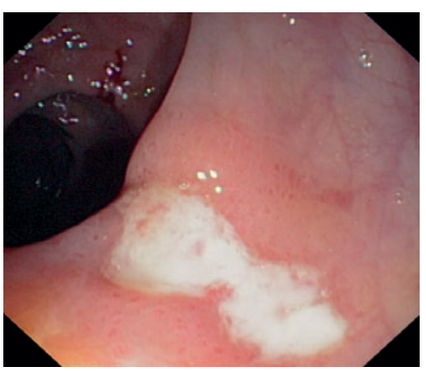


e Giant aphthae with punched-out appearance or mini-ulcers in right transverse colon.

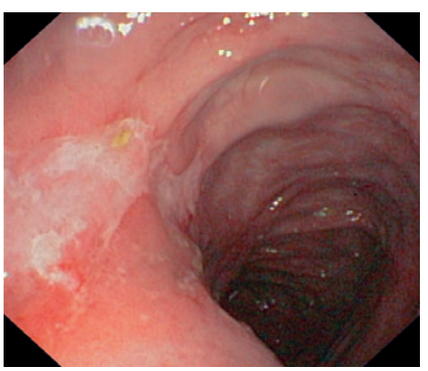


f Attack in terminal ileum, more aphthous character.

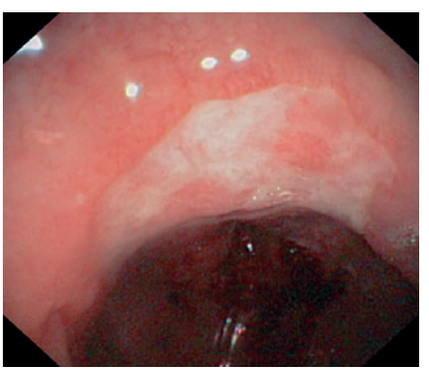
12.7 Ulcers in Crohn disease



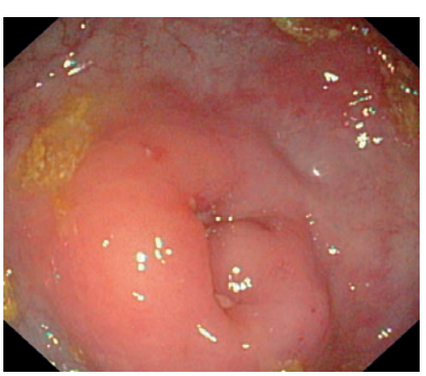
a Elongated ulcer in otherwise normal area of sigmoid colon.



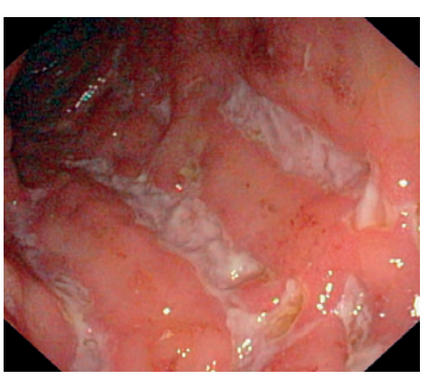
b Serpiginous ulcer, surrounding area quite normal (ascending colon).



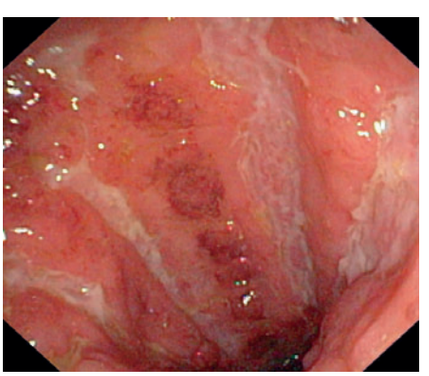
c Oval ulcer at hepatic flexure.



d Appendix involvement: appendix edges are completely swollen, two small ulcers inside.

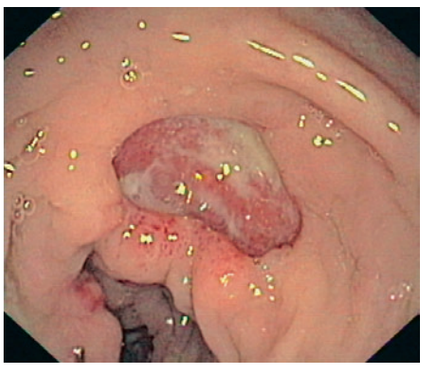


e Long, vaguely serpiginous, parallel ulcerations in descending colon; typical CD finding.

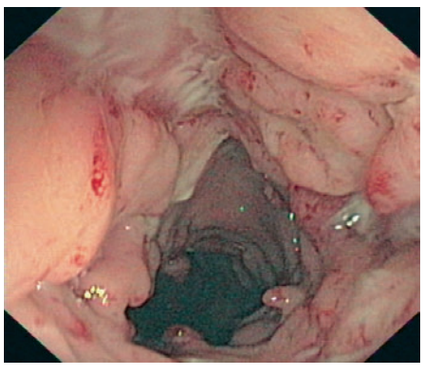


f Typical elongated ulcer in descending colon, extending toward upper sigmoid colon.

12.7 cont.

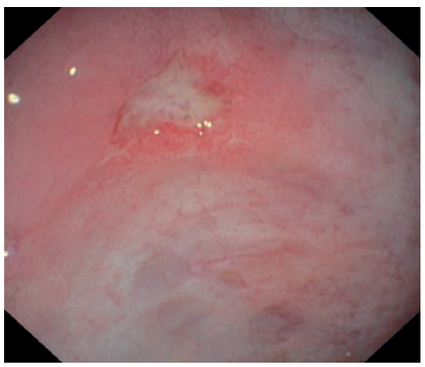


g



h

g, h Recent diagnosis. **g** Recent ulcer, pitlike appearance (ascending colon). **h** Longitudinal ulcers and jagged mucosa; the totality of these changes enables diagnosis.



i Solitary mini-ulcers in Crohn disease in ascending colon.

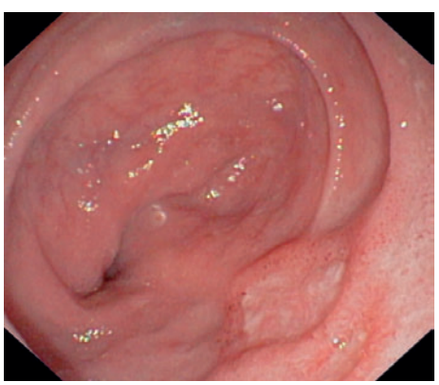


Fig. 12.**19** **CD in terminal ileum with a "skip lesion,"** i. e., an eccentric ulcer, a relatively wide ileal segment and low or moderate inflammation of the rest of the ileal wall.

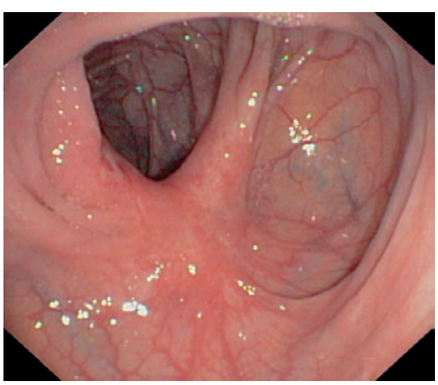


Fig. 12.**20** **Scarring and discrete inflammatory change** (vaguely aphthous), eccentric, i. e., "skip lesion" at the splenic flexure.

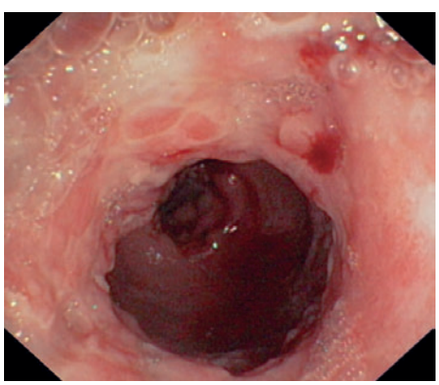


Fig. 12.**21** **Cobblestoning beginning to form in CD** in the ascending colon.

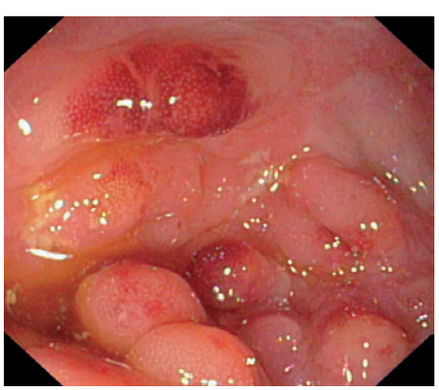


Fig. 12.**22** **Mixture of cobblestoning, pseudopolyp formation, and relative stenosis in the sigmoid colon related to CD.** Several typical findings can appear simultaneously at varying locations in the intestine.

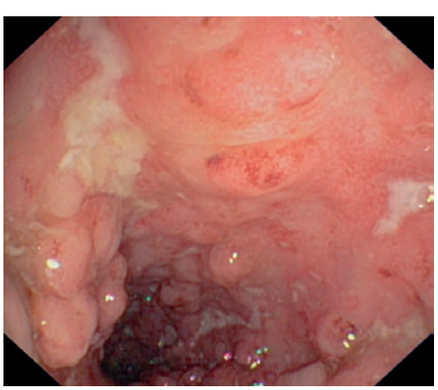


Fig. 12.**23** **Pancolitis Crohn with varied appearance:** aphthous erythema, pseudopolyps, and small ulcers at the same time in ascending colon.

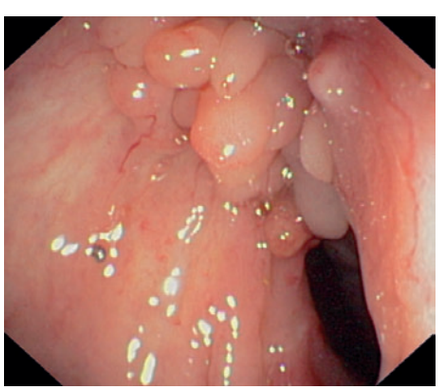


Fig. 12.**24** **Relative stenosis and pseudopolyp bouquet in sigmoid colon.** There is also a fistulous tract ending here, originating in the neoterminal ileum (diagnosed at resection, not endoscopically identifiable).

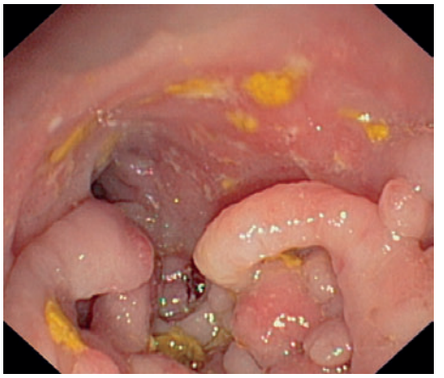


Fig. 12.**25** **Pronounced pseudopolyps and chronic inflammatory stenosis in sigmoid colon.** Adjacent are aphthae and patchy inflammation focals typical of CD.

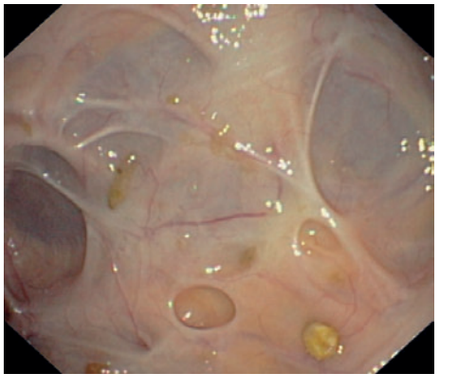


Fig. 12.**26** **Pronounced mucosal scarring after intense immune suppression** (steroids, azathioprine).

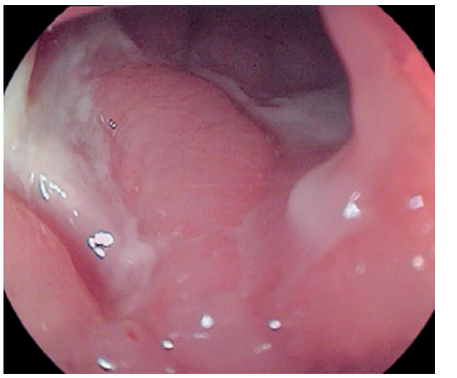


Fig. 12.**27** **View into terminal ileum with pronounced ulcerations** (finding largely unchanged one year later).

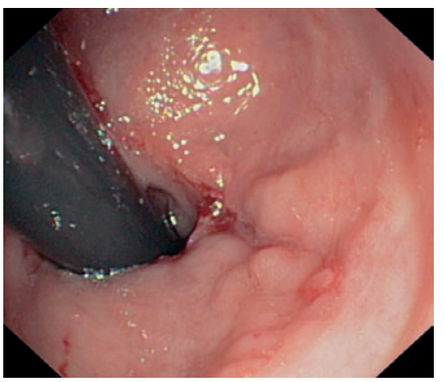


Fig. 12.**28** **Invasion of anal canal and lower rectum** with vaguely cobblestone appearance and fissurizing ulcerations as well as relative narrowing of anal canal (retroflexed instrument).

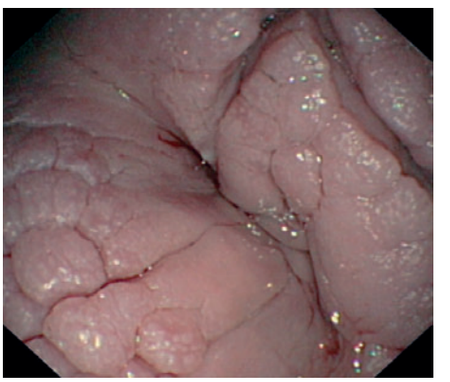


Fig. 12.**29** **Inspection of perianal region** revealing extreme thickening of skin folds (chronic inflammation infiltration), an outward early warning sign of a massive CD attack in the anal canal.

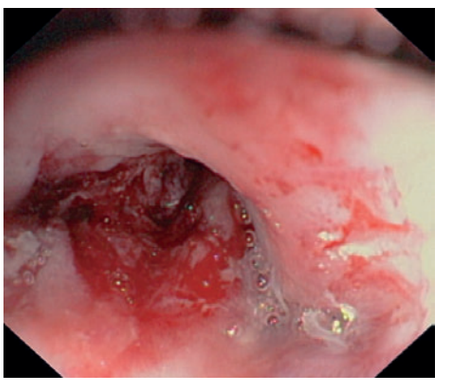


Fig. 12.**30** **View into the anal canal, tinged with blood,** immediately after bougienage in anal canal attack with severe stenosis.

Examining the terminal ileum

An attack involving the ileocecal valve frequently results in stenosis caused by ulceration and scarring, which in turn often prevents passage of the colonoscope into the terminal ileum (▣12.**8**). If passage is possible, aphthous erosions or ulcers of various sizes (sometimes stenosing) are typically seen in the ileum. In the distal terminal ileum, changes can affect the entire circumference; in the proximal ileal segment, changes can also be focal (Fig. 12.**27**). Especially among younger patients, differential diagnosis should exclude lymphoid hyperplasia in its polypoid form.

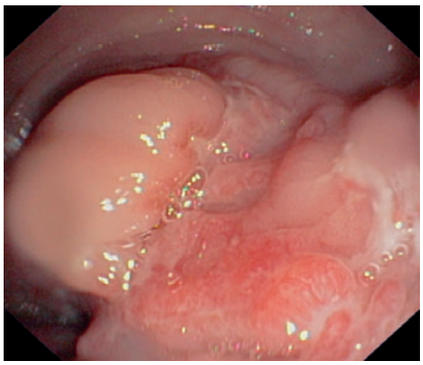
Examination in patients of prior ileocecal resection

CD typically recurs in the anastomosed region with erosions, ulcerations, and increasing stenosis (▣12.**9**). It is usually found very close to the anastomosis in the neoterminal ileum. Deep ulcerations near the anastomosis can be precursors of a high-grade stricture. If the strictured area is less than 5–8 cm long, it is probably passable using balloon dilation (see Chapter 21). Rutgeerts et al. (6) have proposed that clinical relapse can be predicted by severity of inflammation as evaluated endoscopically. This view has not found wide acceptance since a postoperative routine evaluation of the anastomosis is not considered necessarily indicated, but instead is decided on a case-to-case basis, in particular, if there is doubt concerning indications for remission-maintaining drugs.

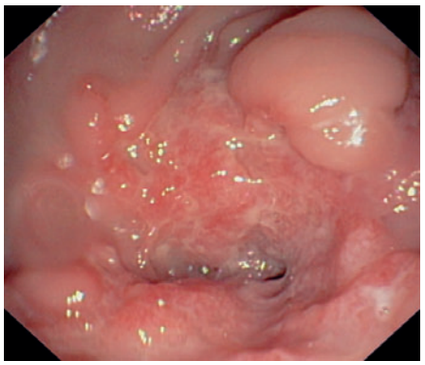
Examining the anal canal

The anal canal is sometimes affected by inflammatory infiltrates, ulcerations, and ultimately abscesses and scarring which can cause strictures (Figs. 12.**28**, 12.**29**). The use of a small-caliber endoscope or even bougienage—either digitally or using an endoscope—is sometimes necessary (Fig. 12.**30**), under appropriate sedation, in order to enable examination of rectal segments. Attack in the rectum does not necessarily accompany anal attack, but is often present.

12.8 Inflammatory atrophy and stenosis in the ileocecal region in CD



a



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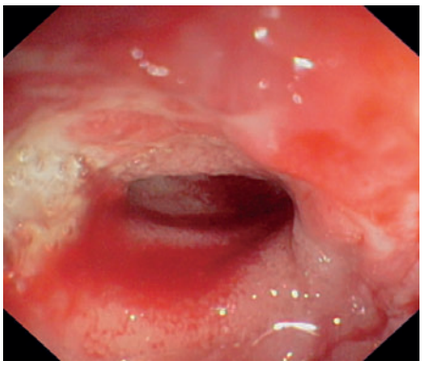
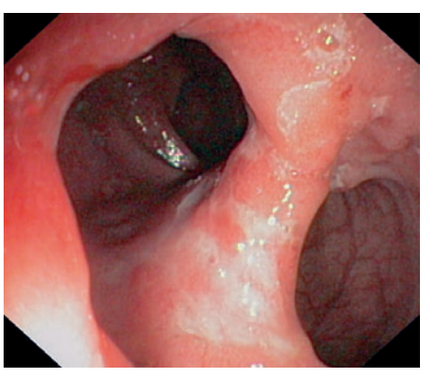


a and **b** Inflammatory atrophy of the cecum. **a** With wide, irregular ulcerations. **b** Atrophy of the cecum, viewed from ascending colon.

c Narrow, inflammatory atrophied ileocecal valve, cannot be intubated, initial flat ulcerations.



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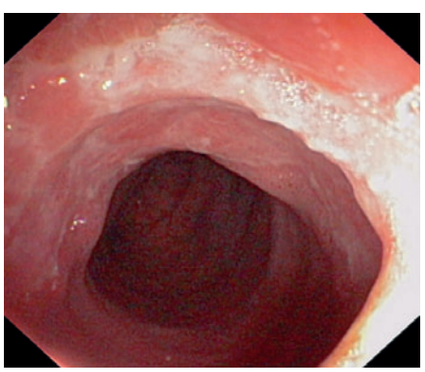

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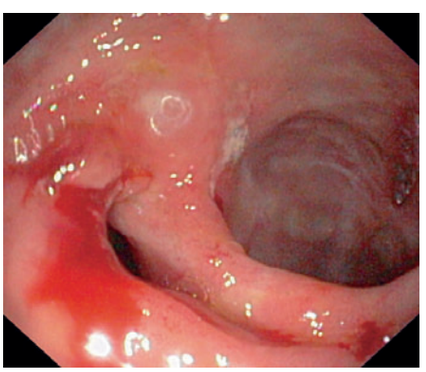
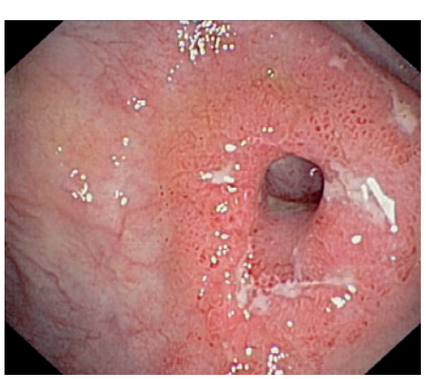


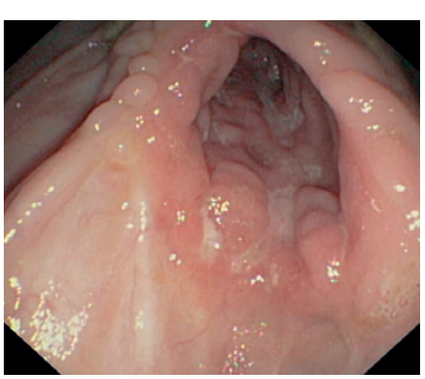
d and **e** Rigid ileocecal valve. **d** View into the valve. **e** Further in the terminal ileum: narrowing with semicircular ulcer, no longer readily passable, behind it again a broader segment partly with “colonlike” mucosa and vessels.

f Ileocecal valve with inflammation and atrophy, bleeding after unsuccessful intubation attempt.

12.9 Recurrent CD near anastomosis following ileal resection



a



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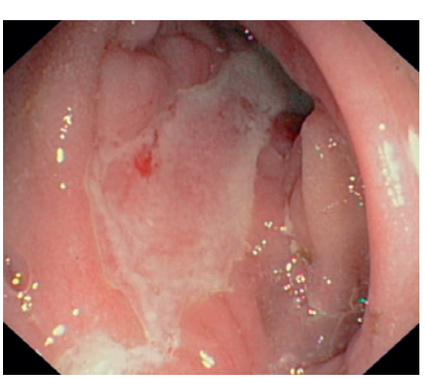
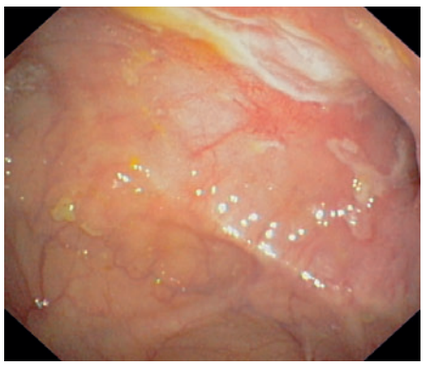


c

a Stenosed ileocolonic anastomosis of ileum and ascending colon, not passable with instrument. Inflammatory changes (aphthae, mini-ulcers) visible in and around the stenosis. This is a frequent occurrence; changes are not usually limited to scarring in this region, and are most likely after intense immunosuppression.

b, c Typical attack in the neoterminal ileum. **b** Stenosed ileocolonic anastomosis of ileum and ascending colon. Here it is passable, elongated ulcer typical of CD. **c** Wide ulcer in neoterminal ileum.

■ **12.9 cont.**



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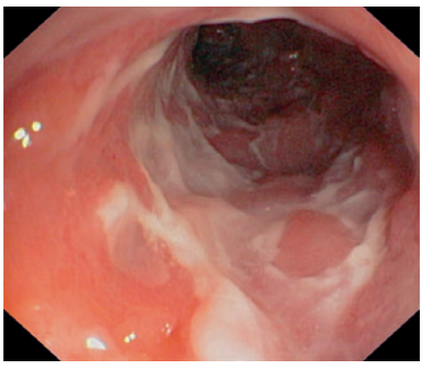
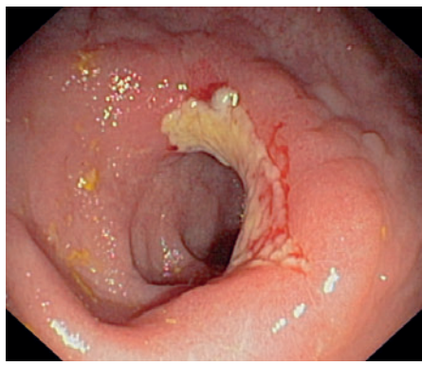


e

d, e Florid inflammation in anastomotic region. **d** View into the ascending colon toward anastomosis. **e** View into neoterminal ileum, highly florid attack, but without significant narrowing (which, however, is imminent in the course of disease).



f Chronic ulcer at ileocolonic anastomosis, no stenosing tendency for years.

Differential Diagnosis of Chronic IBD Types and Other Forms of Inflammatory Bowel Diseases

■ Differential Diagnosis: Ulcerative Colitis vs. Crohn Disease

Tables 12.**1**, 12.**2** display major characteristics for distinguishing between the two main types of chronic IBD.

Table 12.**1** Endoscopic differential diagnosis CD–UC (based on reference 8)

Factors supporting a diagnosis of CD	Factors supporting a diagnosis of UC
▶ key finding: patchy, discontinuous and segmental spread of inflammation	▶ continuous, symmetrical, diffuse inflammation
▶ areas of normal mucosal interspersed with ulcerations; or asymmetrical distribution of ulcerations in a given segment	▶ mucosa in affected segment noticeably different from the surrounding mucosa, sharp demarcation to proximal, uninvolved mucosa
▶ rectum is spared	▶ diffuse rectal involvement
▶ ulceration surrounded by relatively normal mucosa, no marked increase in vulnerability of surrounding mucosa	▶ ulceration on a background of diffuse abnormal mucosa with reddening, vulnerability, and granularity
▶ deep, widespread ulcerations ▶ ulcerations and stenosis or distortion of the Bauhin valve	▶ attention: these factors usually support a diagnosis of UC, but there are always exceptions!

Table 12.**2** Colonoscopy features for differential diagnosis of UC and CD (modified according to reference 8)

Feature	Ulcerative colitis	Crohn disease
Continuous inflammation	always*	extremely rarely
Patchy manifestation	no*	frequent
Rectal involvement	almost always	often spared
Vascular pattern	distorted or lost	often normal
Diffuse bleeding	widespread	rare
Vulnerability	widespread	uncommon
Spontaneous petechiae	widespread	rare
Granularity	widespread	less widespread
Erythema	typical	less typical
Edema	present	present
Cobblestoning	no	typical
Aphthous erosions	no	typical
Surface ulcerations	occasionally	frequent
Large ulcers > 1 cm	in severe cases	common
Long, deep ulcers	rare	common
Linear ulcers	rare	common
Serpiginous ulcers	rare	common
Pseudopolyps	not infrequent	occasionally
Mucosal bridging	occasionally	occasionally
Mucosa surrounding ulcer	abnormal	normal
Sharp demarcation (circular) to normal tisse	yes	no
Stricture	no	common, frequent
▶ This difference is vital for the patient: stricture related to UC may be malignant!		

* For exceptions, see sections concerning UC.

Biopsy techniques
Biopsy samples are generally taken with colonoscopy forceps with and without a needle, so that mucosal layers and parts of the muscularis mucosae can be sampled. The submucosa normally cannot be sampled.
It is advisable to take at least two to four excisional biopsies from various bowel segments in order to increase the likelihood of differential diagnosis. Separate excision, description, and investigation of individual localizations are worthwhile, especially for differentiating between chronic and acute inflammatory bowel diseases. Granulomas characteristic of CD can be found in 30–40 % of patients. Other criteria for CD include discontinuity of the inflammatory infiltrates and transmural inflammation. A diagnosis of UC is supported by multiple crypt abscesses and uniform contiguous inflammatory processes.

Surveillance strategies and endoscopic surveillance (with biopsies) for ulcerative colitis (DGVS guidelines, 2001)
For ulcerative pancolitis (< 8 years) or left-sided colitis (< 15 years):
- annual total colonoscopy with multiple biopsies (2–4 biopsies every 10–12 cm = 40–50 biopsies = (recommendation grade/level of evidence B II-2),
- discuss with the patient proctocolectomy as an option,
- more intense surveillance if PSC (recommendation grade C).

For Crohn disease, there are no recommendations for systematic endoscopic surveillance.

Other Differential Diagnoses

In the absence of characteristic appearances, other differential diagnoses, as described in the following, should be considered. The key to definitive diagnosis is usually clinical course of disease over time and patient medical history. Blood and bacteriological evaluation play a smaller role. Colonoscopic appearances may be the deciding factor in unclear cases. Figure 12.**31** provides a schematic illustration of the varying extent of colonic involvement in different types of colitis. ▣12.**10** shows endoscopic features for differential diagnosis of ileitis.

Indeterminate Colitis

A certain percentage of patients (5–10 %) can be successfully diagnosed as chronic IBD, but, despite chronicity, it remains impossible to determine IBD type either clinically or endoscopically. These are generally cases of pancolitis, whereby the biggest difficulties in differential diagnosis occur with intermittent course.

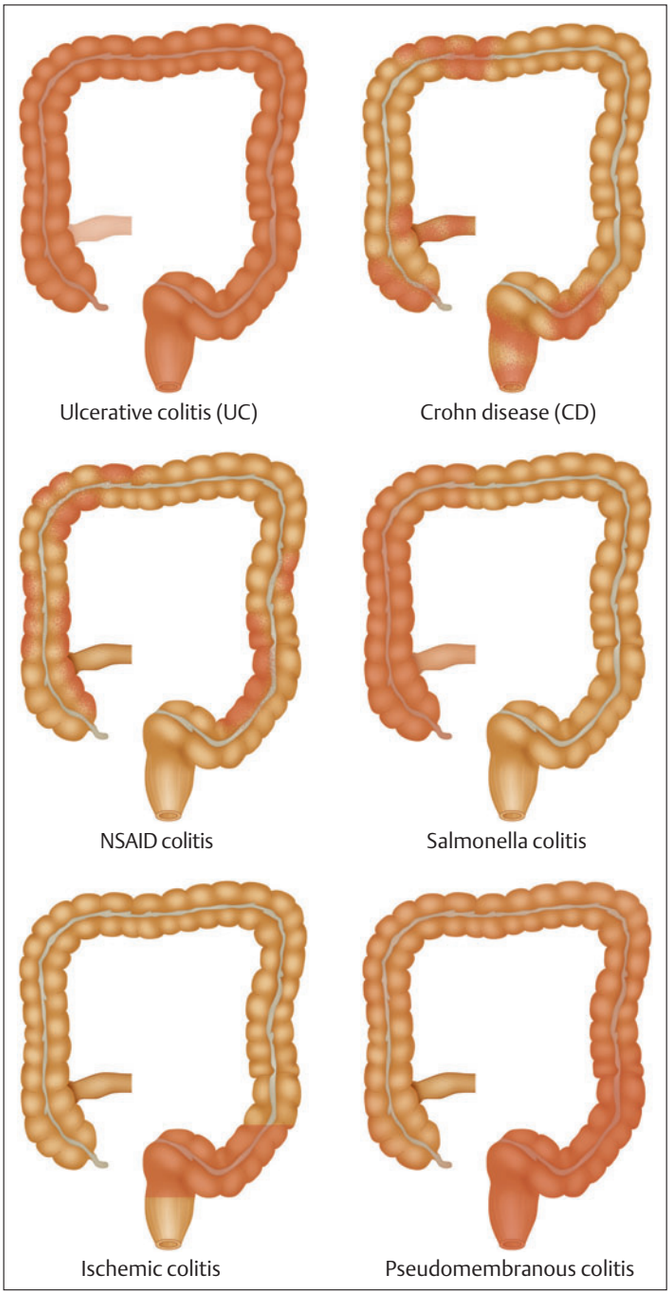
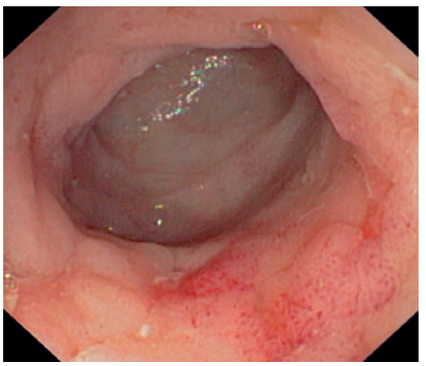


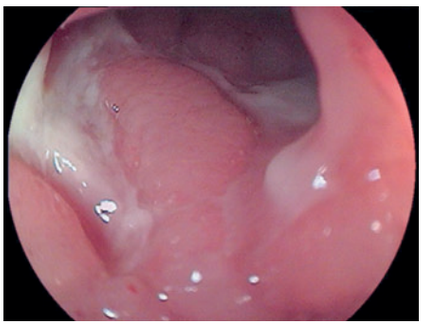
Fig. 12.**31** **Typical extent of colonic involvement in various types of colitis.**

Inflammation tends to be continuous and morphology is similar to UC with evenly distributed ulcerations. The frequent occurrence of pseudopolyps (Fig. 12.**32**) makes differentiation even more difficult. As shown in this example, lesions may on the one hand correspond to UC (Fig. 12.**33 a**) but on the other to CD (Fig. 12.**33 b–d**). The majority of these cases develop over the years into Crohn pancolitis, without necessarily involving the small intestine. More recent serological investigations can assist in classification. The distinction is important: in Crohn disease, the attachment of an ileoanal pouch is avoided.

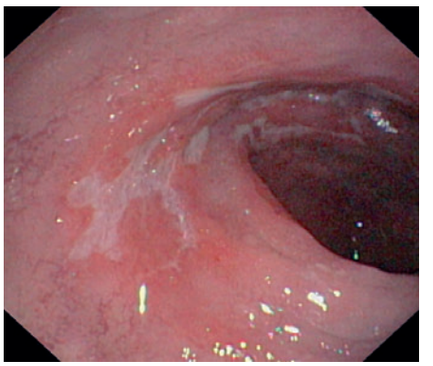
12.10 Differential diagnosis: ileitis



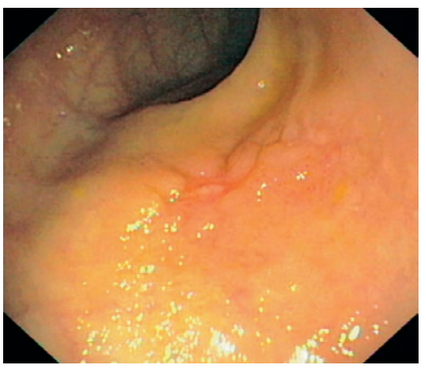
a Crohn disease: terminal ileitis.



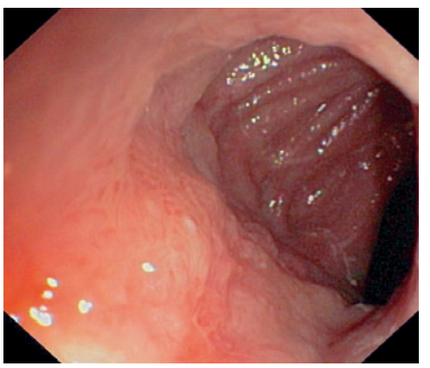
b Crohn disease: terminal ileitis.



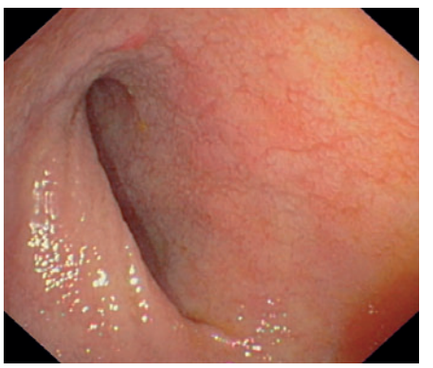
c Ileitis caused by 5-FU chemotherapy.



d Terminal ileitis related to yersiniosis.



e Backwash ileitis in ulcerative colitis.



f Backwash ileitis in ulcerative colitis.

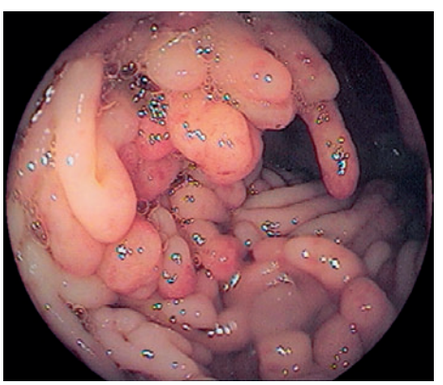


Fig. 12.**32** **Pronounced pseudopolyps.** These appear in UC but can also appear in CD.

Endoscopic appearances are varied. The proximal right side of the colon is usually affected, but disseminated disease also can occur. In the acute phase, there are often patchy erythemas, sometimes partly brown–red in color, intramucosal bleeding, and mucosal edema (Fig. 12.**34**). Vascular pattern usually remains intact or at least partially visible and erosions (including aphthous erosions) may be present. Ulcers are usually small and deep ulcers are rare. Thus, the mucosal "architecture" may seem to appear intact. Difficulties arise in that some longer term infectious colitis types can mimic IBD morphologically (Crohn disease, in particular). This is particularly true of *Campylobacter jejuni* colitis and amebiasis. Suspicious cases must therefore be serologically investigated. Diagnosis can also be difficult when there is a bacterial superinfection related to IBD (Fig. 12.**35**).

Infectious Colitis

These are forms of self-limiting colitis, caused by acute microbial triggers that generally manifest with acute diarrhea. Deciding clinical factors are thus acute onset of symptoms, short time period (less than one week), sometimes rapid onset of fever, travel abroad, or local outbreak of diarrhea. Regarding diagnosis, it is important to remember that stool cultures are often negative in patients with infectious colitis. Chronic changes can be expected after six weeks. Thus, if symptoms persist and there are signs of the condition becoming chronic, surveillance colonoscopy after three months is recommended.

Histopathological analysis can help distinguish between acute infectious enterocolitis vs. early stages of a chronic inflammatory bowel disease. A very high degree of neutrophilic infiltration and severe edema tend to indicate infectious colitis. Severe crypt distortion is more indicative of UC, while discontinuous, focal cryptitis supports a diagnosis of CD. Granulomas may be absent with CD, but, alternatively, they may be found in tuberculosis and yersiniosis.

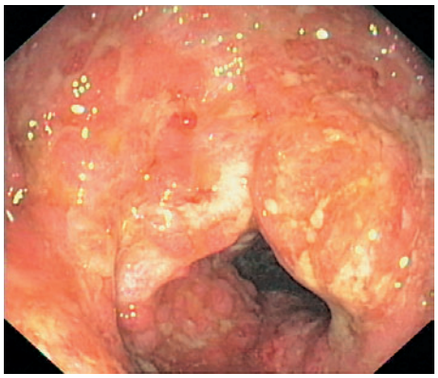
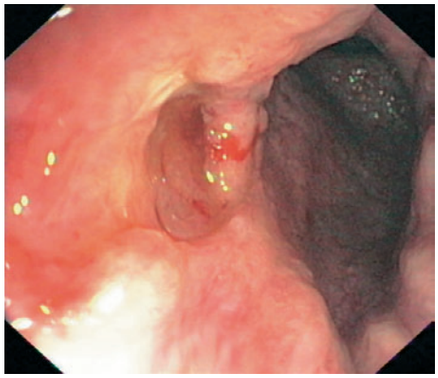
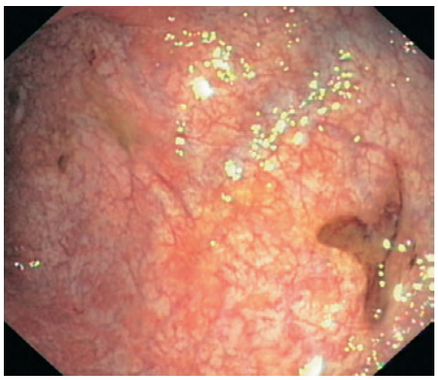
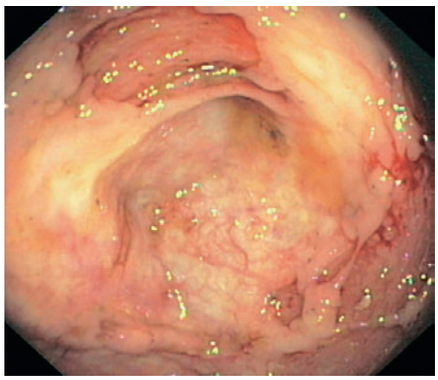





Fig. 12.33 **Differential diagnosis of UC–CD**
a High-grade fibrinous–ulcerous inflammation with granularity; this segment of the transverse colon rather supports a diagnosis of UC.
b Ulcer pocket in anal canal, which does not support diagnosis of UC, rather CD.
c Distorted vascular pattern and patchy erythema in rectum, but no fibrin or ulcers; also does not support diagnosis of UC (NB: no local therapy in past eight months).
d In cecum, partially normal vascular pattern, bizarre, recent ulcers that better support diagnosis of CD.

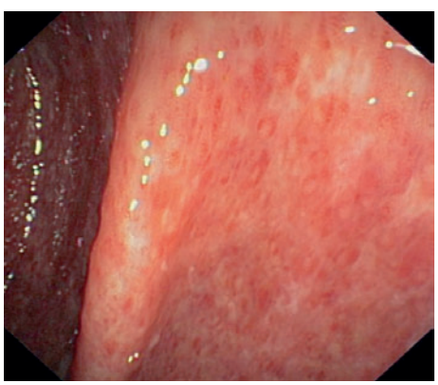
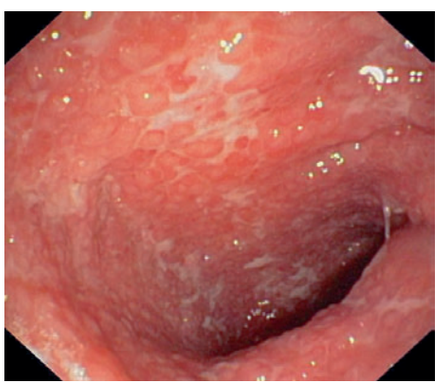
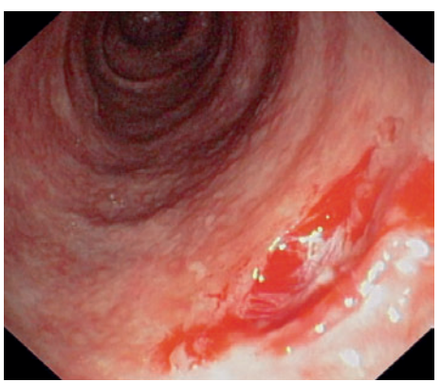
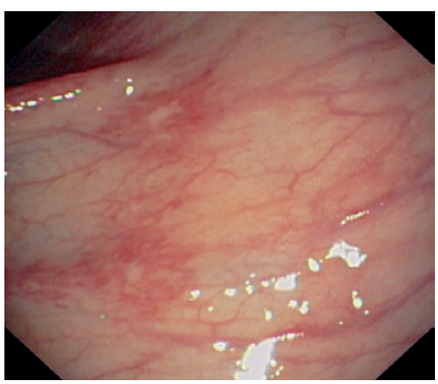





Fig. 12.34 **Infectious colitis, unidentified pathogen.**
a Erythema, erosions, quite continuous in right colon, clearly decreasing to left (ascending colon).
b Very tiny ulcers (ascending colon).
c Mucosal edema became more visible at biopsy.
d Only a few small manifestations in left colon.

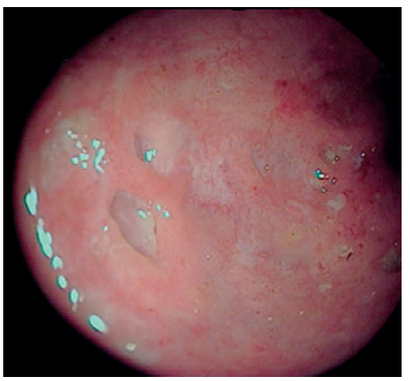


Fig. 12.**35** **Crohn disease, current superinfection with *Chlamydia trachomatis.*** This organism could be responsible for the small pitlike ulcerations in the ascending colon, which the patient had in addition to his Crohn-related lesions; no longer detectable six weeks later at surveillance.

Campylobacter jejuni colitis. Normal endoscopic appearances. Severe cases present variously, with patchy erythema, edema, and scattered ulcerations (▣ 12.**11**). Distal attack occurs more frequently and is more likely to mimic UC.

Yersinia enterocolitica enterocolitis. Small, punched-out mini-ulcers can be visualized endoscopically. Attack is usually segmental and tends to occur on the right side. The terminal ileum and cecal region are often affected. Greater similarity to CD due to localization and morphology.

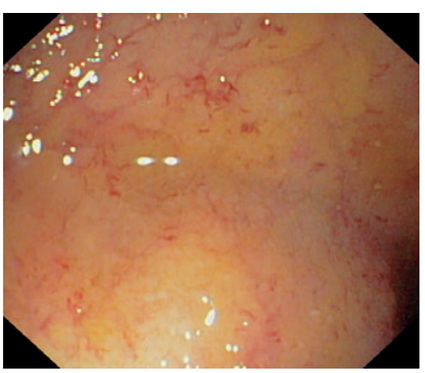
Salmonella enterocolitis. The attack is primarily right-sided and varies greatly in terms of extent and severity (▣ 12.**12**). However, symptoms usually abate quickly.

Amebiasis. Manifests mainly in cecum and rectosigmoid. In chronic courses, ulcerations may resemble those in CD. Protozoans can be found in biopsy samples.

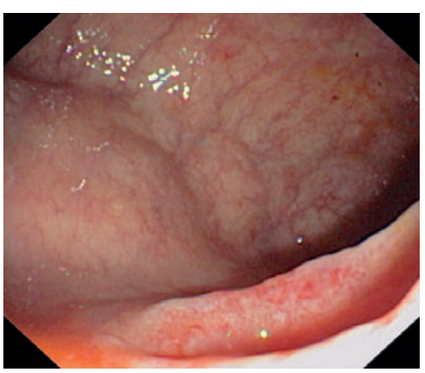
Cytomegalovirus colitis. Patchy areas, but also contiguous reddening and wide ulcerations may create an appearance very similar to UC.

Rotavirus-induced colitis. Morphological progress usually does not include significant colitis. However, pronounced erythema of the colonic mucosa also occurs, though without larger ulcerations (Fig. 12.**36**).

▣ **12.11 Infectious colitis: *Campylobacter jejuni* colitis**



a



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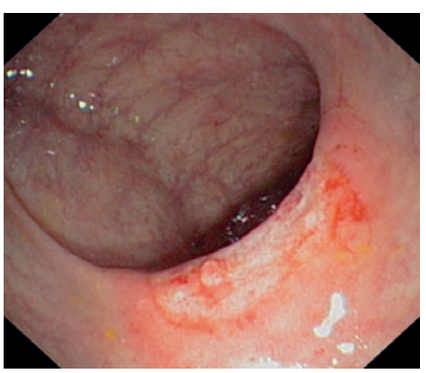
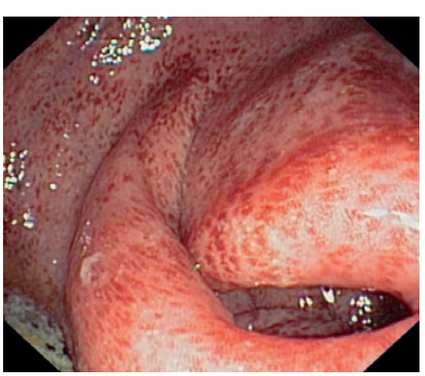


c

a–c Terminal ileum and ileocecal valve involvement. **a** Vascular changes and mild edema in terminal ileum. **b** Circular ulceration on the ileocecal valve toward small intestine **c** Ulceration spreading in half-moon shape on the outer side of the ileocecal valve. The infection was already present for almost three months and thus had a tendency toward chronicity.



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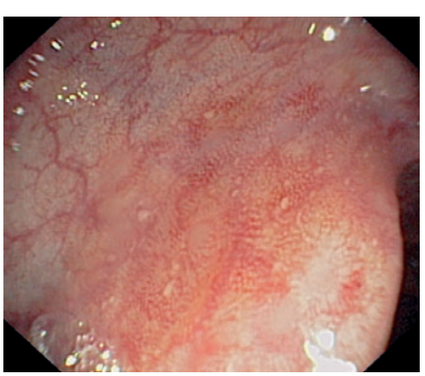
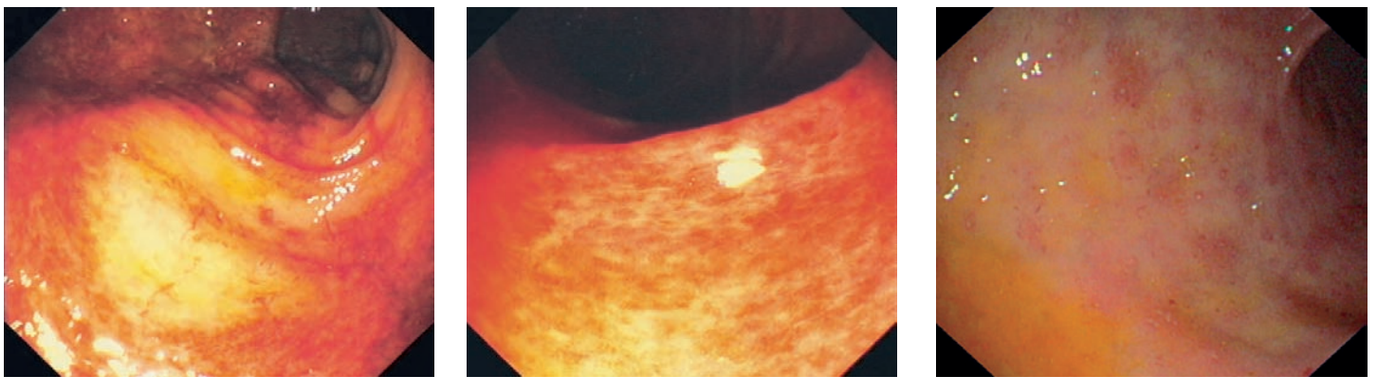


e

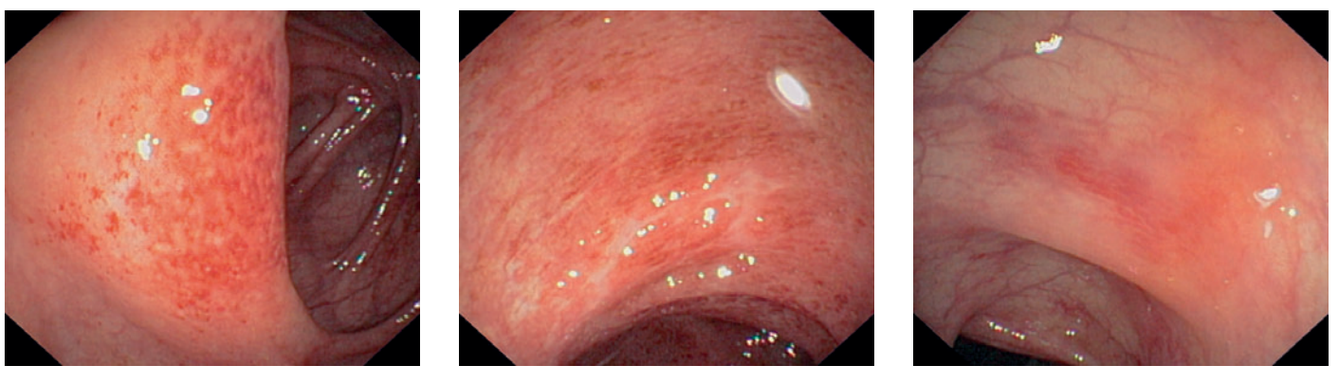
d, e Characteristic appearance of infectious colitis. **d** Pronounced patches of erythema and mucosal edema. **e** Aphthous changes at another location, here with patchy reddening of the surrounding area, differentiating this finding from CD.



a b c

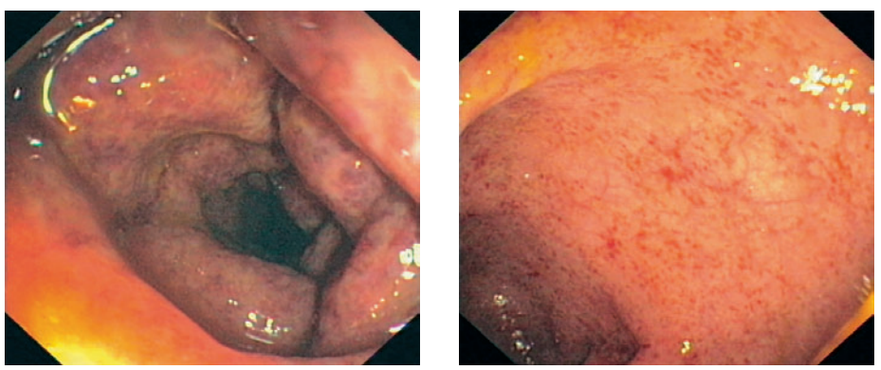
Fig. 12.**36** **Rotavirus-induced infection.**
a Hemorrhagic colitis, primarily right sided, with pronounced vessel fragility. Rare appearances as most rotavirus-induced infections do not have an endoscopic Korrelat (ascending colon).
b Pronounced patches of erythema (transverse colon).
c Numerous tiny aphthae in descending colon.

12.12 Infectious colitis: salmonella colitis



a b c

a–c *Salmonella enteritidis*. **a** Right-sided colitis beginning at ileocecal valve. **b** Circumscribed erosion in ascending colon, relatively mild case. **c** Lightly erythematous patches in transverse colon.



d e

d, e *Salmonella typhimurium*. **d** Attack primarily in right colon with severe edema and patchy hemorrhaging, but no erosions. **e** Sigmoid colon involved to a lesser extent.

Radiation Colitis

Radiation colitis traditionally occurs after brachytherapy for cervical carcinoma. However, in recent years, there has been a significant rise in frequency following adjuvant or neoadjuvant therapies of rectal carcinomas, affecting remaining or anastomosed intestinal segments. In isolated cases, it can also occur as proctitis after percutaneous radiation therapy of prostate carcinoma. Unlike acute radiation necrosis (Fig. 12.**37**), symptoms typically occur after the disease has been latent for a number of years. Symptoms include peranal bleeding and, later, diarrhea and possibly even symptoms of obstruction (sigmoid colon attack). Sometimes components of diversion colitis also occur, if the patient has a temporary surgical diversion of the fecal stream.

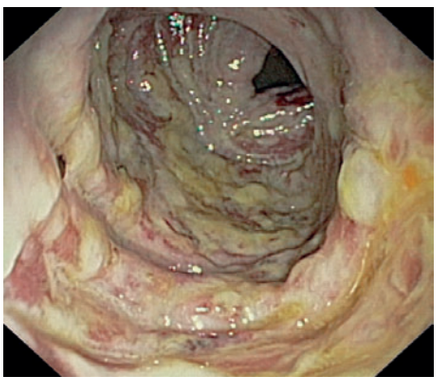
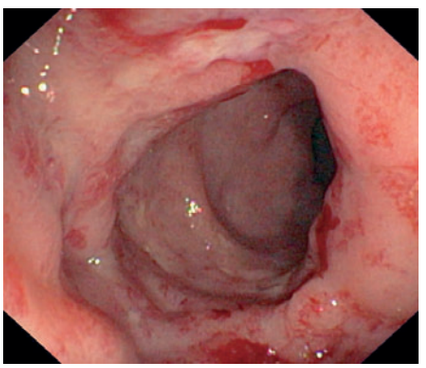


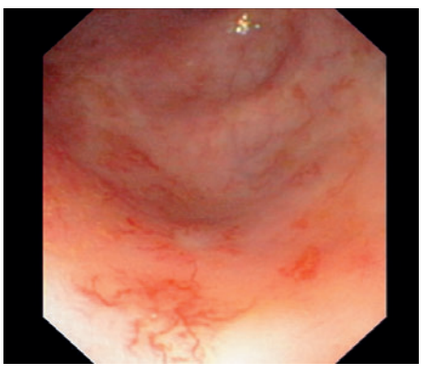
Fig. 12.**37** **Acute radiation necrosis of the mucosa** (in this case in an ileoanal pouch because of ileoanal carcinoma): complete, whitish necrosis.

Endoscopy enables early diagnosis, surveillance of progression, and provides a therapeutic option for bleeding and stenosis. Endoscopically, the disease is characterized by early appearance of edematous mucosa with erythema (▣ 12.**13 a**), and, later, distortion of normal vascular pattern and formation of characteristic neovessels, sometimes like spider veins in appearance (▣ 12.**13 b**, **c**). These atypical vessels are also expanded like telangiectasia and are potential (and sometimes current) sources of bleeding. Bleeding can be controlled with careful (!) application of low coagulation current using an argon beam coagulator (max. 45 W). However, a long healing process and ulcerations may ensue (▣ 12.**13 d**) in the fibrosed bowel wall, and these are difficult to treat with medication.

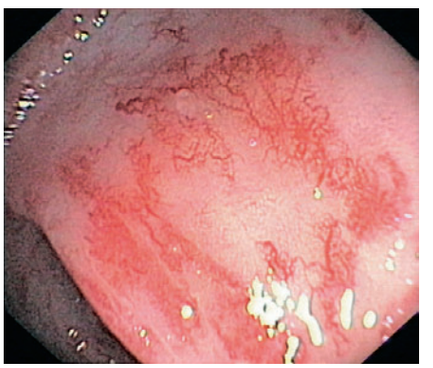
▣ 12.13 Radiation colitis



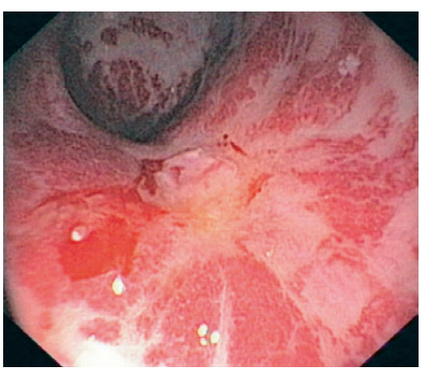
a Clear chronic reaction to radiation therapy, telangiectasia, small ulcer, and bleeding tendency (upper rectum).



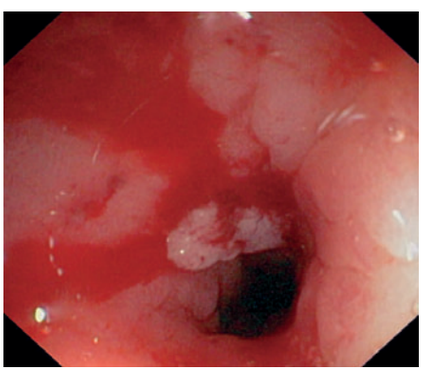
b Moderate delayed reaction of mucosa with spidery angiectasia (lower rectum).



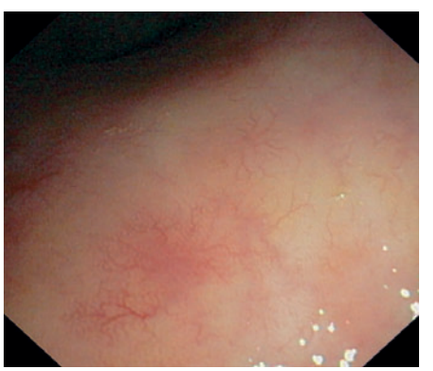
c Pronounced radiation colitis following cervical carcinoma; typical vessel garlands.



d Pronounced radiation colitis: significant vascular changes in lower rectum and chronic radiation ulcer.



e Mucosal edema, chronic inflammation, atrophy, and increased vulnerability during unsuccessful attempted passage, prior cervical carcinoma (rectosigmoid junction).



f Mild vascular changes in lower rectum (following prostate carcinoma).

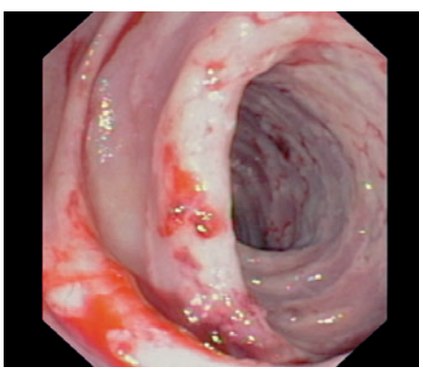
Ischemic Colitis

Ischemic colitis usually affects older patients. However, it can also be found in younger patients, where it is often only pronounced in a small segment of bowel. In the latter case, vasculitis must also be considered as a possible diagnosis. Ischemic enterocolitis is a manifestation of antiphospholipid antibody syndrome, generally affecting only the small intestine. Among older patients, absolute arrhythmia, prior embolism, and other manifestations of arteriosclerotic vascular diseases may be signs of ischemia. Embolic genesis or—more frequently with colitis—nonocclusive mesenteric ischemia (NOMI) should be considered. Ischemic colitis typically proliferates around the splenic flexure—which is a transitional zone between the inferior mesenteric artery and the superior mesenteric artery—potentially extending all the way to the rectosigmoid junction. The rectum is spared.

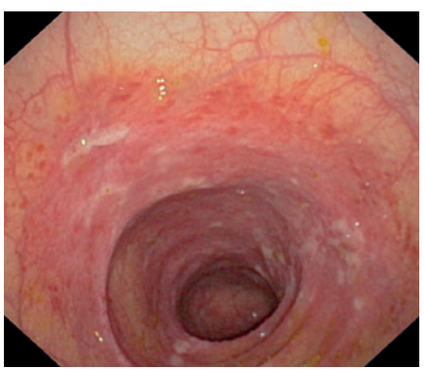
Endoscopy has replaced earlier radiological imaging techniques. Initial examination may occur based on the patient's medical history—sudden onset of peranal bleeding, mostly fresh and seldom older blood without significant pain—sonography with detection of isolated thickening of the intestinal wall, especially on the left side. This should be quickly followed by colonoscopy.

To the extent that early colitis is detected, endoscopic features include a very pale mucosa and intestinal wall (▣ 12.**14 a**). This is rapidly followed by mucosal edema and then very quickly a brown–red or even quite livid coloration and swelling of the mucosa appears, followed by widespread mucosal necrosis (▣ 12.**14 c–g**), which at first can be semicircular (▣ 12.**14 g**). The mixture of edema, livid coloration, and fibrinoid necrosis only lasts a few days before quickly regressing, possibly giving rise to small ulcerations and scarring (▣ 12.**14 f–i**). Atypical localization can also occur, mostly in the ascending colon near the ileocecal valve (Fig. 12.**38**). Occasionally, ischemic colitis presents as an atypical ulcer (Fig. 12.**39**). A rare differential diagnosis that may be considered is also segmental organ attack by a non-Hodgkin lymphoma (Fig. 12.**40**).

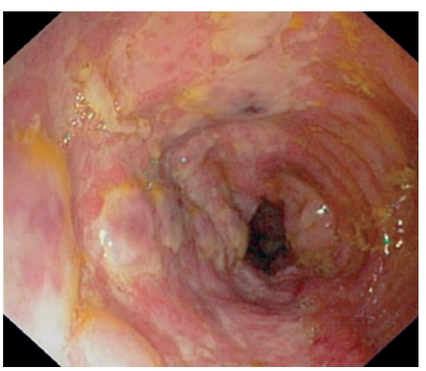
▣ **12.14 Clinical course of ischemic colitis**



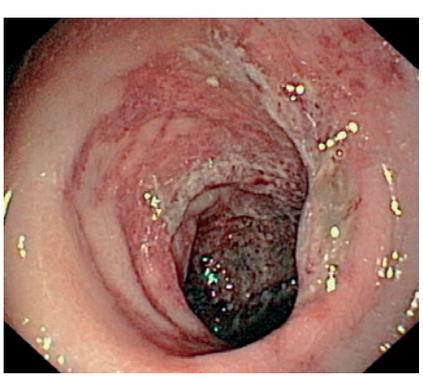
a Early stage: ischemia: very pale mucosa (neoterminal ileum).



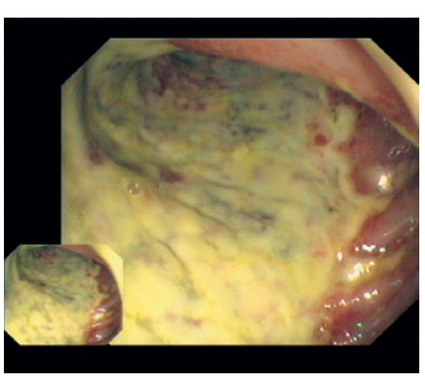
b Widespread segmental involvement: adjacent to regular bowel segments (sigmoid colon).



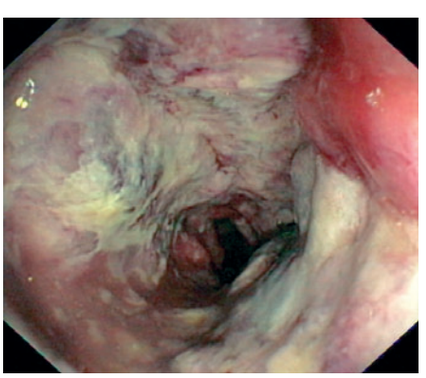
c Widespread mucosal necrosis, almost two-thirds of circumference (descending colon).



d Clear demarcation to normal mucosa of lower sigmoid colon.

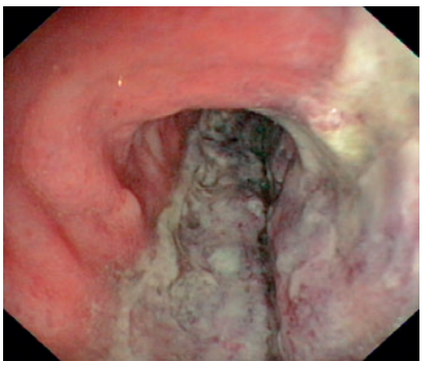


e Mucosal necrosis can develop into complete fibrin coating.

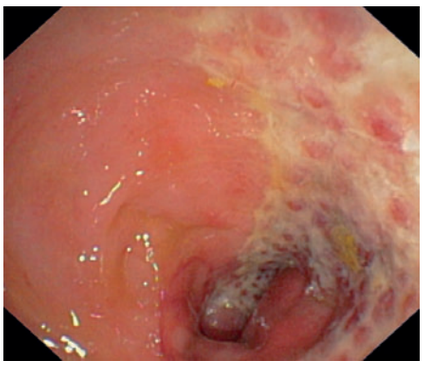


f–i Progression to healing. **f** Severe attack, almost circular, widespread mucosal edema, and partial mucosal necrosis with typical livid appearance: Second stage (descending colon).

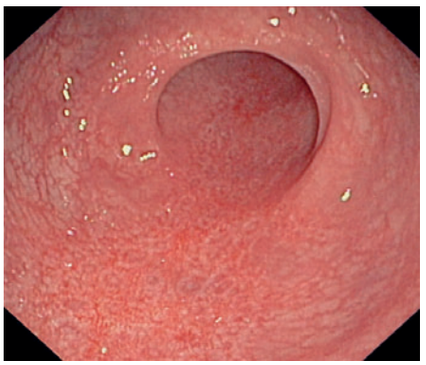
12.14 cont.



g Semicircular necrosis at another location in the descending colon.



h Signs of healing after eight days (patient already asymptomatic).



i Almost completely healed after two months, small bowl-shaped mucosal scars remain.

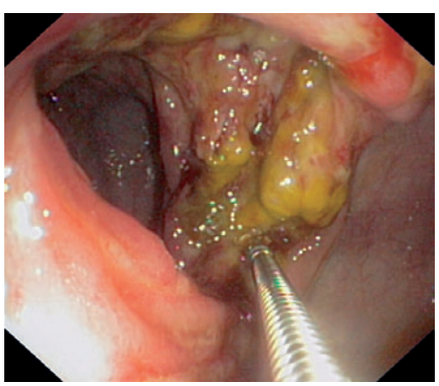


Fig. 12.**38** **Ischemic colitis.** Circumscribed segmental attack in ascending colon (!) just above ileocecal valve (confirmed operatively: did not invade beyond wall).

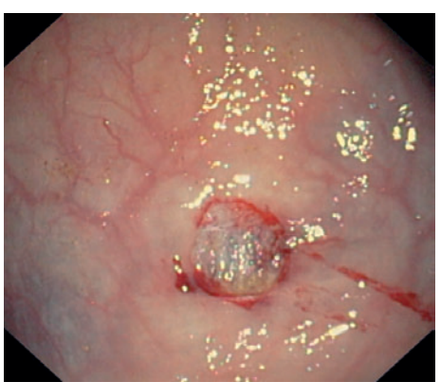


Fig. 12.**39** **Atypical ulcer on base of cecum without wider endoluminal reaction in the surrounding area.** At the base of the ulcer, visible fibers of the lamina propria of the cecal wall. A perforation later developed at this location after colonoscopy. Intraoperative and pathological–anatomical widespread ischemia with hemorrhaging of outer colon wall.

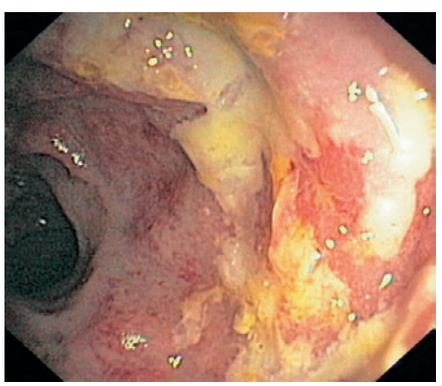


Fig. 12.**40** **Differential diagnosis (ischemia vs. non-Hodgkin lymphoma):** similar to ischemia, maplike appearance with surface ulcerations, but also with nodular elements: segmental organ involvement in sigmoid colon of non-Hodgkin lymphoma (low-grade, follicular type).

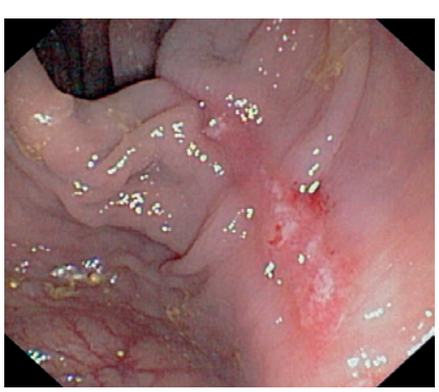


Fig. 12.**41** **Erosive changes** in ascending colon in NSAID colitis.

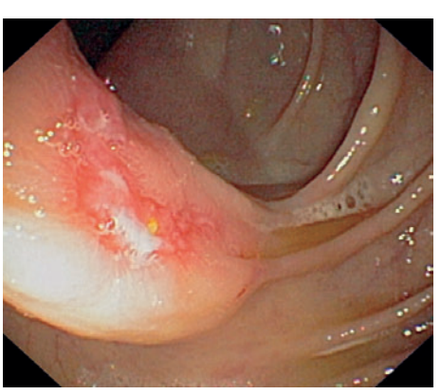


Fig. 12.**42** **Solitary ulcer** in ascending colon: NSAID colitis.

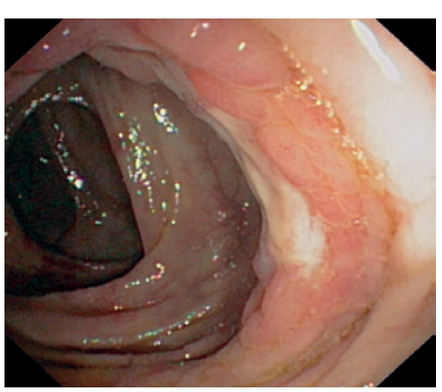


Fig. 12.**43** **Ulceration on the ileocecal valve** (= typical predilection location) in NSAID colitis, surrounding area unaffected.

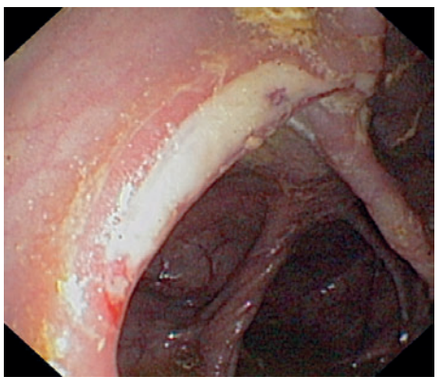
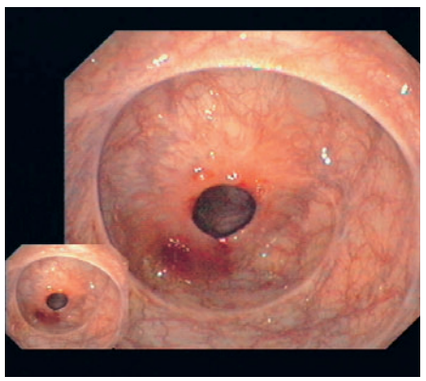


Fig. 12.**44** **Ulcer on ileocecal valve in NSAID colitis.** Ulcer is characteristically flat, without well-delineated margin or reaction at margin.



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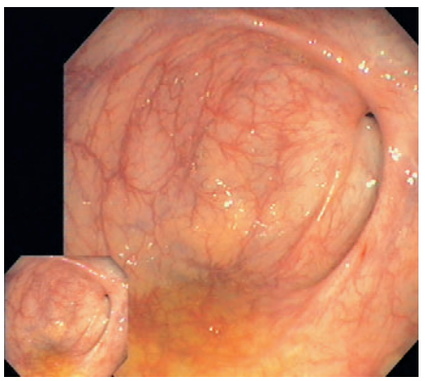


b

Fig. 12.**45** **Atrophy due to NSAID colitis.**
a Residual condition after prior chronic NSAID colitis (right transverse colon), bizarre spiral-shaped scarring with segmental stenoses.
b Atrophy processes could lead to "buttonhole" stenoses (hepatic flexure).

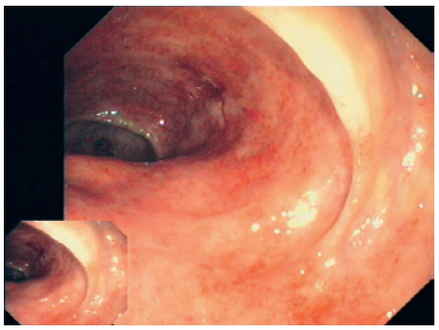


Fig. 12.**47** **Diversion colitis:** moderate reddening and loss of normal vessel pattern, mild edema (ascending colon).

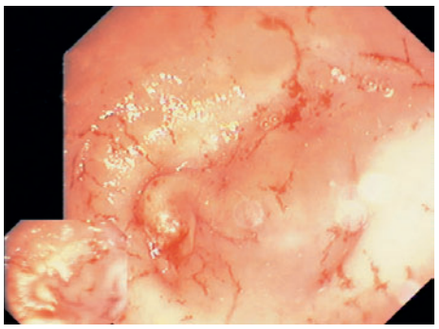


Fig. 12.**48** **Diversion colitis:** atypical vessels (ascending colon).

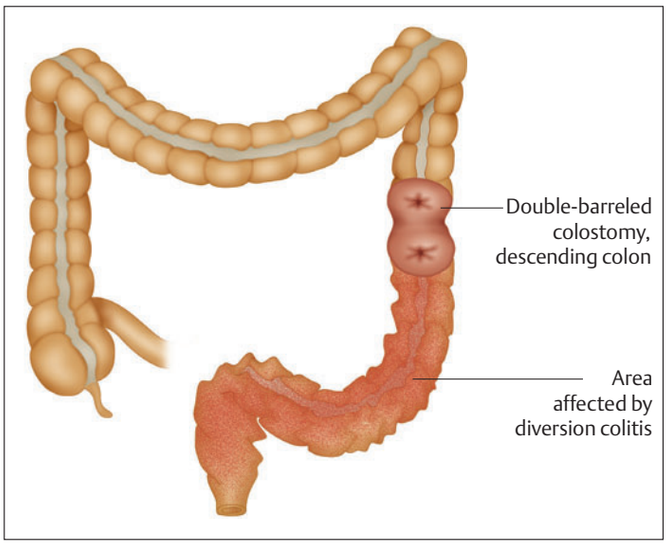


Fig. 12.**46** **Localization of diversion colitis.**

NSAID Colitis

Like ischemic colitis, the incidence of NSAID colitis is on the rise. The increase is attributed to a significant rise in long-term use of nonsteroidal anti-inflammatory drugs and salicylates. Consideration of NSAID colitis as a possible diagnosis is a vital when diagnosing IBD, especially among older individuals.

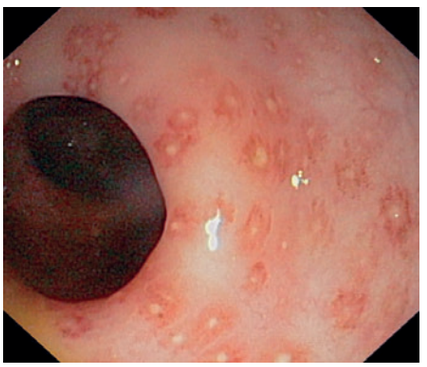
NSAID colitis typically affects the right side. Lesions can range from aphthous erosions to broad-based ulcerations, without deep infiltration of the wall (Figs. 12.**41**, 12.**42**). The area around the ileocecal valve is quite often affected, in many cases by a broad, solitary ulcer (Figs. 12.**43**, 12.**44**). The surrounding area often appears normal and the ulcer margins do not show any significant reaction. However, there can be inflammation in areas that appear virtually unaffected macroscopically. Multiple biopsies from the entire colon are therefore advisable, since right-sided proliferation can be further confirmed histologically. Pronounced scarring may be present (Fig. 12.**45**). Today, NSAID colitis is the most important alternative diagnosis (differential diagnosis) to Crohn disease, especially among older patients.

Diversion Colitis

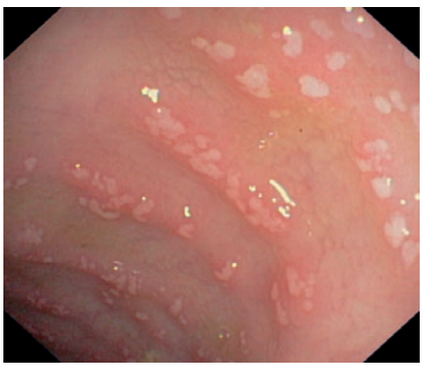
Diversion colitis occurs in so-called shut down large intestinal segments (Fig. 12.**46**). The pathogenesis is not clear. Diversion colitis can be totally asymptomatic, only discovered when, for example, the colon is inspected before restoration following temporary diversion of the fecal stream. Recurrent bleeding can also occur.

The appearance can be similar to UC, with areas of lymphoid hyperplastic tissue (Figs. 12.**47**, 12.**48**). Only very rarely does stenosis appear in diversion colitis. Its presence usually prevents endoscopic therapy.

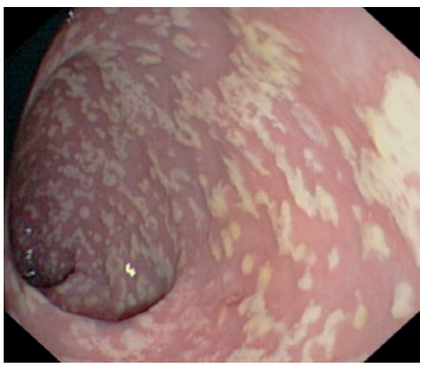
12.15 Pseudomembranous colitis



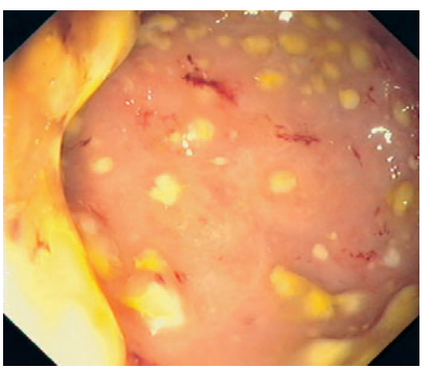
a Initial plaque formation, shown here with reddish "halo," evidence of underlying inflammation.



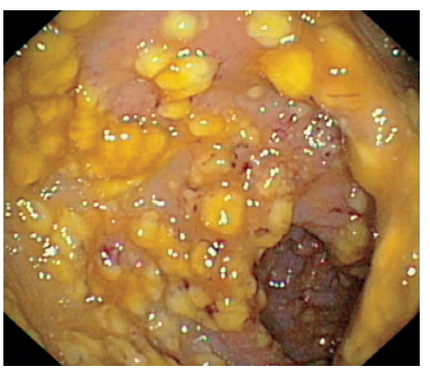
b Tiny plaques.



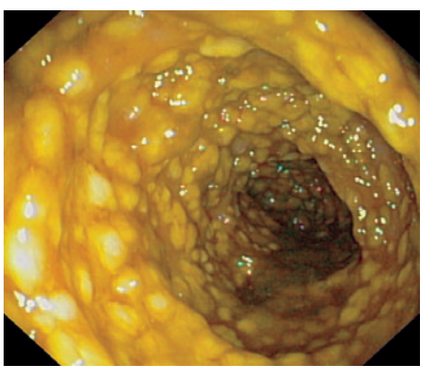
c Smaller plaques, but already confluent.



d Yellowish plaques, medium grade (sigmoid colon).



e Massive confluent plaque formation, chronic course.



f Massive confluent pseudomembranes that were so thick that stenosis was suspected in the sigmoid at radiological examination.

Pseudomembranous Colitis

Pseudomembranous colitis is caused by an overgrowth of *Clostridium difficile*. Diagnosis can be made based on detecting *Clostridium difficile*. However, severity and extent of attack are better evaluated using endoscopy.

Pseudomembranes are yellowish, elevated plaques on an inflamed mucosa. Surrounding mucosa can be normal. In severe stages, inflammation can be confluent (▣ 12.**15**). Ulcerations are not often found. Attack usually involves primarily the rectosigmoid and often examination of the rectosigmoid is sufficient. However, there are also cases that first manifest themselves in the descending colon and then progress proximally. Thus, if there is clinical suspicion and a negative sigmoidoscopy, total colonoscopy should follow.

Other Rare Forms of Colitis

Gold colitis. Gold colitis is an extremely rare occurrence these days due to decreased used of gold salts in the treatment of rheumatoid arthritis.

Behçet colitis. Behçet colitis is characterized by pitlike ulcerations that are numerous and occur frequently in the ileocecal region. Diagnostic confirmation must ultimately be provided by other manifestations (Middle Eastern heritage, oral–genital ulcers, uveitis).

Nonclassifiable Types of Colitis

These colitis types involve acute symptoms (diarrhea, bleeding) accompanied by inflammation of the intestinal mucosa. In terms of appearance and distribution in the bowel, inflammation does not correspond to any of the colitis types discussed in earlier sections. Distribution is mostly diffuse or segmental without predilection. The decision as to whether an attack is an initial manifestation of chronic IBD or an acute reaction to mi-

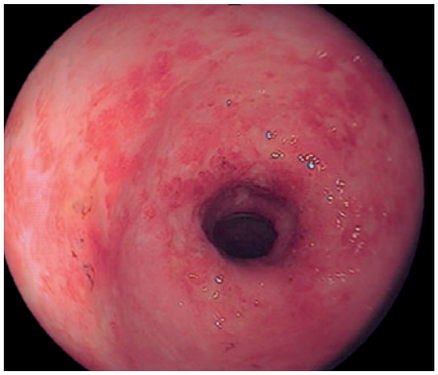


Fig. 12.**49** **Erythema, small ulcers, and intramucosal bleeding tendency** resemble UC; this could not be confirmed during the course of attack. No indication of infectious agents (ascending colon).

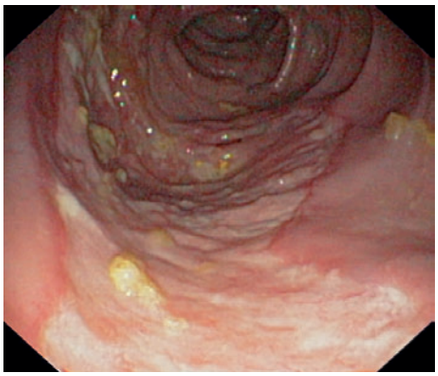


Fig. 12.**50** **Very wide ulcerations distributed over large segments of the colon** in nonreactive surrounding, heparin-related bleeding. No risk factors other than chronic steroid use could be confirmed. Finding unchanged over months of surveillance (descending colon).

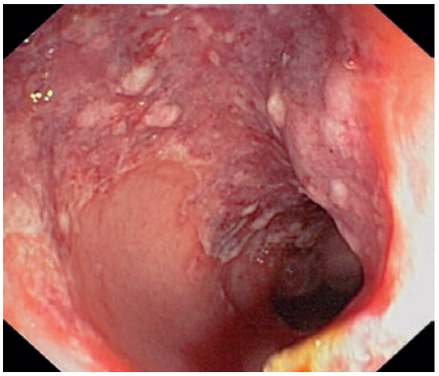


Fig. 12.**51** **Quite acute segmental colitis,** resembling ischemia in terms of morphology but not in terms of extent of colonic involvement, all the way to the upper rectum. Segmental, infectious genesis is leading DD.

crobial or chemical irritants is usually made based on disease course or possibly endoscopic findings. It is rare that such forms of colitis become chronic without being classifiable. If a pathogen cannot be detected, possibilities include cytomegaloviral infections, e.g., among transplant patients or other patients who are immunosuppressed. Ulcerations are usually nonreactive, surrounded by normal mucosa, and healing occurs slowly. Regarding differential diagnosis, possible misuse of medication, especially salicylates or nonsteroidal anti-inflammatory drugs must be considered. As with ulcers of the upper gastrointestinal tract, one must be aware that misuse will not always be acknowledged and cannot be proved in all cases. Figures 12.**49**–12.**51** show cases that as of yet could not be classified.

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13 Acute and Chronic Lower Gastrointestinal Bleeding

J. Barnert

Definitions

Lower gastrointestinal bleeding. Lower gastrointestinal bleeding is defined as acute or chronic blood loss from a source distal to the ligament of Treitz. The following sections will focus on bleeding from sources in the colon and anal canal.

Acute lower gastrointestinal bleeding. Acute lower gastrointestinal bleeding is defined (rather arbitrarily) as a bleeding situation in which blood loss has been occurring for less than three days and is causing hemodynamic instability, anemia, or the need for a blood transfusion.

Chronic lower gastrointestinal bleeding. The definition of chronic lower gastrointestinal bleeding is rather broad, encompassing longstanding or intermittent blood loss of smaller amounts of blood through rectum or melena, but also fecal occult blood loss (often not discovered until the patient seeks medical attention for anemia).

Epidemiology

Incidence of acute lower gastrointestinal bleeding is reported in the literature at 20–27 per 100 000 per year (20). A recent study from the Netherlands has reported lower figures (8.9 per 100 000 per year) (33). Acute lower gastrointestinal bleeding occurs less frequently than acute upper gastrointestinal bleeding (100–200 per 100 000 per year). The small intestine is the bleeding site in only 0.7–9% of patients with acute lower gastrointestinal bleeding (63).

Table 13.**1** Patient medical history for diagnosis of lower gastrointestinal bleeding

Current symptoms	▶ Abdominal pain ▶ Altered bowel habits ▶ Fever ▶ Urge to evacuate bowels, tenesmus ▶ Weight loss ▶ Fainting, syncope, dizziness
Medical history	▶ Endoscopic intervention (e.g., recent polypectomy) ▶ Trauma ▶ Ulcers ▶ Chronic inflammatory bowel disease (e.g., chronic IBD, polyposis) ▶ Radiation therapy ▶ Surgical interventions (e.g., vascular surgery) ▶ Concomitant diseases (heart, kidney, liver, vascular inflammation, rheumatic diseases) ▶ coagulation disorder ▶ gastrointestinal bleeding
History of drug therapy	▶ NSAID, acetylsalicylic acid ▶ Anticoagulants ▶ Calcium antagonists

Prognosis and Clinical Course

Acute lower gastrointestinal bleeding. Spontaneous cessation of acute lower gastrointestinal bleeding occurs in ca. 80% of patients (63). The mortality rate among patients hospitalized with acute lower gastrointestinal bleeding is 2.4%; if bleeding occurs during hospital stay, the rate increases dramatically to 23.1% (34). A more recent study on patients with acute lower gastrointestinal bleeding found six predictive factors which increase the likelihood of severe course or recurrence of bleeding: tachycardia above 100/min, systolic blood pressure below 115 mmHg, syncope, loss of tenderness to pressure on the abdomen, blood loss in the first four hours after hospitalization, and acetylsalicylic acid use (51).

Chronic lower gastrointestinal bleeding. The prognosis for patients with chronic lower gastrointestinal bleeding depends primarily on the bleeding source. Among older patients with iron-deficiency anemia, the most frequent cause of bleeding is a colon carcinoma. Further prognosis is determined by the carcinoma.

Diagnosis

Medical history. Patient medical history can supply valuable information for diagnosing lower gastrointestinal bleeding (Tab. 13.**1**). Stool color is very important: If the patient's vital signs are stable, loss of larger amounts of fresh blood (hematochezia) indicates with high probability that the bleeding site is the colon the while "tarry stool" (melena) indicates a site higher in the gastrointestinal tract.

Physical examination. Physical examination should focus first on the patient's vital signs, e.g., pulse and blood pressure. Marked tachycardia accompanied by a drop in blood pressure, tachypnea, and drowsiness is a sign of blood loss of more than 1500 mL. Acute lower gastrointestinal bleeding causes anemia or faintness/dizziness in more than half of all patients, though its course is less dramatic than upper gastrointestinal bleeding.

Aspiration of gastric contents and laboratory tests. Aspiration of stomach contents using a nasogastric tube has a high predictive value with regard to bleeding proximal to the ligament of Treitz, though it cannot exclude it with total certainty in the negative case. Hemoglobin count can provide information on the severity of bleeding. In actual practice, the creatine kinase (CK) urea ratio, which has been recommended for differentiating between upper and lower gastrointestinal bleeding, is not of great assistance for diagnosis in actual bleeding situations (39).

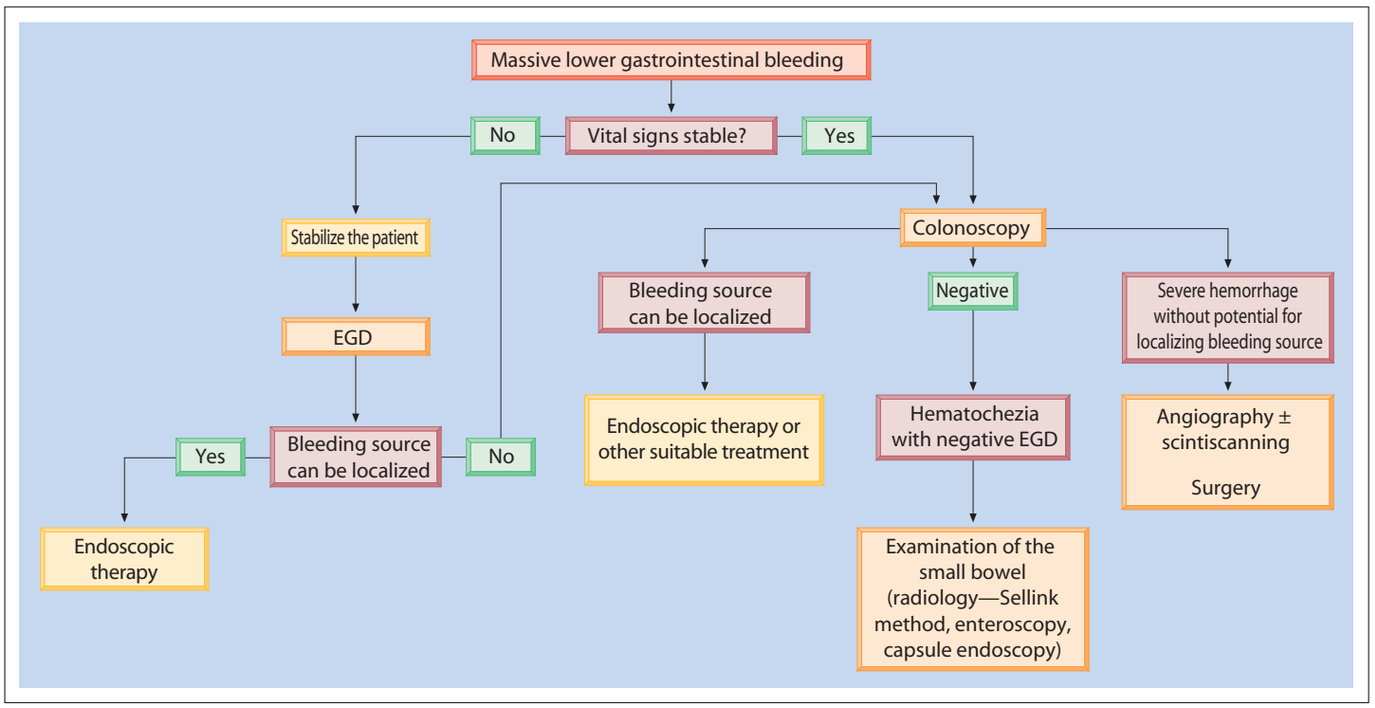


Fig. 13.**1** **Algorithm for diagnosis and therapy** in acute lower gastrointestinal bleeding with hematochezia.

Endoscopic Diagnosis

Chronic Lower Gastrointestinal Bleeding

Occult gastrointestinal bleeding. Occult gastrointestinal bleeding in patients over 50 years of age is most frequently caused by a neoplasia in the colon. Regardless of whether rectal examination and rectoscopy/proctoscopy play a role in initial diagnosis, total colonoscopy is essential for further diagnosis. This applies to patients who have unexplained iron-deficiency anemia as well as those with positive fecal occult blood tests. Results have clearly shown that colorectal carcinoma mortality can be reduced by consistent use of colonoscopy in patients with positive fecal occult blood tests. Compared with diagnosing adenomas or carcinomas, other diagnoses (e.g., chronic inflammatory bowel diseases, angiodysplasia, and damage from radiation therapy) play a lesser role. If no pathology is found during colonoscopy in a patient with occult gastrointestinal bleeding or anemia, endoscopy of the upper gastrointestinal tract is indicated. According to a study by Zuckerman and Benitez, the upper gastrointestinal tract more often causes occult blood loss than does the lower gastrointestinal tract (62). If the upper gastrointestinal tract (to the ligament of Treitz) also fails to yield any useful findings, the small intestine must be examined.

Intermittent melena. Intermittent melena ("tarry stool") is an indication for gastroesophageal duodenoscopy as it is highly likely that the lesion can be found in the area examined. If there are no findings, the procedure is the same as for occult gastrointestinal bleeding, i. e., small and large bowel are investigated.

Bleeding into the rectum. Chronic or intermittent loss of smaller amounts of fresh blood is typical for a bleeding source in the colon. In the majority of patients, the source is a lesion in the anal canal or a distal colon segment. Patient medical history is useful for diagnosing lesions in the anal canal. If the patient is over 50 years of age, total colonoscopy should be attempted regardless. Colonoscopy is not necessary in younger patients with a bleeding source in the anal canal (3).

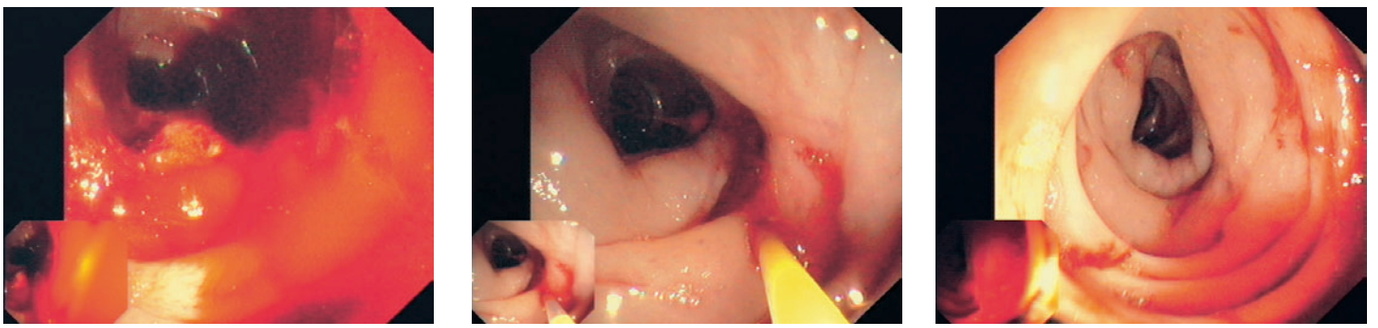
Acute Lower Gastrointestinal Bleeding

Severity of bleeding. Procedures for acute lower gastrointestinal bleeding depend on whether bleeding is moderate or heavy. Severity of bleeding is determined by amount of blood being lost and also by the stability of the patient's vital signs. If bleeding is heavy, stabilizing the patient and replacing lost blood have priority over diagnosis. Figure 13.**1** summarizes the diagnostic and therapeutic algorithm for acute lower gastrointestinal bleeding.

Excluding upper gastrointestinal bleeding. In up to 11 % of all patients with rectal bleeding in whom lower gastrointestinal bleeding is initially suspected, the bleeding source turns out to be located in the upper gastrointestinal tract (31, 38). Potential upper gastrointestinal bleeding sources must especially be considered among patients presenting with hematochezia and unstable vital signs.

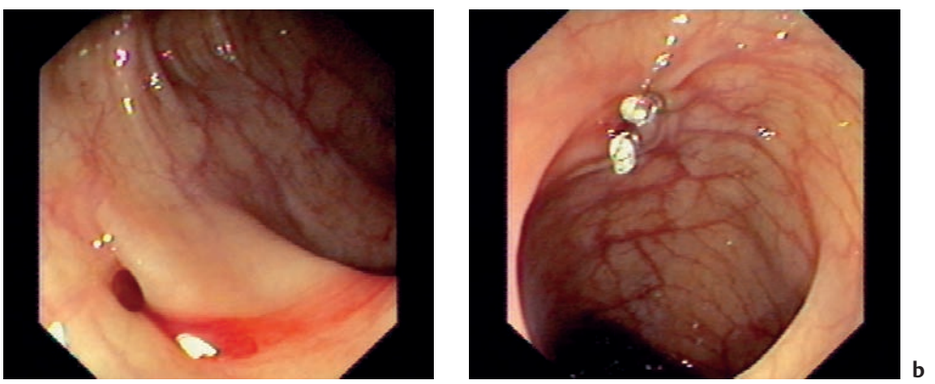
Urgent colonoscopy in acute lower gastrointestinal bleeding

There are two main ideas underlying urgent colonoscopy: first, the desire to quickly determine type and localization of bleeding source and, second, the potential for endoscopic intervention. A further, more recently recognized aspect is



a b c

Fig. 13.**2** **Bleeding diverticulum.**
a Acute diverticular bleeding.
b An epinephrine solution (1:10 000) is injected into the edge of the diverticulum (hidden by a fold).
c Cessation of bleeding after injection; edematous swelling of the mucosa where the epinephrine was injected.



a b

Fig. 13.**3** **Bleeding from a small diverticulum.**
a Bleeding from a small diverticulum, barely recognizable by a streak of red blood.
b Cessation of bleeding after application of two hemoclips (Olympus), closing the diverticular orifice.

the ability to identify patients with longstanding bleeding or with a risk of rebleeding—similar to patients with bleeding ulcers. Jensen et al. (30) and Grisolano et al. (24) have shown that, similar to acute upper gastrointestinal bleeding, evidence of active bleeding (Figs. 13.**2**–13.**4**), visible vessels (Figs. 13.**5**–13.**8**), and adherent clots (Figs. 13.**9**–13.**11**) are associated with severe course or high rate of rebleeding.

According to the literature, colonoscopy is usually performed within 12–24 hours after hospitalization (63). However, it has also been shown that earlier colonoscopy, i. e., within 12 hours after hospitalization, produces a significantly higher number of definitive diagnoses than colonoscopies performed after a longer period of time (52). The number of endoscopic interventions was also higher among earlier colonoscopies according to this (retrospective) study.

Another concern in urgent colonoscopy is patient preparation. Chaudhry et al. (13) showed that in patients with acute lower gastrointestinal bleeding a high diagnostic yield (97 %) was achieved even without bowel preparation and that hemostasis could also be effectively achieved. The authors were able to control active bleeding in 17 of 27 patients (63 %) using endoscopic intervention. The results are on the whole—especially concerning diagnostic yield in urgent colonoscopy—similar with and without bowel preparation (overview provided in 63). One argument for performing urgent colonoscopy without bowel cleansing is that the localization (height) of blood found in the colon can provide information about the bleeding site—though it should be noted that colonic motility could move digestive contents forward or backward. In general, current recommendations (3) rather advise cleansing the colon as thoroughly as possible, even in situations of acute lower gastrointestinal bleeding. This improves evaluation of the mucosa, which in turn enhances recognition of smaller lesions and minimizes the risk of complications resulting from poor visualization.

Bowel cleansing is usually performed with an electrolyte solution (polyethylene glycol basis). For optimal preparation of the colon, the patient must consume 3–4 L of solution. The solution should be drunk quickly, i. e., within a few hours so that colonoscopy can be performed as soon as possible. Patients generally tolerate consumption of 1–2 L of this salty tasting solution per hour. It may be helpful to administer a prokinetic antiemetic such as metoclopramide or to administer the solution using a nasogastric tube (2). Concerns about reactivating a bleeding source by forced large bowel cleansing have not been confirmed.

The endoscopist should attempt total intubation of the colon, i. e., he should try whenever possible to reach the base of the cecum. This is important for two reasons: first, a substantial proportion of bleeding sites are located in the

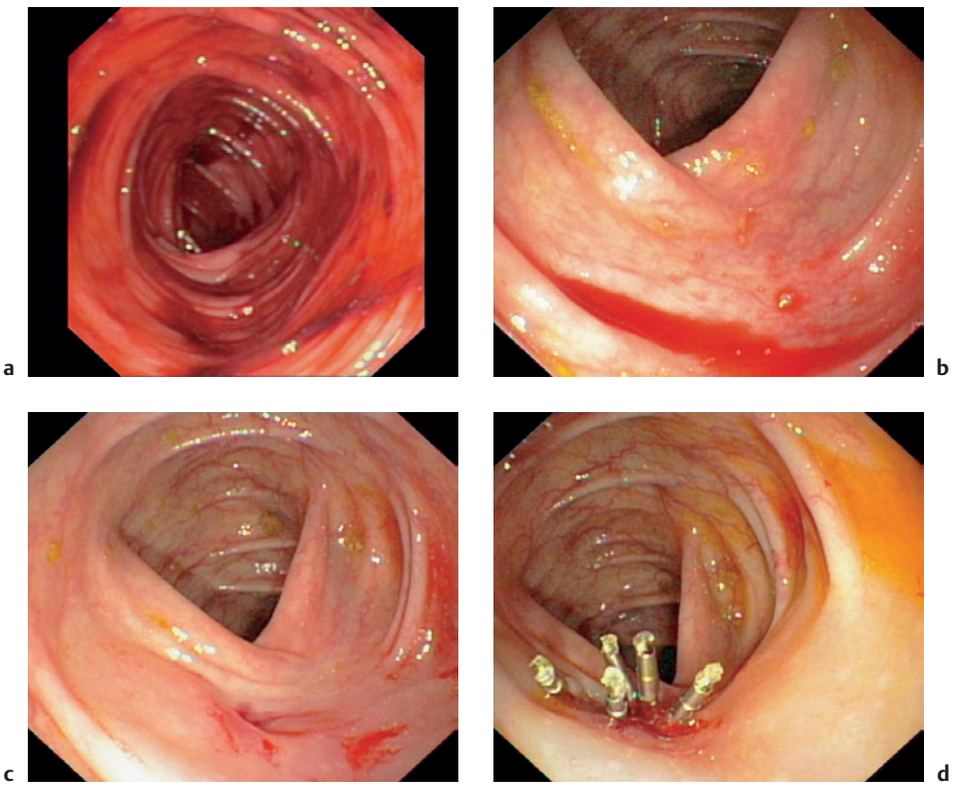


Fig. 13.4 **Diverticular bleeding.**
a Colonic mucosa covered with fresh blood.
b A sea of blood visible behind a fold after irrigation.
c After further irrigation and suction a diverticulum is visible with a slightly eroded mucosa near the orifice.
d Several hemoclips (Olympus) are applied to the suspected bleeding source.

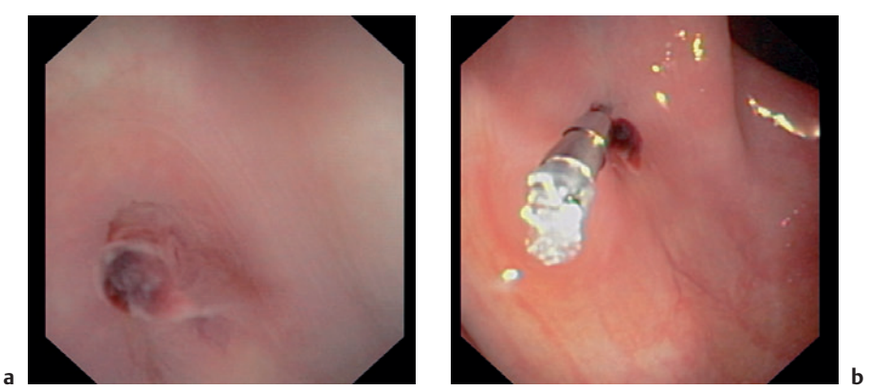


Fig. 13.5 **Visible vessel in a diverticular orifice.**
a Visible vessel protruding from a small diverticular orifice making it unrecognizable.
b Visible vessel is closed mechanically using a hemoclip (Olympus).

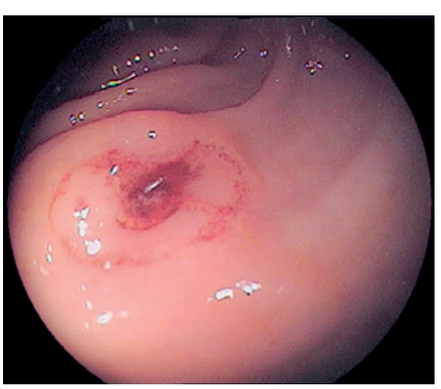


Fig. 13.6 **Visible vessel** protruding from a diverticulum. Edematous swelling of the mucosa on the edge of the orifice.

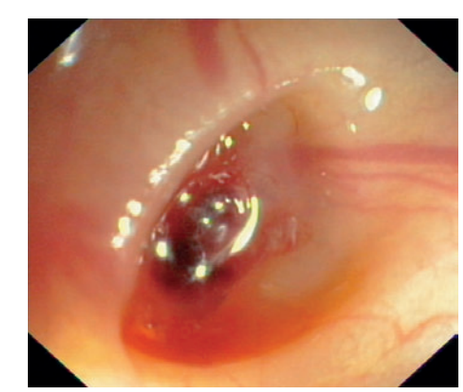


Fig. 13.7 **Visible vessel** on the base of a diverticulum.

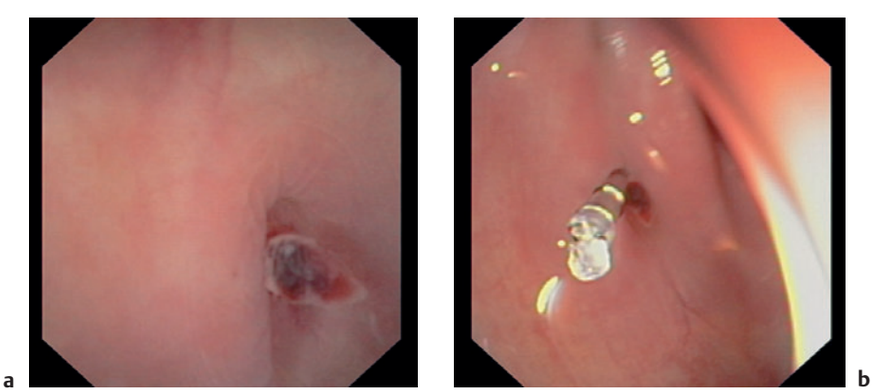


Fig. 13.8 **Visible vessel.**
a Visible vessel, protruding near the edge of a small diverticulum.
b Closed with a hemoclip (Olympus).

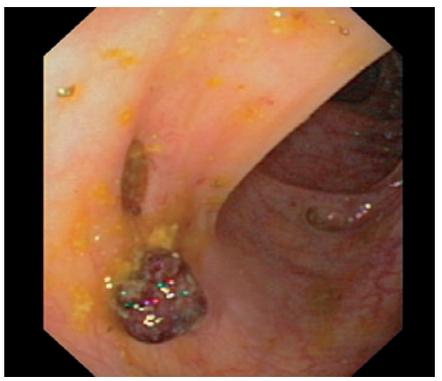


Fig. 13.**9** **Adherent clot** on a diverticular orifice. An open diverticular orifice is visible next to this one.

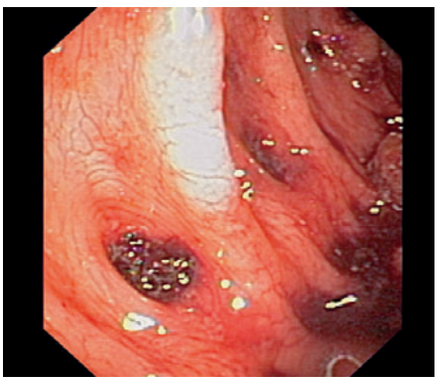
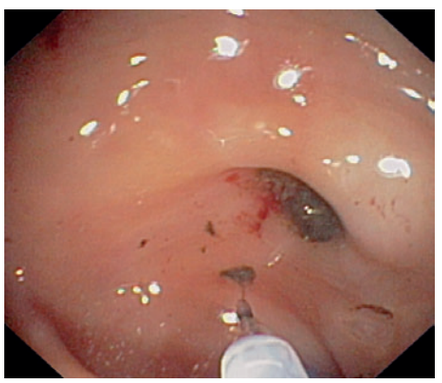


Fig. 13.**10** **Clot**, inside a diverticulum, but not adherent. Unlike an adherent clot (Fig. 13.**9**), it can be easily washed off.



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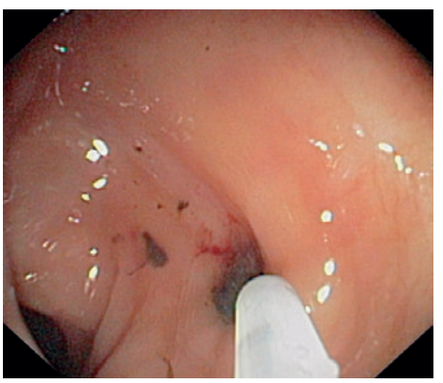

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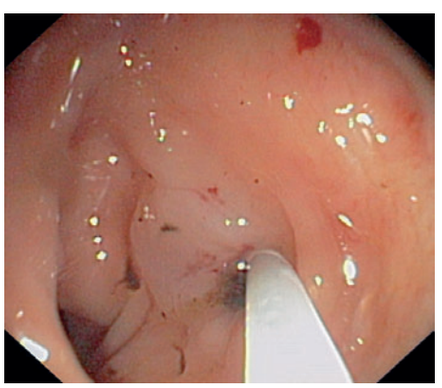

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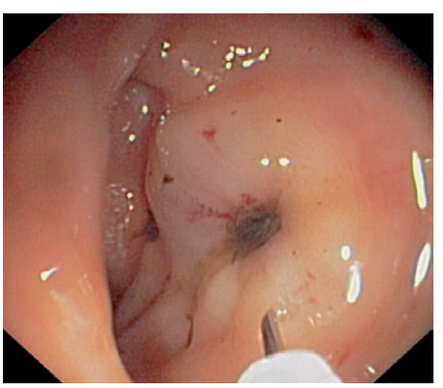


d

Fig. 13.**11** **Blood clot on a diverticulum.**
a Adherent clot on a diverticulum, not removable with irrigation.
b, c Needle injection of epinephrine (1:10 000) into the wall of the diverticular orifice.
d The orifice is swollen afterward and the mucosa is whitish in color from the vasoconstrictive effect of the epinephrine. The clot was subsequently removed by irrigation.

Table 13.**2** Localization of bleeding sites in emergency colonoscopy (*n* = 282 patients) (based on reference 36)

Bleeding site	Frequency in %
Rectum	15.1
Sigmoid colon	15.1
Descending colon	19.7
Transverse colon	12.7
Ascending colon	15.1
Cecum	2.6
Terminal ileum	1.7

right hemicolon (Tab. 13.**2**) and, second, insertion up to the terminal ileum can help determine whether blood is flowing from above, a clear indication of a more proximal bleeding site. Ohyama et al. (36) report that even under conditions of urgent colonoscopy, the cecum was inspected in 56% of patients and that terminal ileum insertion was achieved in 27% of patients.

For diagnosing hemorrhoidal bleeding or locating a bleeding site in the anorectal region, it is important to inspect the anal transitional zone with a retroflexed instrument. Proctoscopy (anoscopy) can also be performed, if necessary.

Diagnostic yield for urgent colonoscopy in acute lower gastrointestinal bleeding is reported in the literature at 48–90% (3, 38, 63). Two recent publications report diagnostic yields of 89–97% (13, 36), which perhaps is a reflection of more consistent use of urgent colonoscopy.

The frequency of colonic bleeding source reported varies from one publication to the next. One reason could be that studies often fail to differentiate between probable and definite sources of bleeding. In addition, the definition of acute lower gastrointestinal bleeding is far from uniform. Moreover, differences in patients' overall health statuses also certainly play a role (Tab. 13.**3**). Table 13.**4** provides an overview of the frequencies of bleeding sources cited in the literature. Age can provide a clue to the cause of acute lower gastrointestinal bleeding: younger patients tend to bleed from hemorrhoids, vascular malformation, and rectal ulcers, while older patients tend to bleed from diverticula, vascular malformation, and neoplasias.

In the diagnostic report, the examiner should make clear whether diagnosis of the bleeding source is definite or probable. Only if there are clear indications of active or prior bleeding (Tab. 13.**5**) should the finding be reported as a bleeding source. Therapy is only indicated when the bleeding source can be clearly identified. Reported figures for endoscopic interventions range between 3 % and 62 %. Success rates of endoscopic therapy in urgent colonoscopy are currently around 70 %. The rate of complications is low for colonoscopy in acute lower gastrointestinal bleeding (1.3 %) (overview in 63).

Table 13.**3** Comparison of results of emergency colonoscopy in acute lower gastrointestinal bleeding among patients in intensive care units with a control group (recently hospitalized inpatient patients) (40)

Diagnosis	Noninten-sive-care unit patients (*n* = 77)	Intensive-care unit patients (*n* = 12)
Diverticula	38 %	17 %
Ischemic colitis	13 %	50 %
Angiodysplasia	6 %	8 %
Postpolypectomy rebleeding	6 %	–
Rectal ulcer	5 %	8 %
NSAID colitis	5 %	8 %
Carcinoma	4 %	–
Misc.	22 %	1 %

Table 13.**4** Overview of the distribution of sources of hematochezia reported in the literature (compiled based on reference 61)

Source of hematochezia	Frequency (%)
Diverticula	17–40
Arteriovenous malformation	2–30
Colitis (ischemic, infectious, chronic inflammatory bowel disease, radiation colitis)	9–21
Neoplasias, postpolypectomy bleeding	11–14
Anorectal sources (incl. hemorrhoids, rectal varices)	4–10
Upper gastrointestinal tract bleeding (incl. ulcers, varices)	0–11
Small bowel (incl. Crohn disease, arteriovenous malformation, Meckel diverticula, tumors)	2–9

Differential Diagnosis of Acute Lower Gastrointestinal Bleeding

Diverticula

Diverticula are the reported source of gastrointestinal bleeding in 17–40 % of patients (Tab. 13.**4**). An estimated 3–5 % patients with colonic diverticula experience bleeding once in their lifetime. However, the correlation may not always be causal since diverticula are often cited as the bleeding source in the colon for lack of evidence of another source.

A recent study identified colon diverticula as the bleeding source in 22 % of patients with acute lower gastrointestinal bleeding, whether based on active bleeding (Figs. 13.**2**, 13.**3**), or based on stigmata such as visible vessels (Figs. 13.**5**–13.**8**) or adherent clots (Figs. 13.**9**–13.**11**) (30). This study also showed that findings on stigmata identified as related to an increased risk for rebleeding peptic ulcers could also be applied to diverticular bleeding. Among patients in the group in which bleeding source was actively treated with endoscopic therapy (Figs. 13.**2**–13.**5**, 13.**8**–13.**11**), there was no rebleeding, compared with 53 % of those who did not undergo endoscopic intervention. Epinephrine injection and bipolar coagulation were used.

Table 13.**5** Diagnostic criteria for urgent colonoscopy

Definite criteria for a bleeding source in the large intestine in urgent colonoscopy
▶ Active bleeding from a lesion
▶ Nonbleeding visible vessel
▶ Adherent clot
Stigmata of bleeding in the colon or in a particular colon segment
▶ Fresh blood in a colon segment
▶ Ulceration on a diverticulum with fresh blood in surrounding area
▶ No sign of fresh blood in the terminal ileum

These excellent results are contradicted, however, by another current study (8) in which a retrospective analysis of diverticular bleeding was conducted. Using the same endoscopic intervention measures, this study found earlier rebleeding in 38 % of patients and late rebleeding in 23 %. At first glance, the results of these two studies appear contradictory. Yet, a closer look reveals that Jensen et al. (30) consistently advise their patients to discontinue use of nonsteroidal anti-inflammatory drugs and acetylsalicylic acid and to follow a high-fiber diet. It is therefore entirely possible that these additional factors help explain differing results and that nonendoscopic factors also play an important role in treatment outcome.

Endoscopic therapy methods. There is no consensus on which therapeutic measure offers the most optimal treatment for diverticular bleeding. Systematic comparative studies are lack-

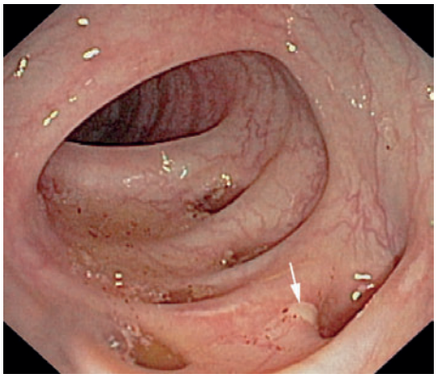


Fig. 13.**12** **Oval erosion (arrow) with fibrinous exudate** on the edge of a diverticulum, protruding into the diverticulum neck.

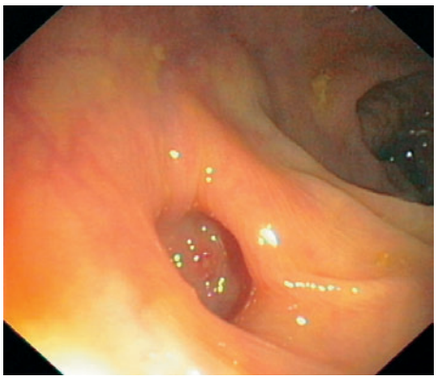


Fig. 13.**13** **Small, round, erosion with fibrinous exudate** on the base ("dome") of a diverticulum.

ing and publications on the principles of endoscopic therapy tend to have a casuistic character. Studies have reported on the success of injection therapy (Figs. 13.**2** , 13.**11**) using epinephrine and fibrin glue, as well as thermocoagulation by means of laser, heater probes, and bipolar coagulation. An interesting report has also been written on mechanical hemostasis of diverticular bleeding using hemoclips (27) (Figs. 13.**4**, 13.**5**, 13.**8**).

Endoscopic therapy

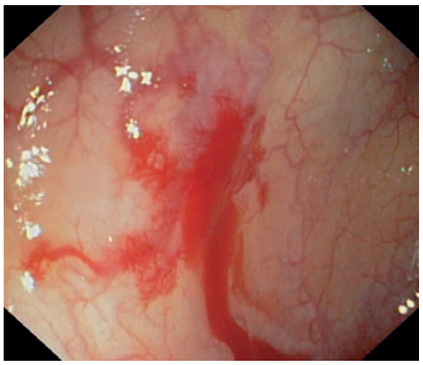
- As a rule, the methods used for achieving endoscopic hemostasis must be adapted individually. The size of diverticula and exact localization of bleeding relative to the diverticulum opening are important factors. It is also important to remember that the diverticulum wall is very thin and that thermocoagulation thus is associated with an increased risk of perforation.
- If the diverticulum itself is the source of bleeding, epinephrine solution (1:10 000) can be injected into the submucosa of the four quadrants of the diverticulum neck. In addition to the vasoconstrictive effect of epinephrine, compression of the supplying vessel also assists in achieving hemostasis. Alternatively, if the vessel or bleeding source is localized in a wider diverticulum, epinephrine injection can be made directly into the mucosa of the tip of the diverticulum, which occasionally elevates the bleeding source, enabling better visualization of the source.
- Adherent clots should be injected with epinephrine solution (Fig. 13.**11**) and carefully removed, in order to evaluate the underlying lesion better. This can be done either with a powerful blast of water or carefully with a snare.
- If bleeding is localized at the edge of the diverticulum, thermocoagulation may also be used. Laser is less suitable since the depth of coagulation is difficult to calculate. When using a bipolar coagulation probe, it is important that power be kept as low as possible (10–15 W) and that each application be brief (one second). A balance should be achieved in terms of applying pressure with the probe: on the one hand, the greater the pressure, the better the vessel is sealed, while on the other hand, greater pressure also increases coagulation depth and thus perforation risk (32)

In the midst of the discussion on optimal endoscopic treatment of diverticular bleeding, one should keep in mind that spontaneous cessation of bleeding occurs in over two-thirds of diverticular bleeding cases and that rebleeding occurs frequently in the course of disease. In one study (34) the risk of rebleeding was 9% in the first year, 10% in the second year, 19% in the third year, and 25% in the fourth year.

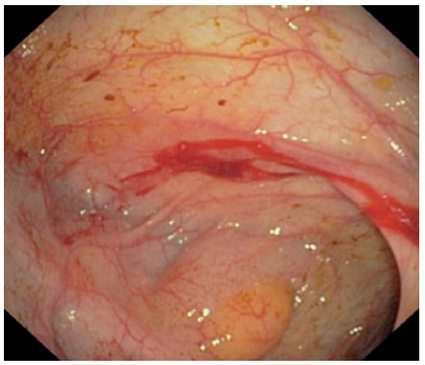
Pathogenesis of diverticular bleeding. Diverticular bleeding characteristically occurs when a vessel ruptures either near the tip of the diverticulum or in the diverticulum neck on the side opposite the mesentery. The blood vessels near the diverticulum are located very close to the surface, separated only by a thin layer of mucosa from the lumen. Foutch (23) made an important observation when he noted the presence of ulcerations near bleeding diverticula. The pathogenesis of these ulcers or erosions (Figs. 13.**12**, 13.**13**), which may erode surface vessels, remains unclear. It has often been suggested that they are caused by mechanical lesions from coproliths or digestive material, though there is lacking evidence to support this explanation. Another possibility is chemically induced ulcerating lesions. As early as 1990, Wilson et al. (58) reported that use of nonsteroidal anti-inflammatory drugs could promote myriad complications related to diverticular disease of the colon. Foutch's observations (23) point in the same direction.

In the western hemisphere, diverticula appear mostly in the left hemicolon, especially in the sigmoid colon (up to 90%). Yet, for unexplained reasons, it is diverticula in the right hemicolon that have a greater bleeding tendency. One hypothesis suggests that mucosal lesions induced by use of nonsteroidal anti-inflammatory drugs occur more frequently in the right hemicolon (41).

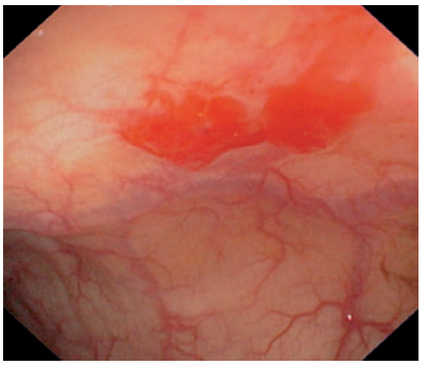
13.1 Angiodysplasia bleeding



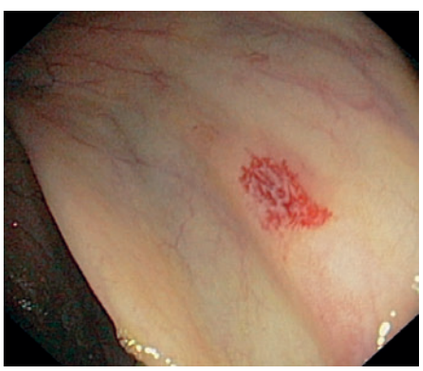
a Bleeding, starshaped angiodysplasias in ascending colon. Note the clearly dilated veins resulting from the arteriovenous shunt.



b Bleeding angiodysplasia, located in the cecum opposite the Bauhin valve.



c Extensive angiodysplasia in ascending colon. In addition to numerous angiodysplasias in the ascending colon, the 68-year-old patient had similar, smaller, arteriovenous malformations in the small intestine.



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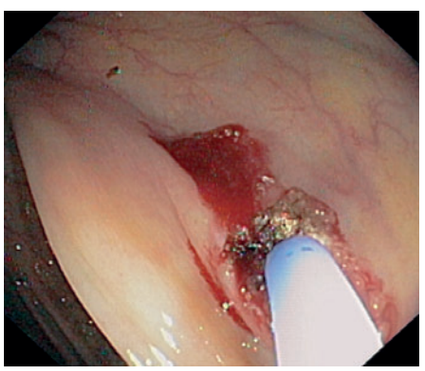

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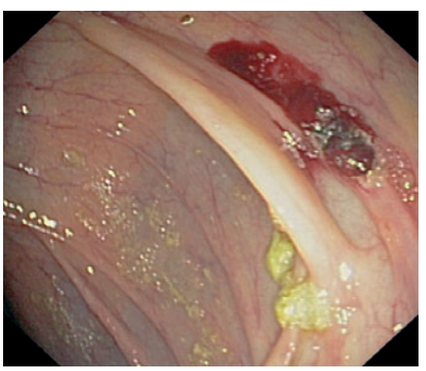


f

d–f Angiodysplasia in ascending colon (**d**). Coagulation with APC (Erbe) (**e**). Afterward, angiodysplasia was no longer detected, and coagulated mucosa is in its place (**f**).

Vascular Causes

Angiodysplasias. Angiodysplasias (▣ 13.**1**) are cited as the source of lower gastrointestinal bleeding in up to 30% of patients, though a rate of 3–12% is probably more realistic (64).

The majority of angiodysplasias (62%) are located in the right hemicolon, often occurring several at a time. The vast majority of affected individuals do not bleed (22, 42) and therapy is not always indicated for every angiodysplasia detected by colonoscopy. In addition, an angiodysplasia detected during urgent colonoscopy is not automatically the source of bleeding. The angiodysplasia must either be clearly actively bleeding (▣ 13.**1 a**, **b**) or have stigmata such as adherent clots or submucosal bleeding.

The risk of rebleeding after an initial, untreated bleeding episode is high and increases over the years. In a study by Richter et al. (42) the risk of rebleeding increased from 26% in the first year to 46% after three years. Prognosis is especially poor for angiodysplasias related to hereditary hemorrhagic telangiectasia (Osler–Rendu–Weber disease). After endoscopic therapy of these vascular malformations, rebleeding occurs almost as a rule (46). The reason is perhaps that the entire lower gastrointestinal tract is usually affected.

It is not clear why angiodysplasias bleed. However, histological analyses reveal mucosal thinning underneath the angiodysplasia and occasional ulcerations (4). Nonsteroidal antiinflammatory drugs and acetylsalicylic acid can play a role in pathogenesis.

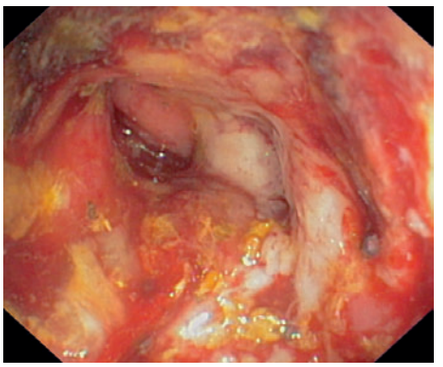


Fig. 13.**14** **Bleeding in ulcerating radiation proctitis.** The 65-year-old patient had undergone radiation therapy for prostate cancer. Histology was compatible with radiation damage; there was no evidence of infiltration of the prostate carcinoma in the rectum.

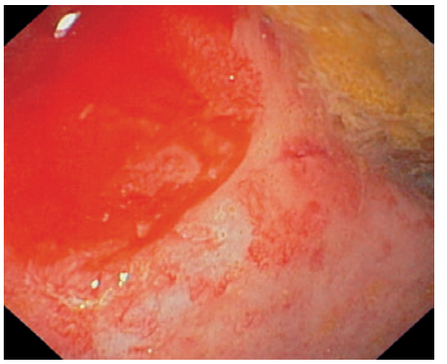


Fig. 13.**15** **Bleeding angiodysplasias** in the rectum of a 75-year-old female patient. The angiodysplasia was caused by radiation therapy of an endometrial carcinoma.

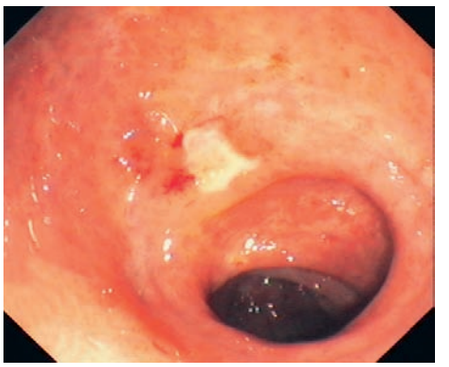


Fig. 13.**16** **Bleeding per rectum caused by previous radiation therapy** for prostate cancer. Reddened mucosa and neovascularization. An ulcer resulting from radiation-induced inflammation (covered with fibrinous exudate) can also be seen.

Endoscopic therapy

- It is important to avoid use of opiates (10, 19) and cold-water lavage of the mucosa (9) during colonoscopy as they reduce blood flow in the mucosa, decreasing diagnostic yield in angiodysplasia.
- Endoscopic therapy employs various methods of thermocoagulation. Successful use of heater probes, monopolar and bipolar probes, Nd:YAG laser, and argon plasma coagulation (APC) has been reported.
- Three things should be noted with regard to practical application of thermocoagulation: First, as little power as necessary should be used, especially in the cecum and ascending colon and each application should be brief in order to limit depth of coagulation and avoid perforation. Laser coagulation in these regions is not without risks (46). Second, larger vascular malformations should be coagulated around their periphery and the supplying vessel should be obliterated if possible. Not until after this has been done can the center of the angiodysplasia be treated. Contact thermocoagulation procedures involve a risk of bleeding as adherent tissue can be torn on withdrawal of the probe from the coagulated area. Noncontact procedures, such as APC, have a distinct advantage in this regard.

Radiation proctitis. Radiation proctitis due to radiation therapy of small tumors in the pelvis (Figs. 13.**14**, 13.**26**–13.**28**) can also lead to blood loss, but bleeding generally does not present a problem. A more serious problem is neovascularization resulting from tissue ischemia in radiation-induced endarteritis obliterans (Figs. 13.**15**–13.**17**). This can lead to considerable morbidity from recurrent blood loss. Following radiation therapy of prostate cancer, 13% of patients report more or less pronounced rectal blood loss over a period of 4–41 months (55). Another publication has reported a lower rate of 4%; Crook et al. (18) reported that 5% of patients who underwent radiation therapy complained of daily blood loss and 9% of weekly bleeding. Resulting anemia can become problematic.

Chronic radiation injury usually presents endoscopically with multiple telangiectasias (Fig. 13.**17**) often extending into the anal canal. The mucosa is pale, lacking vessels, and vulnerable. In severe cases, there can also be ulcerations (Figs. 13.**14**, 13.**16**, 13.**26**) and massive hemorrhage.

Endoscopic therapy

- As with other angiodysplasias endoscopic thermocoagulation has proved effective. A study of 18 men and four women demonstrated that, among contact procedures, bipolar probes and heater probes were equally successful (29). After four sessions, the frequency of heavy rectal bleeding decreased from 75% to 33% among those treated with a bipolar probe and from 67% to 11% among those treated with a heater probe. No complications were observed.
- There are a number of reports on laser use for these indications. Lasers included KTP lasers (53), argon lasers (54), and Nd:YAG lasers (28, 56). Laser therapy is also successful in significantly reducing the number of bleeding episodes and transfusions. The frequency of complications reported in these studies was between 0–9%. In order to avoid perforation, energy delivery should be as low as possible (Nd:YAG laser e.g., < 30 W) and the length of the pulse as short as possible (e.g., one second).
- A recent and promising therapy option is (noncontact) argon plasma coagulation. Its success in radiation-induced vascular malformation in the rectum has been

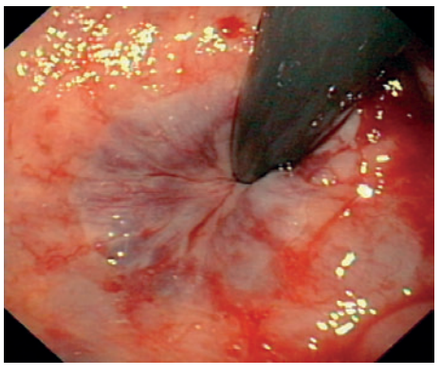


Fig. 13.**17** **Neovascularization in rectum.** View of rectum (retroflexed instrument) with a view of the upper margin of the anus and the instrument shaft. Marked neovascularization can be seen, partly affecting the anal region. This is also a delayed effect of radiation for prostate carcinoma. Bleeding ceased after thermocoagulation of the angiodysplasias using APC.

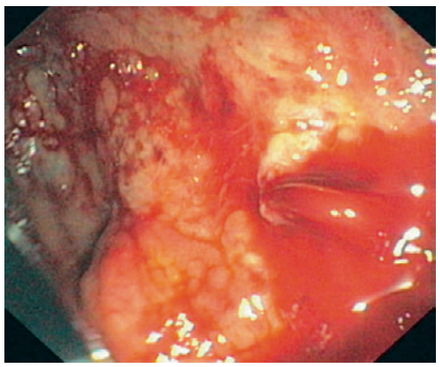


Fig. 13.**18** **Spurting hemorrhage from a rectal varix.** The 35-year-old patient had portal hypertension as a result of alcoholic liver cirrhosis.

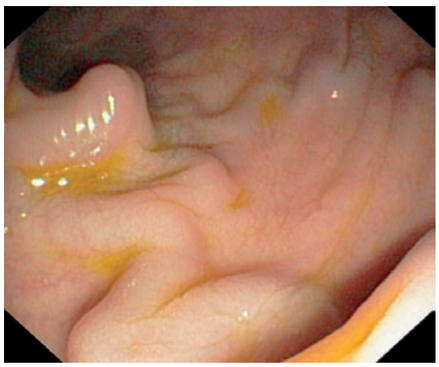


Fig. 13.**19** **Rectal varices** in a 31-year-old man with portal hypertension associated with liver cirrhosis resulting from sclerosing cholangitis.

repeatedly reported (16, 21, 47–49). The setting used for coagulation varied in these reports. Reported rates of argon gas flow were 0.6–3 L/min and electrical power ranged from 40–70 W. It should be noted that only the endoscopist who reported complications (rectal strictures) had used the highest power setting (70 W) (47). Thus, it is apparently advisable to keep energy delivery as low as possible. Gas flow should also be kept low because of the rigidity of the rectal wall in patients with radiation damage, which lowers compliance (flexibility). Success rates for APC are good. In most cases reduced rectal bleeding and increased hemoglobin levels are reported, though complete relief of symptoms can only be achieved among a minority of patients. Endoscopic therapy must be repeated due to new formation of telangiectasias.

Colon varices. Bleeding from colon varices (Figs. 13.**18**, 13.**19**) in patients with portal hypertension is not uncommon.

In the vast majority of cases, varices are limited to the rectum, where they present as tortuous gray–blue lesions running perpendicular to the folds. If only briefly glimpsed, they can be confused with solid polypoid structures, especially in situations involving acute bleeding, obscuring visualization. The risk of bleeding is 8–9 %.

Endoscopic therapy

- Endoscopic therapy options include (based on treatment of varices in the upper gastrointestinal tract) sclerotherapy, band ligation, and intravariceal injection of acrylic glue.

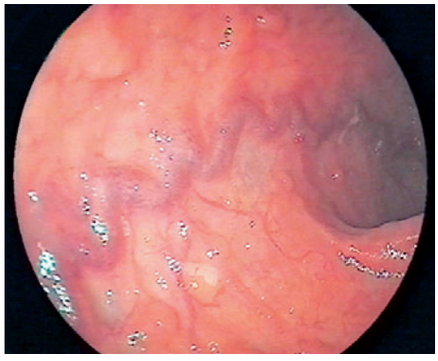


Fig. 13.**20** **Vascular ectasia in ascending colon** in a patient with Turner syndrome. The patient was suffering from extreme lower gastrointestinal hemorrhage.

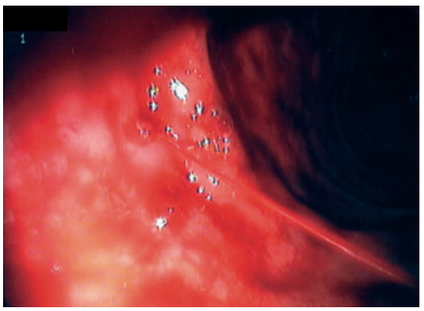
- In principle, choice of therapy depends on the actual situation and one's own experience. Based on our own experience with rubber band ligation, this method is not advisable.

Vascular ectasia. Vascular ectasia in the colon without portal hypertension (also called phlebectasia) is uncommon. It is described in conjunction with cavernous hemangiomas in Turner's syndrome (Fig. 13.**20**). In our own clinical files, such a case led to severe lower gastrointestinal rebleeding. The vascular ectasias were primarily in the cecum, ascending colon, and transverse colon.

Endoscopic therapy

- Despite reports on endoscopic therapy of cavernous hemangiomas (1, 25), the safety of endoscopic intervention remains unclear. A further endoscopic therapy option is injection of sclerosing agents, similar to procedures for skin hemangiomas (59).
- The therapy of choice for larger cavernous hemangiomas thus remains surgical intervention.

13.2 Bleeding from a Dieulafoy ulcer in the intestine



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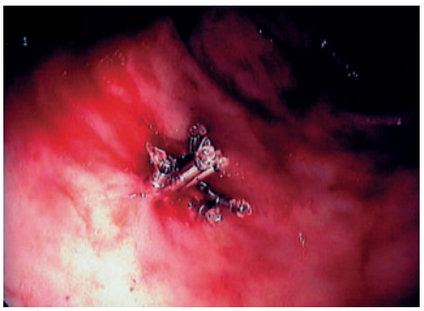
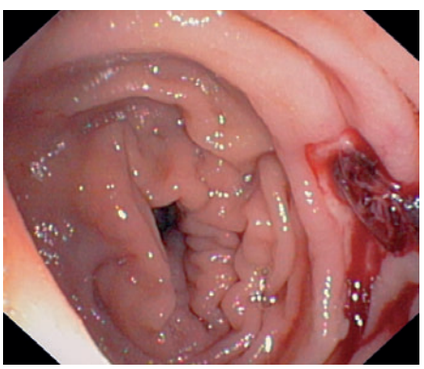


b

a, b Spurting blood from a Dieulafoy ulcer in the sigmoid colon **(a)**. Bleeding stopped immediately after application of a hemoclip (Olympus) **(b)**.



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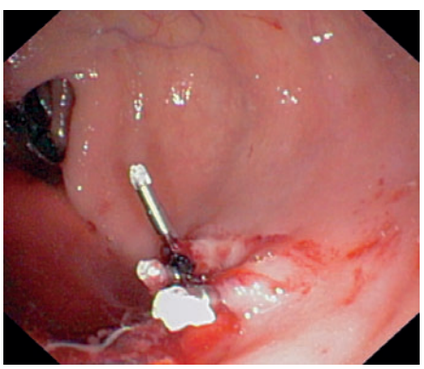
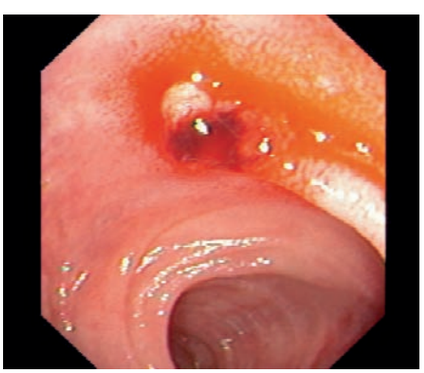


d

c, d Adherent clot on a Dieulafoy ulcer **(c)**. After washing off the clot the visible vessel underneath is clipped (Olympus) **(d)**.



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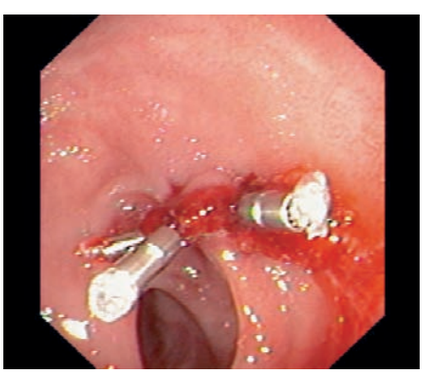


f

e, f Dieulafoy ulcer in the small bowel (ileum) with visible vessel and slight hemorrhagic oozing (**e**). The lesion was reached endoscopically through an ileostomy and bleeding was stopped mechanically with a hemoclip (Olympus) (**f**).

Dieulafoy Ulcer

Bleeding from a Dieulafoy ulcer in the stomach is not an unusual finding for the endoscopist, but it is an unexpected cause of colonic bleeding.

Dieulafoy lesions are caused by unusually large, tortuous vessels (arterioles) that are located at the affected area in the mucosa or submucosa. Small mucosal lesions can lead to massive spurting hemorrhage (▣ 13.**2 a**, **b**). Viewed endoscopically, one sees an adherent clot on a small lesion (▣ 13.**2 c**, **d**) or a visible vessel protruding from the mucosa (▣ 13.**2 e**, **f**), which is not due to arteriovenous malformation. It has been postulated that the ruptured vessel is caused by fibrosis of the intima and loss of elastic fibers in the vessel wall.

Endoscopic therapy

- Endoscopic therapy of Dieulafoy lesions in the colon is based on experience with these lesions in the stomach.
- Successful achievement of endoscopic hemostasis using injection of sclerosing agents, band ligation, thermocoagulation, and hemoclips has been (casuistically) reported (▣ 13.**2**).
- A comparative study demonstrated that mechanical methods of hemostasis (hemoclip and band ligation) were clearly more effective in treating Dieulafoy ulcers (8 % vs. 33 %) than injection therapy (15).

Ischemia (see also Chapter 12)

Hematochezia is not infrequently caused by colonic ischemia (Tabs. 13.**3**, 13.**4**). The resulting lower gastrointestinal bleeding does not usually cause hemodynamic compromise. The patient complains of cramplike abdominal pain and usually diarrhea. In most cases, clinical picture is self-limiting and does not require any special treatment. Thus, one also speaks of transient ischemic colitis. However, there are also chronic forms and these can be difficult to differentiate from chronic inflammatory bowel syndrome (Tab. 13.**6**).

Endoscopic appearances vary in the course of disease. Submucosal hemorrhage and mucosal nodularity are typical endoscopic findings in early stages (▣ 13.**3 a–c**). Alternatively, the mucosa may not be edematous, but livid (▣ 13.**3 d**) or pale (▣ 13.**3 e**). In later stages, when blood and edema have been resorbed, appearances are less characteristic. The mucosa may be red (▣ 13.**3 f**, **g**), vessel pattern may be obscured, and ulcerations (▣ 13.**3 h**, **i**) can appear. Changes are sometimes only segmental. In more serious forms the colon wall is covered by gray–white membranes (similar to pseudomembranous colitis) (▣ 13.**3 i**) or gray–black membranes. After the membrane is sloughed off, ulcers appear. Endoscopic therapy options are limited unless a circumscribed bleeding source can be identified and treated.

Ischemic colitis lesions may also occur in association with vasculitis (Fig. 13.**21**). These are relatively rare and are usually only found in advanced forms of systemic vasculitis.

Table 13.**6** Characteristics of colon ischemia (based on reference 35)

Age	> 60 years old	< 60 years old
Cause	Idiopathic Aortic surgery Distal obstruction Hypotension	Vasculitis Oral contraceptives Medication Drugs Thrombophilia Infection Pseudo-obstruction Marathon running Pheochromocytoma
Clinical picture	Acute	Acute/recurrent
Localization	Splenic flexure Sigmoid colon	Ascending colon Cecum Rectum

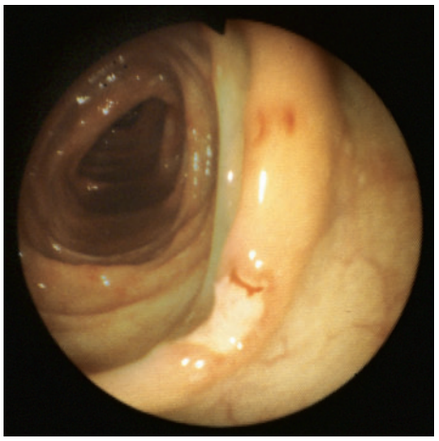
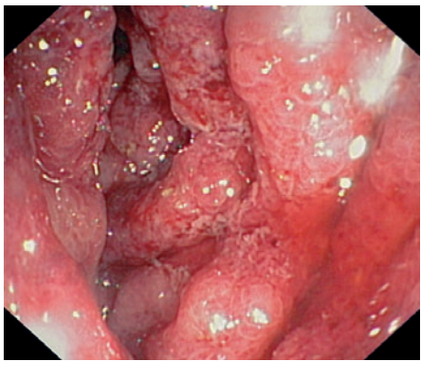


Fig. 13.**21** **Colon ulcer in vasculitis** due to chronic polyarthritis.

▣ **13.3 Ischemic colitis: various appearances**



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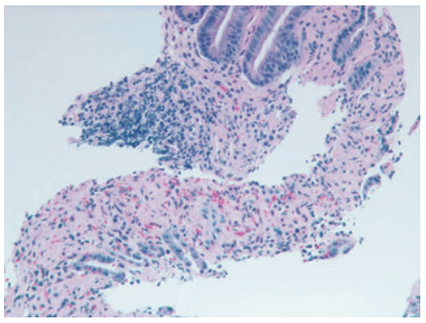

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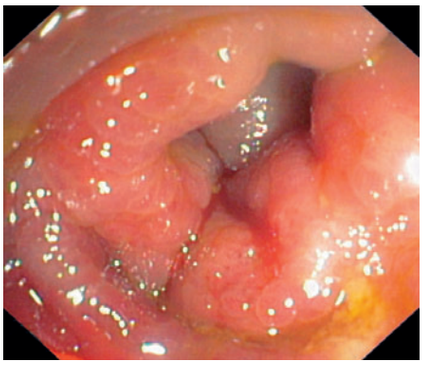
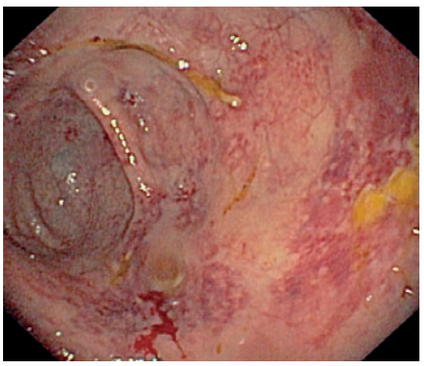


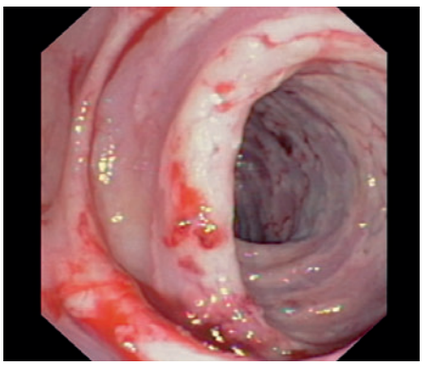
a, b Ischemic, nontransmural colitis, limited to the hepatic flexure in a 50-year-old female patient. **a** Endoscopy: swollen, bloodsoaked mucosa. **b** Histology: "damming" crypts and hemorrhagic inflammatory infiltrate, a typical finding for ischemic colitis (provided courtesy of Dr. Th. Wagner, Institute for Pathology, Augsburg Clinic). The patient recovered with conservative therapy.

c Ischemia of the sigmoid mucosa in an 88-year-old female patient. The mucosa was reddened, swollen, vulnerable, and demonstrated submucosal bleeding at multiple locations; histology showed this also to be ischemic colitis.

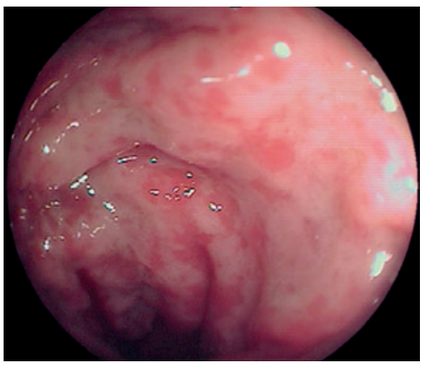
13.3 cont.



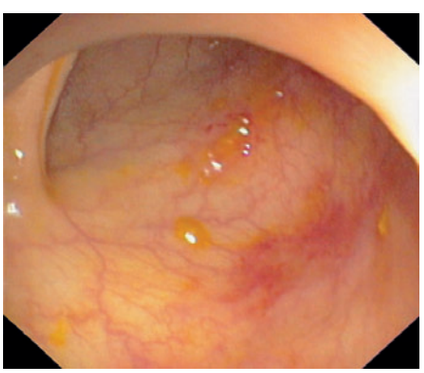
d Ischemic colitis in the ascending colon of a 79-year-old patient. There is some patchy submucosal bleeding, while the surrounding mucosa appears pale and livid. There are also small erosions with fibrinous exudates in the mucosa.



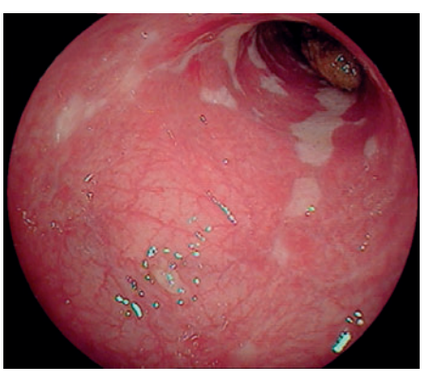
e Ischemia in the neoterminal ileum after prior right hemicolectomy for carcinoma. The mucosa is pale and has multiple bleeding foci.



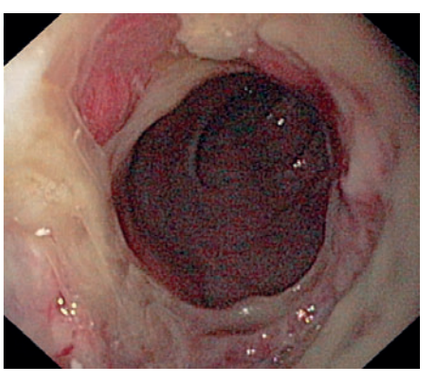
f Circumscribed ischemic colitis affecting the sigmoid and descending colon in a 72-year-old woman. The mucosa is reddened, has signs of submucosal bleeding, and is slightly polypoid (endoscopic equivalent of thumbprinting seen on radiographs).



g Low-grade pronounced ischemic colitis. Adjacent to this lesion the patient had pronounced ischemic colitis. This is presumably nonocclusive ischemic colitis.



h Flat ulcers on a reddened mucosa. Nontransmural ischemia in the descending colon.



i Ischemic proctitis with mucosal membranes presenting as pseudomembranous. *Clostridium difficile* was not detected.

Colitis (see also Chapter 12)

Ulcerative colitis and Crohn disease. Heavy bleeding is responsible for 6–10% of emergency surgical procedures in patients with ulcerative colitis; ulcerative colitis is the cause of lower gastrointestinal bleeding in 2–8% of patients (overview in 64). Nevertheless, massive lower gastrointestinal bleeding is not a frequent occurrence in chronic inflammatory bowel diseases.

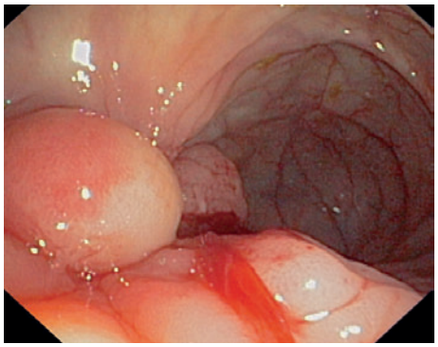
Massive hemorrhaging leads to hospitalization in 0.1% of patients with ulcerative colitis and 1.2% of patients with Crohn disease (37) (Figs. 13.**22**, 13.**23**). Among Crohn patients, bleeding localization has been said to be evenly distributed among the small bowel, ileocolonic junction, and colon (37). Two studies have contradicted this, however. One cited the colon (6) and the other the ileocolonic junction (17) (Figs. 13.**22**, 13.**23**) as the sites of predilection for gastrointestinal bleeding in Crohn disease.

Endoscopic therapy

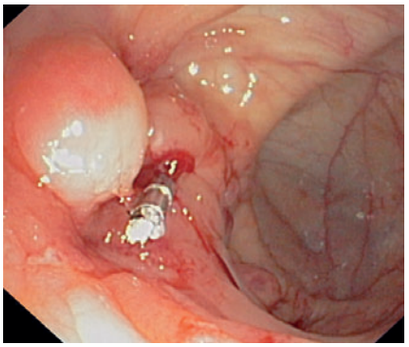
- In half of all patients with bleeding related to chronic inflammatory bowel diseases, cessation of bleeding is spontaneous. However, the rate of rebleeding is 35% (44). In about half of patients in whom bleeding does not cease spontaneously, operative intervention is required (37) and is associated with an elevated risk.
- In the majority of cases of hemorrhaging induced by chronic inflammatory bowel diseases, bleeding is diffuse; there are, however, circumscribed bleeding sources which are endoscopically treatable. Pardi et al. (37) used epinephrine injection as well as bipolar coagulation.
- Hirana et al. (26) treated bleeding in a patient with ulcerative colitis endoscopically, using injection of a mixture of absolute alcohol and 1% polidocanol. Hemoclips have also has been shown to successfully achieve hemostasis (60) (Fig. 13.**22**).

Infectious colitis. Though infectious colitis can present with bloody diarrhea, life-threatening hemorrhage is rare. Hemorrhaging has been observed in colitis caused by *Salmonella typhi*, *Salmonella typhimurium*, *Escherichia coli* 0157:H7 (EHEC), and cytomegalovirus. Pseudomembranous colitis can also manifest as acute lower gastrointestinal bleeding.

▣ 13.**4** shows varying degrees of infectious colitis attack due to various causes. Endoscopic intervention is generally not necessary.



a



b

Fig. 13.**22** **a Bleeding in Crohn disease.** Bleeding from the mucosa on the Bauhin valve in Crohn disease. **b** Cessation of bleeding after clip application.

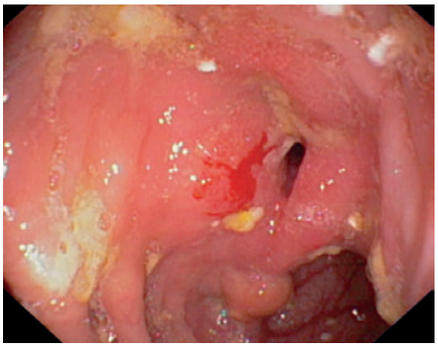
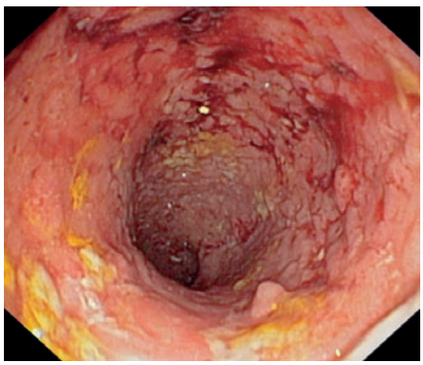
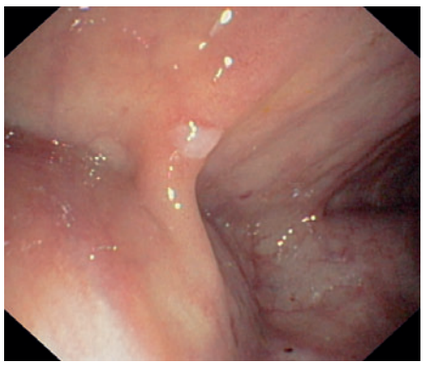


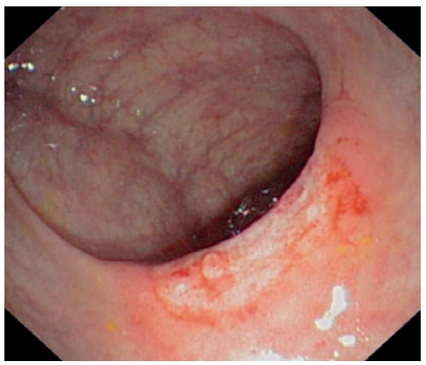
Fig. 13.**23** **Low-grade bleeding** from the mucosa on the Bauhin valve in Crohn disease. The lumen of the Bauhin valve is distorted by scarring.



a Bleeding in infectious (self-limiting) colitis in the descending colon. No pathogen detected.

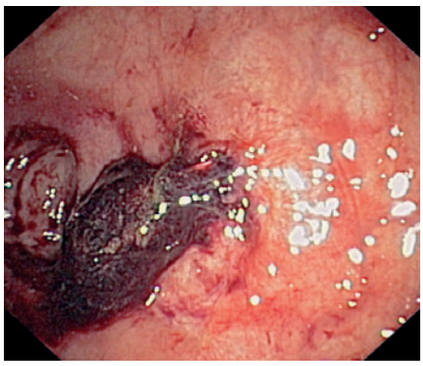


b

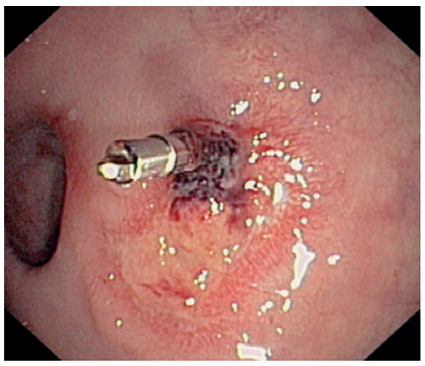


c

b, c *Campylobacter jejuni* infection of the colon. Ulceration (**b**) and bloody diarrhea led to hospitalization. Patchy ulceration on the Bauhin valve with small visible vessel on the ulcer periphery (**c**).

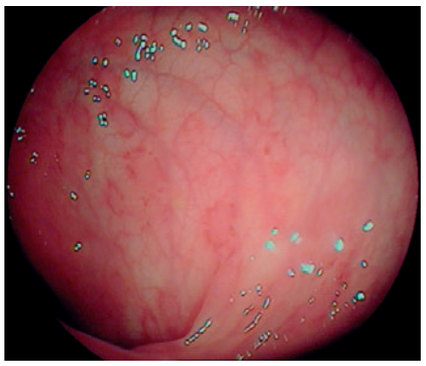


d



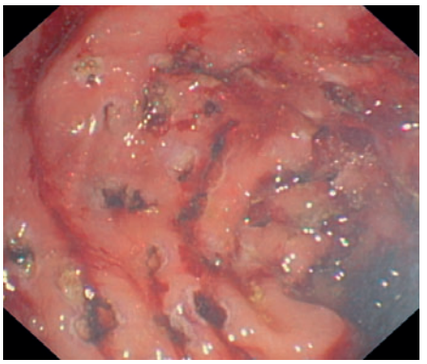
e

d, e Acute ulcerative colitis of uncertain genesis; presumed cytomegalovirus infection was ruled out. Colon ulcer with adherent clot (**d**). Visible vessel was seen after irrigation and removal of the clot and was closed with a hemoclip (Olympus) (**e**).

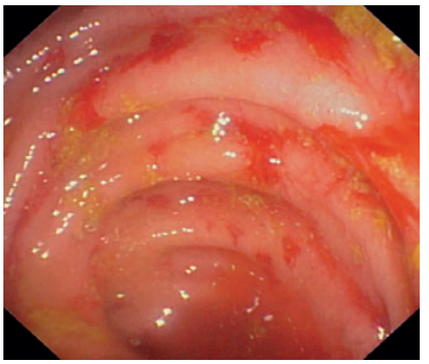


f Mild, acute hemorrhagic colitis in left hemicolon. No pathogen isolated.

13.4 cont.

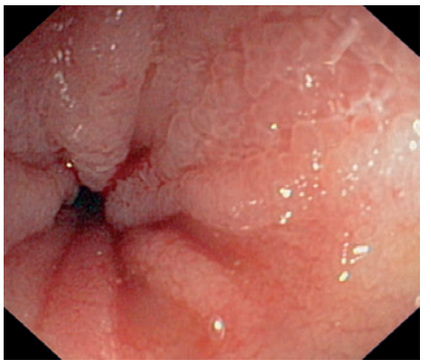


g



h

g, h Acute colitis with hemorrhagic erosions (**g**) and submucosal hemorrhages (**h**). No pathogen detected.



i Acute colitis with massive mucosal edema, isolated hemorrhaging, and small, erosive defects in a 64-year-old patient who was treated with immunosuppressants following a kidney transplant. Cytomegalovirus infection was excluded and no other pathogen was detected.

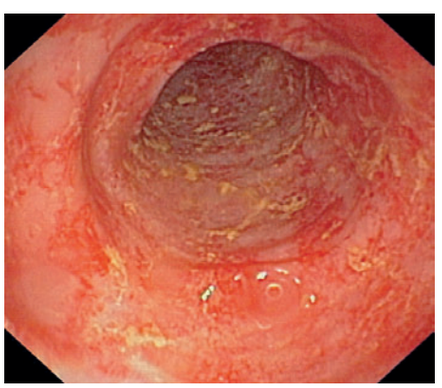


Fig. 13.**24** **Diversion colitis** after attachment of an ileostomy due to peritoneal carcinosis in ovarian cancer.

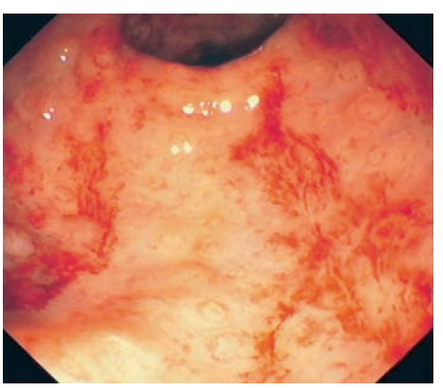


Fig. 13.**25** **Diversion proctitis in a rectal stump in a patient who had undergone a Hartmann procedure.** The patient presented with recurrent anal bleeding.

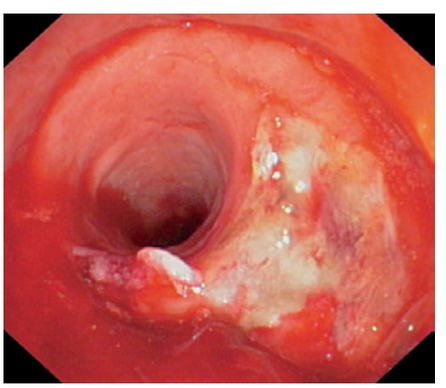


Fig. 13.**26** **Rectal ulcer in radiation proctitis.** The 64-year-old female patient had undergone radiation therapy for uterine cancer.

HIV infection. The causes of lower gastrointestinal bleeding in patients with HIV differ from those in other patients. The most common are cytomegalovirus colitis (25%), lymphoma (12%), and idiopathic (unidentifiable) colitis (12%) (7). The first two causes are especially pronounced in patients with a CD4 lymphocyte count below 200/mm³. If cell count is greater than 200/mm³ the most common bleeding sources are idiopathic colitis, diverticula, and hemorrhoids. Rebleeding is not uncommon. Thirty-day mortality related to bleeding is around 14%, whereby patients with concomitant medical problems, rebleeding and those requiring operative intervention are especially at risk. In a study by Bini et al. (7), bleeding was controlled endoscopically in nearly all patients by means of bipolar thermocoagulation probes, with or without epinephrine injection. In a study by Chalasani et al. (12), the most common cause of bleeding was also cytomegalovirus infection, followed by hemorrhoids and anal fissures. Thrombocytopenia was a particular risk factor for hemorrhoid bleeding. Further bleeding sources in patients with HIV are histoplasmosis of the colon, Kaposi's sarcoma in the colon, and bacterial colitis.

Diversion colitis. Diversion colitis (Figs. 13.**24**, 13.**25**) sometimes manifests clinically as blood loss. This type of colitis is caused by bacterial imbalance of the colon mucosa resulting from lacking fecal stream after the attachment of a stoma (Fig. 13.**24**) or a Hartmann procedure (Fig. 13.**25**). Diminished levels of short-chain fatty acids in the colonic lumen, which nourish the mucosa, are blamed etiopathogenically.

Radiation colitis. Radiation therapy can cause acute and subacute radiation colitis (Figs. 13.**26**–13.**28**) in the colon, particularly in the rectum and sigmoid colon, and occasionally heavy

loss of blood. The cause of radiation colitis is disrupted cellular proliferation and regeneration as well as induction of inflammatory processes in the colonic mucosa. Possibilities for endoscopic therapy are usually limited.

Nonsteroidal anti-inflammatory drugs (NSAID). Nonsteroidal anti-inflammatory drugs can promote bleeding from any number of possible lesions in the gastrointestinal tract.

Nonsteroidal anti-inflammatory drugs can also induce colitis, which may not be visibly discernible from infectious colitis or chronic inflammatory bowel disease (Figs. 13.**29**, 13.**30**). However, its endoscopic aspect can also include flat and usually irregularly bordered erosions and ulcerations, which are surrounded by an otherwise normal appearing mucosa (▣ 13.**5**). Individual lesions may bleed.

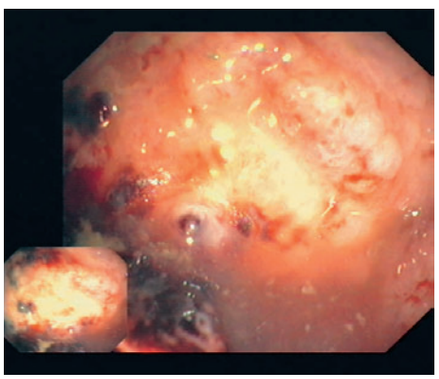


Fig. 13.**27** **Ulceration at rectosigmoid anastomosis** with prior resection of a sigmoid carcinoma and chemoradiotherapy. Several visible vessels can be seen on the edge of the ulceration.

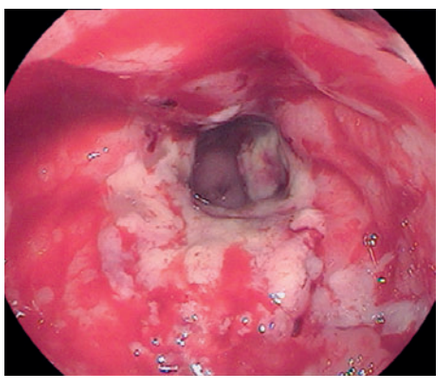


Fig. 13.**28** **Severe radiation-induced hemorrhagic inflammation of the mucosa at a rectosigmoid anastomosis** following prior resection of an adenocarcinoma and chemoradiotherapy. Recurrent blood loss from massively inflamed mucosa in this region, which was difficult to treat endoscopically.

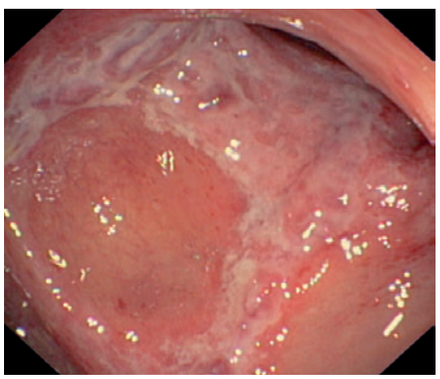
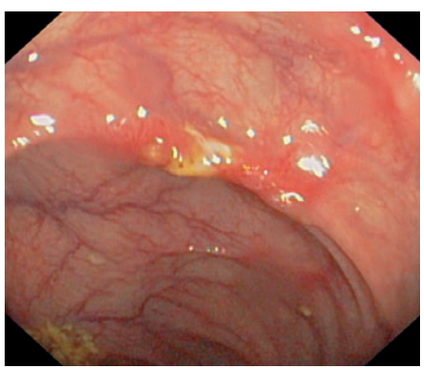
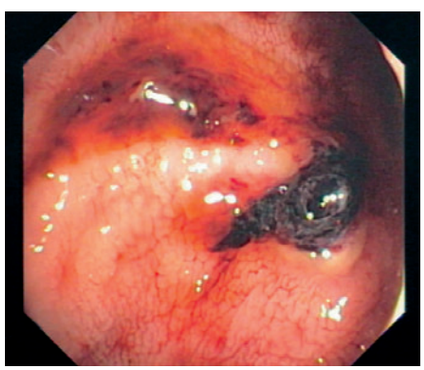


Fig. 13.**29** **Pronounced colitis** with reddened mucosa and fibrinous exudates, primarily affecting descending colon, related to use of nonsteroidal anti-inflammatory drugs.

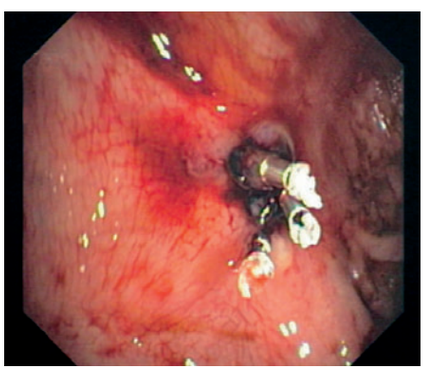
▣ 13.5 Ulcers in use of anti-inflammatory drugs



a Diffuse ulcers affecting the entire colon.



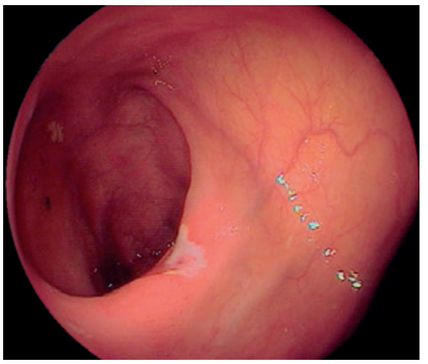
b



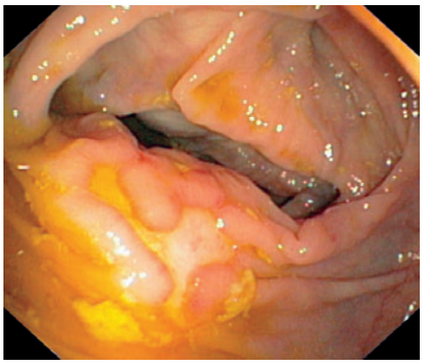
c

b, c Ulcer with adherent clot near an ileocolic anastomosis in a 46-year-old patient using anti-inflammatory drugs (**b**). The visible vessel underneath the clot was hemoclipped (Olympus) (**c**).

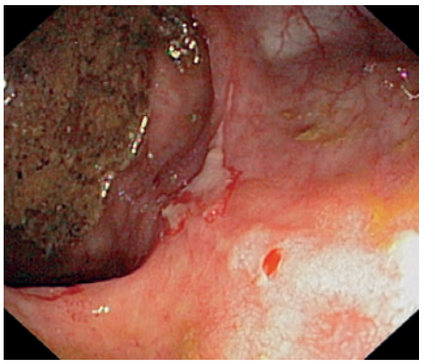
13.5 cont.



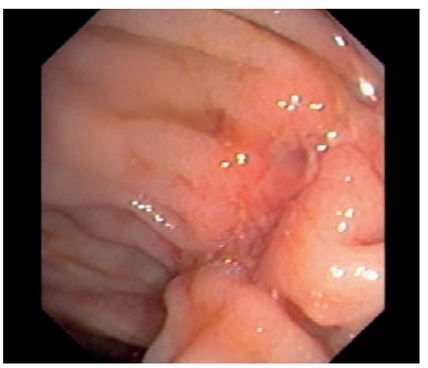
d Flat ulcer on a haustrum in the ascending colon; the surrounding area shows no reaction. The patient was using Diclofenac.



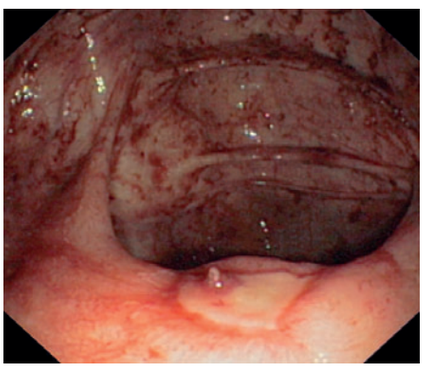
e Bizarre ulceration on the Bauhin valve.



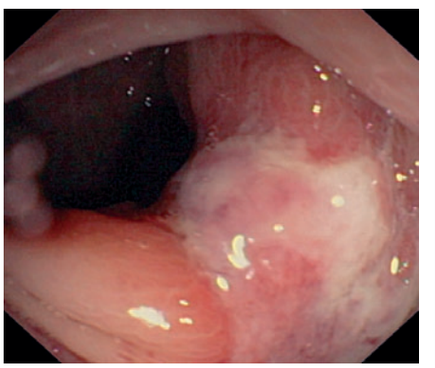
f Bizarre ulcer in the cecum with blood-covered borders.



g Depressed linear ulceration with swollen margins.



h Larger, flat ulcer on the Bauhin valve, with a visible vessel on its margin.



i Large ulcer with swollen borders in the descending colon.

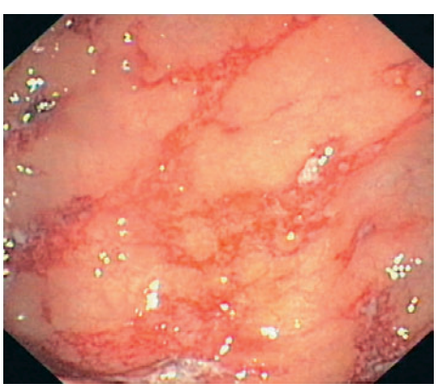


Fig. 13.**30** **Hemorrhagic and erosive defects** in swollen colon mucosa. The patient was using nonsteroidal anti-inflammatory drugs.

Neoplasias (see also Chapter 10)

Carcinomas. Carcinomas were cited in one study in 21% of patients as the source of hematochezia (45). More probable figures range from 2–9% (14, 34, 36, 40, 43, 52, 57**).**

Bleeding from a carcinoma (Figs. 13.**31**–13.**35**) is the result of erosions and ulcerations on the surface of the tumor, which can be exacerbated by use of anti-inflammatory drugs. Carcinomas in the sigmoid colon often lead to early rectal bleeding. In the right hemicolon, in contrast, they do not manifest with rectal bleeding until after they are clearly ulcerated.

Endoscopic therapy

- Endoscopic therapy must be determined on an individual basis. Based on our experience, injection therapy and mechanical hemostasis using hemoclips have proved to be effective methods (▣ 13.**5 b**, **c**).

Endoscopic therapy

- Both laser and APC methods allow endoscopic hemostasis of the usually superficially bleeding carcinomas by means of noncontact thermocoagulation. APC has proved especially effective based on individual reports (50, 11) and also in our own experience.

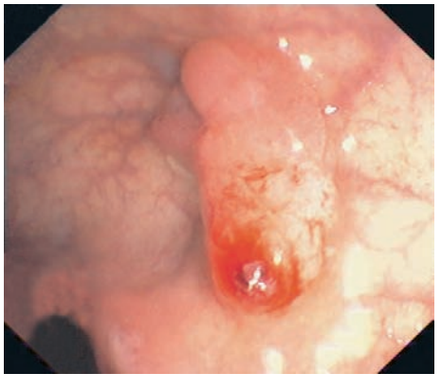


Fig. 13.**31** **Polyp in the rectum with erosion of the tip and a small visible vessel.** Histology revealed an adenocarcinoma.

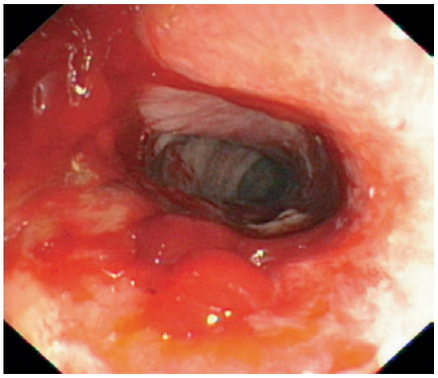


Fig. 13.**32** **Rectal carcinoma with hemorrhagic oozing.**

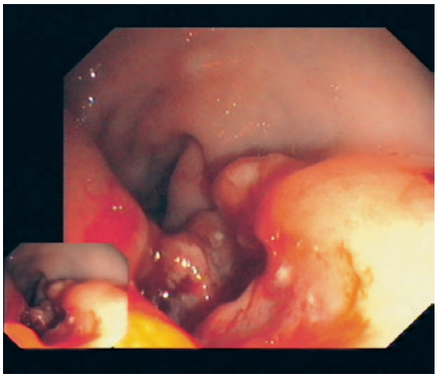


Fig. 13.**33** **Ulcerated rectal carcinoma with hemorrhagic oozing.**

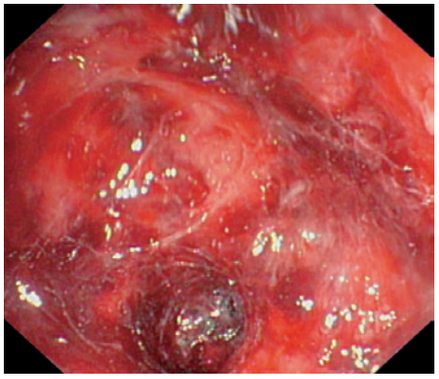


Fig. 13.**34** **Bleeding caused by a bladder carcinoma protruding into the rectum.**

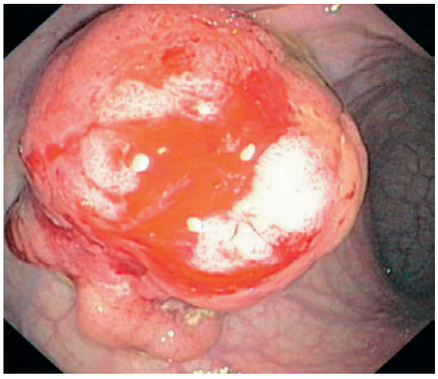


Fig. 13.**35** **Polyp at rectosigmoid junction** with eroded and bleeding surface. The polyp was already transforming into an adenocarcinoma.

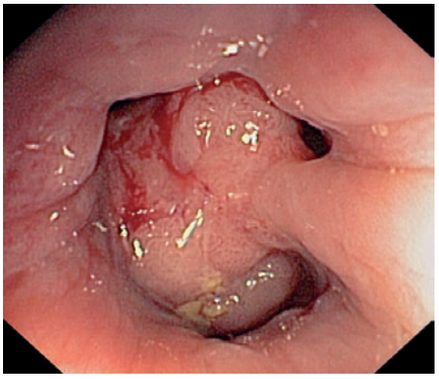


Fig. 13.**36** **Stalked polyp covered with a layer of blood.**

- Thermocoagulation using contact methods is less suitable because tearing of tissue after completing coagulation can cause hemorrhagic oozing.
- Injection of absolute alcohol into a tumor has reportedly been successful in achieving hemostasis (5).
- In circumscribed bleeding sources, especially with visible vessels, mechanical methods such as hemoclipping can also be used.

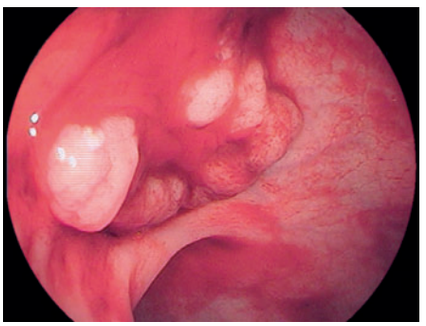
Colon polyps. Colon polyps are cited in 5–11 % of patients as the source of acute lower gastrointestinal bleeding (overview in 64). However, polyps are more commonly involved in chronic intermittent bleeding.

Generally, it is larger polyps with a diameter greater than 1 cm that bleed (Fig. 13.**36**). The polyp surface demonstrates lesions of uncertain genesis, though they could be caused by hardened waste eroding the vulnerable polyp surface. Anti-inflammatory drugs or acetylsalicylic acid probably play a role. The most common cause of lower gastrointestinal bleeding from benign polyps is polypectomy (▣ 13.**6**, 13.**7**, Fig. 13.**37**). Bleeding may occur immediately after resection (▣ 13.**6**, Fig. 13.**37**), though the time between polypectomy and bleeding can vary, and can occasionally be a few days (▣ 13.**7**).

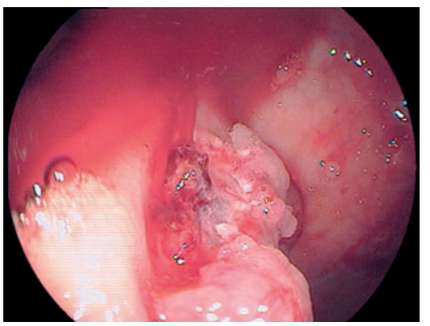
Endoscopic therapy

- Bleeding can generally be controlled endoscopically. Preferred methods of hemostasis include epinephrine injection or hemoclip application (▣ 13.**6**, 13.**7 a–c**), or a combination of the two (Fig. 13.**37**).
- Therapy with hemoclips seems to be the more reliable method based on our own experience in that they guarantee mechanical closure of the vessel if applied correctly.

13.6 Bleeding after resection of colon polyps, hemostasis with clips



a



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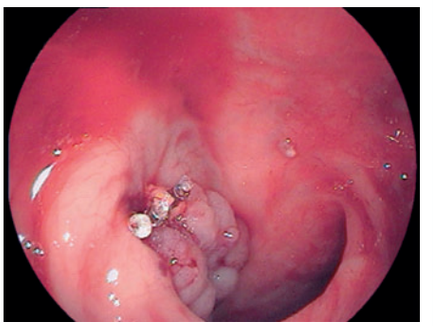
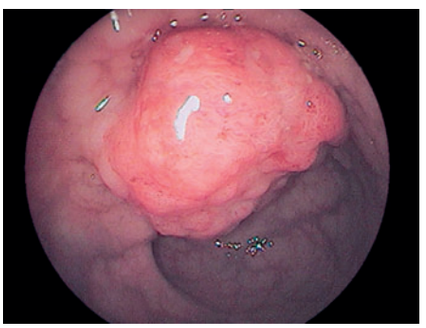


c

a–c Bleeding after partial resection of a large, broad-based polyp at the rectosigmoid junction (**a**). Visible vessel can be seen after irrigation (**b**) at the base of the resection wound. Bleeding was definitively controlled by application of three hemoclips (Olympus) (**c**).



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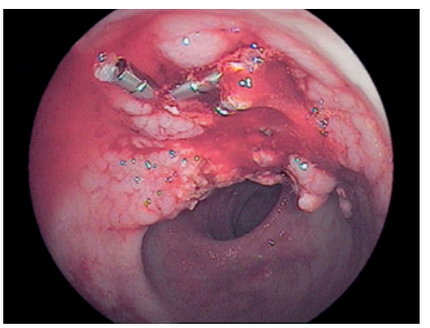
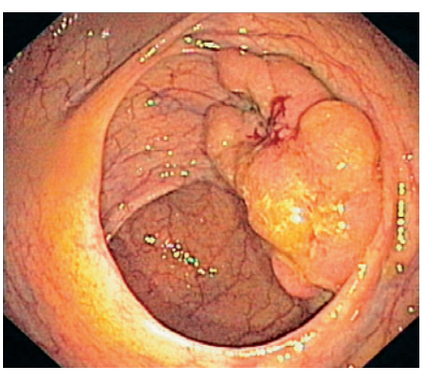


e

d, e Broad-based sessile polyp (histology: tubular adenoma) (**d**). After resection, spurting bleeding stopped with two hemoclips (Olympus) (**e**).



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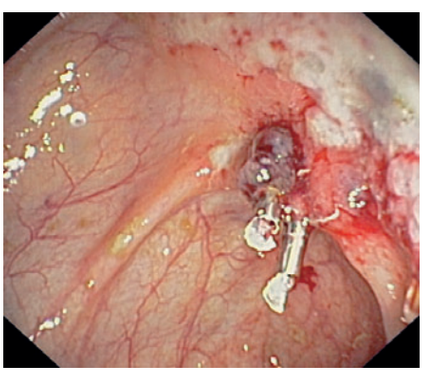


g

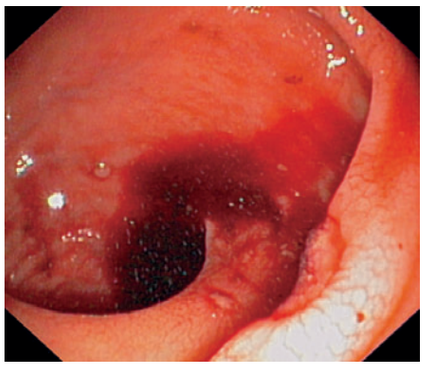
f, g Broad-based, sessile polyp (opposite the Bauhin valve) (**f**). After polypectomy by means of mucosectomy, bleeding occurred from a vessel. The visible vessel on the edge of the resection site was closed with two hemoclips (Olympus) (**g**).

Although it does not belong to the topic of polyps, rebleeding after forceps biopsy (Figs. 13.**38**, 13.**39**) should be mentioned here. Not infrequently, the cause of rebleeding lies in anticoagulant use, which is resumed too soon or not discontinued before the biopsy.

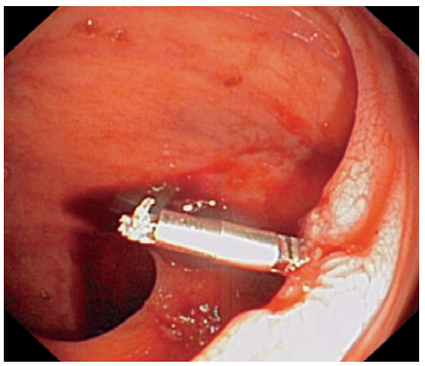
Anorectal Diseases

Anorectal causes of acute lower gastrointestinal bleeding can be detected in fewer than 10% of patients, whereby hemorrhoids and anal fissures are the main sources of bleeding. The exact prevalence is difficult to determine, as some studies of acute lower gastrointestinal bleeding do not include anorectal causes.

13.7 Rebleeding after polypectomy, hemostasis with clips



a



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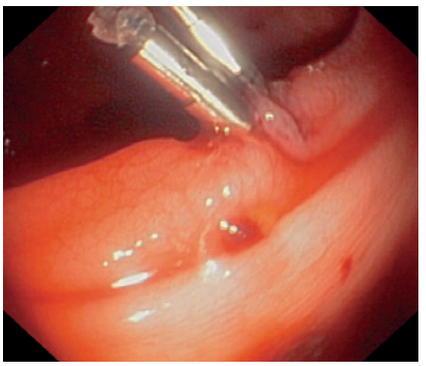
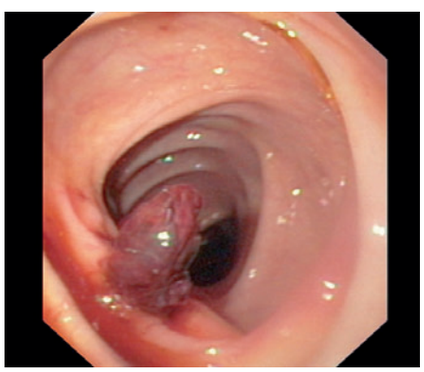


c

a–c Polypectomy site in the ascending colon. A visible vessel can be seen on the upper edge of the resection defect (**a**), which led to massive rebleeding. Hemostasis is attempted with a single hemoclip (Olympus) (**b**). An additional clip is used to definitively compress and close the visible vessel (**c**).



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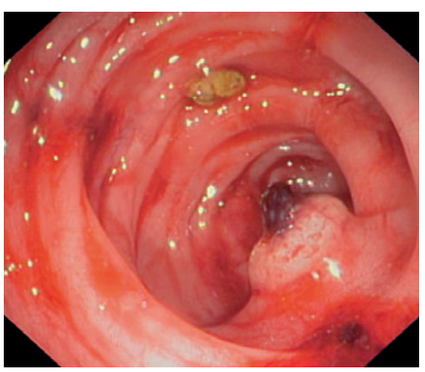
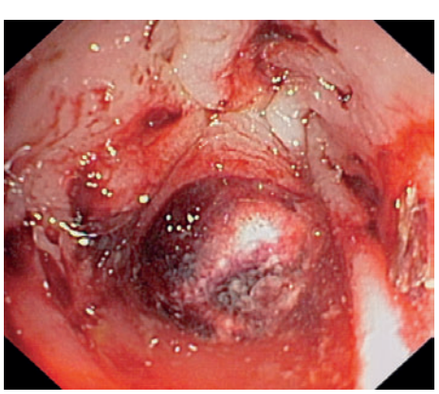


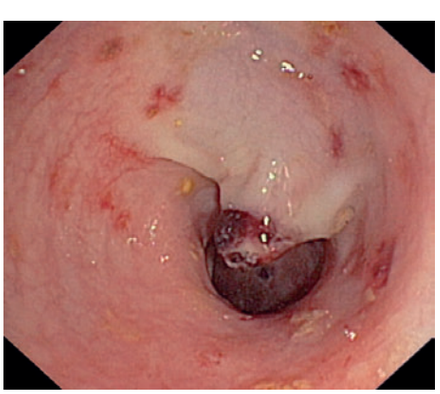
e

d Adherent clot on the resection site; rebleeding after polypectomy in the sigmoid colon.

e Visible vessel protruding from the remaining stalk of a resected polyp in the sigmoid colon.



a



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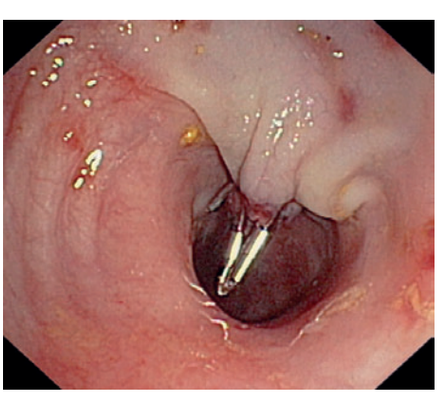


c

Fig. 13.**37** **Bleeding from a visible vessel of a resected polyp.**

a Visible vessel, protruding from the remaining stalk of a resected polyp in the sigmoid colon, causing massive rebleeding.

b Epinephrine injection (1:10 000) at the resection site.

c Afterward the visible vessel is clipped and closed with two hemoclips (Olympus).

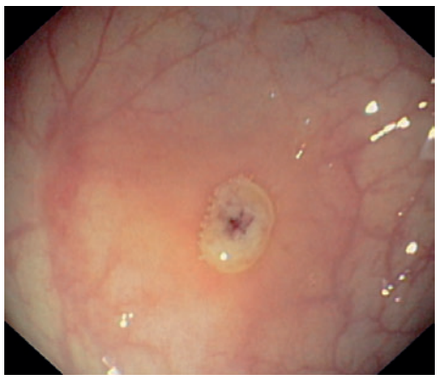


Fig. 13.**38** **Small visible vessel** at the site of an excisional biopsy of the colon mucosa.

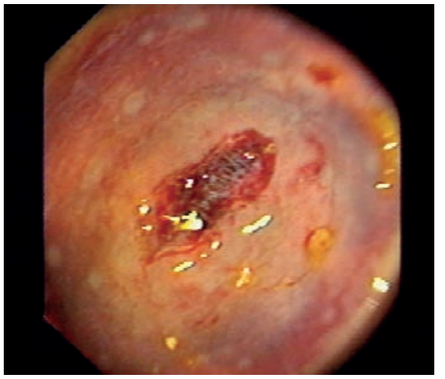


Fig. 13.**39** **Visible vessel** at the site of an excisional biopsy of the colon mucosa. The mucosa is slightly elevated around the biopsy site as a result of submucosal bleeding.

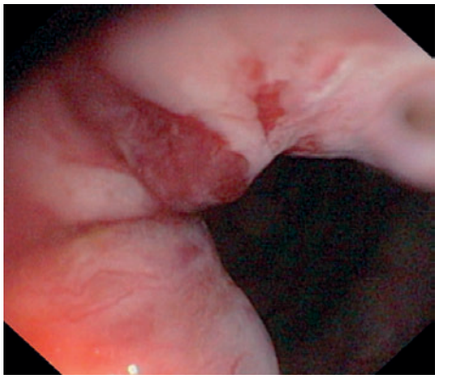


Fig. 13.**40** **Bloodsoaked anal fissure.**

Hemorrhoids. Hemorrhoids are the source in 2–9% of patients with acute lower gastrointestinal bleeding (overview in 64). In particular, among patients with HIV they seem to be a not uncommon cause of bleeding (12).

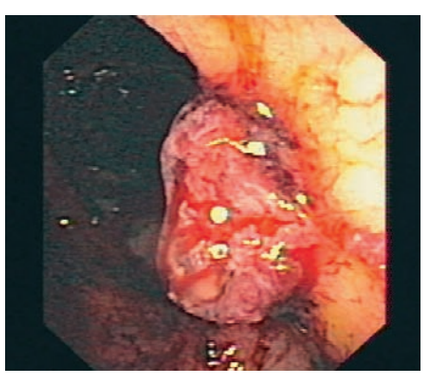
Endoscopic therapy

- Hemorrhoid management long belonged to the domain of surgical intervention. In recent years, however, endoscopic alternatives have established themselves in the treatment of symptomatic internal hemorrhoids and hemorrhoidal bleeding. Ligation of internal hemorrhoids has proved an especially effective and easy-to-learn method for treating internal hemorrhoid bleeding.
- Jensen (29) compared BICAP (bipolar coagulation) and heater probe therapies of bleeding internal hemorrhoids. Pain was more often reported with heater probe use, yet the success of therapy compared with BICAP was more evident and appeared more quickly.

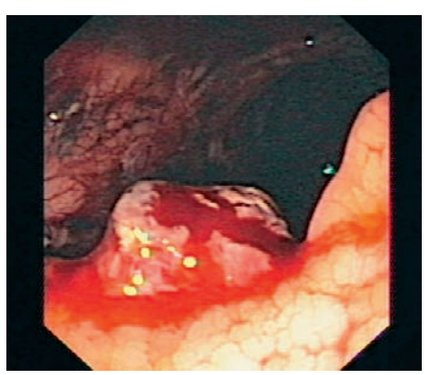
According to our own clinical files, acute hemorrhoidal bleeding occurs less frequently than bleeding following ligation of internal hemorrhoids (▣ 13.**8 a–c**). Rebleeding after hemorrhoid operation is also seen on occasion (▣ 13.**8 f**, **g**). Mechanical hemostasis using hemoclips has proved effective.

Anal fissures. Though anal fissures (Fig. 13.**40**) often cause bloody stools, acute bleeding is rare. Fissures are relatively easily diagnosed by inspecting the anus. The patient typically has severe pain upon spreading the anus, but the lesion can be carefully and painlessly inspected after injecting a few milliliters of local anesthesia. Bleeding from fissures usually ceases spontaneously. If bleeding is detectable at the time of examination, however, hemostasis can be attempted with injection of an epinephrine solution. Hemostasis can also be attempted with a swab soaked in epinephrine placed in the anus.

▣ **13.8 Rebleeding after ligation or surgical intervention for hemorrhoids**



a



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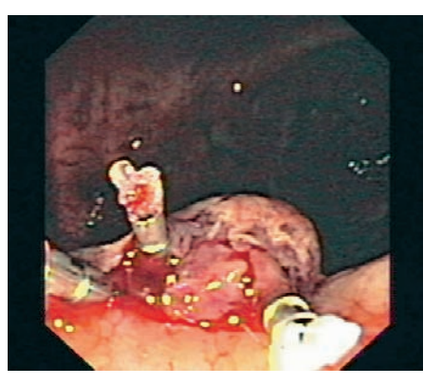
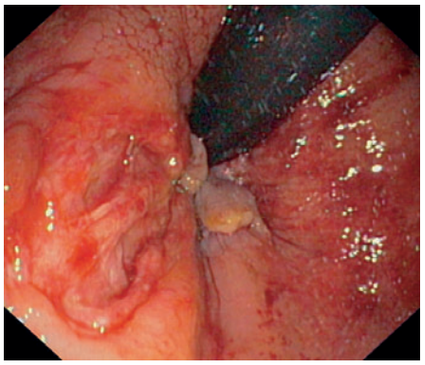


c

a–c Bleeding after ligation of an internal hemorrhoid. A bloodsoaked polypoid form is visible with a slight ring around the base where it was ligated (**a**). Following removal of the elevated tissue, hemorrhagic oozing ensued (**b**), and bleeding was stopped mechanically using three hemoclips (Olympus) (**c**).

13.8 cont.



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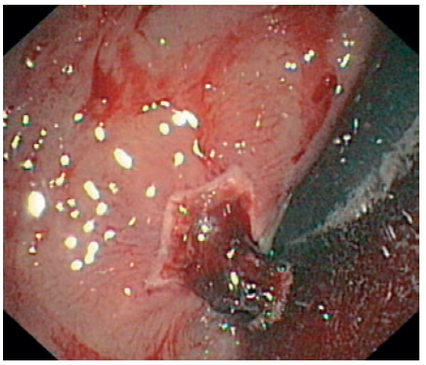
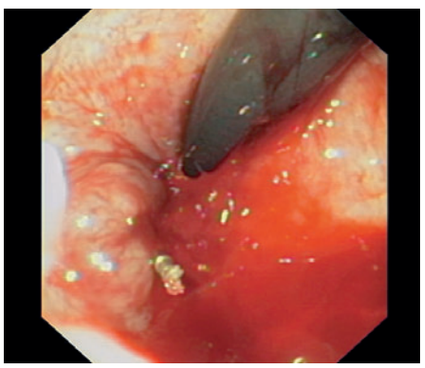


e

d Diffuse bleeding from an anorectal ulcer which formed after a ligated internal hemorrhoid fell off. The image was taken with a retroflexed colonoscope.

e Thick visible vessel protruding from the resection site (band ligation) of an internal hemorrhoid at the upper border of the anal canal. The ligated hemorrhoid fell off. The image was taken with a retroflexed instrument.



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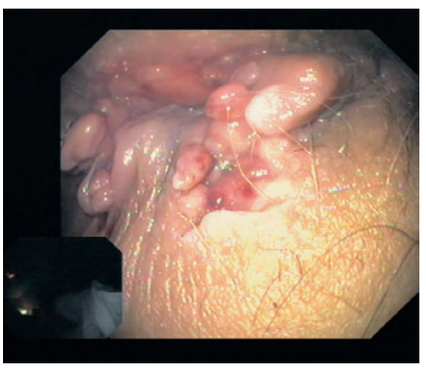


g

f Rebleeding from an operative wound four days after hemorrhoidectomy. A clip has already been applied, but cannot stem the flow of arterial bleeding. Hemostasis was not achieved until two further hemoclips (Olympus) were applied. The image was taken with a retroflexed colonoscope in the rectum—view from above to the upper edge of the anus.

g Ulcer with visible vessel on the outer margin of the anus, causing anal bleeding. A hemorrhoid operation was performed fourteen days prior. The patient was known to suffer from Crohn disease.

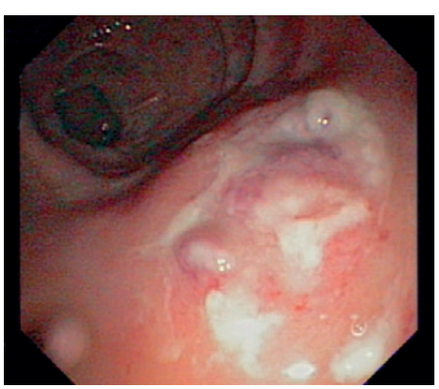


Fig. 13.**41** **Solitary rectal ulcer.**

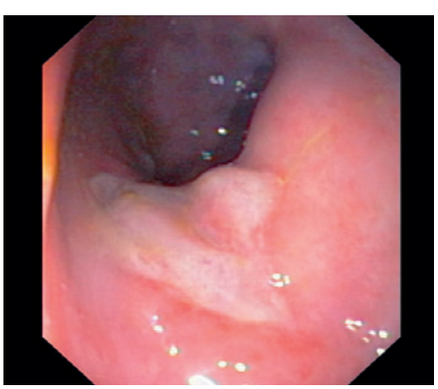


Fig. 13.**42** **Solitary rectal ulcer** in a patient who complained of low-grade anal rebleeding. He was previously treated with rectopexy to relieve severe rectal constipation.

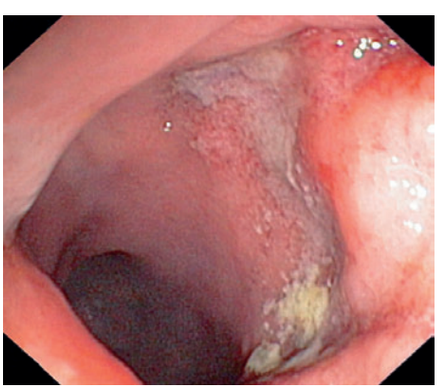
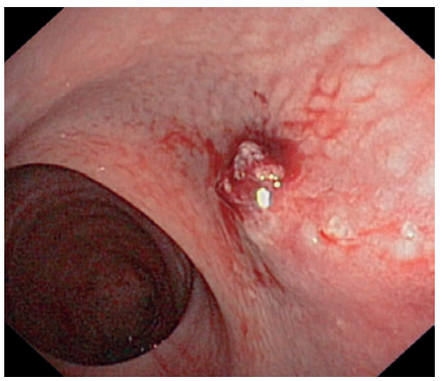


Fig. 13.**43** **Relatively large, rectal ulcer with fibrinous exudate** of uncertain genesis.

Solitary rectal ulcer. Local ischemia appears to play a role in the pathogenesis of solitary rectal ulcers (Figs. 13.**41**–13.**43**). Intussusception (internal rectal prolapse) causes excessive straining which, through repeated compression of the anorectal mucosa leads to necrosis and ulceration. The ulcer usually becomes symptomatic with pain and a layer of blood on the stool. Heavy bleeding is rare.



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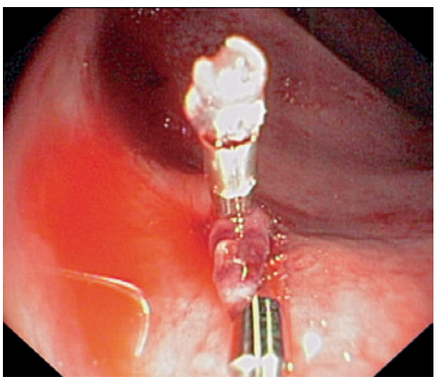

b

Fig. 13.**44** **Injury to the rectal wall and hematochezia.**
a Injury from a transrectal prostate biopsy.
b Bleeding source closed with two hemoclips (Olympus).

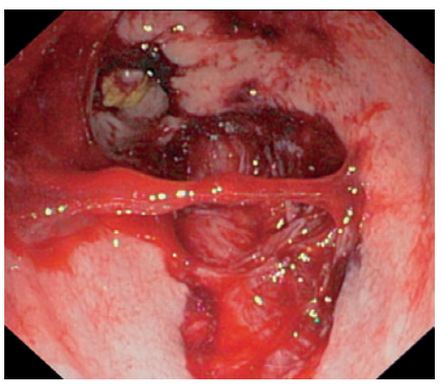

Fig. 13.**45** **Transmural laceration (perforation) of the rectal wall.** The lesion was caused by balloon inflation during a proctometrogram (to determine rectal sensation) as part of anal manometry. Injury to the wall manifested clinically with anal bleeding immediately after examination. The lesion healed with conservative therapy and without any further problems.

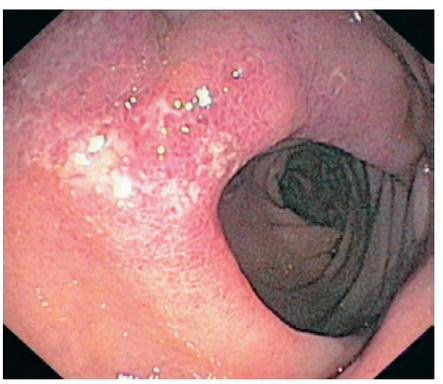

Fig. 13.**46** **Submucosal rectal wall bleeding from anticoagulation with Marcumar.** The patient presented with massive hematochezia. Prior to endoscopy, coagulation was normalized by administering clotting factors.

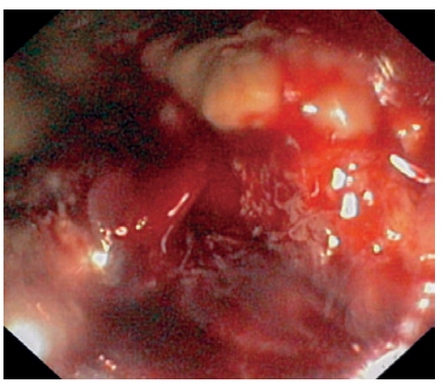

Fig. 13.**47** **Arterial bleeding from a rectal stump from a Hartmann procedure.** Bleeding occurred some time after operative intervention for carcinoma. The patient was treated preoperatively with neoadjuvant chemoradiotherapy. Bleeding could not be controlled using hemoclips and did not stop until after injection of acrylic glue.

Endoscopic therapy

- There are no comparative studies on optimal endoscopic therapy. Hemoclips can be applied or local injection of epinephrine solution may be used for circumscribed bleeding sources.
- Thermocoagulation (APC) can be attempted for diffuse bleeding.

Mechanical lesions of the rectal mucosa. Such lesions are not an entirely rare cause of acute lower gastrointestinal bleeding. Rectal manipulation on the part of the patient can cause quite serious damage to the mucosa. Injuries from thermometer use are less frequent nowadays as rectal temperature measurement is no longer common. Treatment principles are analogous to those for other circumscribed bleeding sources.

Rectal bleeding following diagnostic procedures such as transected prostate biopsy (Fig. 13.**44**) or functional diagnostic investigations (Fig. 13.**45**) is rare. Rectal wall hemorrhage due to anticoagulation drugs (Fig. 13.**46**) is uncommon. The treatment of choice is to correct clotting. Postoperative rectal bleeding (Fig. 13.**47**) is not particularly uncommon, especially if the mucosa has been damaged by prior radiation therapy.

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14 Vascular Malformations and Other Vascular Lesions

J. Barnert

Definitions

When describing vessel dilation in the gastrointestinal tract, the literature makes a distinction between angiodysplasia (telangiectasias) and hemangiomas. Angiodysplasia is defined as a dilation of superficial vessels while hemangiomas are defined as benign vascular tumors. However, the question as to whether the difference between the two is merely gradual and whether differentiation reflects differing pathogenesis remains unanswered.

Angiodysplasia (Telangiectasia)

Epidemiology and Pathogenesis

The pathogenesis of angiodysplasia is not entirely clear. In addition to hereditary forms, such as hereditary hemorrhagic telangiectasia (HHT), there also appear to be acquired forms, as suggested by the more frequent occurrence of angiodysplasias in the colon with increasing age. In a colonoscopic study, Richter et al. (29) found angiodysplasias in the large bowel in 1.4% of patients examined; Foutch et al. found them in 0.83% of patients (12).

Degeneration of blood vessels. Based on pathological studies, Boley et al. (6) hypothesized that angiodysplasias in older individuals are a result of degenerative processes of the veins in the colon wall. They suggest that dilation of submucosal veins is caused by hypertrophy of the longitudinal and transverse colon musculature. Hypertrophy constricts the points of entry of the veins in the muscle layer, obstructing blood flow. Obstruction would first cause dilation of the veins surrounding the crypts and later the submucosal veins, followed by dilation of the capillaries and incompetence of the precapillary sphincters, ultimately resulting in an arteriovenous short circuit. This pathogenesis would also explain why 60–90% of angiodysplasias are found in the right hemicolon (12, 29) where the wider lumen leads to greater wall tension (according to the Laplace law) and thus increased obstruction of venous outflow.

Ischemia. Another hypothesis suggests that angiectasias are caused by chronic ischemia. This almost certainly applies to radiation proctitis in which multiple telangiectasias are caused by mucosal ischemia.

In the pathogenesis of HHT, **genetic factors** are the most important determinant.

Histology

Early-stage angiectasias present histologically as dilated, tortuous submucosal veins. Later, marked clinical presentation includes dilated veins, venoles, and capillaries with a thin lining of endothelium and a small amount of smooth muscle in the vessel wall (Fig. 14.**1**). Pathological changes in HHT are identical to those observed in older individuals.

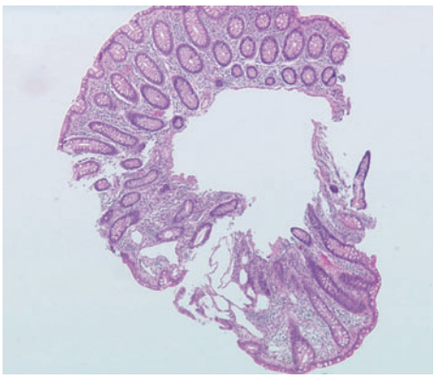


Fig. 14.1 **Microscopic features of angiodysplasia in the colon mucosa** with hollow areas lined with endothelium (courtesy of Dr. Th. Wagner, Institute for Pathology, Augsburg Clinic).

Clinical Course and Prognosis

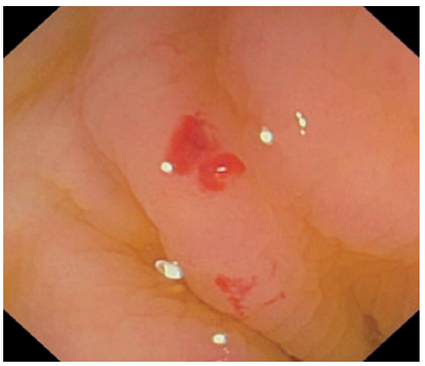
In older patients, angiodysplasia has a benign clinical course. Hemorrhage is uncommon (12, 29, 30), and an incidental finding during colonoscopy in a patient without a history of bleeding does not require therapy.

Telangiectasias related to HHT have a different prognosis and course. Around one-third of cases result in gastrointestinal bleeding which can be related to substantial morbidity (18, 35).

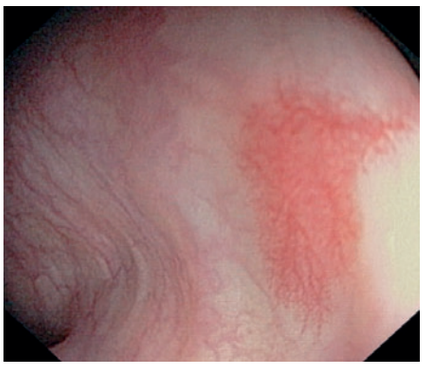
Diagnosis

Angiectasias appear endoscopically as red, circumscribed mucosal lesions with a diameter of one millimeter to a few centimeters (▣ 14.**1**, 14.**2**). About half of the lesions are smaller than 5 mm (31, 38). Small angiectasias usually present as red patches with either a round, circumscribed border or a slightly irregular margin and generally are not elevated. Larger lesions usually lie above the level of the mucosa (▣ 14.**2 d**) and have irregular borders (▣ 14.**1 b–d**, 14.**2 a**), resulting from communication with neighboring capillaries. Very large angiodysplasias have also been reported (24). Some angiodysplasias can resemble the spider naevi found in patients with liver cirrhosis (▣ 14.**1 f**). The number of angiodysplasias per patient can vary and the number of patients with solitary angiodysplasias is about equal to the number of those with multiple lesions (31, 38). One endoscopic study reported an average of 1.4 lesions in the colon per patient among patients with angiodysplasias (29).

14.1 Various appearances of angiodysplasias in the lower gastrointestinal tract

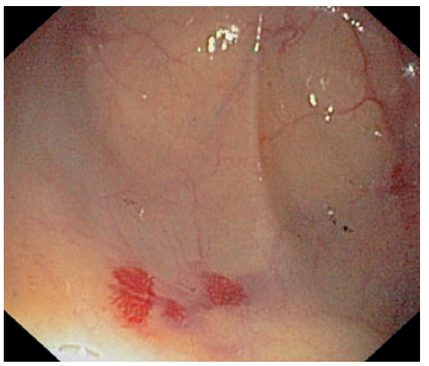


a

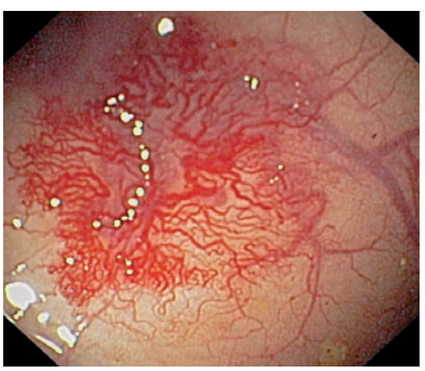


b

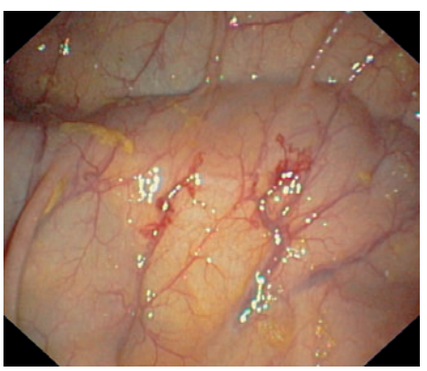
a, b Angiodysplasias in a 68-year-old man with multiple angiodysplasias in the colon and small bowel, but with no evidence of HHT. The patient was taking anticoagulants due to mitral valve endoprosthesis and had had several gastrointestinal bleeding episodes. Small angiodysplasia in the ascending colon (**a**). Large, patchy angiodysplasia also in the ascending colon (**b**).



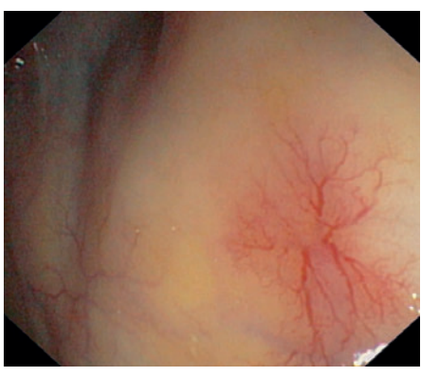
c Angiodysplasia in the ascending colon in an older man undergoing colonoscopy due to anemia.



d Patchy angiodysplasia in ascending colon. Dilated and branchlike capillaries as well as a dilated vein are readily visible.



e Small angiodysplasias in ascending colon that are clearly related to discretely dilated veins.



f Spidery angiodysplasia in ascending colon with dilated vein and two dilated capillaries that are fanning out peripherally like branches.

Older patients with angiodysplasias in the colon rarely have vascular malformations in the upper gastrointestinal tract. In HHT and among patients under 50 years of age, angiodysplasias are usually distributed throughout the entire gastrointestinal tract. Thus, examination of stomach and small intestine is essential.

Endoscopic therapy

- Angiodysplasias are treated with thermocoagulation (▣ 14.**2**). Injection of epinephrine or application of hemoclips may also be necessary if there is bleeding.

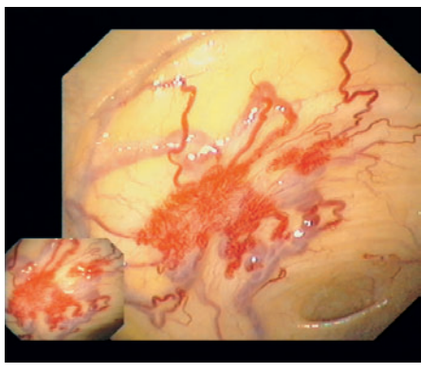
Telangiectasias in radiation proctitis. In 5–10% of patients, patients who have had radiation therapy in the pelvis develop chronic radiation injury to the rectal mucosa within 4–41 months following treatment. Endarteritis of the mucosal vessels causes both chronic mucosal ischemia and neovascularization (formation of telangiectasias).

Identification of telangiectasias in the rectum in radiation proctitis (Figs. 14.**2**–14.**4**) is generally straightforward given localization, appearance, and patient medical history. Endoscopic appearance is characterized by a pale and vulnerable mucosa that is covered by multiple telangiectasias often extending to the anal canal (Fig. 14.**5**).

Endoscopic therapy

- The therapy of choice for symptomatic patients with anemia and/or blood loss is thermocoagulation using APC (Fig. 14.**4**).

14.2 Thermocoagulation of angiodysplasias



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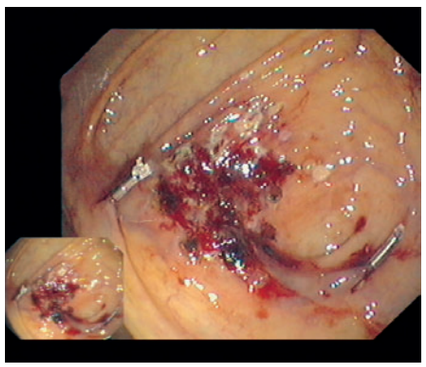

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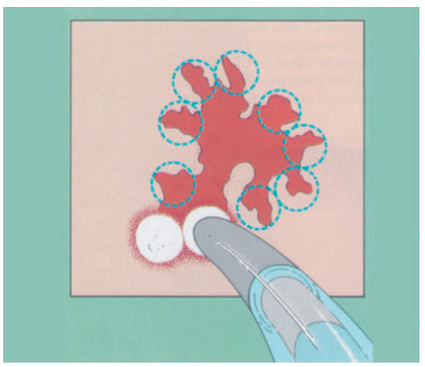
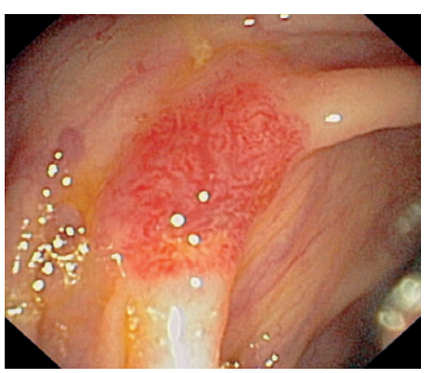


c

a, b Angiodysplasia in the cecum of an older woman who underwent endoscopy due to anemia. One can see dilated veins and a markedly red center formed by dilated capillaries that are branching out into the surrounding area (**a**). After ligation of the veins using hemoclips (Olympus) the angiodysplasia is coagulated point-by-point using APC (**b**).

c Illustration of thermocoagulation of an angiodysplasia (based on 11). Beginning at the periphery of the angiodysplasia, one then moves toward the center. It should be taken into account that bleeding caused by coagulation may obscure vision.



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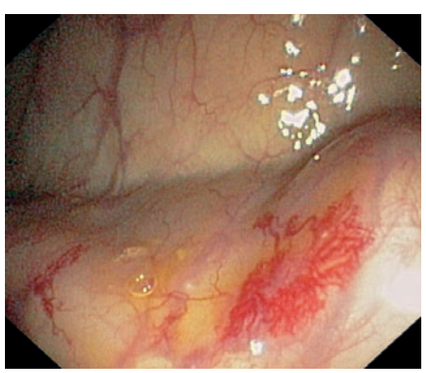

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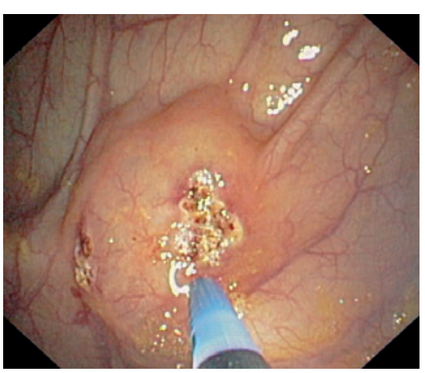

f

d–f Patients with chronic anemia and numerous angiodysplasias in the ascending colon. An angiodysplasia, ca. 6 mm in size and slightly elevated above the mucosal surface (**d**). An angiodysplasia in the ascending colon (**e**), coagulated using APC (point coagulation) (**f**).

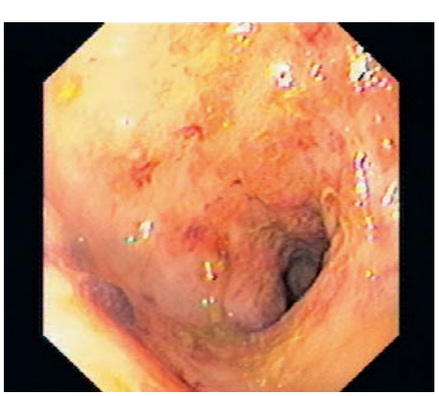

Fig. 14.2 **Rectum in a female patient with prior radiation therapy for an endometrial carcinoma.** One can see the pale mucosa with scattered angiodysplasias, a finding typical of late onset of radiation injury (radiation proctitis).

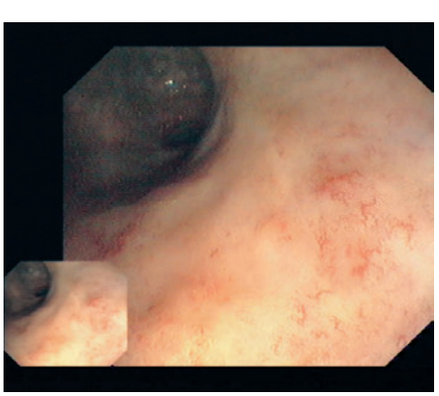

Fig. 14.3 **Rectal mucosa after prior radiation therapy of an endometrial carcinoma.** Tiny, newly forming vessels visible in the pale rectal mucosa.

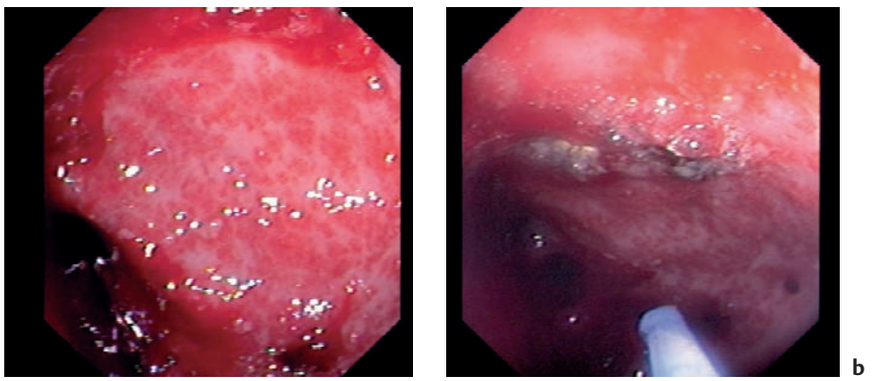

Fig. 14.4 **Rectum in a patient after neoadjuvant radiation of a rectal carcinoma.**
a Angiodysplasias almost completely covering the mucosa.
b Point coagulation (APC) is used to coagulate newly forming vessels. Only a portion of the angiodysplasias should be coagulated to avoid risk of deep ulcerations or stenosis caused by scarring.

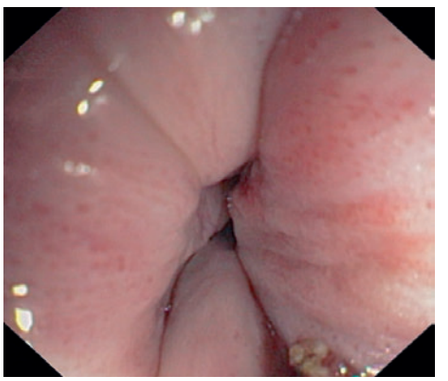

Fig. 14.5 **Anal canal in a patient with radiation-induced angiodysplasia** in the rectum. Newly forming vessels are also visible on the surface of the hemorrhoids.

Table 14.1 Criteria for clinical diagnosis of hereditary hemorrhagic telangiectasia (HHT)

Findings
▶ Epistaxis (spontaneous or recurrent) ▶ Telangiectasias on skin and mucosa: multiple, characteristic localizations (face, lips, oral cavities, and fingers) ▶ Visceral arteriovenous malformations (lung, brain, liver, spine) or gastrointestinal telangiectasias (with or without bleeding) ▶ Family history: immediate family member with HHT (according to above criteria)
Probability of diagnosis
▶ Definitive diagnosis: ≥3 criteria ▶ Probable diagnosis: 2 criteria ▶ Improbable: < 2 criteria

Differential Diagnosis

It is important to distinguish angiodysplasias from artifacts (i. e., mucosal bleeding) caused by the instrument (Fig. 14.**6**). This is often difficult, particularly with small, clearly bordered lesions. Localization can sometimes provide a clue: a circumscribed red patch in the sigmoid colon, for example, is more likely to be an artifact than a vascular malformation.

Biopsy of smaller lesions is only of further assistance in positive cases. Despite typical colonoscopic appearances, pathologists in two separate studies observed classic histological appearances (Fig. 14.**1**) in only half of the patients (16, 36). This could be explained either by pinching of the mucosa during biopsy, causing vessel collapse, or by compression of the vessels as a result of bleeding into the surrounding tissue, rendering diagnosis impossible. Endoscopic aspect thus remains the essential diagnostic criterion. Larger vascular malformations should not be biopsied, given the risk of bleeding.

Syndromes/Vascular Malformations in Other Diseases

Hereditary hemorrhagic telangiectasias. Angiodysplasias manifest in the colon in association with numerous syndromes, the most well known of which is hereditary hemorrhagic telangiectasia, also known as Osler–Weber–Rendu syndrome. HHT is a hereditary, autosomal dominant disease characterized by the formation of abnormal blood vessels. Clinical picture involves attack in various organs, primarily the brain, lungs, skin, nose, liver and gastrointestinal tract. Two clear genetic defects related to HHT have been identified.

Unlike usual angiectasias, HHT affects younger people as well, with involvement of the entire gastrointestinal tract. Diagnosis should be made based on Curaçao criteria (Tab. 14.**1**). Gastrointestinal bleeding occurs in one-third of patients with HHT (18) and patients over age 60 are especially at risk. The most common localization of HHT telangiectasias is the stomach and proximal small bowel; the colon is less often affected (22).

Lesions can become symptomatic as a result of acute lower gastrointestinal bleeding or due to anemia from chronic bleeding. Endoscopic intervention promises little long-term success (32), perhaps in part because the lesions in the small bowel are difficult to reach endoscopically if at all.

Portal colopathy. Angiodysplasias can also be detected in the colon (analogous to the stomach) in patients with portal hypertension (Figs. 14.**7**, 14.**8**). Prevalence reported in the literature ranges between 12–89 % (5, 9, 13, 14, 19, 34, 37). Risk of bleeding from these angiodysplasias appears to be low (between 0–9 %) (9, 13). However, hemorrhaging can become problematic in rare cases, and can only be controlled by means of a transjugular intrahepatic portosystemic shunt (TIPPSs) (4).

Patients with portal hypertension often also have dilated veins (varices), primarily in the rectum (Fig. 14.**9**). Prevalence related to portal hypertension reported in the literature ranges from 8–89 % (8–10, 13–15, 34, 37, 40). Frequent detection of

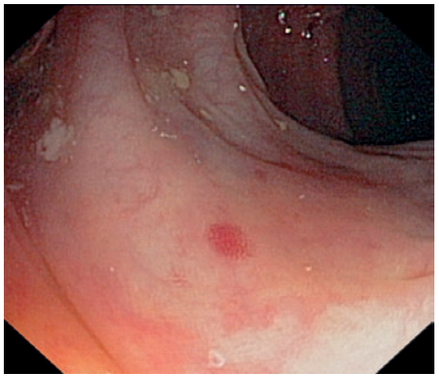

Fig. 14.6 **Artifact in sigmoid colon: submucosal bleeding.** Red, textured pattern which can be clearly distinguished from angiodysplasia.

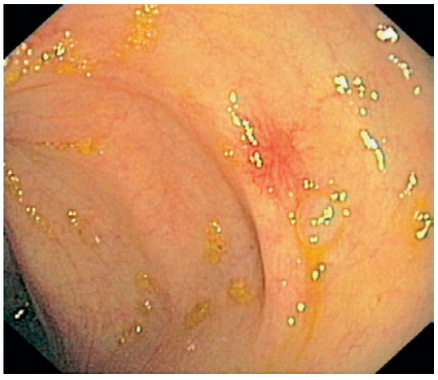

Fig. 14.7 **Angiodysplasia in ascending colon** in a patient with portal hypertension due to liver cirrhosis in chronic hepatitis. The angiodysplasia looks similar to a spider nevus.

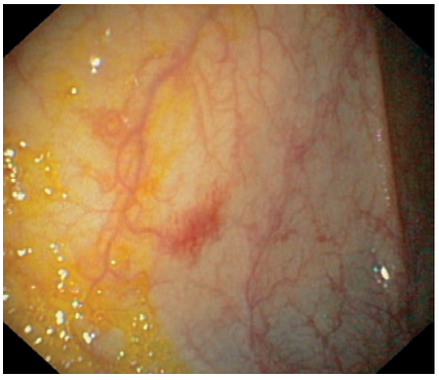

Fig. 14.8 **Small angiodysplasia in the right hemicolon** in a patient with portal hypertension due to liver cirrhosis.

colonic angiodysplasias and varices in patients with portal hypertension has led to use of the term "portal colopathy."

Other diseases. Increased frequency of angiectasias is also postulated in other diseases such as von Willebrand disease, aortic stenosis (Heyde syndrome), and terminal renal insufficiency, although data are sometimes inconsistent.

Hemangiomas

Epidemiology

Hemangiomas are a much less common finding in the colon than angiodysplasias. Hemangiomas are benign dysontogenetic vascular neoplasms, with an incidence of 1:1400 according to one study based on autopsy reports (1).

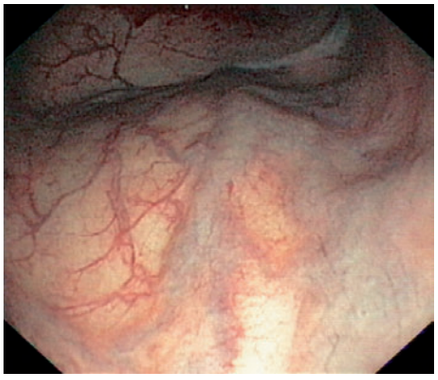

Fig. 14.9 **Rectal varices** in a patient with portal hypertension due to liver cirrhosis. This was an incidental finding.

Histology

Histologically, hemangiomas are comprised of wide, bloodfilled endothelium-lined sinuses, separated by varying amounts of connective tissue. Hemangiomas occur in capillary, cavernous, and mixed forms. Hemangiomas in the gastrointestinal tract are mainly the cavernous type. Histology shows large, bloodfilled, endothelium-lined cavities separated from each other by thin connective tissue septa.

Kaposi sarcoma (KS) occurs as a multicentric vascular proliferation. Histology reveals small, endothelium-lined vessels, between which are spindle cells; erythrocytes can be detected in small tissue cracks (not lined with endothelium). Kaposi sarcoma is primarily found in patients with AIDS.

Course and Prognosis

Hemangiomas have a tendency to bleed and patients become symptomatic through hematochezia or chronic anemia. Therapeutic potential for large and infiltrative hemangiomas is limited, though endoscopic removal can be attempted with smaller lesions. Children are very often affected by bleeding. Complications related to Kaposi sarcoma include abdominal pain, gastrointestinal bleeding, and intestinal obstruction.

Diagnosis

Hemangiomas have a predilection for the rectum or sigmoid colon and more often occur as single lesions than multiple. A distinction is made between polypoid and infiltrative types.

- *Polypoid cavernous hemangiomas* present as single or multiple broad-based, sessile, deep red, or livid lesions with a strawberry appearance (Figs. 14.**10**, 14.**11**). They measure 5–10 mm in diameter and are markedly elevated.
- *Infiltrative cavernous hemangiomas* are found primarily in the rectum, appearing as blue–red or black undefined masses, bulging into the lumen. A radiograph of the pelvis typically reveals phleboliths, which are characteristic.

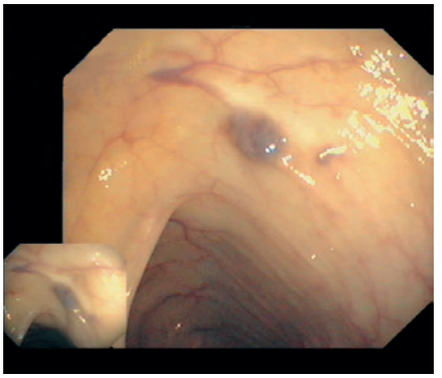

Fig. 14.**10** **Two small hemangiomas in the transverse colon.**

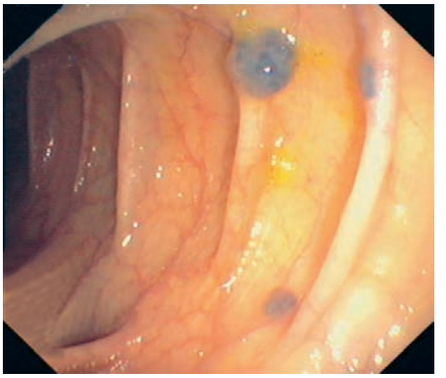

Fig. 14.**11** **Three hemangiomas in the transverse colon** near the hepatic flexure.

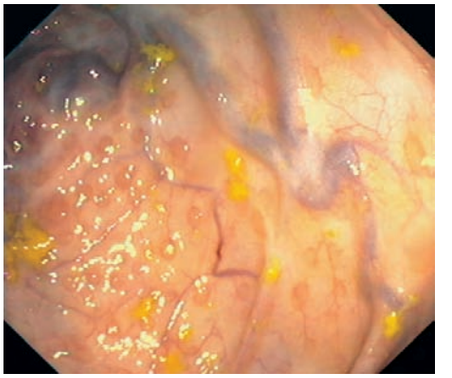

Fig. 14.**12** **Vascular ectasia (phlebectasia) in the ascending colon** in a patient with Turner syndrome and recurrent hematochezia. Incidental finding: multiple small, glassy lymphoid hyperplasias on the colon mucosa.

- *Multiple phlebectasia,* dilated mucosal vessels are another type of hemangioma (Fig. 14.**12**).
- *Kaposi sarcoma* is found in patients with AIDS with roughly the same prevalence in the upper gastrointestinal tract and colon. KS presents either similarly to angiodysplasia with red foci with a slightly elevated center and starlike branches or as a small red polyp, often ulcerated in the center.

Syndromes/Vascular Malformations in Other Diseases

Hemangiomas of the gastrointestinal tract are often associated with those in other parts of the body.

Blue rubber bleb nevus syndrome. BRBNS is an autosomal dominant hereditary disease.

Small mucosal nodules with a blue–red sac, not unlike a rubber nipple, present in the rectum and left hemicolon. Flat, livid mucosal changes have also been reported (overview in 27). Lesions occur in multiple numbers, affecting the upper gastrointestinal tract, peritoneum, liver, spleen, oropharynx, eyes, lungs, heart, CNS, skeletomuscular system, bones, and urogenital system. Gastrointestinal lesions tend to bleed and gastrointestinal hemorrhage is the leading cause of death among affected individuals.

Endoscopic therapy

- Therapy depends on the size and extent of the lesions. Resection of the affected bowel segments and endoscopic intervention using thermocoagulation have been reported.
- After removing the lesions, new ones may develop elsewhere.

Maffucci syndrome. In Maffucci syndrome, blue rubber bleb nevus syndrome is associated with cerebral medulloblastomas, chronic lymphoid leukemia, hypernephromas, pulmonary hypertension, thrombocytopenia, and consumption coagulopathy. Joint involvement, bone malformation and pathological fractures have also been reported (overview in 27).

Klippel–Trenaunay–Weber syndrome. Gastrointestinal hemangiomas are also seen in association with Klippel–Trenaunay–Weber syndrome (2, 3, 17, 21, 25, 41). Even among children, it can already be a source of massive gastrointestinal hemorrhage. Klippel–Trenaunay–Weber syndrome is characterized by a triad of symptoms: asymmetric limb hypertrophy, varicose veins, and nevus flammeus of the skin.

Turner syndrome. Turner syndrome is caused by X-chromosome monosomy or mosaicism (X0/XX). The result is gonadal dysgenesis with hypergonadotropic hypogonadism. Patients also exhibit pterygium colli, lymphedema, dwarfism, a flat, "shieldshaped" chest, and facultative abnormalities such as congenital cardiac defects. Hemangiomas (Fig. 14.**13**) and phlebectasia (Fig. 14.**14**) of the colon (7, 20, 23, 26, 28, 33, 39) have also been reported in patients with Turner syndrome. There are no standardized recommendations for therapy in the literature. Treatment must be determined on an individual patient basis (Figs. 14.**13**, 14.**14**).

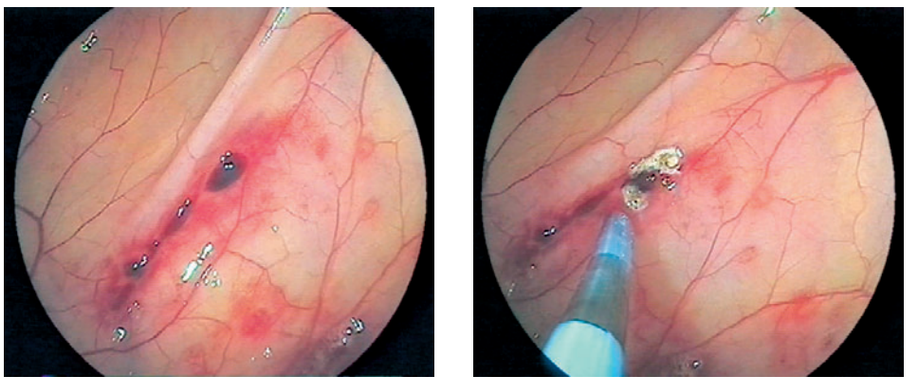

Fig. 14.**13** **Hemangiomas in Turner syndrome.**
a Several hemangiomas in the ascending colon mucosa in a patient with Turner syndrome (same patient as in Fig. 14.**12**).
b Obliteration of the hemangioma using APC. Coagulation of the adjacent area first (due to potential risk of bleeding) and then the hemangioma itself.

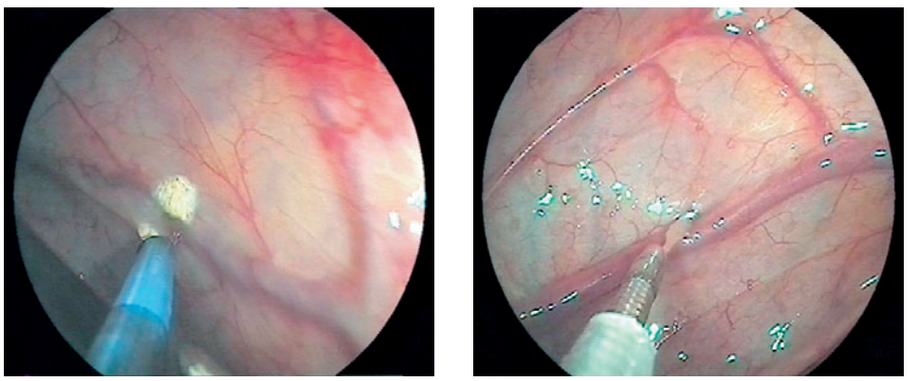

Fig. 14.**14** **Vascular ectasia in the ascending colon in a patient with Turner syndrome** (same patient as in Figs. 14.**12**, 14.**13**).
a Obliteration of the vein adjacent to the vascular ectasia using APC.
b In addition to sclerosis with APC, the veins are also closed mechanically using hemoclips (Olympus). This diminished vascular ectasia and caused complete cessation of lower intestinal rebleeding.

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15 Melanosis Coli

R. Fleischmann

Definition

Melanosis coli presents as dark brown pigmentation of the mucosa that may have a zebra-striped or reticulated appearance. The pigment, which is actually not melanin, but rather lipofuscin, is deposited extracellularly in macrophages. Because the pigment is not melanin, this phenomenon is also correctly called pseudomelanosis coli.

Clinical Picture

Melanosis coli is most often found in women with surreptitious heavy use of laxatives and who are seeking medical attention for chronic diarrhea. Low levels of potassium are often evident. The condition begins to reverse 12–18 months after laxative use is discontinued.

Diagnosis

Sigmoidoscopy reveals dark pigmentation of the mucosa (Figs. 15.**1**, 15.**2**). Changes are most pronounced in the right hemicolon and cecum.

Endoscopic diagnostic criteria

- Dark pigmentation of the mucosa, from light brown to black, sometimes with a leopard-skin pattern.
- In advanced cases, the colon is dilated with loss of haustra.
- Tiny polyps and inflammatory mucosa often remain light against an otherwise dark background (Fig. 15.**2**). These should be biopsied.

Additional procedures. A colon contrast enema does not increase diagnostic yield.

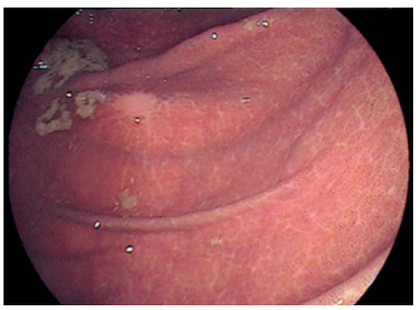

Fig. 15.**2** **Pseudomelanosis coli.** Mosaiclike brown mucosal coloration. Tiny polyps usually remain lighter in color.

Differential diagnosis. Melanosis coli is most often observed following use of anthracene (senna) laxatives, which are seldom prescribed nowadays. Phenolphthalein laxative use can be easily detected by using sodium hydroxide (alkalization causes the stool to turn red). Use of magnesium-containing laxatives can be determined by high magnesium stool content. Anthracene can also be detected in urine.

Therapy

Treating laxative abuse is difficult as most patients deny misuse. Misuse of laxatives is often related to eating disorders, and both behaviors require psychotherapy.

Surveillance

Surveillance is not necessary, as melanosis coli has no malignant potential.

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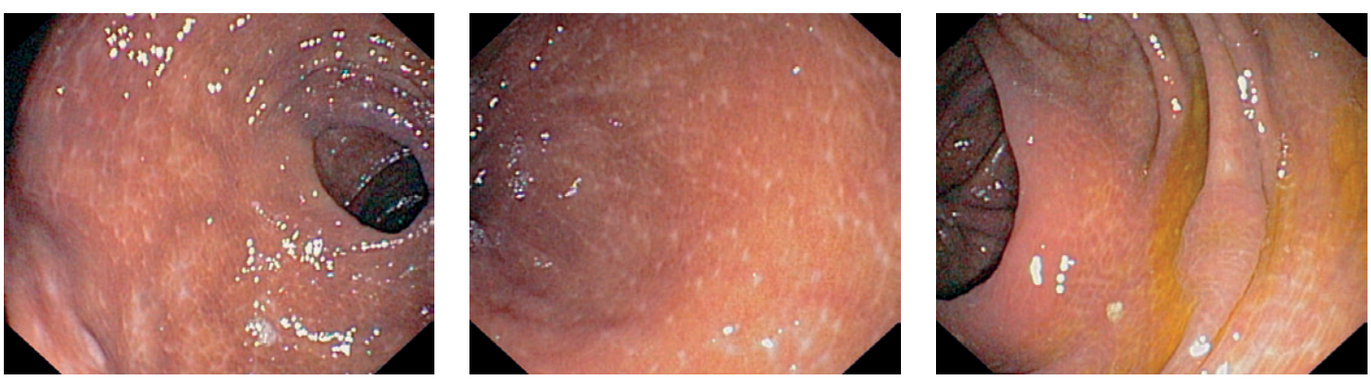

a **b** **c**

Fig. 15.**1 a–c** **Pseudomelanosis coli.** Brown, reticulated mucosal pattern (leopard-skin pattern).

16 Solitary Rectal Ulcer Syndrome

R. Fleischmann

Definition

Solitary rectal ulcer syndrome describes a solitary ulcer surrounded by normal appearing rectal mucosa.

Clinical Picture

Patients present with tenesmus and a feeling of anal obstruction. Blood and mucus appear in stool.

Diagnosis

Using either a rigid rectoscope or a flexible sigmoidoscope the rectum should be inspected up to the rectosigmoid junction. An ulcer 0.5–2.0 cm in size can usually be found between 6 and 13 cm from the anus on the anterior rectal wall, surrounded by normal appearing mucosa.

Endoscopic diagnostic criteria

- flat ulcer with smooth border, usually covered with fibrinous exudate (Figs. 16.**1**, 16.**2**).

Endoscopic examination procedure

- perianal inspection,
- digital examination,
- rectoscopy/sigmoidoscopy,
- smear from the base of the ulcer,
- biopsy.

Causes. Solitary rectal ulcers have various causes, but they are most often caused by rectal or anal prolapse. Full-thickness rectal prolapse—protrusion of the full thickness of the rectal wall through the anal ring—is always an indication for surgical intervention.

Anal prolapse is an eversion of hyperplastic anal corpus cavernosum into the anal lumen. Unlike rectal prolapse, the mucosa has radial folds and a starshaped appearance (Fig. 16.**3**). The stages in anal prolapse are:

- partial,
- semicircular, and
- circular.

It is not always possible to detect mucosal prolapse. Nonetheless, the presumed cause of a rectal ulcer is often a protrusion of the anterior rectal wall into the anal canal due to excessive pressure. This in turn leads to local mucosal ischemia and ultimately ulcer formation. Severe constipation and chronic excessive straining during bowel evacuation are the most common causes of rectal ulcers.

Occasionally a solitary rectal ulcer is caused by self-performed digital manipulation of the ampulla to relieve chronic constipation (Fig. 16.**2**). Solitary rectal ulcers are seldom caused by medication (i. e., ergotamine or NSAID suppositories).

Histology. Histology reveals a nonspecific inflammatory change with smooth muscle components extending to the lamina propria of the mucosa. Differential diagnosis should first exclude carcinoma or a Crohn-related ulcer (Tab. 16.**1**).

Treatment

Asymptomatic patients do not require treatment. Symptomatic patients should be advised to avoid excessive straining during bowel evacuation. Measures for softening the stool are recommended (e.g., drinking more fluids, avoiding black tea, Indian Plantago, etc.).

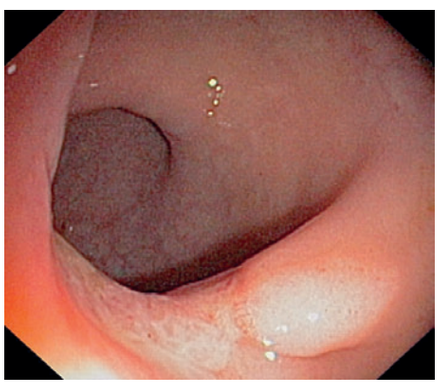

Fig. 16.**1** **Flat rectal ulcer** at 5 cm above the anus in mucosal prolapse.

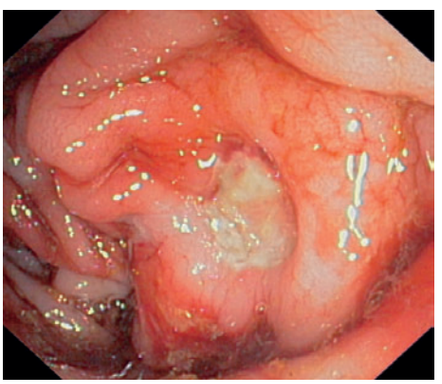

Fig. 16.**2** **Moderately deep rectal ulcer covered with fibrinous exudate:** mucosal prolapse and digital manipulation (surrounding mucosa is edematous).

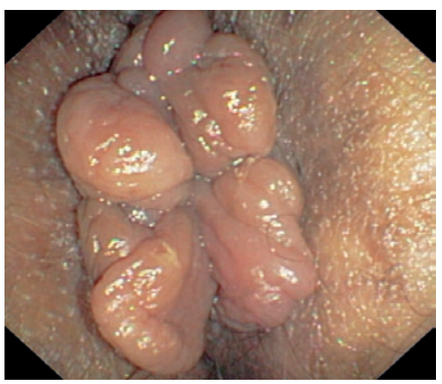

Fig. 16.**3** **Anal prolapse (complete).**

Table 16.**1** Differential diagnosis of chronic solitary rectal ulcers

Diagnosis	Features
Chronic solitary rectal ulcer	Anterior rectal wall 6–13 cm from the anus Possible rectal or anal prolapse
Radiation ulcer (delayed radiation proctitis)	Three months to several years after exposure
Crohn disease	Seldom solitary
Rectal carcinoma	Usually elevated
Lymphoma	Histology for differential diagnosis
NSAID ulcer	Localization in rectum with suppository use
Ischemic colitis	Mid third of rectum to splenic flexure Obstruction of inferior mesenteric artery
Stercoral ulcer	In patients with severe constipation, often bedridden perforation risk!
Prior polypectomy	Ulcer appears up to four weeks after polypectomy
Inguinal lymphogranuloma (Durand–Nicolas–Favre disease)	Tropical STD (viral) Narrowing of rectal lumen Anal swelling
Syphilis, primary syphilitic sore	Three weeks after infection can be localized in anal or rectal area, dark-field microscopic examination of exudate, serology
Gonorrhea	Acute proctitis with numerous ulcers and discharge Gram stain
Herpes	Areas dotted with very small ulcers in the rectum ranging from tiny perianal blisters to ulcers PCR from smear and biopsy
Chancroid ulcer (soft chancre)	1–2 cm large painful ulcer with ragged, undermined borers Smear for detection of *Hemophilus ducreyi*

In severe cases of rectal prolapse with rectocele (detected by means of defecography) transabdominal rectopexy with or without resection is indicated. Resection is especially indicated for complete rectal prolapse.

Less severe cases of anal prolapse can be treated with band ligation or sclerotherapy. The most successful procedure appears to be transanal staple operation (Longo procedure).

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17 Rare Diseases and Disorders

R. Fleischmann

Extrinsic Compression (Stenosis)

Extrinsic narrowing of the colon lumen can be caused by retroperitoneal tumors (kidneys, adrenal glands, pancreas, ovaries, uterine and cervical carcinoma or lymphoma) or by retroperitoneal abscesses (Fig. 17.**1**). Though peritoneal carcinoma often causes adhesions—which especially make endoscope passage in the sigmoid difficult because of lacking compressibility of the abdomen ("splinting technique")—it does not cause narrowing of actual lumen.

A small-caliber therapeutic colonoscope is usually necessary to achieve total colonoscopy if there is stenosis of the bowel. Additional use of a colon contrast enema or virtual computed tomography (CT) imaging of the colon is rarely necessary for examination.

Postoperative Strictures and Suture Granulomas

Postoperative strictures. Postoperative strictures are commonly observed in the anastomosed region of the lower rectum after rectal resection. They are occasionally the result of dehiscence after resection of very low rectal carcinomas. Therapy with balloon dilation can help dilate the stricture. Dilation should be performed step-by-step in intervals of three to four days (balloon diameter: 15–25 mm). Intestinal resection in Crohn disease can also lead to inflammatory stenosis or scarring (Fig. 17.**2**), the latter of which is endoscopically treatable with balloon dilation, if a shorter bowel segment is affected.

Suture granulomas. The surface of the often-granulated mucosa is smooth so that it is unlikely that a suture granuloma will be mistaken for a sessile polyp or early carcinoma, even based on visible appearances alone. If there is any doubt, a biopsy should be performed. Sometimes suture remnants are visible (Fig. 17.**3**).

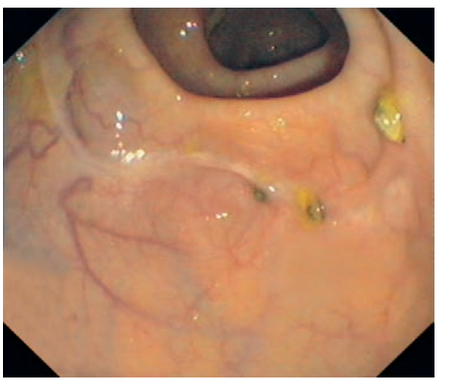


Fig. 17.**3** **Suture granulomas.** Three dark suture granulomas at the anastomosis after sigmoid resection (not clinically relevant).

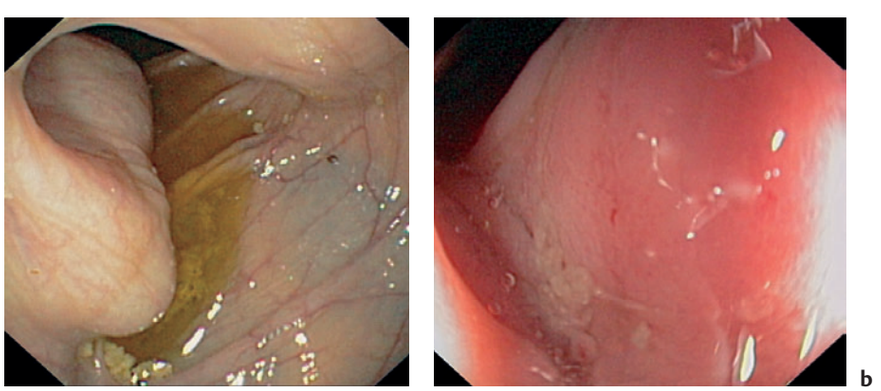
Lumen Dilation and Pseudo-obstruction

Definition

Megacolon. The term megacolon describes both congenital and acquired forms of colon dilation. In all younger patients with megacolon, the possibility of Hirschsprung disease should be excluded. Hirschsprung disease manifests in the first years of life caused by absence of parasympathetic ganglion cells in myenteric (Auerbach) and submucosal (Meissner) plexuses in the rectum. The absence of ganglion cells results in constipation and bowel obstruction.

Clinical Picture

Congenital megacolon (Hirschsprung disease). Congenital megacolon manifests from birth as persistent constipation. If the aganglionic intestinal segment is short, it is usually possible to empty the bowel using enemas and laxatives. If the affected seg-



a b

Fig. 17.**1** **Extrinsic compression (stenosis).**
a Compression in ascending colon from retroperitoneal lymph nodes.
b Compression in ascending colon due to kidney tumor; mucosa is reddened.

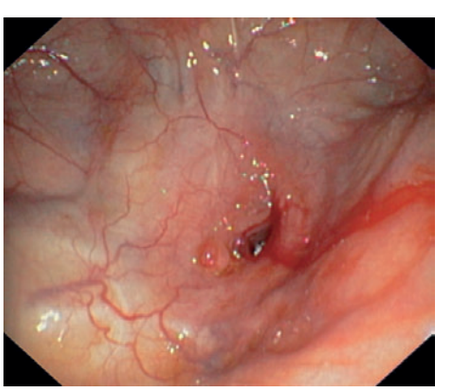


Fig. 17.**2** **Postoperative stricture.** Extreme narrowing of ileocolonic anastomosis in Crohn ileitis.

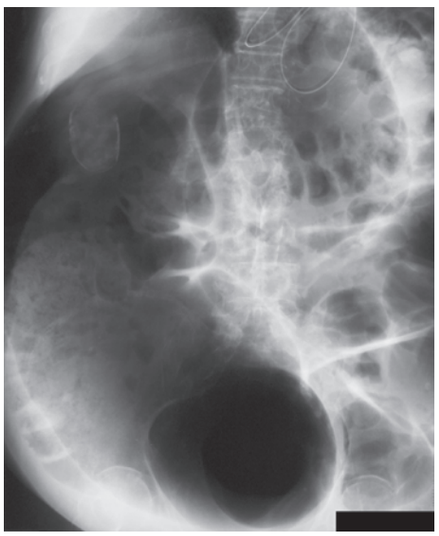


Fig. 17.**4** **Dilation of colon: pseudo-obstruction.** Radiograph image shows considerable colon distention, especially in the ascending colon, and spare or lacking haustration. Increased vulnerability of the mucosa and risk of perforation (courtesy of Dr. Volker Remplik, Institute for Radiology, Augsburg Clinic).

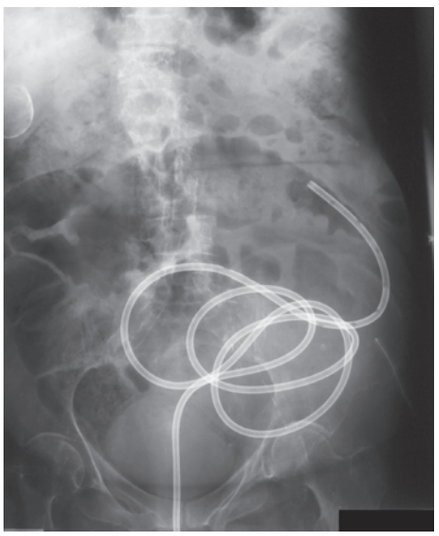


Fig. 17.**5** **Decompression of colon distention by means of a decompression tube.** Very large gallstone presenting as a rounded calcification at the upper left edge of the image (courtesy of Dr. Volker Remplik, Institute for Radiology, Augsburg Clinic).

ment is longer, there is complete blockage of the bowels and the infant will die without early surgical intervention.

Acquired megacolon (secondary). Acquired (secondary) megacolon develops at any age as a result of stenosis in the rectum or sigmoid caused by a tumor, scarring, or spasms related to chronic anal fissures.

Functional megacolon (actual pseudo-obstruction). In functional megacolon (no anatomical cause), the colon is dilated up to the rectum. Unlike in Hirschsprung disease the rectum is filled with stool. Functional megacolon can be constitutional and may be familial. Distention usually affects the entire sigmoid colon, though it can also involve the whole colon.

Diagnosis

The colon is distended and elongated. Stagnation of bowel contents can lead to secondary colitis.

Endoscopic diagnostic criteria

- In Hirschsprung disease, the rectum is empty and narrowed from increased muscle tone in the rectal wall.
- A full thickness rectal biopsy is performed with special dye techniques for detecting the absence of ganglion cells in the submucosal plexus. The frozen biopsy is sent to a special laboratory for evaluation (staining technique) of acetylcholinesterase (AChE) activity, which is increased in Hirschsprung disease. Using the safer method of suction biopsy, the mucosa can be biopsied and the specimen evaluated using enzyme histochemical staining to determine (only) cholinesterase activity.
- Pseudo-obstruction appears as a dilated colon/rectum rather than as a narrow rectal segment. Clinical picture can be acutely worsened by volvulus (an ischemic and/or edematous dark-red narrowed segment in the sigmoid colon).

Additional examinations. An abdominal radiograph can demonstrate the exact extent of bowel distention in the acute phase as well as decompression after placement of a decompression tube (Figs. 17.**4**, 17.**5**).

In Hirschsprung disease, manometry can be used to detect defective relaxation of the internal rectal sphincter. Disease can be eliminated by resection of the aganglionic rectal segment.

Radiographic contrast studies are essential in all forms of megacolon for further treatment planning.

Differential diagnosis. Hormonal and medication-induced causes must be excluded. These include: hypothyroidism, acromegaly, diseases of the brain and spinal cord, morphine abuse, vitamin B_1 deficiency, acute potassium deficiency, and anticholinergic use.

Treatment

In Hirschsprung disease, resection of the aganglionic rectal segment is unavoidable. Other forms of megacolon ("slow transit") are treated conservatively.

If there is massive dilation with lacking intestinal tone and distention of the transverse diameter exceeding six centimeters, placement of a decompression tube (e.g., Cook, Wilson–Cook Medical Inc., Winston–Salem, NC, USA) using a long guidewire inserted through the endoscope (4.80 m long, 0.035 inches wide) is necessary. Decompression occurs immediately (Fig. 17.**5**). After carefully building up food intake, beginning with tea and plain toasted bread, after a few days the patient may have foods higher in fiber along with ample amounts of water. In some cases, high fiber bulk laxatives (Indian Plantago, etc.) must be prescribed. Laxatives should only be used in severe cases. Osmotic laxatives (magnesium sulfate, lactulose, lactitol, macrogol) have limited side effects. Macrogol (polyethylene glycol) does not cause gas and thus does not increase bowel distention.

If the patient has obstructive constipation (e.g., rectocele) surgical intervention is the only option.

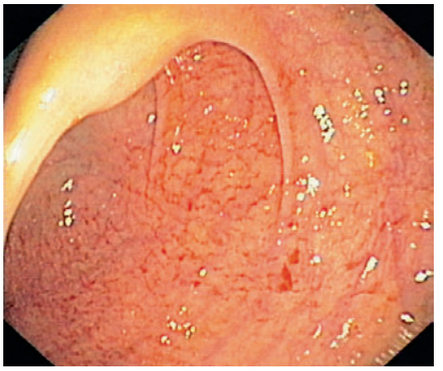


Fig. 17.**6** **Segmental hemorrhagic colitis.** Forty-year-old patient with highly acute lower abdominal pain (rightsided) five days after taking amoxicillin. Mucosal hemorrhaging in ascending colon.

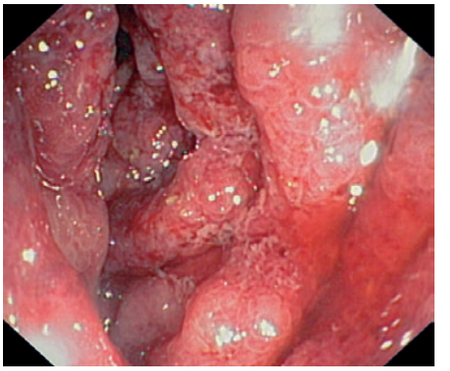


Fig. 17.**7** **CMV colitis.** Severe segmental swelling and bloodsoaked mucosa at the hepatic flexure following polychemotherapy in a patient with B-cell lymphoma. Often also ulcerous.

Surveillance

Follow-up surveillance should be performed every 4–6 months to monitor the continued effectiveness of treatment and, if necessary, treatment should be adjusted. Attention should be paid to regularity of bowel movements.

Acute Segmental Hemorrhagic Colitis

Definition

Acute segmental hemorrhagic colitis presents clinically with acute lower and middle abdominal pain (usually rightsided) and hematochezia.

Clinical Picture

Onset is sudden and occurs about four to five days after oral use of penicillin or amoxicillin. Typical signs include spontaneous pain and considerable local tenderness in the right abdomen. Lab tests show increased levels of CRP (C-reactive protein) and leukocytes. Fever is not present or is only mild; diarrhea is not typical.

Diagnosis

Sonography reveals lacking layering of the intestinal wall in the affected segment of the thickened ascending colon. The thickened colon wall is asymmetrical, unlike in pseudomembranous colitis, where the wall is symmetrical.

Colonoscopy shows typical hemorrhagic mucosal changes in the ascending colon (Fig. 17.**6**). The rest of the colon is totally unremarkable with normal capillarization of the mucosa. Even macroscopically, it is possible to differentiate acute segmental hemorrhagic colitis from Crohn disease or ulcerative colitis.

Differential diagnoses. Differential diagnosis should exclude *Yersinia* enterocolitis, *Campylobacter jejuni*, EHEC (Enterohemorrhagic escherichia coli) or *Klebsiella*, all of which are accompanied by pronounced and sometimes watery diarrhea. In immunosuppressed patients cytomegalovirus (CMV) colitis (Fig. 17.**7**) can be excluded with biopsy (CMV PCR); the chief symptom of CMV colitis is hematochezia.

Antibiotic-associated colitis can be ruled out based on a negative *Clostridium* test and endoscopic biopsy.

Rare causes, such as amyloidosis and ischemic colitis, can be excluded using endoscopy and histological evaluation. NSAID (nonsteroidal anti-inflammatory drug) colitis usually presents with ulcers in the ileocecal region. Symptoms usually include hematochezia, but seldom pain or signs of inflammation.

Treatment

Clinical symptoms improve rapidly after discontinuing antibiotic use.

Surveillance

Endoscopic surveillance is not necessary. Reduced inflammation can be observed using sonography.

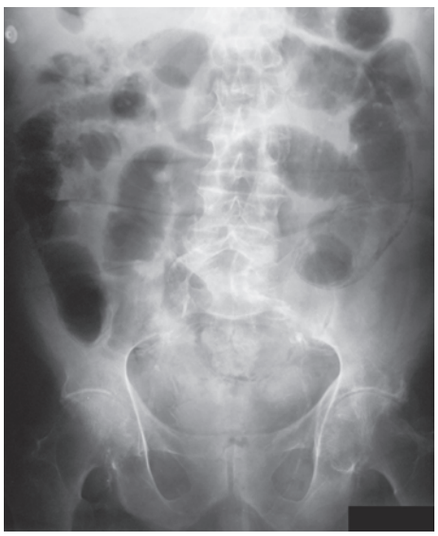
Pneumatosis Cystoides Intestinalis

Definition

In this rare disease, multiple gasfilled cysts ranging in size from the head of a pin to as large as a cherry form in the wall of the small bowel or colon. Cysts occur in clusters, mainly in the submucosa or subserosa, but occasionally also in the retroperitoneum. The gas is either air or it may contain nitrogen. Pneumatosis cystoides intestinalis is a rare phenomenon that has been observed in numerous gastrointestinal diseases (with and without obstruction of the lumen), for example, in Crohn disease, but also in obstructive diseases of the airways such as pulmonary fibrosis.

Clinical Picture

The patient has either uncharacteristic abdominal pain or, occasionally, acute pneumoperitoneum due to cyst rupture. Most patients complain of severe constipation and less frequently of colitislike symptoms. Three-fourths of patients have other abdominal diseases or disorders (ulcers, carcinomas, stenosis, abdominal tuberculosis, etc.). Pneumatosis cystoides intestinalis has also been found in obstructive diseases of the airways. In some cases, there is no causal explanation, however.



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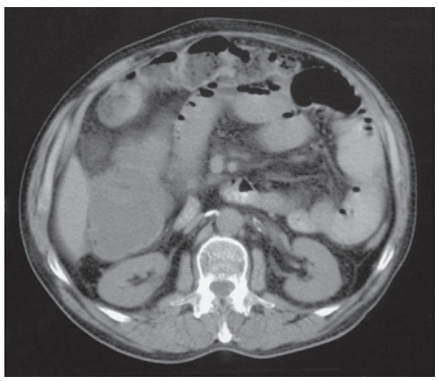

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Fig. 17.**8** **Pneumatosis cystoides.** Radiograph may show a pearl-shaped or lamellar double contour (image provided courtesy of Dr.Volker Remplik, Institute for Radiology, Augsburg Clinic).

a Abdominal radiograph: double contour of colon wall in ascending and descending colon in intestinal ischemia resulting from an occlusive carcinoid tumor in the sigmoid colon.

b Abdominal CT: a row of gas bubbles lined up like a string of pearls in transverse colon and descending colon in necrotizing enteritis caused by an occlusive tumor.

Diagnosis

Submucosal pneumatosis cystoides intestinalis presents in colonoscopy as clusters of flat, domeshaped bubbles on a smooth mucosa.

Endoscopic diagnostic criteria (submucosal pneumatosis cystoides intestinalis)

- uniform, domeshaped bubbles 3–5 mm in size are typical,
- no signs of mucosal inflammation.

Additional examinations. Diagnosis is assisted by abdominal radiograph and CT (Fig. 17.**8**). Radiography is the only possible means of diagnosis for both pneumatosis cystoides intestinalis in the subserosa and acute pneumoperitoneum. Both are usually found in association with intestinal malignancies or ischemic changes.

Differential diagnosis. Familial adenomatous polyposis (FAP) can present with multiple, uniform small polyps in the distal colon and rectum. The surface structure is not smooth, but instead has a "pit pattern" structure, which enables FAP to be differentiated from pneumatosis cystoides intestinalis. If necessary, zoom endoscopy can be of assistance.

Treatment

Treatment is generally not necessary. Oxygen therapy, using an oxygen mask to administer 80% oxygen for 30 minutes per day for 14 days promotes more rapid cyst resolution, as the gas usually contains nitrogen. Metronidazole can also be helpful. Concomitant disorders, such as bowel obstruction or intestinal ischemia, must be treated operatively.

Surveillance

Surveillance is not necessary, but is advised for ischemic causes (nonobstructive intestinal ischemia).

Kaposi Sarcoma

Definition

Kaposi sarcoma (KS) is a neoplastic proliferation of endothelium-like cells containing modified smooth muscle cells, which occurs in four forms:

- classic KS,
- African endemic KS,
- KS in patients using immunosuppressants,
- KS in patients with HIV.

Kaposi sarcoma related to HIV occurs primarily among homosexual men. KS is localized in the skin, mucous membranes, lungs, lymph nodes, gastrointestinal tract, and liver.

Diagnosis

Diagnosis of KS in the gastrointestinal tract can only be made with the assistance of endoscopy and biopsy.

Endoscopic diagnostic criteria

- KS presents endoscopically as multiple flat, red–brown lesions. Occasionally these resemble capillary hemangiomas in which case it is impossible to distinguish between the two macroscopically. Lesions are 3–15 mm in size and may be ulcerated and bleeding (Fig. 17.**9**).
- Persistent bleeding after biopsy.
- Stages:
  - Stage I = solitary, limited,
  - Stage II = disseminated (more than 10 foci).

Endoscopic examination procedure

- Colonoscopy and EGD are indicated.
- If there is occult bleeding, small bowel examination is necessary using "push enteroscopy" and capsule endoscopy.

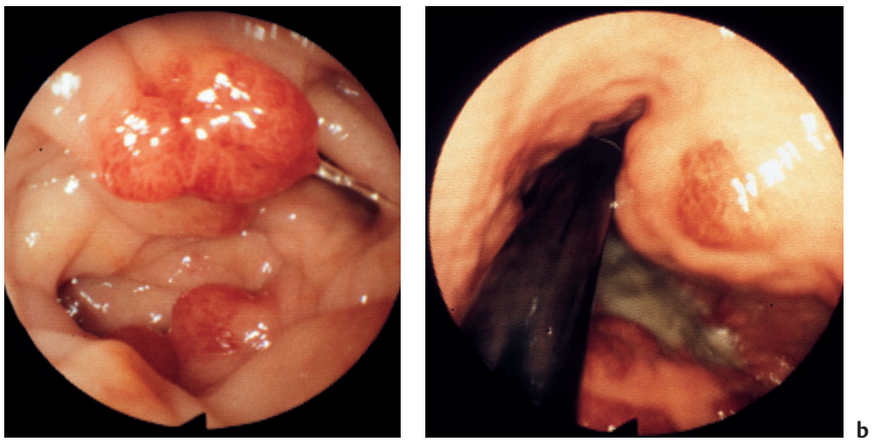


Fig. 17.**9** **Kaposi sarcoma** (provided courtesy of Dr. Walter Heise, Herzberge Evangelical Hospital, Berlin).
a An ulcerating Kaposi sarcoma in the rectum.
b Kaposi sarcoma causing stenosis in the sigmoid colon.

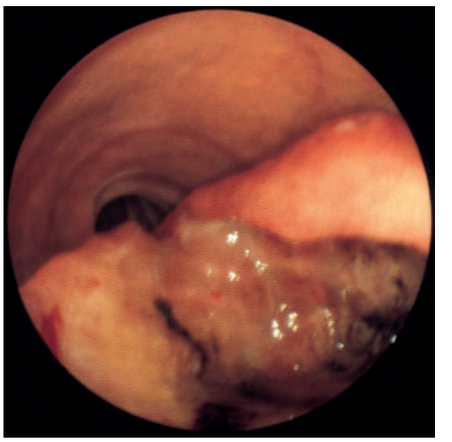


Fig. 17.**10** **AIDS-related lymphoma.** Large, ulcerating, highly malignant B-cell rectal lymphoma (provided courtesy of Dr. Walter Heise, Herzberge Evangelical Hospital, Berlin).

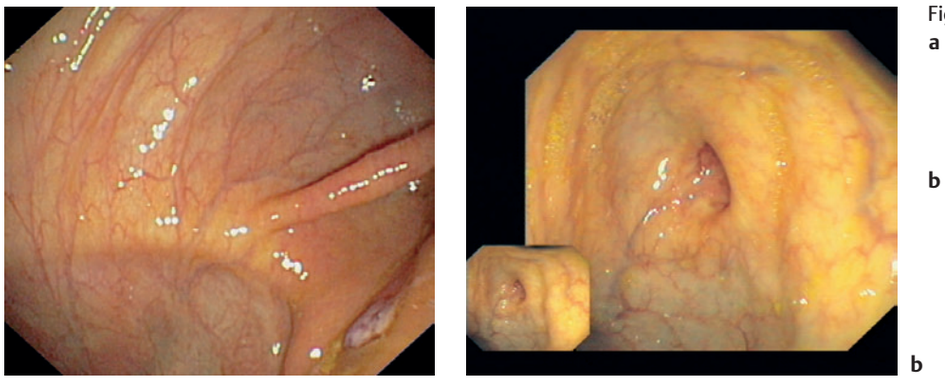


Fig. 17.**11** **Appendix.**
a Acute appendicitis, clear inflammatory swelling of appendiceal orifice (at lower right edge of image) distinguishing it from Crohn ileitis (diagnosis confirmed histologically following appendectomy).
b Normal appendiceal orifice, with smooth, excavated form.

Additional examinations. Diagnosis is confirmed by biopsy. Histology reveals an angiosarcoma with proliferation of thin-walled vessels.

Treatment

Treatment of hemorrhage consists of argon beam coagulation or laser therapy.

This disease is now uncommon since the introduction of HAART therapy for HIV-infected individuals.

Surveillance

Surveillance sigmoidoscopy is recommended at three to four weeks following HAART therapy (intensified HIV treatment).

HIV-Related Lymphoma

Colorectal lymphoma can also (rarely) occur in individuals with HIV (Fig. 17.**10**).

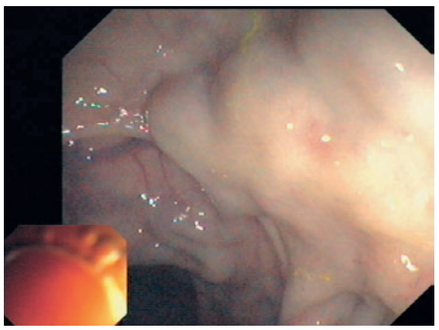
Ileitis

The ileum can be affected by Crohn disease or by a number of other cancerous and infectious diseases (Tab. 17.**1**). Differential diagnosis from acute appendicitis can also be difficult. Alongside sonography and CT, colonoscopy can often successfully confirm appendicitis (Fig. 17.**11**). Colonoscopic aspect includes redness and swelling of the appendix opening; the normally smooth excavation is lost.

Fistulizing cecal carcinomas are easily mistaken macroscopically for Crohn disease and can spread to the terminal ileum. All ileocecal conglomerate tumors must be histologically evaluated and classified. Resection is desirable in the absence of certain histology and signs of stenosis.

Colon Varices and Portal Colopathy

Sharp increases in portal hypertension related to liver cirrhosis or portal vein thrombosis can cause formation of varices throughout the entire colon (Fig. 17.**12**). Lymphedema is not present. Bleeding is acute and can be controlled with fibrin glue and hemoclips. Long-term prophylactic measures such as lowering



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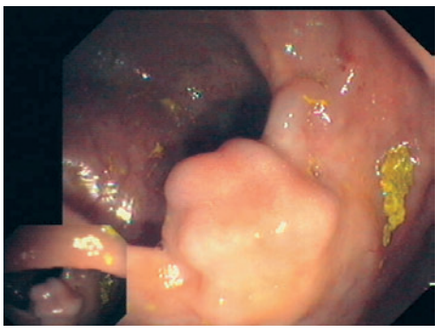

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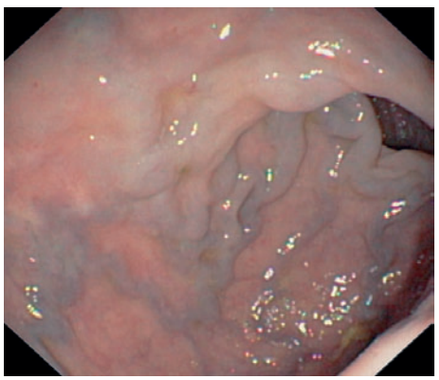
Fig. 17.**12** **Portal colopathy.**
a Thick strands of varices throughout the entire colon.
b Varix on the Bauhin valve in the same patient.



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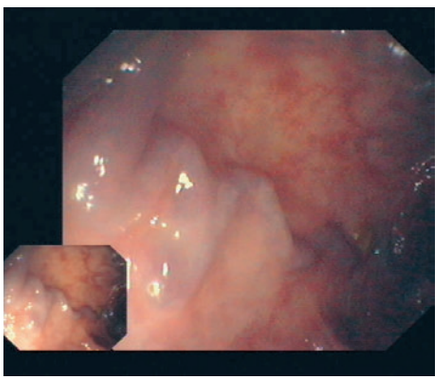

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Fig. 17.**13 a, b** **Rectal varices.** Dilated submucosal veins in older patients.

portal hypertension, best achieved using beta-blockers, should be mentioned. The success of shunt placement or, for example, TIPPS, needs to be evaluated.

Rectal Varices

Dilated submucosal veins in the rectum are frequently found among older patients (Fig. 17.**13**) and may occur without portal hypertension. The presence of rectal varices sometimes leads to discovery of cryptogenic cirrhosis. In rare cases, submucosal venous thrombosis can occur following polypectomy (Fig. 17.**14**).

Iatrogenic Lesions

These days perforations and ulcerations should no longer result from poor examination technique. Radiation proctitis can be difficult to treat if mucosal changes cause rebleeding or chronic ulcerations. Radiation-induced angiectasias can be successfully treated by repeated plasma sessions with argon coagulation. Strictures affecting shorter bowel segments can be dilated using balloon dilation. Ulcers resulting from polypectomy persist for two to three weeks (Fig. 17.**15**).

Lipomas and Carcinoid Tumors

Lipomas. Lipomas are benign submucosal tumors. Yellowish in color with a smooth, polypoid bulging surface, they are most frequently found on the Bauhin valve. Unlike with adenomas, microscopic inspection with zoom endoscopy does not reveal a

Table 17.**1** Differential diagnoses: ileitis

Tumors	**Features**
Carcinoid tumor (70 % in ileocecal region)	▶ Diarrhea is a complication ▶ Bleeding and obstruction are uncommon ▶ Increased 5-HIAA in urine
Lymphosarcoma	▶ Obstruction
Lymphoma	▶ MALT lymphoma ▶ Burkitt lymphoma (Africa)
Leiomyoma	▶ Endoscopic removal possible up to 2 cm (> 2 cm often malignant) ▶ Obstruction ▶ Bleeding
Adenoma	▶ Obstruction ▶ Bleeding
Lipoma	▶ Incidental finding
Hemangioma	▶ Obstruction
Cecal carcinoma with fistulas	▶ Often initially mistaken for Crohn disease
Inflammation	**Features**
Lymphoid follicles of the terminal ileum	▶ More common among young children ▶ Yersinias, adenoviruses, measles, toxoplasmosis, Tbc, etc.
Appendicitis	▶ Highly acute with fever
NSAID colitis	▶ Melena ▶ Hematochezia
Actinomycosis	▶ Fistulas
Radiation ileitis	▶ Occult bleeding ▶ Diarrhea
Sarcoidosis	▶ Usually involves additional organs

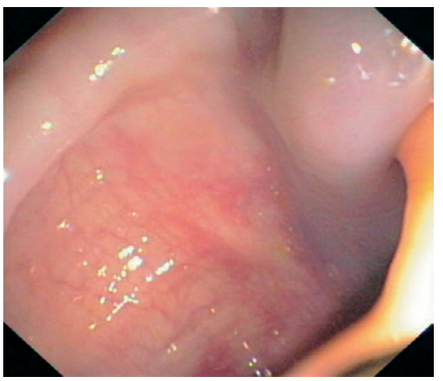


Fig. 17.**14** **Venous thrombosis (submucosa)** following polypectomy (not clinically relevant).

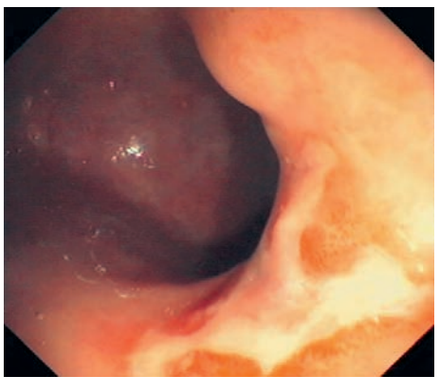


Fig. 17.**15** **Iatrogenic lesion.** Polygonal ulcer after rectal mucosectomy.

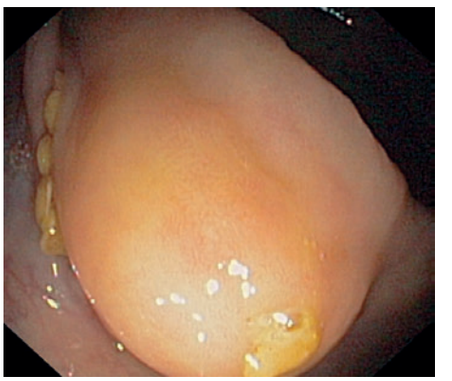
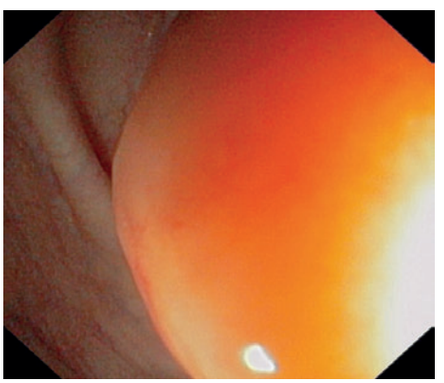


Fig. 17.**16** **Lipomatous valve.** Polyplike form on the Bauhin valve with smooth surface and yellowish color.



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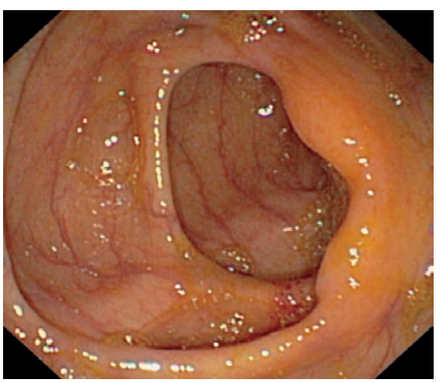

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Fig. 17.**17** **Carcinoid tumor in the cecum.**
a Domeshaped growth behind the Bauhin valve, confirmed with biopsy as a highly differentiated carcinoid tumor.
b A carcinoid tumor behind the Bauhin valve. The tumor, which is located in the cecum cannot be identified from this instrument position.

"pit pattern." For the experienced examiner, certain diagnosis of a lipoma is possible based on endoscopic appearances alone. Removal is not necessary. Risk of bleeding or perforation from snare biopsy is low, but nonetheless avoidable (Fig. 17.**16**).

Carcinoid tumors. Up to 70 % of carcinoid tumors are located in the ileum and cecum. Occasionally they occur in the rectum and, in principle, they can present anywhere in the gastrointestinal tract.

Diagnosis. Given that carcinoid tumors and leiomyomas may resemble lipomas macroscopically (submucosal localization) and are only distinguishable by color (red–brown), if there is doubt, the lipoma should be biopsied using a deep forceps biopsy, as shown in Fig. 17.**17**. Lipomas are incidental findings; leiomyomas and carcinoid tumors are often detected when investigating chronic anemia, hematochezia, or melena.

Endometriosis

Endometriosis is caused by metaplasia of endometrial tissue. In only 3 % of patients does it occur in the intestine (small intestine, appendix, colon, rectum), most often in the rectosigmoid. Endometriosis is frequently in the subserosa and is rarely submucosal. If localized in the rectum, there may be narrowing of the lumen with normal mucosa or there may be a polypoid tumor. In extremely rare cases, endometriosis may penetrate the bowel wall (intramural) to the mucosa, causing menstruation-related mucosal bleeding. In these cases, a bluish submucosal tumor can be seen shimmering through the mucosa. Diagnosis is usually made, however, by a gynecologist during laparotomy.

Amyloidosis

Amyloidosis does not cause any visible changes to the colonic mucosa. Diagnosis is possible with histological evaluation using a deep biopsy, in which the rectal submucosa is also sampled. Macroscopic changes in the small intestine can be seen in some patients as swelling of the valvulae coniventes or circumscribed tumorlike amyloid deposits.

Collagenous Colitis and Microscopic Colitis

In microscopic colitis, inflammatory changes to the colonic mucosa can be only detected microscopically (thus the mucosa must be routinely biopsied in patients presenting with chronic diarrhea). Collagenous colitis occasionally presents with discrete hyperemia and obscured vessel pattern. Microscopically, the normally 3 µm wide collagen layer in the submucosa swells up to 100 µm. Histological changes are more pronounced in the proximal colon than in the rectosigmoid region.

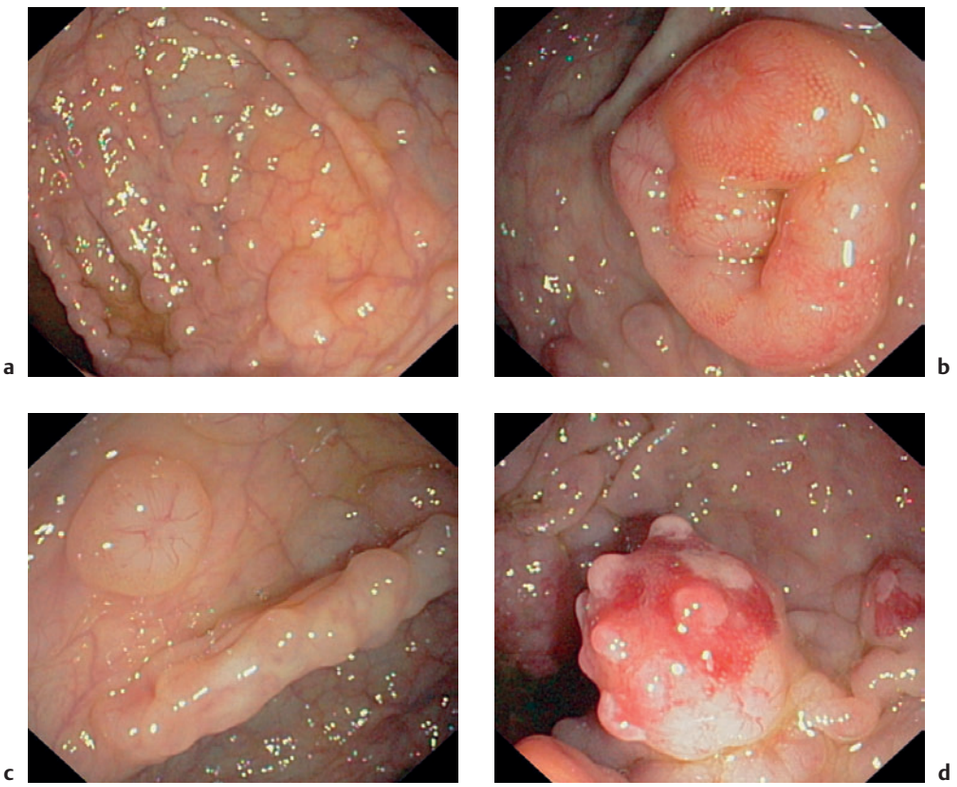

Fig. 17.**18** **Mantle cell lymphomas.**
a Multiple flat lymphoma infiltrates in the ascending colon.
b Lymphatic infiltrates, forming a ring-shaped swelling around the appendiceal orifice.
c Large, pale polyp with starlike vascular pattern.
d Stalked lymphatic polyp with a villous surface in the rectum (histologically confirmed lymphoma).

Colon Involvement in Non-Hodgkin Lymphoma

Gastrointestinal tract involvement in non-Hodgkin lymphoma (NHL) varies greatly and is rather rare. Involvement of the gastrointestinal tract in mantle cell lymphoma, by way of contrast, may be 100%, according to some American authors. Lymphoma infiltrates in the colon are often very numerous leading to confusion with FAP. The surface of lymphoid polyps is, however, often very smooth, sometimes demonstrating a typical starlike vessel pattern. The mostly broad-based polypoid structures are paler than the unaffected colon mucosa. Larger polyps can present as stalked villous adenomas (Fig. 17.**18**).

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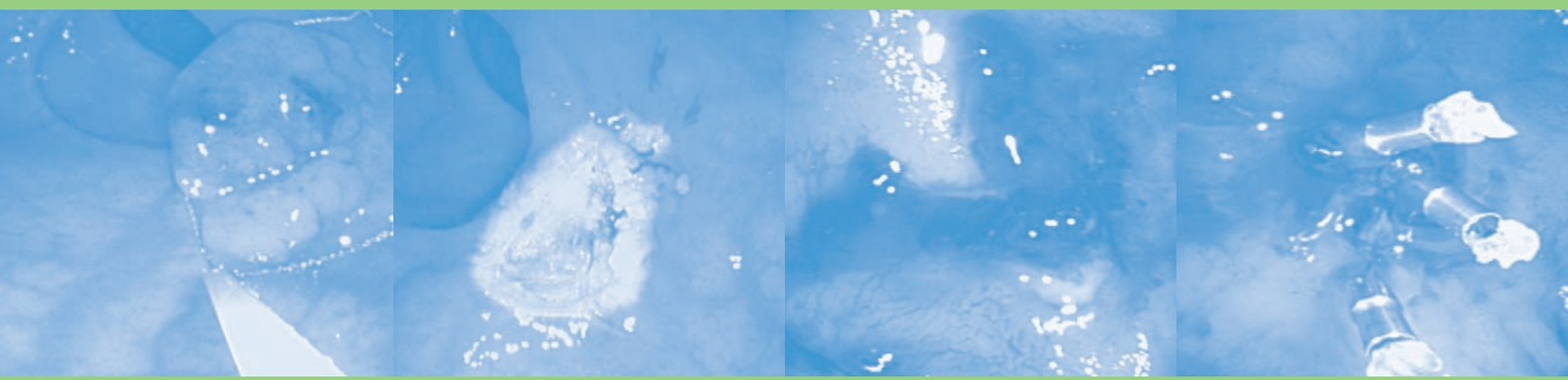
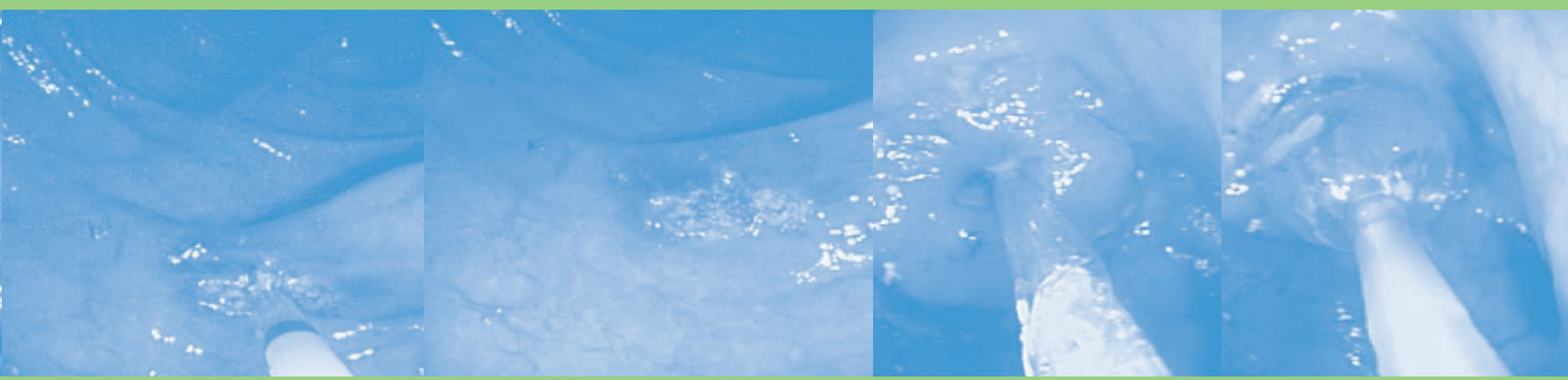


IV Endoscopic Intervention



18 Polypectomy and Mucosectomy

T. Eberl

Indications and Contraindications

Endoscopic polypectomy of colorectal polyps is indicated by clinical symptoms (to the extent that they are present) such as bleeding or occlusion and/or early detection or prevention of cancer. Table 18.**1** lists indications and contraindications for endoscopic polypectomy. If the patient has any contraindications, the potential risks of polypectomy must be weighed against expected diagnostic or therapeutic benefit (3). In such cases, procedures must be performed on an in-patient basis or with appropriate postoperative surveillance. Colonoscopic polypectomy involves significantly lower morbidity, mortality, and costs than comparable surgical resection. If an experienced endoscopist is unable to remove safely or completely a polyp, then laparotomy or laparoscopic polypectomy are indicated (3). The aim of polypectomy can be viewed as primarily diagnostic: complete removal of colorectal polyps for definitive histological diagnosis, evaluation of their nature, and thus assessment of further therapeutic measures.

Table 18.**1** Indications and contraindications for endoscopic polypectomy

Indications	Contraindications
▶ Polyps with potential for malignant transformation ▶ Cancer prevention ▶ Clinical symptoms (bleeding, occlusion)	▶ Lacking informed consent ▶ Grave concomitant disease (heart insufficiency, coronary heart disease, liver cirrhosis) ▶ Ileus, peritonitis, florid inflammatory bowel diseases ▶ Coagulation disorders, hemorrhagic diathesis ▶ Limited life expectancy (malignancy)

Preparing for Polypectomy and Mucosectomy

Patient preparation for colonoscopy and polypectomy, including bowel cleansing, is performed according to standardized procedures (see also Chapter 1 “Preparing for the Examination”. The patient should be prepared for an i. v. line before intervention.

Clotting status, platelet aggregation inhibitors, and anticoagulants. In addition to relevant patient medical information for colonoscopy (see also Chapter 1), knowledge of the patient’s current clotting status is also essential. According to DGVS (German Society of Digestive and Metabolic Diseases) guidelines, prothrombin time/INR should be above 50 %; platelet count should be above 50 000/mm³; and partial thromboplastin time should not be longer than double the norm (3). It should be noted that patients with thrombocytopenia are at risk of bleeding even at levels considerably higher than 50 000 thrombocytes/mm³.

Patients taking platelet aggregation inhibitors and patients receiving dialysis treatment have an increased bleeding tendency even when clotting parameters are normal. This is not a contraindication. For routine polypectomies, patients should discontinue use of platelet aggregation inhibitors including substances containing acetylsalicylic acid or clopidogrel one week prior to the procedure (8). In individual patients, however, where the risk of discontinuing use is greater or where medication is absolutely necessary due to underlying disease, continued use may be considered after discussion with the patient.

Patients taking Marcumar for a limited period of time should postpone elective intervention until after discontinuation of anticoagulation therapy. If long-term Marcumar use is necessary, the patient must be switched to unfractionated heparin in an in-patient procedure. Heparin should not be administered less than six hours (at the latest, four hours) before the procedure begins. Use can be resumed two to six hours after examination. Low molecular weight heparin should not be used at all on the day of examination.

Antibiotic prophylaxis. Indications for prophylactic antibiotics are determined based on overall risk of endocarditis and bacteremia resulting from polypectomy. It should be noted that polypectomy is associated with only a low risk of bacteremia. Patients with a high risk of endocarditis should receive an antibiotic prophylaxis prior to polypectomy. Prophylactic antibiotics are not mandatory for patients with a moderate risk of endocarditis.

Instrumentation

Endoscopic polypectomy techniques using a high frequency current (high frequency diathermy) with a larger patient plate placed on the thigh (neutral electrode) and a second (active electrode), smaller one (polypectomy snare), are highly standardized (3, 8). Electrical isolation of the patient must be ensured; the patient may not come into contact with any metal parts of the examining table during polypectomy. Patients with pacemakers require ECG monitoring and a defibrillator must be immediately available and ready for use. A pacemaker check should be performed as soon as possible following endoscopic therapy (i. e., same day). An overview of necessary accessories and technical requirements for polypectomy and mucosectomy of colon polyps is provided in the following box (8).

Necessary instrumentation for endoscopic polypectomy and mucosectomy

- proper equipment and connection to the neutral electrode,
- functioning high frequency electrosurgery unit,
- for pacemaker/antidefibrillator patients: ECG monitoring, ready-to-use defibrillator and magnet (if necessary, prior consultation with cardiology),
- intravenous medication (analgesic, antispasmodic with Buscopan),
- polyp snare and proper cord, special snares if needed for problem polyps or mucosectomy (▣ 18.**1 a**, **b**),
- special suction caps for mucosectomy (▣ 18.**1 c**),
- grasping forceps (▣ 18.**1 d**), trap device (▣ 18.**1 e**), polyp net (▣ 18.**1 f**),
- Endoloop (▣ 18.**1 g**, **h**),
- hemoclips (▣ 18.**1 i**),
- injection needle with 0.9% NaCl solution and epinephrine (dilution 1:100000 or 1:10000),
- small tube or larger polyp container (filled with formalin) for preserving resected polyps,
- if necessary, container with compresses soaked in saline for rapid histological diagnosis.

Polypectomy

Polyps are routinely removed during colonoscopy. Thus, around 95% of all polyps are removable without invasive surgical procedures. Polyps smaller than 5 mm are removed with forceps; all others are removed with a snare (3). Despite safe and effective techniques for removing almost all colon polyps, deciding whether a particular polyp should be endoscopically resected and actually performing the procedure require a great deal of experience.

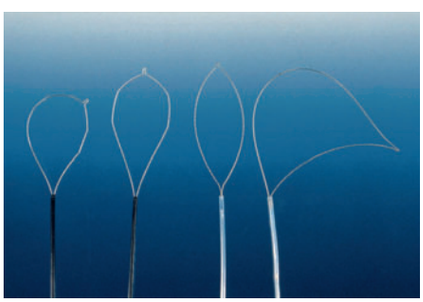
Complications are to be expected for endoscopic polypectomy of polyps with a base measuring > 3 cm in diameter. Polyp size, however, is only a relative contraindication for endoscopic resection; larger polyps can be removed with so-called "piecemeal" techniques in some cases. Prior to endoscopic polypectomy, the polyp must be closely inspected in its entirety with base and stalk. This may require manipulation with a biopsy forceps, especially in the case of a malignant polyp in order to test whether it can be depressed against the bowel wall and thus check the potential for endoscopic polypectomy. If the polyp is localized in one of the flexures, or another area where visualization is difficult, it may be helpful to change the patient's position.

If there are several polyps, they can usually be removed in one session. Large numbers of polyps may have to be resected one area at a time over several sessions; for several larger polyps, numerous sessions may help to avoid massive complications such as simultaneous perforations at various resection sites.

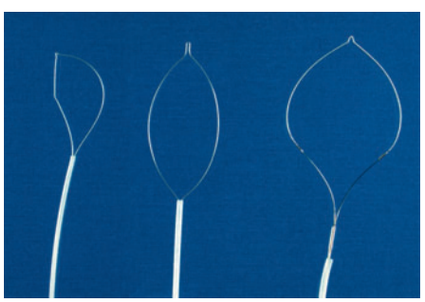
In general, proximally located polyps are resected and retrieved first. The colonoscope is then withdrawn to the more distal polyp, which is then removed. If the retrieval of a large, proximal polyp requires complete withdrawal of the colonoscope, the instrument must again be advanced to the height of the resected polyp and then slowly withdrawn to the deeper polyp and positioned.

Histological evaluation of all polyp fragments is necessary for precise diagnosis. If retrieval of all polyp pieces is not complete at the end of the examination, remaining polyp pieces can be retrieved from evacuated fluid with a sieve (Fig. 18.**1**).

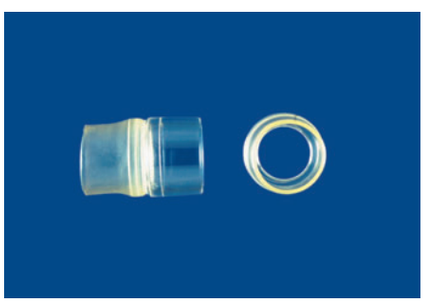
▣ **18.1 Instrumentation for endoscopic polypectomy and mucosectomy**



a Various types of polypectomy snares (left to right): symmetrical, hexagonal, oval, extra large snare. These snares have a diameter of 2.5 cm and are selected according to the type of polyp being resected. The extra large snare is for giant polyps; maximum snare diameter is 6 cm.



b Mucosectomy snares (left to right): for stenosis (cap technique, 2.5 cm diameter), monofilament snare for flat polyps (2.5 cm diameter), reinforced snare (5 cm diameter).



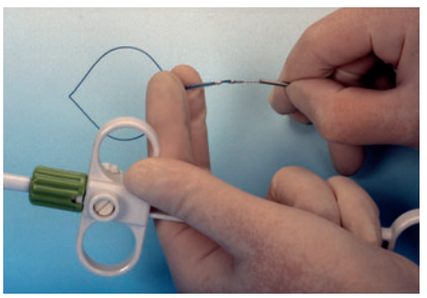
c Special suction caps for "suck-and-cut" method. The diameter of the suction caps is 17.2 mm.

18.1 cont.

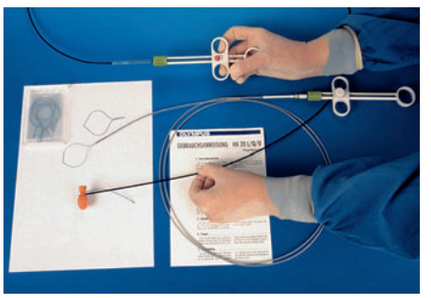
d Instruments (left to right) for polyp retrieval: normal polypectomy snare, grasping forceps, four-pronged grasping forceps, dormia basket.

e Polyp trap device. The specimen bag is placed in the trap so that suctioned polyp fragments or small polyps remain hanging in the bag. The bag is cut open with scissors to retrieve the polyp fragments.

f Polyp retrieval net. The net is placed over the working channel of the instrument and the polyp or polyp pieces are caught in the net. The polyps are retrieved on withdrawing the instrument.

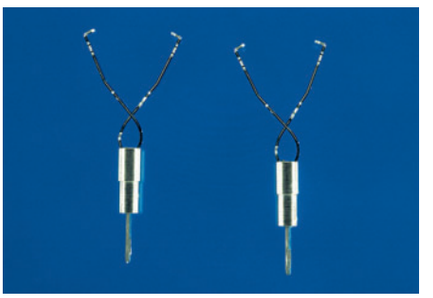


g



h

g, h Endoloop (detachable nylon snare). The detachable snare is tightened after being placed on the polyp stalk, closed, and later falls off.



i Hemoclips for mechanical hemostasis.

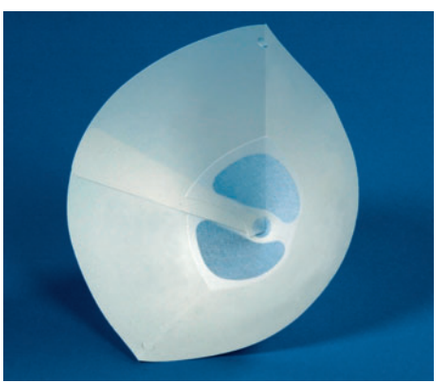
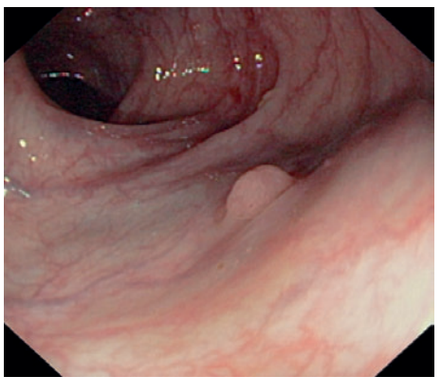


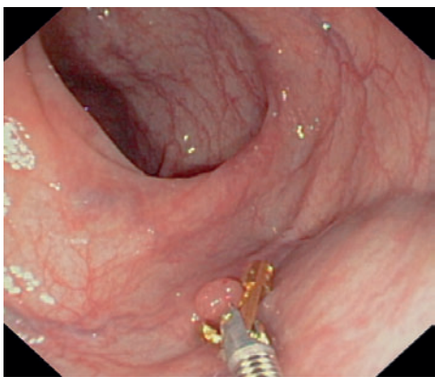
Fig. 18.**1** **Polyp sieve.** By allowing stool to pass through a sieve, polyps or polyp pieces lost during endoscopic retrieval can be retrieved after examination.

Principles of polypectomy

- thorough inspection of the entire polyp (size, test depression against the bowel wall), possible changing of patient position,
- determine number of sessions (according to number and size of polyps),
- polypectomy and polyp retrieval from proximal to distal,
- histological evaluation of all polyp fragments,
- potentially remaining polyp pieces should be retrieved from fluid evacuated from the bowels using a polyp sieve (Fig. 18.**1**).

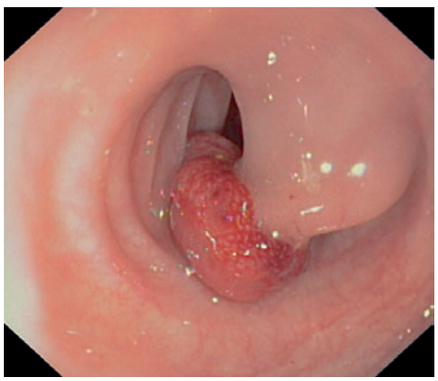


a



b

Fig. 18.**2 a, b** **Resection of a diminutive polyp (< 5 mm) with grasping forceps.**



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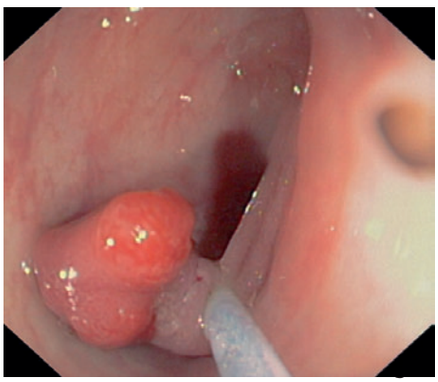


Fig. 18.**3 a, b** **Polyp with prominent stalk.** Positioning the polypectomy snare around the polyp stalk. The level of transection or coagulation of the stalk is closer to the polyp head than to the bowel wall.

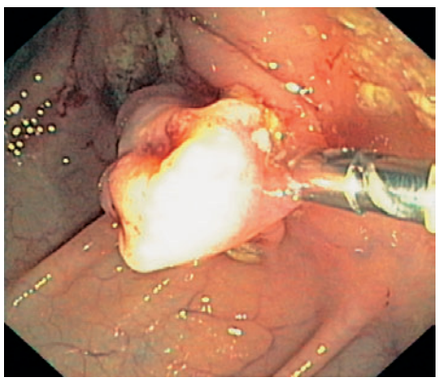


Fig. 18.**4** **Polyp stalk treated with hemoclips after rebleeding.**

Small Polyps

According to guidelines of the German Society of Digestive and Metabolic Diseases (DGVS) (3) polyps < 5 mm are removed using biopsy forceps; attempted snare polypectomy usually "burns" the polyp, rendering histological evaluation impossible (Fig. 18.**2**). The small polyp should be repeatedly grasped with the forceps until it appears macroscopically to have been completely removed. This method entails the risk, however, that macroscopically undetectable polyp pieces may remain and could cause residual polyps. For this reason, some authors prefer the hot-biopsy forceps method (8), which enables the retrieval of histologically valuable material while coagulating the base of the resection site. However, this method entails risk of rebleeding and perforation.

Stalked Polyps

It should be possible to remove stalked polyps of any size in a single session using a snare.

Ensnaring the polyp head and positioning the snare. An appropriate snare is selected, looped around the polyp head, and then positioned around the stalk. The level of transection or coagulation should be closer to the polyp head than to the bowel wall (Fig. 18.**3**). Transection too close to the bowel wall may cause perforation; also, if rebleeding occurs from the remaining stalk, endoscopic hemostasis using injection or application of hemoclips is much easier with more remaining stalk (Fig. 18.**4**). The snare should not be closed until it is exactly at the transection line. Closing the snare too firmly before diathermy can "guillotine" the polyp stalk and cause bleeding. Polypectomy must always be performed under visualization: insufficient visualization can result in perforation of the bowel wall. If the polyp head is so large that even a giant snare cannot be looped around it, it should be removed in several pieces or portions ("piecemeal" polypectomy). This can reduce the size of the polyp head so that the snare can then be placed around the stalk and the polyp can be safely and completely removed.

Coagulation of the polyp stalk. Polypectomy is performed using a mixture of coagulation and cutting current (monopolar blended current, 120 W), which is a standard setting in most recent electrocautery units. The snare must be closely encircled around the polyp stalk. When the coagulation effect on the stalk becomes visible, the snare can be closed more tightly. Cutting current may be increased if there is continued resistance against the closed snare, despite sufficient coagulation. Resection of stalked polyps can also be performed using pure coagulation current (60 W) to minimize risk of rebleeding from the polyp stalk.

Bleeding prevention measures. A very prominent stalk or obvious pulsating is usually a sign that the polyp head is being supplied by a thick vein, perhaps even an artery. In such cases, the examiner must consider how to minimize the risk of rebleeding by endoscopic treatment of the stalk prior to polypectomy. There are three possibilities that may be used in combination with each other (8):

1. An Endoloop (detachable nylon loop) may be placed around the polyp stalk. Like a polypectomy snare, a detachable snare is wide enough to be placed around the polyp head and positioned around the stalk. The detachable snare should be positioned closer to the bowel wall, as the polypectomy snare will later be placed above it and sufficient space must be left between the snare and the polyp head for safe and complete polypectomy. The Endoloop is then closed, ligating the stalk and vessel. Positioning the polypectomy snare can be extremely difficult and often optimal positioning is impossible due to the presence of the Endoloop. Following polypectomy, the Endoloop later falls off after necrotization of the stalk. Rebleeding at that time is rare. ▣ 18.**2** shows snare removal of a polyp with a thick, pulsating stalk using an Endoloop for assistance.
2. The polyp stalk can be injected with 0.9% NaCl and/or an epinephrine dilution of 1:100000 (Fig. 18.**5**). Injection technique is described in more detail in the section "Mucosectomy" p. 173. The dilution of epinephrine (1:100000 or 1:10000) varies in the literature (8). The more concentrated form of epinephrine (1:10000) tends to be indicated for achieving hemostasis with rebleeding following polypectomy.

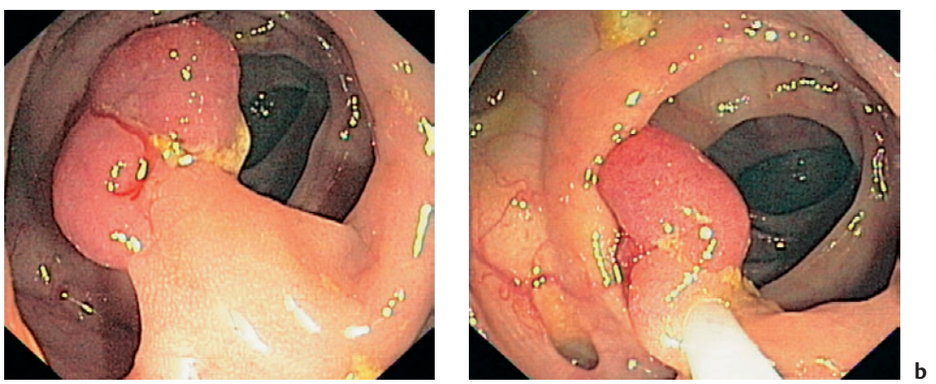
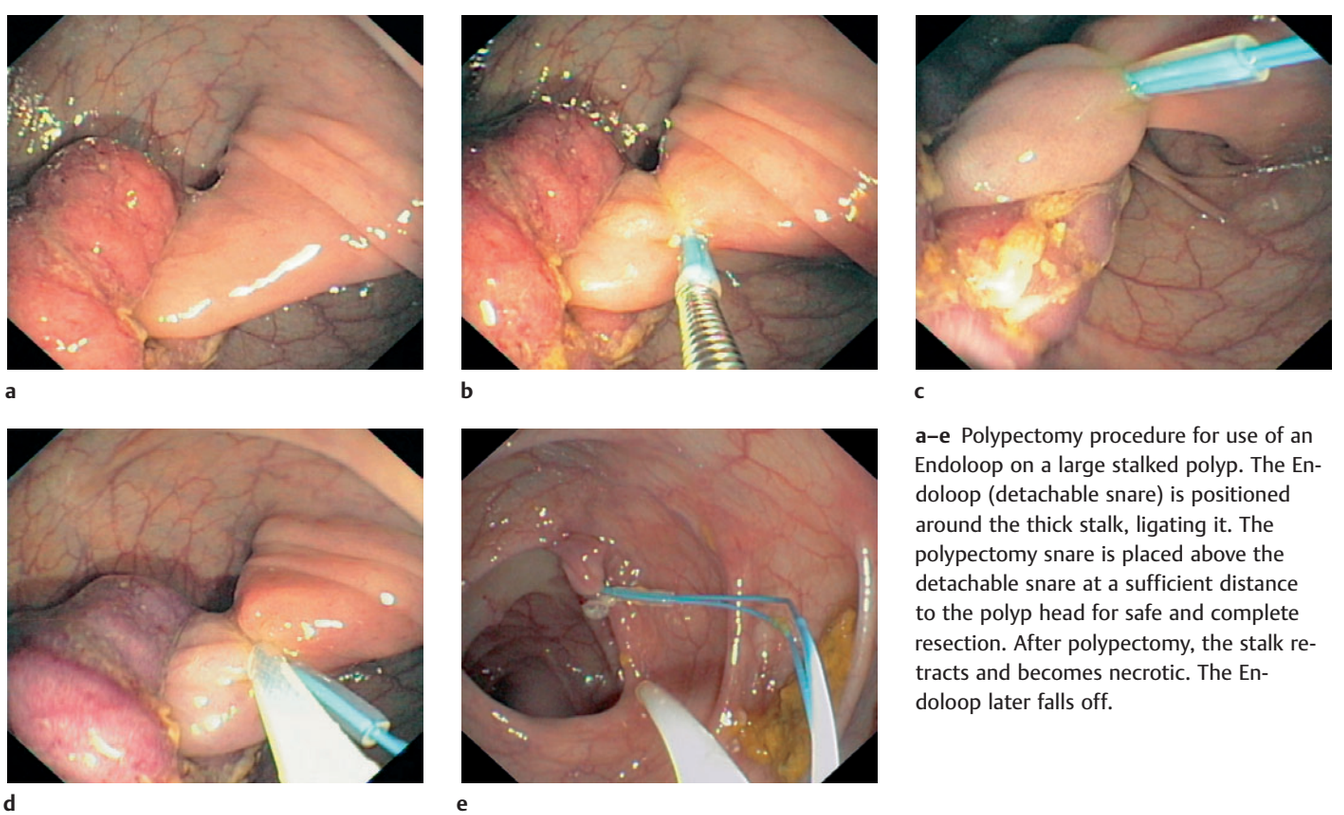


Fig. 18.**5 a, b** **Large stalked polyp.** Injection of the polyp stalk with 0.9% NaCl and/or an epinephrine dilution of 1:100000.

▣ **18.2 Polypectomy procedure using an Endoloop on a stalked polyp**



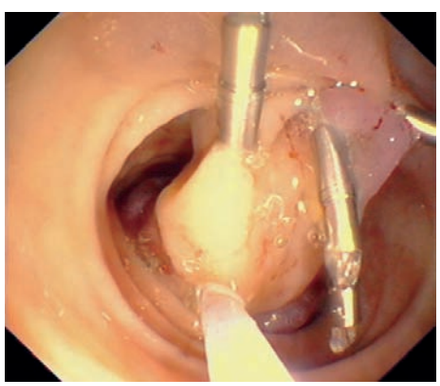
a–e Polypectomy procedure for use of an Endoloop on a large stalked polyp. The Endoloop (detachable snare) is positioned around the thick stalk, ligating it. The polypectomy snare is placed above the detachable snare at a sufficient distance to the polyp head for safe and complete resection. After polypectomy, the stalk retracts and becomes necrotic. The Endoloop later falls off.

3. The polyp stalk can be hemoclipped (Fig. 18.**6**). One or more hemoclips are applied to the polyp stalk close to the bowel wall so that compression of the stalk is complete. This technique must also leave space for securely positioning the polypectomy snare closer to the head of the polyp. Similar to the Endoloop, the effect is considered sufficient when ligation causes the proximal polyp stalk and the polyp head to visibly blanch or turn livid.

Given the lack of randomized studies, there are ultimately no clear recommendations for determining which polyps should be preventively treated with prophylactic measures to prevent bleeding. The examiner must decide which technique he or she prefers.

Sessile Polyps

Sessile polyps are attached directly to the colon wall by a broad base. Polypectomy procedure is determined by their size and localization. Sessile polyps in the left hemicolon up to 1.5 cm in size can be removed with a diathermy snare in one session. The snare is encircled around the base of the polyp, directly resting on the intestinal mucosa, and the polyp is removed using diathermy current (▣ 18.**3 a–d**). If the polyp is localized in the right hemicolon, it should not exceed 1 cm in total size for snare resection, unless submucosal injection of saline (with or without diluted epinephrine) is used to reduce the risk of thermal damage to the colon wall (8). The heat produced by diathermy is transmitted through the submucosa to the serosa of



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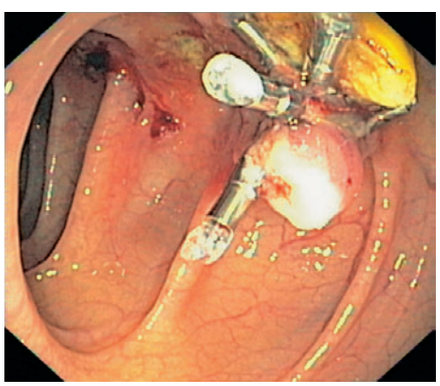
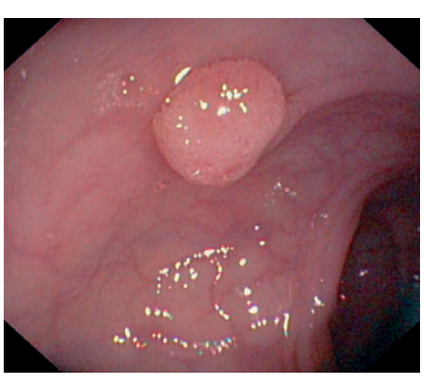


b

Fig. 18.**6 a**, **b** **Large stalked polyp.** One or more hemoclips are applied to the polyp stalk close to the bowel wall so that the stalk is completely compressed and ligated. The polypectomy snare is positioned proximal to the hemoclips close to the polyp head so that the polyp can be resected at a sufficient distance from the hemoclips.

▣ **18.3 Polypectomy of sessile polyps**



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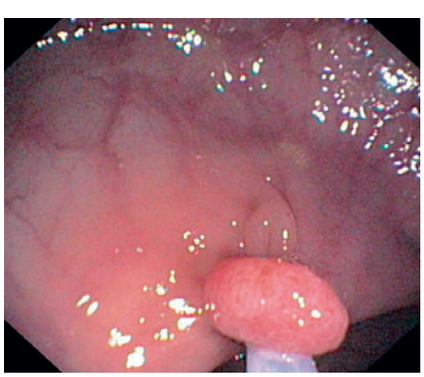

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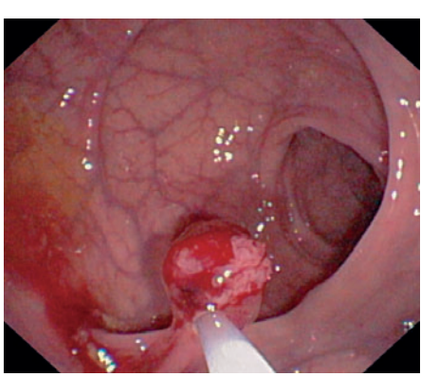

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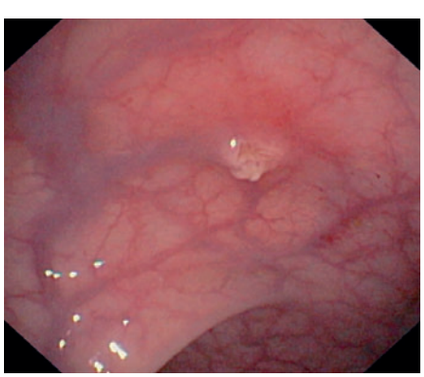
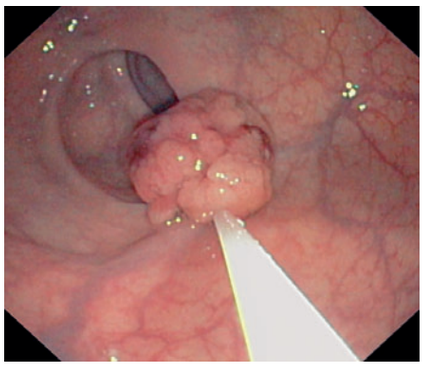


d

a–d Procedure for polypectomy of a sessile polyp up to 1.5 cm in size. The snare is placed around the base of the polyp, directly on the bowel mucosa, and the polyp is removed using electrocautery current. The resection site is smooth.

18.3 cont.



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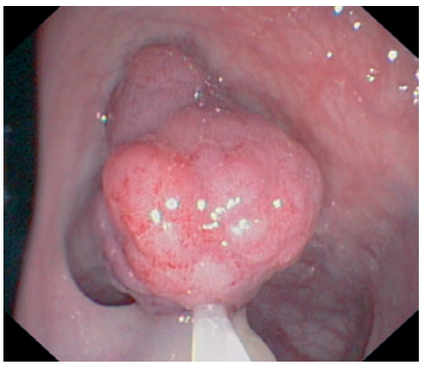


f

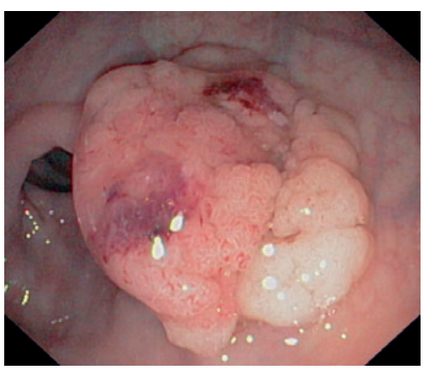
e For polypectomy of sessile polyps, the snare should be positioned around the polyp head so that the snare wire closes exactly around the base, if possible without including normal bowel mucosa.

f The snare is then closed and the polyp lightly lifted in order to avoid contact during diathermy with the neighboring or surrounding bowel wall.

the wall and when resecting voluminous polyps, it can be sufficient to induce necrosis in the bowel wall, ultimately causing perforation. When the snare is closed around the polyp, the thickness of the submucosa and muscle layer is only about 1 mm. Thus, when ensnaring sessile polyps, the snare must be positioned so that the snare wire can close around the base of the polyp without including any normal bowel mucosa (▣ 18.**3e**). The snare is then closed and the ensnared polyp is lifted slightly to avoid contact with the surrounding or opposite bowel wall during diathermy (▣ 18.**3f**).

Broad-based polyps. ▣ 18.**4** shows two approaches for polypectomy of broad-based, sessile polyps. Larger polyps are removed in pieces or portions ("piecemeal" resection technique), if necessary, in several sessions over a period of a few weeks. If a sessile polyp is on a haustra, the distal part of the polyp should be resected first. This can cause tissue retraction during healing of the coagulation ulcer that leads to better positioning of the proximal part of the polyp. Retroflexion of the instrument can be helpful for viewing portions of the polyp located behind a haustrum. It can also assist investigation and removal of polyps

18.4 Procedure for polypectomy of broad-based polyps



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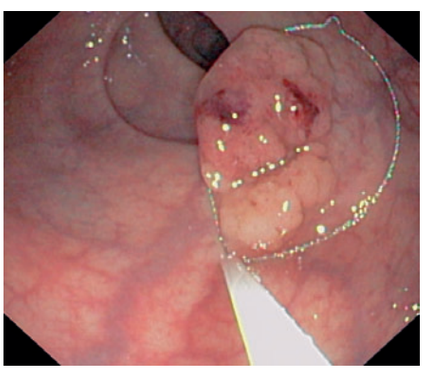

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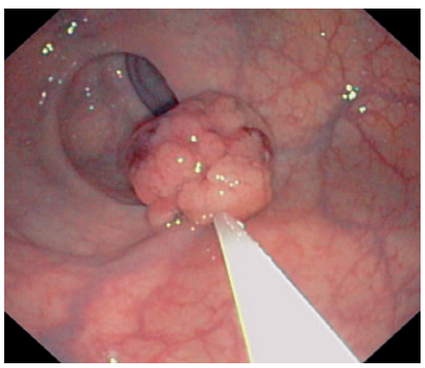

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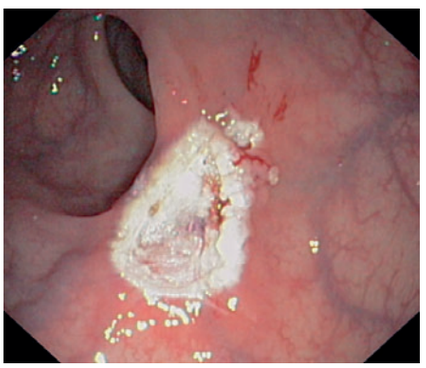
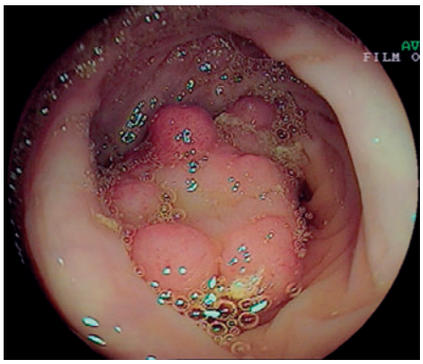


d

a–d Procedure for removing a broad-based sessile polyp about 3 cm in size. The polyp is ensnared around the entire base. The snare is closed around the polyp and the polyp is slightly lifted. This polyp can be removed en bloc.

18.4 cont.



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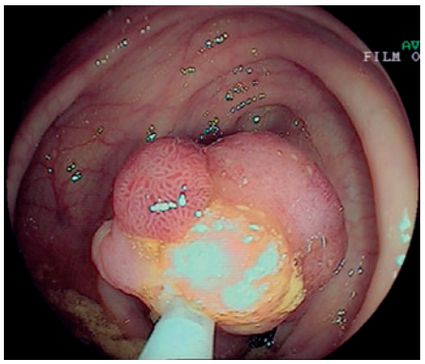

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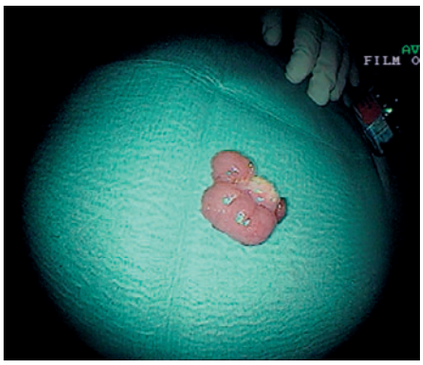

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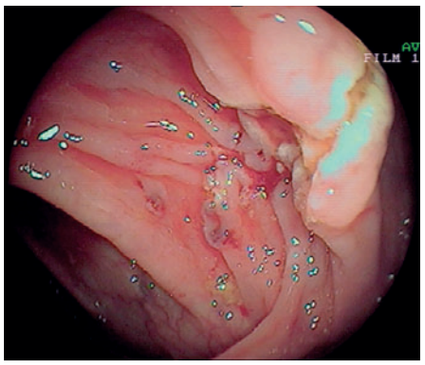


h

e–h Polypectomy of a broad-based sessile polyp: the snare is positioned at the base of the polyp and the entire polyp is removed using electrocautery current.

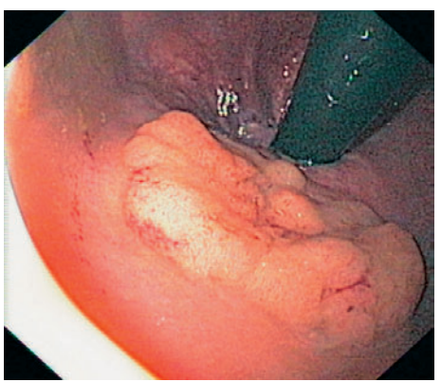


Fig. 18.7 **Retroflexed instrument in the rectum.** Retroflexion allows better evaluation of a flat adenoma located in the distal rectum.

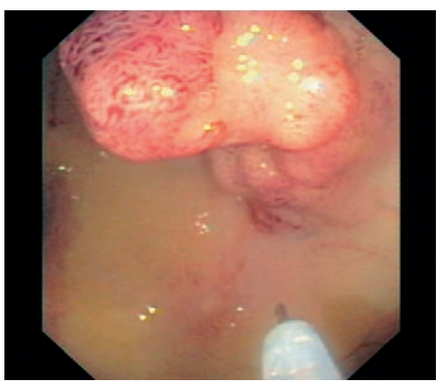


Fig. 18.8 **Submucosal injection of saline with diluted epinephrine** prior to polypectomy of a broad-based polyp. Mucosa is white from vasoconstriction effect.

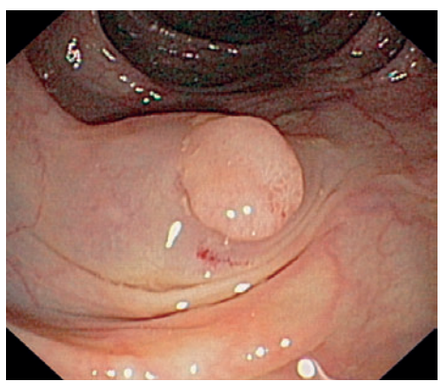
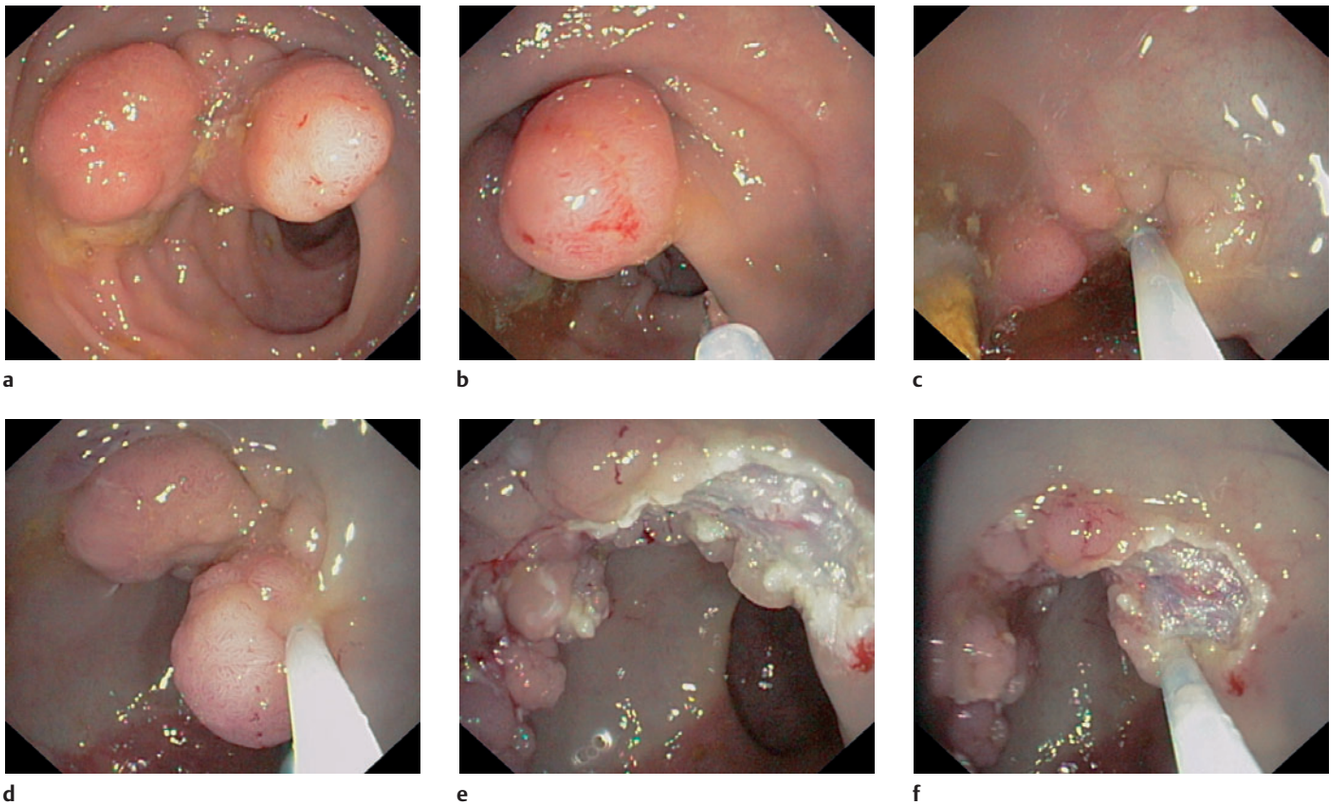


Fig. 18.9 **Creating a fluid cushion in the submucosa.** Increasing the distance between polyp base and muscularis propria by creating a fluid cushion of 10 mL of NaCl in the submucosa, minimizing potential for thermal damage in the entire colon wall.

located in the distal rectum (Fig. 18.**7**). If small adenomatous components remain on the margin of the polyp base, the tissue can be obliterated using argon plasma or laser coagulation, though this should remain an exception. The risk of thermal damage to the bowel wall during polypectomy of larger or flat polyps can be substantially reduced by submucosal injection of saline with or without diluted epinephrine (Fig. 18.**8**). The submucosal fluid cushion increases the distance between polyp base and serosa, reducing the potential for thermal damage to the entire colon wall, and thus perforation (Fig. 18.**9**) (8). ▣ 18.**5** shows piecemeal polypectomy of a polyp with a very broad base after injection. Further details on submucosal injection technique are provided in the section "Mucosectomy" p. 173.

Postpolypectomy changes. After polypectomy with diathermy current, the polyp base appears edematous (Fig. 18.**10**). After a

18.5 Procedure for polypectomy of a broad-based polyp using piecemeal resection technique



a–f Polypectomy of a polyp with a very broad base around 5 cm in size, encircling half of the circumference. After creating a fluid cushion by submucosal injection, the polyp is resected in several portions (piecemeal polypectomy).

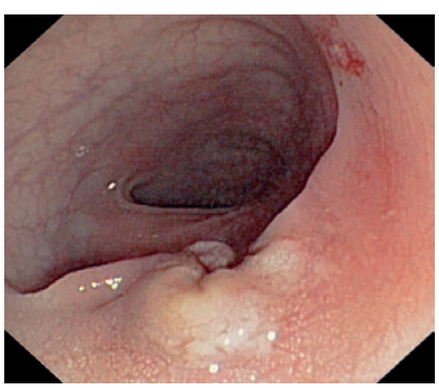


Fig. 18.**10** **Edematous resection site immediately following polypectomy.**

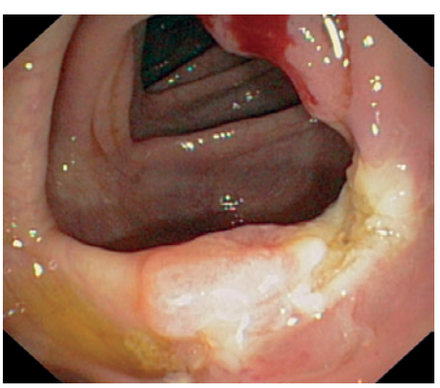


Fig. 18.**11** **Ulcer forming** after sloughing off of necrotic tissue and formation of granulation tissue. Ulcers begin forming three to five days after polypectomy; granulation and scarring are completed within about eight weeks.

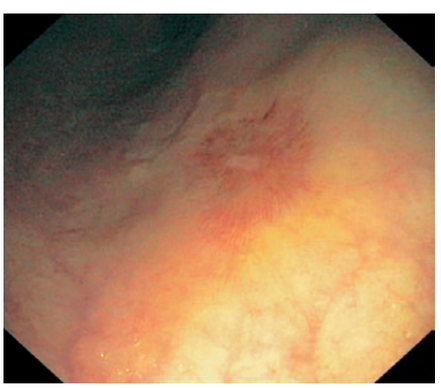


Fig. 18.**12** **Neocapillary formation** between coagulated resection site and surrounding mucosa in the scarring phase.

period of a few days up to several weeks (depending on polyp size), the necrotic tissue is sloughed off and granulation tissue forms (Fig. 18.**11**). During the initial healing phase, neocapillaries form between the coagulated resection base and the surrounding mucosa (Fig. 18.**12**). In the scarring stage, a linear, depressed, whitish scar forms (Fig. 18.**13**). Resection of very large, sessile polyps causes deeper coagulation with extensive scarring and retraction of the folds. If a sessile polyp is not completely removed at its base, residual polyps appear nearby and these must be resected (Fig. 18.**14**). Piecemeal resection technique for very large colon polyps is also an effective method. Frequency of residual polyps after piecemeal polypectomy has been reported at 3 % over a 40-month observation period (1).

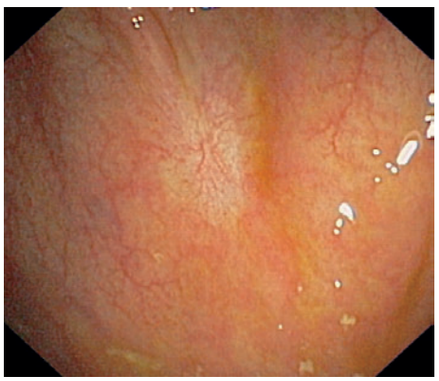


Fig. 18.**13** **Linear, depressed whitish scar following polypectomy.**

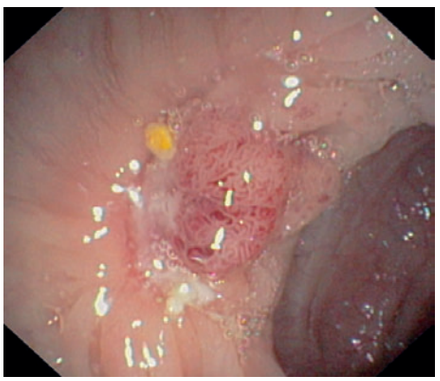


Fig. 18.**14** **Residual polyp** after incomplete resection of the base of a sessile polyp.

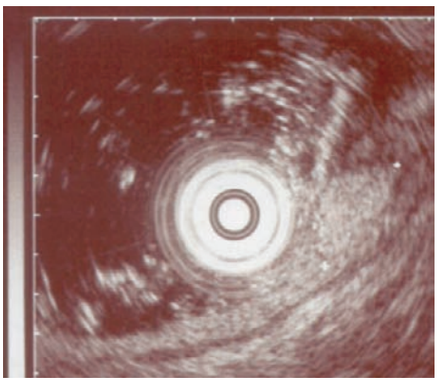


Fig. 18.**15** **Sonography** (20 MHz probe) to determine depth of infiltration of a malignant flat adenoma. The tumor measured 8 mm (white markings). The unbroken continuity of the smooth muscle layer (dark line beneath the tumor) shows that the carcinoma is limited to the mucosa.

Malignant Polyps

The risk of malignancy increases with increased polyp size, proportion of the adenoma's villous components, and degree of dysplasia. The deciding factor is whether the muscle layer has been infiltrated, which signals lymphatic infiltration. If malignancy is first diagnosed during histological evaluation of a polyp that was not biopsied prior to removal, histopathological stage determines further procedures. Resection of a malignant polyp with a healthy margin and which is in stage pT1 with a differentiation grade of G1 or G2 and without vessel infiltration (pT1 carcinoma: "low risk") is followed by curative endoscopic therapy. Lymphatic proliferation is not a major concern, particularly for malignant components in the proximal portion of the polyp at a good distance from the smooth muscle layer.

Patient age and health status determine whether resection of a malignant polyp (confirmed with biopsy) should be endoscopic or surgical. For routine resection of histologically confirmed malignant polyps, submucosal injection to elevate the polyp is indicated, especially for sessile polyps. Easily lifted malignant polyps are a sign of superficial infiltration of the bowel wall; complete removal of the malignancy with a margin of healthy tissue can be expected. If the polyp is not elevated by submucosal injection, the tumor is presumably fixed to the colon wall and the depth of infiltration makes complete removal with a healthy margin difficult. Though submucosal injection theoretically would entail a risk of spreading tumor cells in or through the bowel wall, findings are uncertain. It is vital that resection site of a malignant polyp is marked correctly—e.g., by marking the coagulated base of a resected polyp with a needle—so that the pathologist can determine invasion depth.

■ Mucosectomy

Indications and contraindications. Mucosal resection is indicated for superficial carcinomas or flat adenomas that cannot be resected using conventional polypectomy techniques. If possible, endosonographic staging should be used prior to mucosectomy to ascertain depth of infiltration of the superficial carcinoma in the bowel wall (Fig. 18.**15**). Only those superficial carcinomas that are limited to the mucosa can be treated with endoscopic mucosal resection. If the tumor has infiltrated the submucosa, there is a risk of lymph node metastasis and thus surgical resection (lymph node dissection) is necessary. Table 18.**2** shows the risk of lymph node metastasis related to mucosal and submucosal infiltration of superficial carcinomas (5, 7).

Procedure. Mucosal resection is performed in the following steps (2, 4) (Fig. 18.**16**):

- Using an argon beam coagulator or polypectomy snare, the borders of the polyp or carcinoma are marked. If the borders are not clearly demarcated, chromoendoscopy with indigo carmine can be helpful for emphasizing the surface and margins.
- The lesion is injected with a mixture of 0.9% NaCl and diluted epinephrine (1:100000) or only 0.9% NaCl (Fig. 18.**16 b**). Infiltration to the submucosa helps to lift the lesion (Fig. 18.**6 c**), while the epinephrine allows the fluid cushion to remain longer in the submucosa. Injection volume depends on the size of the lesion, though usually at least 20 mL must be used. For larger flat polyps, a viscous solution of 0.5% hyaluronic acid can be used (9). This solution is isotonic and is absorbed slowly so that the cushion can last for several hours, though the practicability of this technique needs further study.
- Mucosal resection of the lesion is performed with a monofilament polypectomy snare or special suction cap (straight or angled) and the corresponding asymmetrical snare (Fig. 18.**16 a**, **d**). As the colon wall generally measures only 1.5–2.2 mm, the "suck-and-cut" technique using a special

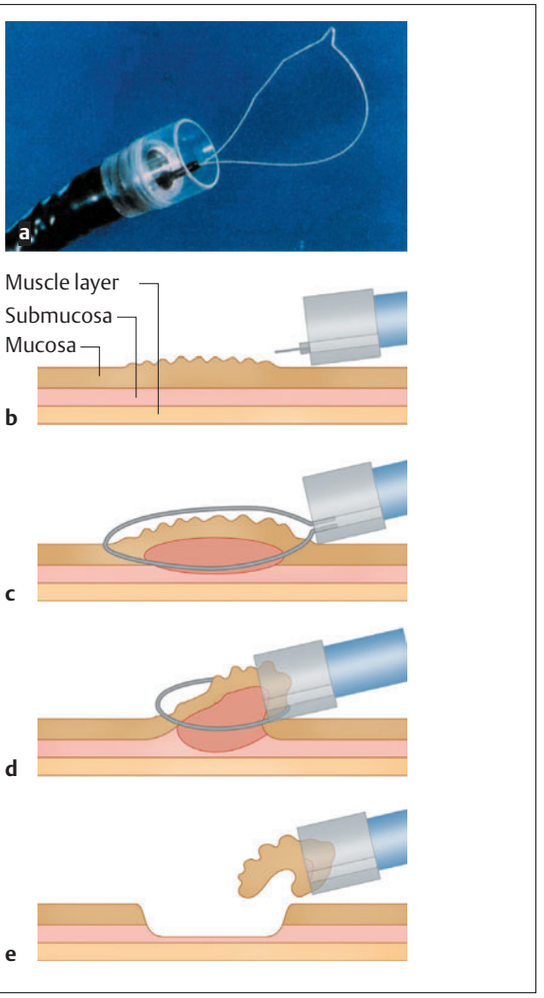


Fig. 18.**16** **Schematic illustration of endoscopic mucosal resection using suction cap technique** (modified based on 4).
a Colonoscope with suction cap and asymmetrical snare.
b Submucosal injection with NaCl solution and epinephrine.
c Lifting the flat lesion after submucosal injection.
d Suctioning the flat lesion into the cap and resection with a snare.
e Recovering the resected lesion by suction into the cap.

Table 18.**2** Lymph node metastases in superficial colon carcinomas related to depth of infiltration (5, 7)

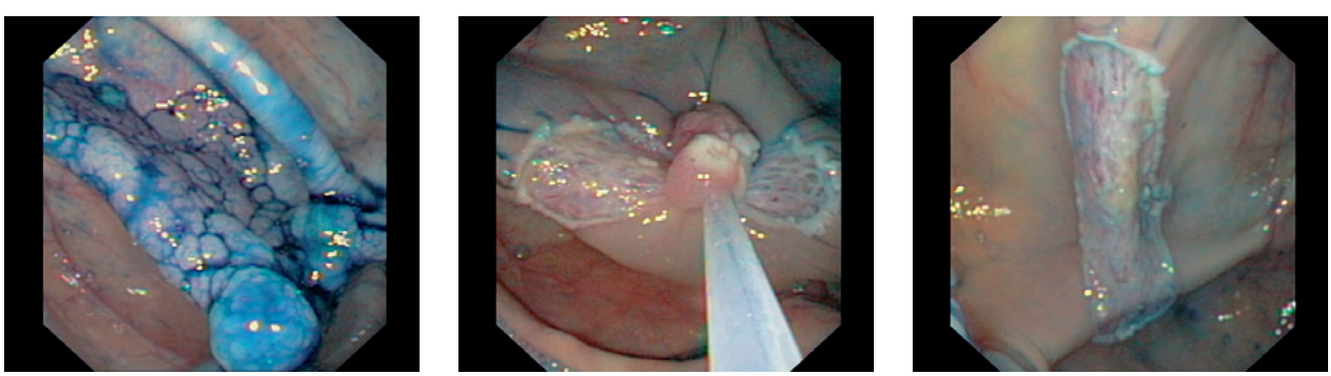
Tumor stage	Frequency of lymph node metastasis
T1: Infiltration of mucosa	
▶ m1: intraepithelial carcinoma	0 %
▶ m2: infiltration of the lamina propria	0 %
▶ m3: infiltration of the smooth muscle layer	0 %
T1: Infiltration of submucosa	
▶ sm1: upper third	2 %
▶ sm2: middle third	up to 30 %
▶ sm3: lower third	up to 30 %

suction cap increases risk of perforation. For this reason, we choose not to use this technique at our clinic for mucosal resection in the colon.

- For larger lesions, it may be necessary to perform resection in several steps.
- Recovering resected material can be done with a polypectomy snare or by suction into the cap in the "suck-and-cut" technique (Fig. 18.**16 e**).
- Possible complications (bleeding) are treated after recovering resected material. If bleeding is light or moderate, recovering resected material has priority.
- Using thin needles, resected material is spread out and fixated on cork, placed in a 10 % formalin solution, and submitted for histological evaluation.

▣ 18.**6** shows mucosal resection of a flat, broad-based polyp. Mucosectomy of a superficial carcinoma is demonstrated in ▣ 18.**7**.

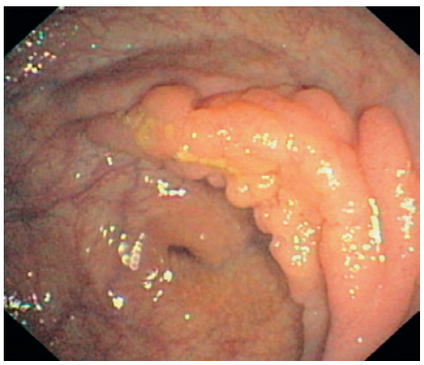
▣ 18.6 Steps in mucosal resection of broad-based polyps using piecemeal resection technique



a **b** **c**

a–c Steps in mucosal resection of a broad-based flat polyp using piecemeal resection. Chromoendoscopy to better visualize borders. After injection, mucosal resection of the polyp using a snare. Smooth resection site.

18.6 cont.



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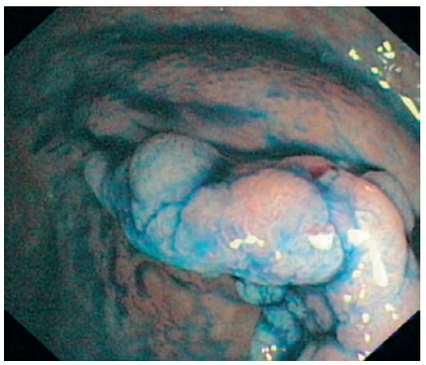

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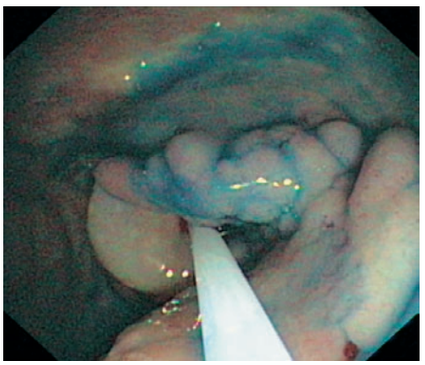

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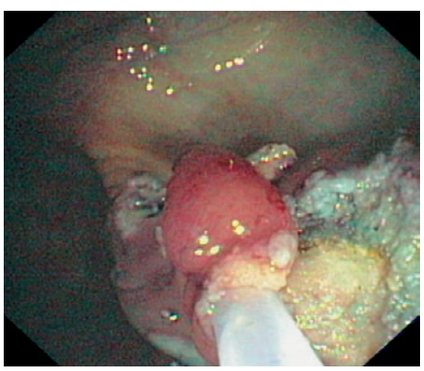

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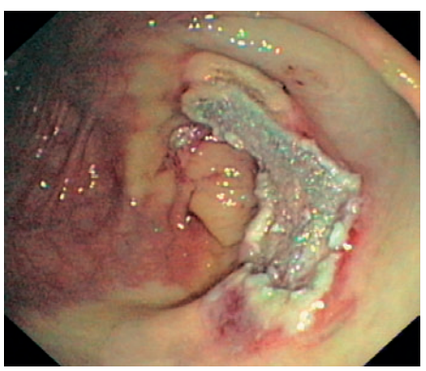
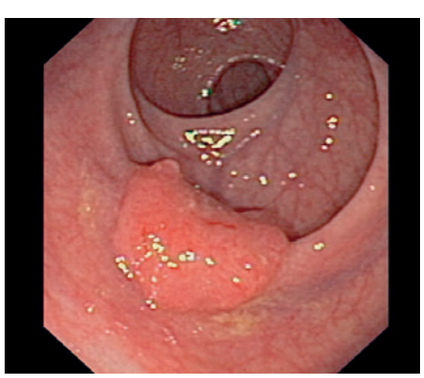


h

d–h Mucosal resection of a broad-based polyp in the cecum (piecemeal resection technique) following chromoendoscopy. Submucosal injection of the polyp enabled removal without complications.

18.7 Steps in mucosal resection of a superficial carcinoma



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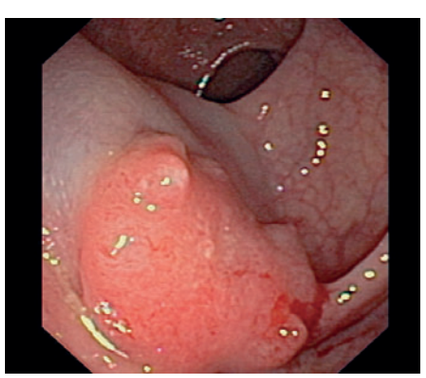

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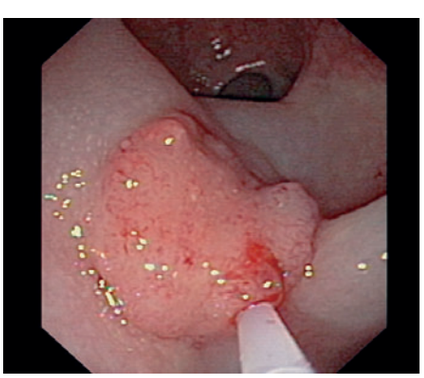

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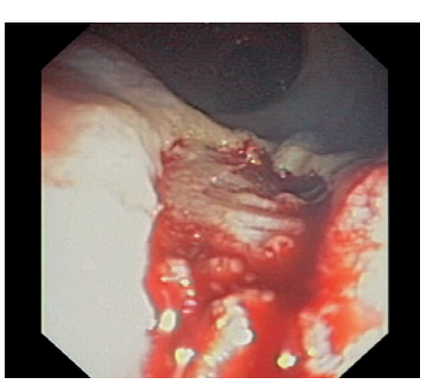

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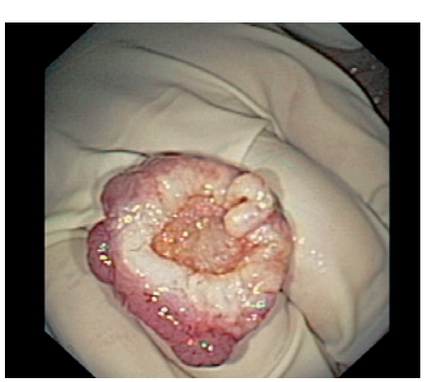


e

a–e Mucosal resection of a superficial carcinoma after submucosal injection. Histology showed an adenocarcinoma limited to the mucosa.

Steps in performing mucosectomy

- mark the borders of the lesion,
- chromoendoscopy if necessary,
- submucosal injection of the lesion to create a fluid cushion,
- mucosectomy with polypectomy snare or "suck-and-cut" technique,
- resection in several steps if necessary,
- recover resected material,
- treat complications (bleeding),
- spread resected material on a piece of cork in, formalin solution,
- histological evaluation.

Recovering Resected Polyps

Complete retrieval of resected polyps and all resected polyp pieces is essential for ensuring thorough histological diagnosis. Smaller polyp pieces can be either suctioned with the instrument or they may be suctioned and then collected in a trap device attached to the working channel (▣ 18.**1 e**). The disadvantage of suction technique is that, upon withdrawing the instrument, visualization is less than optimal and polyp fragments can be lost, especially in passing the sigmoid colon. Larger polyp pieces (larger than ca. 1 cm) can be retrieved by ensnaring the stalk or grasping the polyp head and recovering them upon withdrawal (Fig. 18.**17**). For very large, lobulated polyp heads—generally villous adenomas—the use of a dormia basket can assist in polyp recovery. For retrieving several polyp pieces, a polyp net can be used to capture all fragments at once (▣ 18.**1 f**).

Lost polyps. The risk of loosing a resected polyp ("lost polyp") during instrument withdrawal is not very great (< 5 %). In addition, if the patient "bears down" somewhat, polyps can pass the anal canal without completely falling apart or moving back up the rectum. Polyp pieces lost during retrieval in the colon can be sieved from fluid evacuated from the bowels (Fig. 18.**1**). Preparing the patient again with an enema or a bowel cleansing solution can usually be avoided, sparing him additional discomfort.

Complications following Polypectomy or Mucosectomy

Possible complications related to polypectomy or mucosectomy include bleeding, perforation, and postpolypectomy syndrome.

Bleeding. Bleeding is the most common complication following polypectomy; risk of bleeding is 1.7 % (3, 8). Bleeding can be spontaneous (▣ 18.**8 a**) or it may appear with a delay of 7–12 days (▣ 18.**8 b**). Depending on vessel supply to the resected polyp, bleeding intensity ranges from minimal oozing to arterial, pulsating spurting. Spontaneous bleeding occurs immediately after resection in 1.5 % of patients, while delayed bleeding occurs in around 2 % of polypectomies. In both cases, immediate hemostasis by the endoscopist is the therapy of choice. Even in cases of delayed hemorrhage, immediate hemostasis can be attempted endoscopically as blood stimulates intestinal peristalsis and the colon is cleansed of remaining stool. Polypectomy bleeding is treated with endoscopic hemostasis, blood transfusion, and close clinical surveillance. Surgical intervention is a last resort and used only in rare cases in which hemostasis cannot be achieved endoscopically. There are a number of endoscopic therapy possibilities depending on polyp features and bleeding intensity.

Endoscopic therapy of postpolypectomy bleeding

- Spontaneous bleeding from a polyp stalk can be controlled by again positioning the snare around the stalk, closing it, and holding it closed for ca. five minutes.
- If bleeding does not cease, a second attempt can be made with the snare for five more minutes.
- Alternative methods of hemostasis include injection therapy, application of hemoclips, and thermal methods such as argon plasma coagulation and the use of BICAP electrodes.
- For injection therapy, several milliliters of 1:10 000 diluted epinephrine solution are injected into the bleeding site. The injection of epinephrine reduces blood flow by vasoconstriction and compression (due to the presence of injected fluid), creating a "cushion" effect.
- Only in rare cases is injection of tissue glue (e.g., fibrin glue) indicated for diffuse bleeding at a resection site.
- Polyp stalks or visible vessels at the resection site can be treated with mechanical hemostasis using a hemoclip to compress the stalk or vessel (▣ 18.**8 c–e**).
- In rare cases, an Endoloop can be placed around the stalk to achieve hemostasis.
- For more detail on these techniques, see also Chapter 20 "Hemostasis."

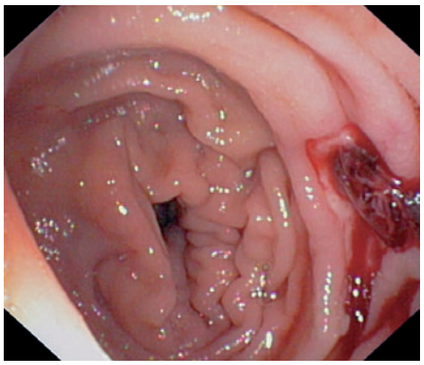
Perforation. Perforation occurs in 0.04 %–2.1 % of colonoscopic polypectomies and is caused by transmural damage to the colon wall from either a snare or thermal necrosis of the bowel wall, which causes perforation within a matter of hours (8). The majority of these perforations occur after mucosectomy or polypectomy of sessile polyps. Pain following polypectomy and signs of peritoneal irritation are cause for immediate investigation and, if necessary, laparotomy. There is no time to lose if the endoscopist observed the perforation, for example, by visualization into the abdominal cavity or of the serosa of intra-abdominal organs (Fig. 18.**18**).

Small leakages can be closed immediately with hemoclips. Based on available research, the presence of intraperitoneal or retroperitoneal free air without remarkable clinical signs ("silent perforation") does not necessitate immediate surgical intervention. The patient should initially be given intravenous antibiotics and fluids; a nasogastral tube should be attached; and he should be kept under close clinical observation by a gastroenterologist and surgeon (watch-and-wait strategy). Small leakages may close spontaneously without requiring further invasive therapy (8).

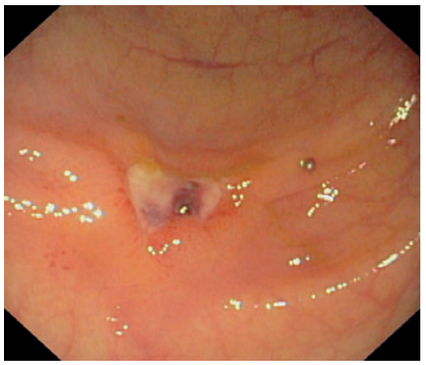
Postpolypectomy syndrome. Postpolypectomy syndrome manifests with localized abdominal pain, brief periods of fever and meteorism. Caused by local peritoneal irritation from transmural thermal damage with irritation of the serosa, the syndrome occurs in 1 % of all polypectomies with onset of symptoms between six hours and five days after polypectomy (8).

The syndrome usually abates spontaneously after two to five days, though antibiotics may be necessary depending on clinical course, fever, and laboratory tests for signs of inflammation. Differential diagnosis should exclude perforation. Perfora-

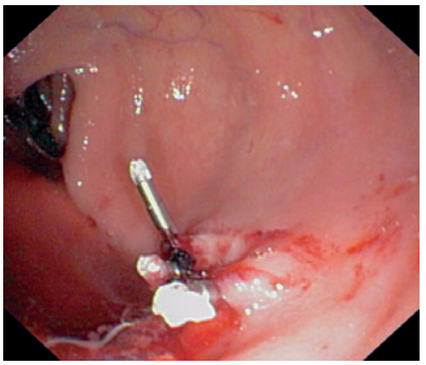
18.8 Rebleeding following polypectomy/mucosectomy and hemostasis



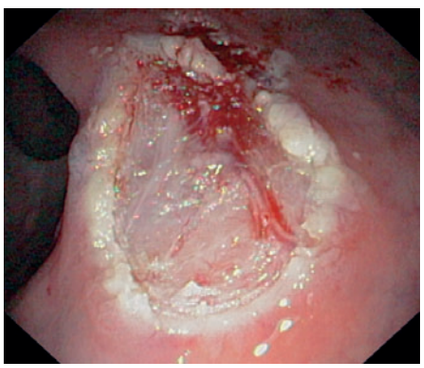
a Spontaneous rebleeding from a visible vessel following polypectomy.



b Delayed rebleeding (after seven days) following polypectomy from a visible vessel at resection site.



c Mechanical hemostasis with hemoclips for rebleeding from a large visible vessel following polypectomy.



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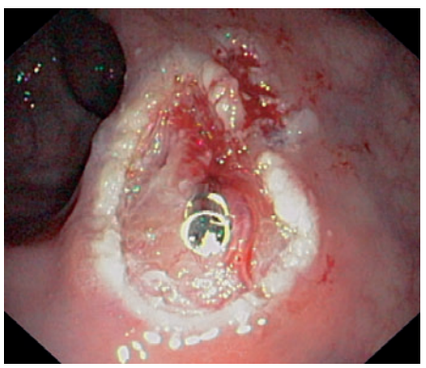
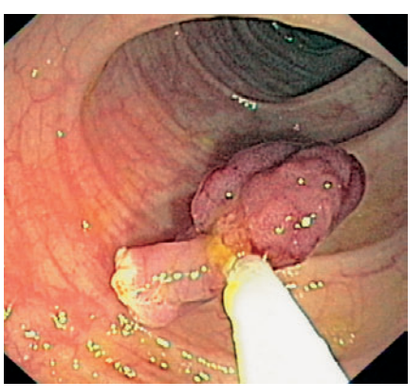


e

d, e Rebleeding after mucosectomy from a visible vessel (upper edge of image). Mechanical hemostasis by means of application of hemoclips to a visible vessel.



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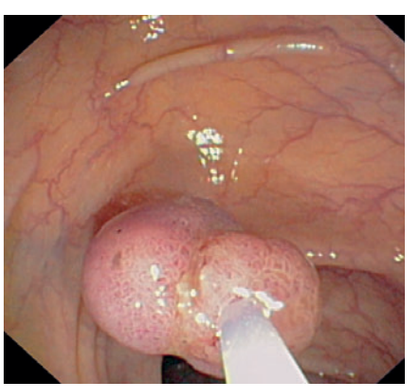

b

Fig. 18.**17 a , b** **Recovery of resected polyps.** Ensnared around the stalk or polyp head.

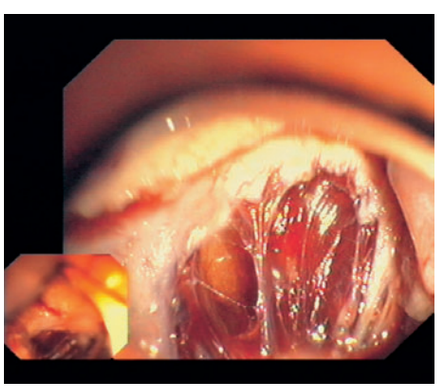

Fig. 18.**18** **Perforation following polypectomy of a broad-based polyp.** Remaining muscle fibers of the muscularis propria still visible with partial view into the abdominal cavity.

tion is distinguished from postpolypectomy syndrome by the presence of intra-abdominal free air. Worsening peritonism following polypectomy can be caused by the development of transmural necrosis resulting from thermal damage to the colon wall and can result in perforation. Patients with signs of local peritoneal irritation must be placed under close clinical surveillance.

Damage to the colon wall opposite the polypectomy site can be caused by contact with the diathermy snare during resection. This can be avoided by slightly lifting the polyp during transection from the colon wall so that the snare is only in contact with the base. Risk is considerably greater for problem polyps or hidden polyps. Maximum visualization must be ensured before electrocautery. “Blind” polypectomies are unacceptable.

Follow-up Surveillance after Polypectomy

The German Society of Digestive and Metabolic Diseases (DGVS) provides guidelines for follow-up endoscopy surveillance after polypectomy (6). After complete polypectomy and complete removal of all visible polyps, the first surveillance endoscopy is performed after three years, after which endoscopic check-ups are performed every five years. If detected adenomas remain, surveillance must be performed shortly after the initial procedure. If larger adenomas remain, polypectomy must be performed as soon as possible, depending on the patient's overall situation. If smaller polyps (< 5 mm) remain, another colonoscopy should be performed within a year.

After a malignant polyp in stage pT1 with a differentiation grade of G1 or G2 has been resected with a healthy margin and without vessel invasion (pT1 carcinoma: "low risk"), endoscopic therapy is considered completed and further follow-ups are indicated after six, 24, and 60 months (6). Following partial removal of an invasive carcinoma or complete removal of a carcinoma with poor differentiation (G3 or G4), further surgical intervention is required.

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19 Interventional Tumor Therapy

R. Fleischmann

Pathology of Malignant Colon Tumors

The vast majority of malignant colon tumors are epithelial adenocarcinomas (ca. 80%), while the remainder (ca. 20%) are mucinous adenocarcinomas. Signet-ring cell carcinomas and undifferentiated carcinomas are very uncommon as are nonepithelial malignant colon tumors (e.g., malignant lymphomas and fibrosarcomas), the latter comprising less than 1% of all colon tumors. Malignant lymphoma is primarily localized in the cecum and is infiltrative or polypoid, growing rapidly, and metastasizing early without signs of stenosis. It is usually detected late when symptoms of pain, fever, and possibly ascites appear. The most common localization of colon carcinoma (adenocarcinoma) is in the rectum (40%). However, colon carcinoma can only be excluded with certainty using total colonoscopy. In nearly 5% of patients, there are secondary or third carcinomas. Metastasis first involves regional lymph nodes, then supplying arteries, and finally occurs as distant spread in the liver, bones, lungs, and peritoneum. Distal rectal carcinoma metastasizes primarily also in the brain, skin, bones, and neighboring organs (bladder, vagina).

Fig. 19.**1** **Laser equipment,** ND:YAG laser (Medilas II, Dornier, Munich).

Indications for Endoscopic Tumor Therapy

Adenomas with severe dysplasia (carcinoma in situ) can be curatively treated by means of endoscopic polypectomy or mucosectomy (for flat adenomas). Rectal T1 carcinomas (T1Sm1, N0, M0) can also be removed by means of endoscopic resection of the intestinal lumen. However, there is a 3% risk of lymph node invasion. Thus, among younger patients and among patients with higher tumor stages, transabdominal resection should be attempted if possible.

The major indications for interventional endoscopic tumor therapy are:

- preoperative hemostasis,
- preoperative removal of an obstruction/ileus,
- palliative tumor therapy in inoperable patients (advanced tumor stages or older comorbid patients).

Hemostasis Methods and Relief of Obstruction

Bleeding tumors can be most expediently treated with argon plasma coagulation (20–50 W). Injection of tissue glue (fibrin or acrylic glue) or application of hemoclips is seldom necessary.

Ileus symptoms can be relieved by inserting a decompression tube, traversing the stenosis with a metal stent, or by using ablative methods such as laser and argon plasma coagulation (APC). These three procedures have all but replaced cryptotherapy. Tumor control using ethoxysclerol or alcohol has not gained acceptance everywhere; PDT and therapeutic ultrasound of the colorectal area are still in the experimental phase.

Palliative Tumor Therapy in Inoperable Patients

Laser

Laser therapy techniques (Nd:YAG laser)
The patient should be sedated with 0–10 mg of Midazolam during laser treatment. The Nd:YAG laser (Medilas I or II, Dornier C., Munich, Germany) (Fig. 19.**1**) is operated at 70–100 W. The laser is aimed at the tumor through an optical fiber (cooled by carbon dioxide) in a noncontact procedure. A helium–neon laser emits a red pilot light. The optical fiber is easily guided through the working channel of the colonoscope, which is protected by a ceramic tip.

Treatment proceeds from the upper tumor edge (proximal end of the colon) (Fig. 19.**2**) to the lower tumor edge (distal end of the colon). If the lumen is severely narrowed, endoscopic passage is ensured using a guidewire for bougienage or balloon dilation (Fig. 19.**3**) and with the assistance of radiographic imaging.

Results. Table 19.**1** shows results achieved in 83 consecutive patients by the working group of headed by J.F. Riemann (5). Median long-term survival rate was nine months (0.25–116 months).

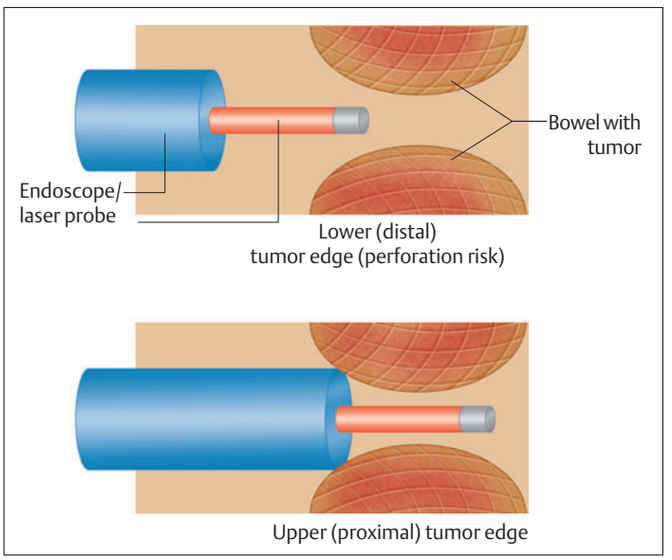


Fig. 19.**2** **Laser ablation/APC tumor ablation,** schematic illustration.

Table 19.**1** Results of palliative laser therapy in 83 patients (5)

	Obstruction	Bleeding	Obstruction and bleeding
Initial success	95 %	100 %	100 %
Sessions	3	2	3
Follow-up treatment (weeks)	12	10	10
Palliative colostomy	25 %	–	10 %

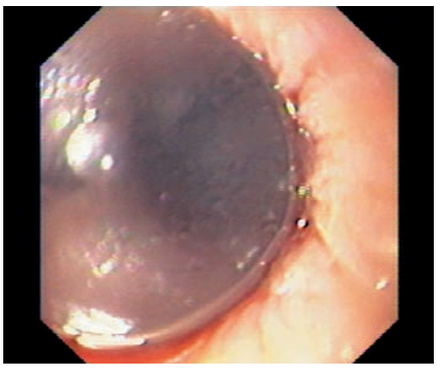


Fig. 19.**3** **Balloon dilation,** tumor stenoses prior to laser/APC application.

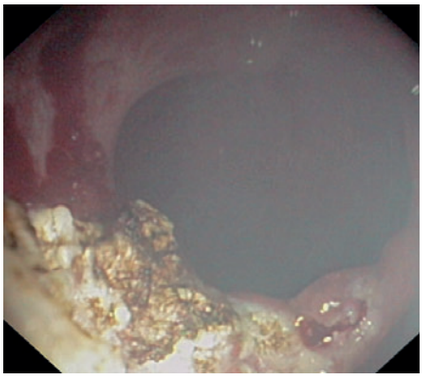


Fig. 19.**4** **Coagulation vapor must be suctioned during APC ablation.**

Complications. The greatest concern in terms of complications is hemorrhage, which can lead to abscess, peritonitis, and fistulization. Hemorrhage occurs in ca. 2–5 % of patients and must be treated with abscess drainage and antibiotics. Colostomy is often unavoidable.

APC (Argon Plasma Coagulation)

APC techniques

The argon beam coagulator has a high frequency generator (ERBE, Tübingen, Germany) with an output of 50–100 W and an argon gas flow of 0.5–2 L/minute. Energy is emitted though a 2-mm-thick Teflon tube in which argon gas flows around an electrode. The ionized argon gas is aimed from the probe tip at the targeted tissue without contact between the probe and the tissue. Because the argon gas is illuminated, it is possible to guide it over the coagulation surface. Energy delivery can be axial, lateral, or radial. The operative distance to the tissue is 2–10 mm; at a greater distance, ionization—and thus coagulation—disappears.

The main advantage over laser coagulation (laser carbonization and vaporization) is low depth of penetration (2–3 mm) that reduces risk of perforation. Bowel distention is also diminished by the gas flow. Argon gas and visibility-blocking coagulation vapor must be intermittently suctioned (Fig. 19.**4**). Further advantages include low equipment cost and the potential for gas sterilization of the delivery catheter.

Results. On average, two to three sessions are necessary for treating an occlusive tumor. More recently, APC colorectal treatment has increasingly been used for recanalizing metal stents occluded by tumor ingrowth or proliferation (Fig. 19.**5**).

Complications. Complications are few. Bowel distention can be prevented by intermittently suctioning the argon gas and coagulation vapor. Submucosal emphysema (Fig. 19.**6**) is extremely uncommon with tumor ablation and can be spontaneously reversed. Perforation rate is reported at up to 1 %.

Metallic Mesh Stents

Metal stent insertion and positioning technique

There are now a number of manufacturers of self-expanding metal stents for traversing colon stenoses caused by tumor growth. The most common are the Wallstent Enteral and the Ultraflex Precision (Boston Scientific), as well as the Z-stent (Wilson–Cook), the latter of which is also available as a covered stent especially designed for traversing fistulas. There are two techniques for applying colorectal stents:

- radiologic positioning,
- endoscopic positioning.

The advantage of endoscopic positioning is better fixation of the rectosigmoid colon with the endoscope, especially if the tumor is causing torsion of the bowel. The Wallstent Enteral can be guided directly through the working channel (3.7 mm) of a therapeutic video gastroscope and deployed

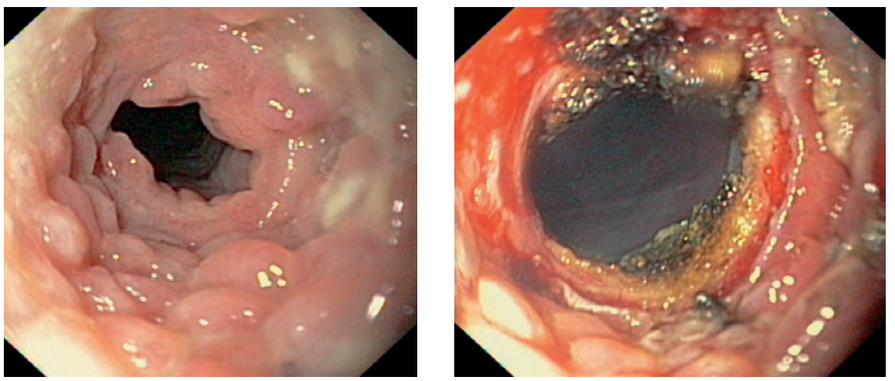


Fig. 19.5 **Tumor proliferation in a metal stent.**
a Before APC treatment.
b Metal stent after APC treatment of tumor ingrowth at the proximal end of the stent.

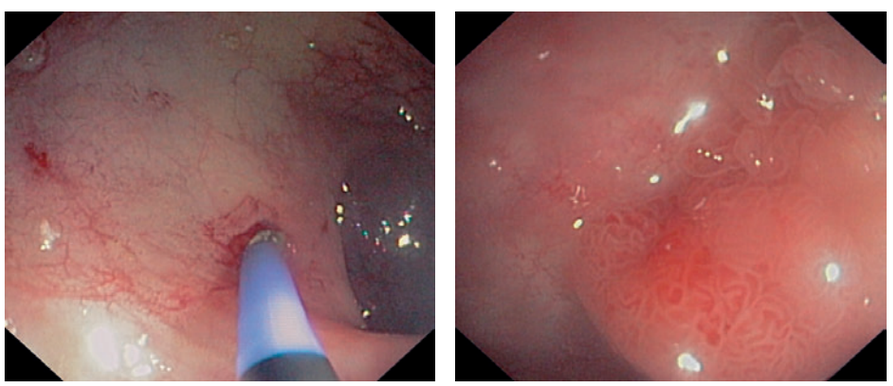


Fig. 19.6 **a, b** Subcutaneous emphysema in APC therapy.

under visualization. The metallic mesh is easily visible on radiographs.

The proximal end of the Ultraflex Precision stent is 30 mm wide and is a good fit with the bowel anatomy. To deploy the stent, the endoscope must be introduced next to the application system (Tab. 19.**2**).

Table 19.**2** Wallstent Enteral vs. Ultraflex Precision

	Wallstent Enteral	Ultraflex Precision
Placement with endoscope	+	–
Radiograph contrast	+++	+
Length of stent	60–90 mm	60–120 mm
Flexibility and lumen maintenance	++	+++

Results. There are two indications for placement of a metal stent:
- preoperative relief from obstruction,
- long-term relief of bowel obstruction.

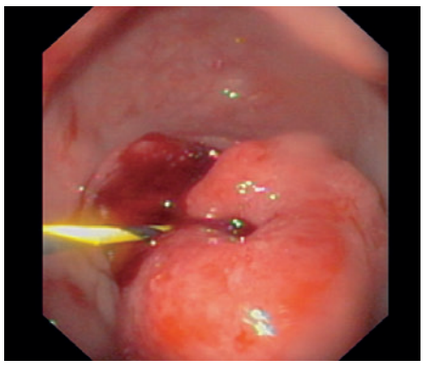
Emergency surgical intervention for a malignant obstruction in the large bowel is associated with a morbidity rate of 60% and a mortality rate of up to 22%. Placement of a metal stent can help avoid a second surgical intervention. Currently reports on over 500 interventions have been published. The technical success rate is 90–100% and the clinical success rate 83–100% (▣19.**1**, 19.**2**).

After collapsing the stent, it can be extracted endoscopically. This requires experience; in particular, the anal sphincter should be protected. After the stent is withdrawn to the rectal ampulla, it should be grasped in the middle with a snare and removed, keeping the snare closed. It is normally not necessary to use an anal dilator and brief sedation.

Complications. The most common complication is proximal or distal stent migration. Potential complications that must be considered are listed in Tab. 19.**3** (▣19.**3**). The number of complications will decrease with increasing experience and advancements in stent technology. Bleeding and tumor ingrowth clogging the stent can be effectively treated with APC. Placement of a metal stent should already be part of acute management of mechanical bowel obstruction due to inoperable malignancies in the rectum and sigmoid. The extent to which these indications can be applied to resectable carcinomas causing acute distal bowel obstruction can only be answered by future clinical studies (improvement of preoperative circulatory and bowel function). Stent placement appears to be preferable to purely ablative techniques (laser, APC, cryptotherapy) as a palliative treatment for chronic bowel obstruction in advanced inoperable colorectal tumors.

There are only a few cases in which high colon tumors have been traversed with a metal stent and a cost–use analysis is not

19.1 Successful placement of a metal stent



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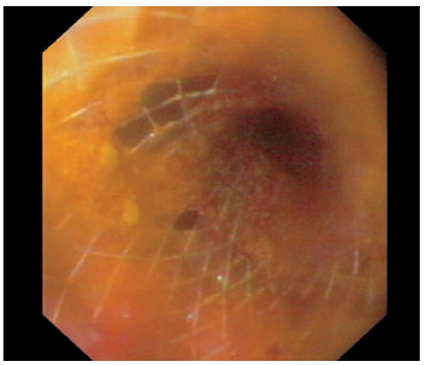

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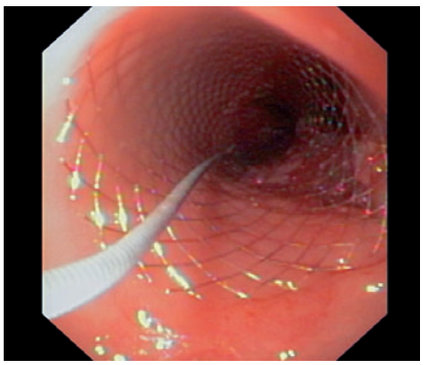
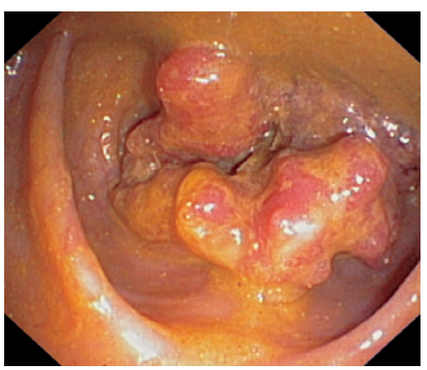


a, b Narrow stenosis caused by rectal carcinoma. Guiding with the rigid guidewire (Jagwire Stiff Shaft, 0.035 inches, 260 cm) (**a**), expansion of the Wallstent Enteral, passage of watery stool (**b**).

c Wallstent Enteral, fully expanded distal end of the stent, near the splenic flexure (60–70 cm from the anus).



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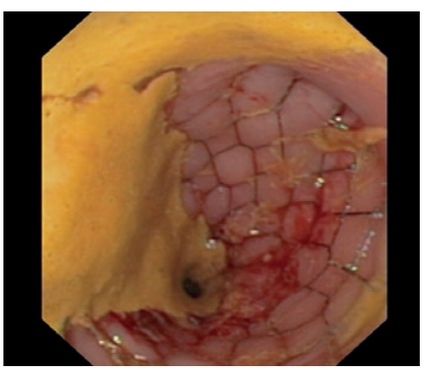
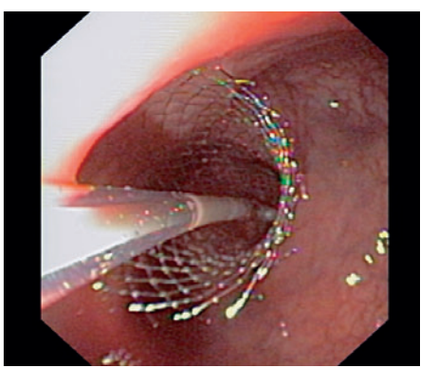
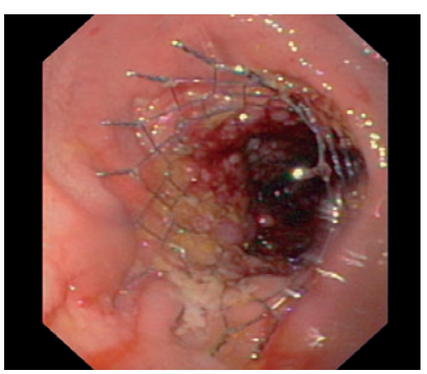


e

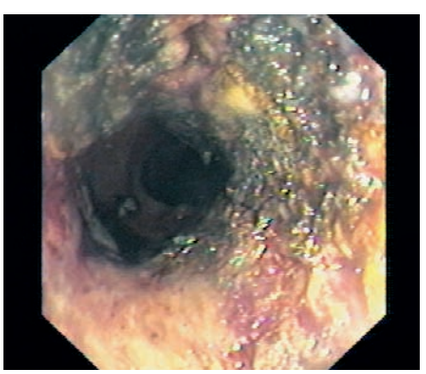


d, e Exophytic rectal carcinoma, partly causing stenosis. No bougienage required prior to stent deployment (**d**). After the stent is opened, immediate passage of watery stool (**e**).

f Wallstent Enteral, distal end of the stent and correct deployment in the rectum.



g



h

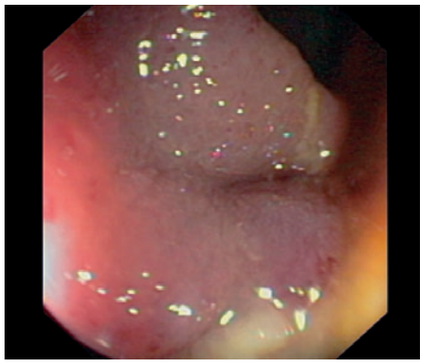
g Ultraflex Precision, distal stent end is open, but not yet completely expanded at the rectosigmoid junction.
h Wallstent Enteral, proximal end of the stent just reaches the tumor edge, APC therapy is necessary.

Table 19.3 Complications of metal stent insertion

Complications	%
Migration	5–6
Pain	10
Mild bleeding	15
Incomplete expansion	3
Ingrowth	4–8

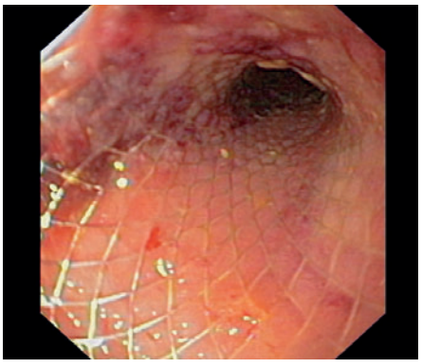
yet possible. Temporary stenting for obstruction (stenosis) caused by a rectal carcinoma until preoperative chemoradiotherapy (down-staging) has been completed can be a viable and sensible alternative.

19.2 Placement of metal stents combined with balloon dilation or bougienage



a

a Stenosis caused by a tumor at the rectosigmoid junction, livid color after balloon dilation.



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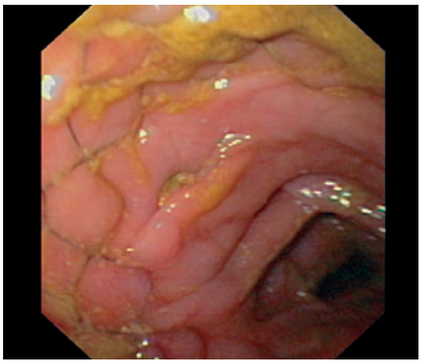
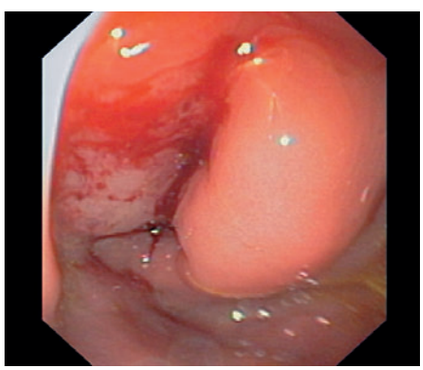


c

b, c Wallstent Enteral, partially expanded (**b**), and fully expanded following balloon dilation (**c**).



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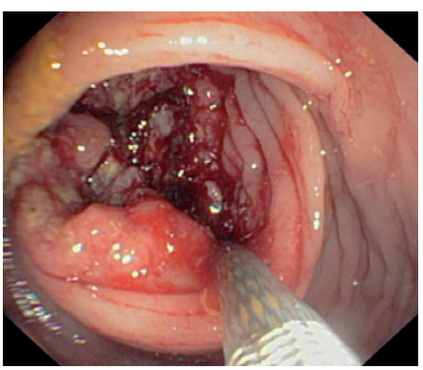

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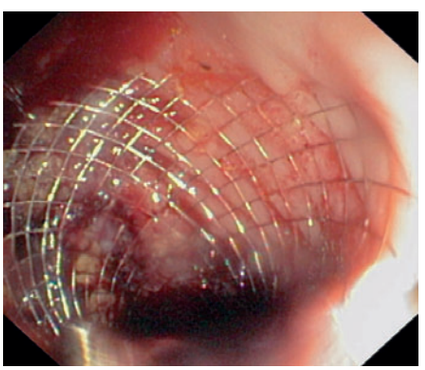
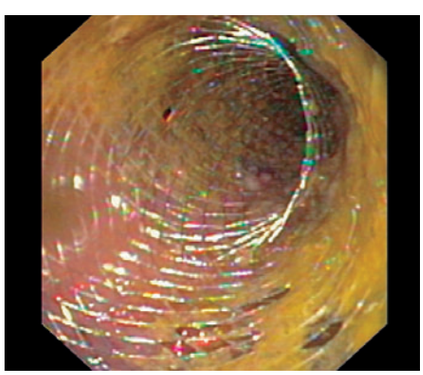


f

d–f 5-cm long stenosis, 12 cm from the anus (**d**). After bougienage, up to 11 mm visualization into an exophytic ulcerating tumor, stent before deployment (**e**). Wallstent Enteral after deployment, distal end fully expanded (**f**).



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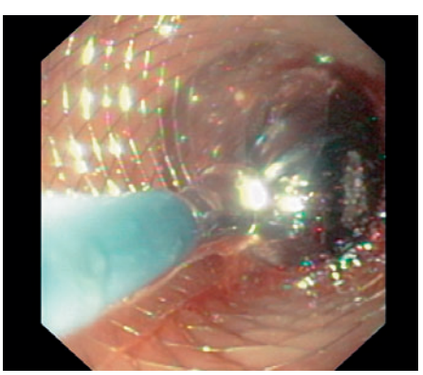
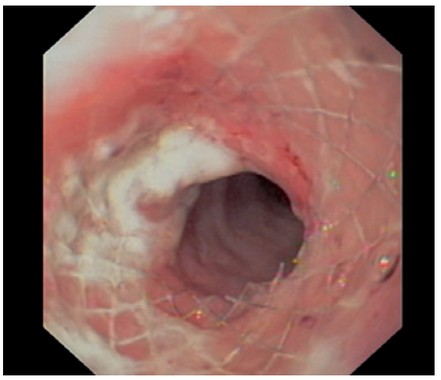


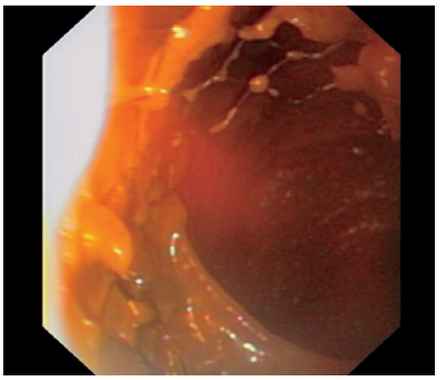
h

g, h Proximal overstenting, stent still insufficiently expanded (**g**), expanded using balloon dilation. Stent after balloon dilation (**h**).

19.3 Stent complications



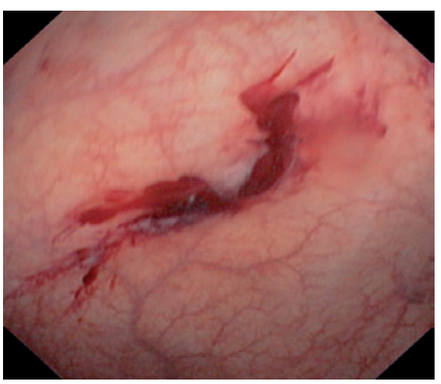
a



b

a Distal migration of Wallstent Enteral; passage with a therapeutic gastroscope impossible.

b Deployment of a 9-cm long Wallstent Enteral to traverse 5-cm long sigmoid carcinoma. The proximal end of the stent (22 mm) does not have complete contact with the dilated intestine.



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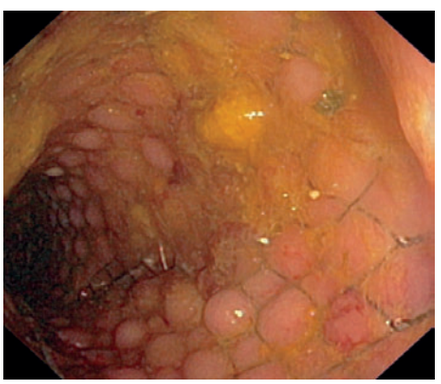

d

c Mucosal tear proximal to the upper end of the stent (Wallstent Enteral).

d Tumor ingrowth, not yet occlusive.

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20 Hemostasis

J. Barnert

The efficacy of endoscopic intervention in treating upper gastrointestinal bleeding and its benefits from a patient perspective have already been shown. Recently, these have also increasingly been demonstrated in lower gastrointestinal bleeding (15). There are basically three methods that are suitable for achieving hemostasis in the lower gastrointestinal tract: thermocoagulation (with and without tissue contact), injection therapy of various agents, and mechanical methods. Regardless which method is used, it is important that before endoscopic intervention, optimal visibility conditions are ensured and, in particular, that the suspected bleeding source is washed off. Some biopolar coagulation probes include an opening at the tip for irrigation as well as the necessary generator have pumps. However, there are also devices (Fig. 20.**1**) that can be attached at the opening of the instrument channel enabling forced irrigation. At the same time, probes can still be introduced into the instrument channel. In situations where vision is obscured, e.g., by stool, blood clots, and heavy bleeding, washing away the stool can be of great help.

Thermocoagulation

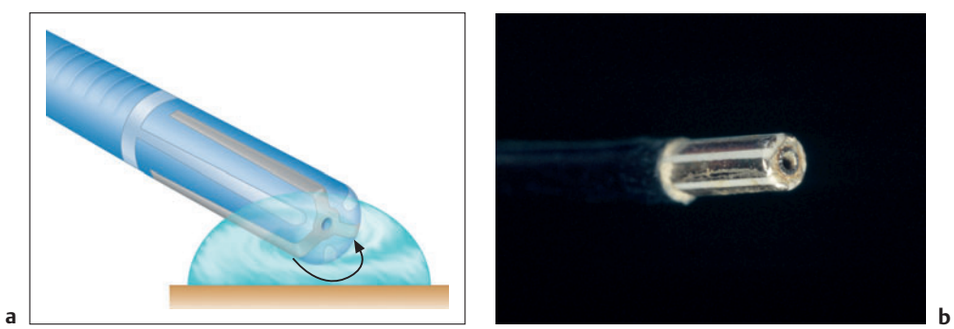
Thermal devices deliver heat directly (heater probe) or indirectly by tissue absorption of light energy (laser) as well as passage of electrical current through tissue (bipolar probe, argon plasma coagulation). Heat application causes edema, coagulation of tissue protein, and contraction of vessels in the tissue and thus hemostasis. Coagulation of tissue occurs at a temperature of at least 70 °C.

Monopolar and Biopolar Electrocoagulation

In biopolar coagulation an electrical current passes through the tissue between the two electrodes contained in the probe tip (Fig. 20.**2**). This requires that the tissue not be desiccated (dried out) as this results in loss of conductivity. Unlike monopolar probe use, the current does not pass through the patient's body, but is limited locally to the targeted tissue area. Loss of conduc-

a **b** **c**

Fig. 20.**1** **Endoscopic irrigation device** (Endowasher, MTW).
a Irrigation from the working channel of the colonoscope.
b Adaptor attachment placed on the working channel to transport irrigation water from the pump into the endoscope.
c Pump.



a **b**

Fig. 20.**2** **Bipolar coagulation.**
a Principle of bipolar coagulation. The electrical current flows (arrow) from the electrode in the probe tip through the tissue to the other electrode.
b ACMI bipolar probe. Steel electrodes lying lengthwise and parallel to one another in the tip.

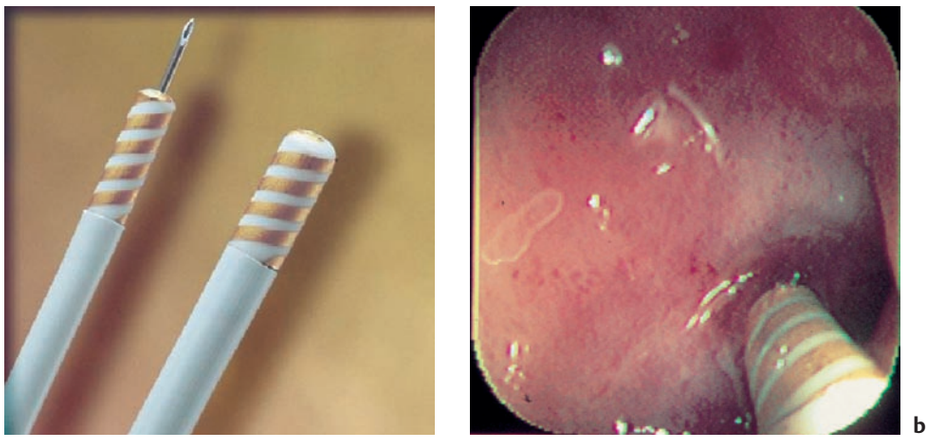


Fig. 20.3 **Injection Gold Probe** (Boston Scientific).
a Probe with electrodes spiraling around the tip. On the left, the built-in needle is extended, allowing, e.g., epinephrine injection to achieve hemostasis and improve visualization. After injection, the needle is withdrawn and coagulation begins.
b Endoscopic view. Injection Gold Probe is placed on the mucosa (images courtesy of Boston Scientific).

tivity as tissue desiccation increases limits depth and breadth of tissue damage as well as maximum temperature (100 °C). Bipolar probes generally have an opening at the tip for irrigation (Fig. 20.**2 b**), the effect of which is two-fold: improved visualization of the bleeding source and formation of a thin film of fluid separating the probe from the tissue and preventing adhesion. If the probe tip sticks to the tissue, the electrical current is interrupted and the probe must be removed from the endoscope and cleaned. Removal of the adhered probe in turn entails the additional risk of tearing tissue and inducing bleeding. One study reports this occurring in 18 % of applications for peptic ulcers (19). Probes are also available which include an extendable needle at the tip (Fig. 20.**3**), allowing quick alternation between injection and coagulation therapy, which can have distinct advantages in critical situations.

Procedure
Bipolar electrocoagulation. When using bipolar coagulation it is advisable to perform point coagulation in a ring around the bleeding source rather than directly placing the probe on the bleeding point, in order to prevent tissue adherence. Bipolar probes offer the advantage of coagulation with the tip as well as tangential coagulation. Coagulation depth depends on the amount of compression force applied with the probe as well as coagulation time. Intensity of current plays a lesser role (9, 12, 18). It should be kept in mind that when treating diverticula or angiodysplasia in the right hemicolon, the bowel wall is thinner and thus increased coagulation effect also increases risk of perforation. Energy must therefore be applied in careful doses. The duration of individual bursts should be limited at the beginning of the procedure to one second and the output of the high frequency generator should be set at 10–15 W. Settings should be adjusted if necessary for desired coagulation effect. The examiner often faces a dilemma between achieving effective hemostasis vs. risk of perforation. Perforation in the right hemicolon occurs in up to 2.5 % of patients using bipolar coagulation (11).

Monopolar electrocoagulation. Monopolar electrocoagulation requires the placement of an indifferent plate (neutral electrode) on the patient's body. The electrical current flows from the probe tip through the patient's body. Some models of monopolar probes also have holes in the probe tip for irrigation, e.g., electrohydrothermal (EHT) probes. In principle, monopolar probes can be attached to any high frequency electrosurgery unit, as in papillotomy and polypectomy. Coagulation depth is greater in monopolar coagulation than in bipolar coagulation (31), though the level of energy application is more predictable in monopolar probes with irrigation at the tip (EHT probes) and it is also less dependent on angle of delivery (30). The problem with monopolar probes is that the electrical current flowing to the neutral electrode "flows away" and thus current density is much higher in the surrounding area compared with the coagulation site. In general, bipolar coagulation appears more appropriate for hemostasis in the colon as perforation risk—at least theoretically—appears lower.

Rebleeding after polypectomy. Postpolypectomy rebleeding can be managed by attempting to ensnare and coagulate the remaining portion of the stalk. This of course requires an adequately long polyp stalk. However, it is a simple method, which does not require extensive preparation and it can be effective in achieving hemostasis even in heavier rebleeding.

Argon Plasma Coagulation (APC)

Argon plasma coagulation (APC) transmits energy from ionized argon gas to the tissue without contact between the probe and tissue. Equipment includes a unit for controlling and regulating the supply of argon gas, a high frequency electrosurgical generator, and a flexible application probe (Fig. 20.**4**). The probe is a 2-mm-thick Teflon tube (Fig. 20.**5**), which is inserted into the endoscope's working channel. Energy delivery from the probe tip can "go around a corner" and there are also probes that emit gas sideways, offering the potential of coagulation parallel to the probe axis.

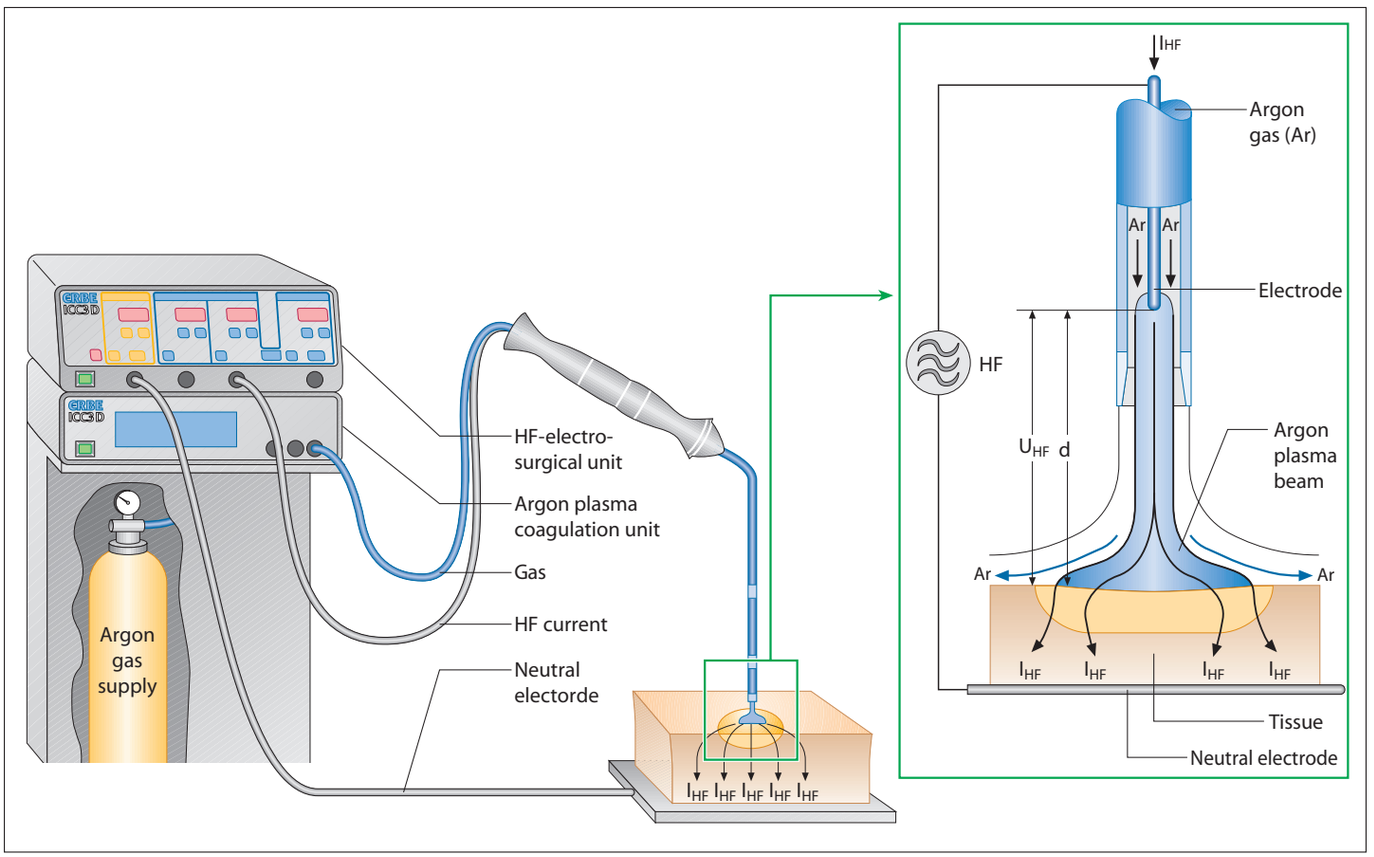
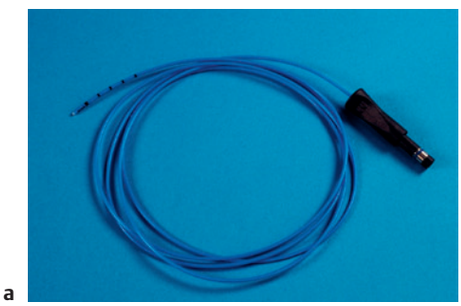


Fig. 20.**4** **APC unit** (Erbe) comprising high frequency (HF) electrosurgical unit, argon plasma coagulation unit, argon gas tank, and probe. The lumen of the Teflon probe contains an electrode surrounded by argon gas. The distal end of the probe has a ceramic tip containing a tungsten electrode. The argon gas is ionized by the high electrical current passing the probe tip, and acts as a conductor. The ceramic tip prevents sticking on inadvertent tissue contact. The high frequency current is applied to the tissue surface via the ionized argon gas.



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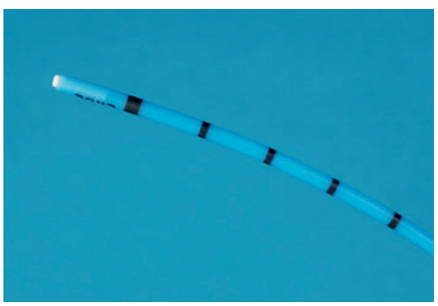


b

Fig. 20.**5** **APC probe** (Erbe).
a Image showing the entire probe device.
b Close-up view of the probe tip.

Procedure
The operative distance is 2–10 mm; at a greater distance, electrical discharge disappears. Energy passes through the body and is returned by a neutral electrode placed on the skin. The argon gas flow blows blood and debris away from the coagulation site, improving visualization. The white–blue arc of light allows targeted and controlled energy application. Coagulation vapor should be continuously suctioned (Fig. 20.**6**). Penetration depth of coagulation is 0.8–3 mm, depending on duration and intensity of energy application. After one second of application, penetration depth is around 2 mm; after five seconds, depth of penetration is around 3 mm. In experiments on swine gastric mucosa, at five seconds of application at all power settings (40–100 W) coagulation remained limited to the mucosa; even at 20 seconds and a power setting of 40 W there was no coagulation effect beyond the mucosa (25). The duration of each individual pulse for clinical use, especially in the colon, should be between 0.5 and two seconds maximum. Depth of penetration is automatically limited by the desiccated tissue layers, which prevent the spread of thermal and electrical energy (29) (Fig. 20.**7**). Nonetheless, APC application should be used with the utmost caution around the particularly thin-walled cecum, in order to avoid perforation (Fig. 20.**8**). Though valid figures on perforation rates are lacking they are likely well below 1 %.

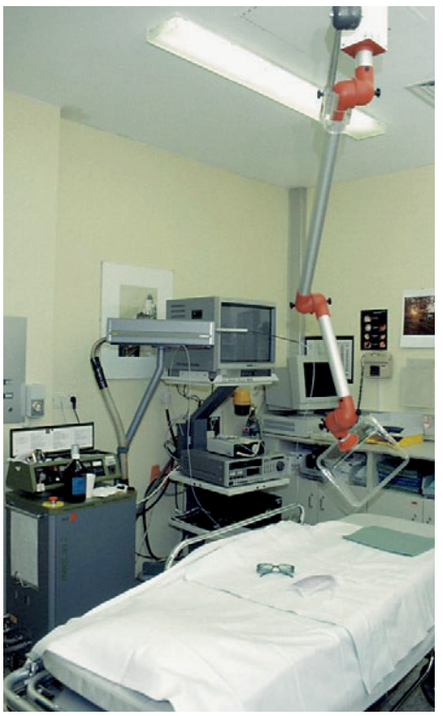


Fig. 20.6 **Device for suctioning vapor** produced during APC or laser application.

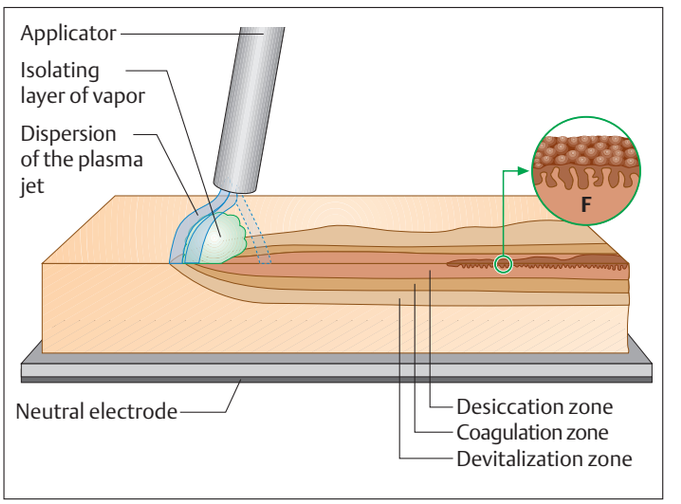


Fig. 20.7 **Depth of devitalization and coagulation zones,** schematic illustration. The evaporating liquid forms a cloud of vapor. Longer-lasting application causes formation of a spongelike dry structure (F) on the surface of parenchymatous organs. The electrically isolating vapor cloud and the formation of a desiccation zone together cause the plasma beam to automatically move over the entire reachable surface (based on 10).

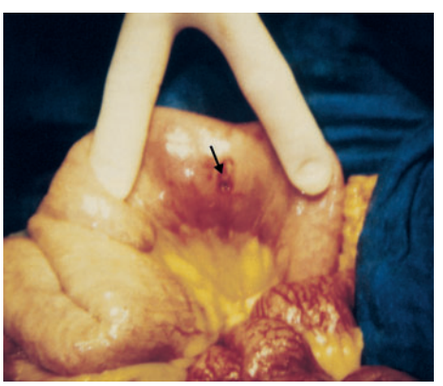
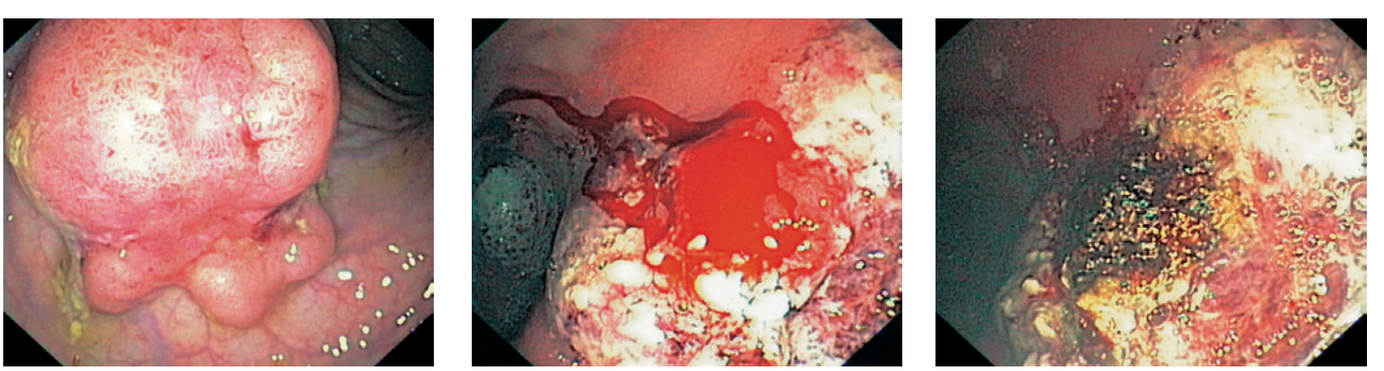


Fig. 20.8 **Perforation from APC application in duodenum.** The perforation site is marked with an arrow.

One drawback to APC is bowel distention caused by argon gas insufflation. This problem can be minimized by reducing the amount of gas flow to below one liter per minute and by intermittent suctioning. Contact between the probe tip and colon wall should be avoided during coagulation as it can force argon gas into the tissue, potentially causing submucosal emphysema.

Complications and potential applications. Even without perforation, argon gas can permeate the colon wall. Occurrence of pneumoperitoneum and pneumomediastinum has been reported. Clinical way of acting is problematic and in such situations excluding perforation is nearly—if not entirely—impossible.

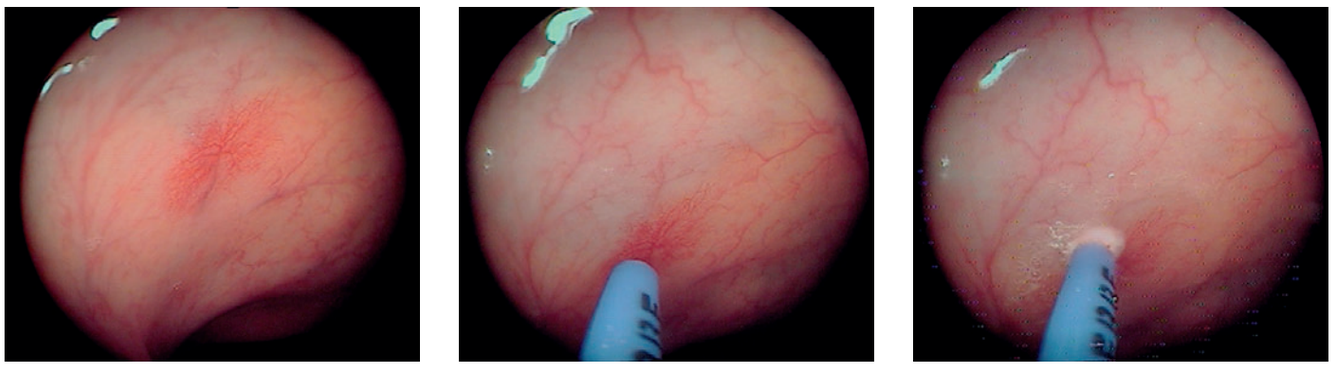
Argon plasma coagulation techniques can be applied in the colon for achieving hemostasis in bleeding angiodysplasias (▣ 20.**1**), diverticular hemorrhage, postpolypectomy rebleeding (Fig. 20.**9**), and tumor bleeding.



a **b** **c**

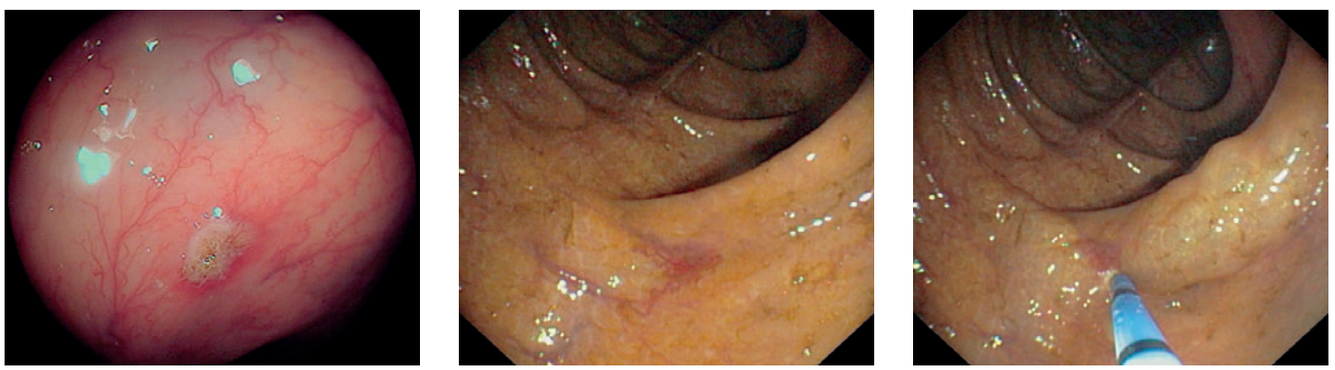
Fig. 20.9 **APC application** (Erbe) in rebleeding after polypectomy.
a Shortstalked rectal polyp.
b Hemorrhage from the polypectomy wound.
c Coagulation at the resection site achieves hemostasis.

20.1 Coagulation of an angiodysplasia using APC (ERBE)

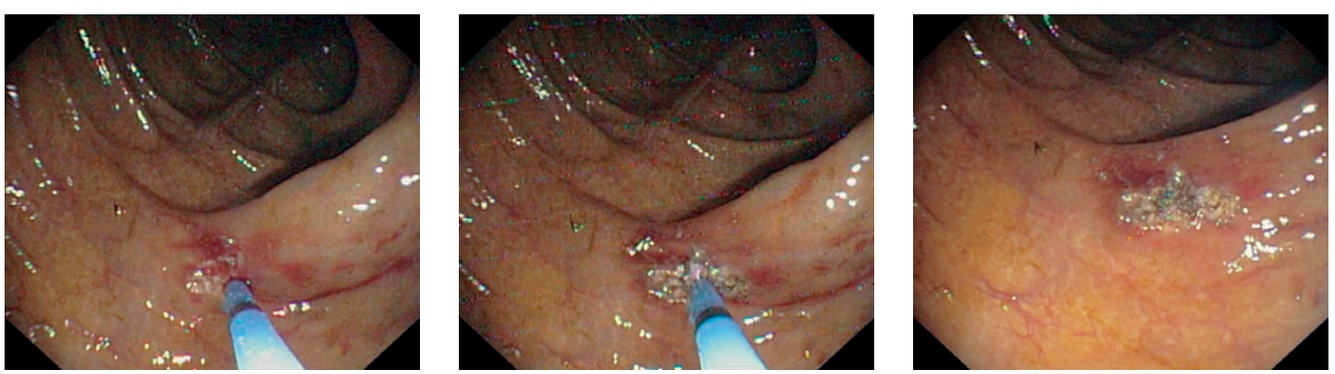


a b c

a–d Coagulation of an angiodysplasia (**a**) in the ascending colon. The probe is extended from the endoscope's working channel to 2–10 mm from the tissue (**b**). Energy application: tissue blanching is a sign of successful coagulation (**c**). After coagulation the angiodysplasia is no longer visible (**d**).



d e f



g h i

e–i Coagulation of an angiodysplasia (**e**) in the ascending colon. Energy application. Starting at the periphery, the vascular malformation is destroyed using brief pulses to effect coagulation (**f–h**). The bluish beam is easily visible at the probe tip (**h**). After coagulation the angiodysplasia is no longer visible (**i**).

Laser

Laser is the acronym for "**L**ight **A**mplification by **S**timulated **E**mission of **R**adiation." A crystal (active medium) is excited by outside energy to emit particularly intense light waves. One side of the crystal is completely reflective while the other is only partially reflective. The reflectivity of the crystal causes a type of chain reaction amplifying the light beam (optical pumping). Only the photons traveling perpendicular to the mirrors are amplified; all others escape to the side. When the beam has reached a specific intensity, it "shoots" through the partially mirrored frontal surface to the outside, creating a nearly parallel band of light which is comprised of only a single color ("monochromatic") and in which the individual light waves vibrate in the same way ("coherent"). The intensity of the beam of light produced is much higher than in normal

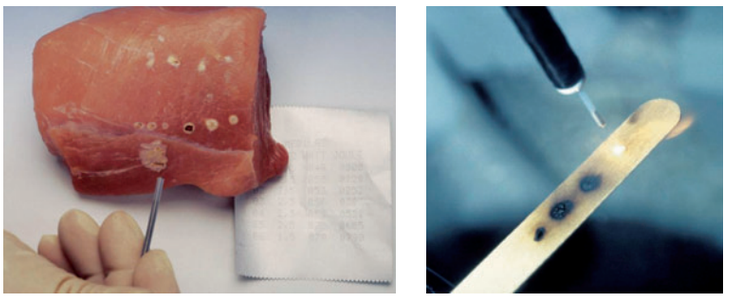


Fig. 20.**10** **Demonstration of the effect of a laser beam.**
a Laser application on a piece of meat. Creation of exact coagulation points.
b Laser application on a wooden stick. Though the beam is invisible, it burns the wood.

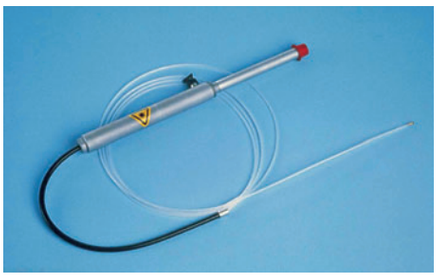


Fig. 20.**12** **Nd:YAG laser optical fiber (diameter: 2.1 mm)** (GE 300 B, MBB). The optical fiber is inserted into the working channel of the endoscope.

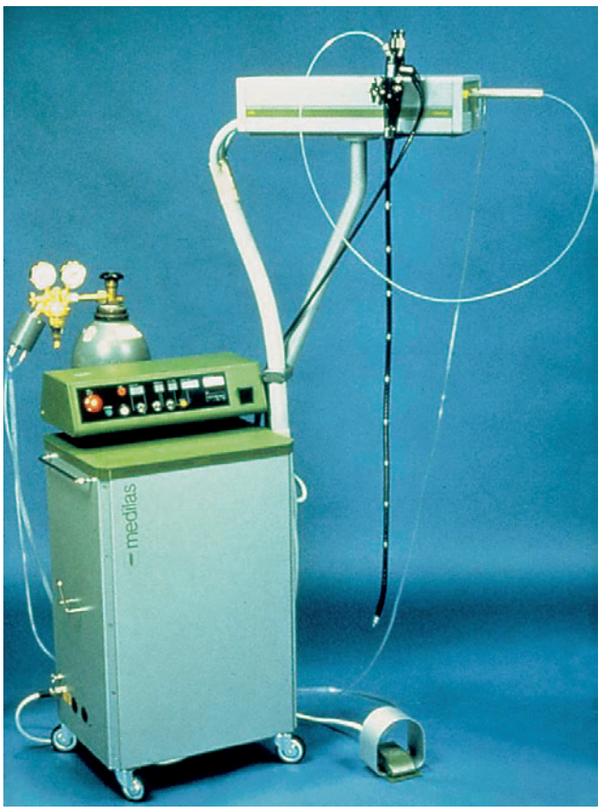


Fig. 20.**11** **Nd:YAG generator** (MediLas, MBB).

mixed-color light (Fig. 20.**10**). A flexible optical fiber transmits the laser beam.

Drawbacks to laser application. Laser application in gastrointestinal hollow organs is not without drawbacks. In the commonly used Nd:YAG laser, one disadvantage is that the optical fiber must be constantly cooled by carbon dioxide. Also, carbon dioxide flow is 0.5 L/min when the laser is inactive and 4 L/min when the laser is activated, which can cause considerable bowel distention.

A further disadvantage is that the laser requires high electrical input and a cool water supply to function and is therefore not mobile. The laser generator (Fig. 20.**11**) is rather large and difficult to transport, clearly limiting its use in emergencies. And, the optical fiber (Fig. 20.**12**) is a relatively sensitive and expensive piece of equipment; the least bit of dust or dirt can diminish output and cause the tip to burn out.

The application of light energy to tissue causes coagulation, which rapidly becomes vaporization without any transition in between. The resulting vaporization of tissue can cause bleeding from opened vessels. Depth of penetration of a single pulse from an Nd:YAG laser is 0.2–6 mm depending on dose compared with 1–2 mm with an argon beam laser (overview in 11). Penetration depth of energy emitted by the argon laser (488–514 nm) is limited by light absorption by the hemoglobin. The deeper necrosis achieved with an Nd:YAG laser may be desirable for tumor ablation, but it makes application in thinly walled hollow organs such as the right hemicolon unpredictable, explaining its not inconsiderable perforation risk. The risk of perforation in Nd:YAG laser treatment of angiodysplasias in the colon, for example, is 6% (24). Larger lesions requiring several pulses can pose greater risk. Rutgeerts et al. (24) recommend limiting energy to 200 J. A study on use of argon beam laser in treating angiodysplasias reported perforation in the right hemicolon in one patient (4). The absorption of light from an argon beam laser makes its application less prudent in bleeding situations.

Another problem with laser application is that the depth of damage increases after a few days, reaching its maximum in animal studies after seven days (17). This can cause bleeding from resulting ulcerations.

Procedure
Laser application can be a contact or noncontact procedure. The sapphire or ceramic tips placed on the end of the optical fiber allow contact with the tissue, which increases intensity of the laser beam. In noncontact methods, the coagulating beam is invisible (Fig. 20.**10**); but a so-called aiming beam allows exact targeting of the Nd:YAG laser.

It is impossible to make general recommendations for laser settings. One important point to remember is that penetration depth depends on power setting (W) and length of laser pulse (seconds), i. e., transmitted energy (amount of heat) (J) whereby 1 Ws = 1 J. The maximum depth of the defect caused by the MediLas Nd:YAG laser (MBB), which we use in our clinic, for example, is 4.5 mm (50 W output, 1.8-mm beam diameter, three seconds). The lowest power setting on this particular model is 15 W.

Fig. 20.**13** **Protective goggles for working with laser.**

Fig. 20.**14** **Heater probe (Olympus)** (image provided courtesy of Olympus).

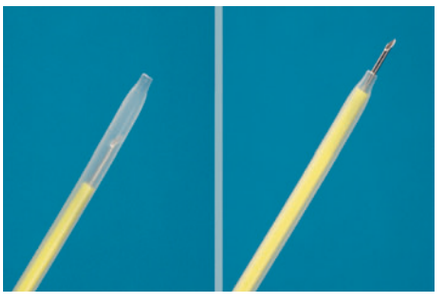


Fig. 20.**15** **Injection needles** (disposable needles) for endoscopic injection therapy. On the left, the tip is retracted into the Teflon sheath; on the right, the needle is extended (needle extension length: 4 mm).

When beginning therapy of vascular malformations, the endoscopist should carefully "approach" the optimal dose. For applications in the colon, attention should be paid to using a low power setting, maintaining sufficient distance between probe tip and tissue (> 5 mm) and avoiding overdistention of the colon with subsequent thinning of the bowel wall.

It should be noted that toxic vapors arising from tissue vaporization should be suctioned (Fig. 20.**6**). Special protective glasses (Fig. 20.**13**) must be worn by the examiner, assistants, and patient in order to prevent accidental retinal burn.

Heater Probe

The heater probe tip consists of a Teflon-coated hollow aluminum cylinder with a heating coil inside (Fig. 20.**14**). The Teflon coating is designed to prevent the probe from adhering to tissue. Temperature at the tip is constant. The most recent model from Olympus (HeatProbe) delivers thermal energy in two strengths (diameters of 3.7 mm and 2.8 mm), at six different heat levels (5–30 J), and includes an opening in the tip for washing off the mucosa. Heater probe therapy has been used successfully for various causes of bleeding in the colon including diverticular bleeding, angiectasia, radiation proctitis, and Dieulafoy lesions. Coagulation depth achieved using a heater probe is similar to bipolar coagulation (30). The mechanism is different, however, as the heat is produced in the probe tip and transmitted directly to the tissue. It should be noted when comparing the two methods that electrocoagulation induces heating of the tissue by electrical current. The heater probe has proved effective for treating diverticular bleeding (15) and angiodysplasias in the colon (16). Use of heater probes vs. bipolar coagulation has only been compared for radiation-induced angiodysplasia in the rectum (14). Efficacy and safety of both methods were equal. Various thermocoagulation methods have been compared for bleeding peptic ulcers (7, 8, 13, 20) and all methods were equally effective.

Procedure
The potential for compressing the bleeding source with the probe is seen as one advantage of using a heater probe. Some recommendations advise pressing the probe firmly against the bleeding source and then beginning coagulation, thus improving "sealing" of the vessel or the blood-filled cavity. Others recommend coagulating a small ring of points around the vessel to avoid tearing adhered tissue when retracting the probe, which can cause rebleeding.

Complications. The perforation rate associated with heater probe application is between 1.8 % and 3 %; the risk of bleeding is reported at 5 % (6, 8, 22). However, these figures are based on bleeding peptic ulcers. There are no corresponding figures for use in the colon.

Injection Therapy

Injection therapy is a simple, inexpensive, and relatively uncomplicated method for achieving hemostasis. Injection needles consist of a Teflon sheath with an extendable needle (21–25 gauge) at the tip (Figs. 20.**15**, 20.**16**). For therapy in the colon, we generally use needles with a needle extension length of 4 mm in order to limit depth of penetration.

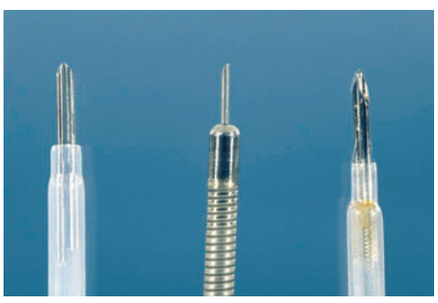


Fig. 20.**16** **Various models of injection needles.** The two outer needles have Teflon sheaths, while the needle in the center has a metal sheath.

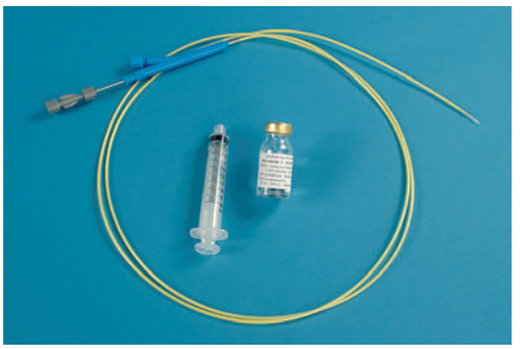


Fig. 20.**17** **Endoscopic accessories for hemostasis methods using injection therapy.** An injection needle, epinephrine solution (1 : 10 000), and a disposable needle are required.

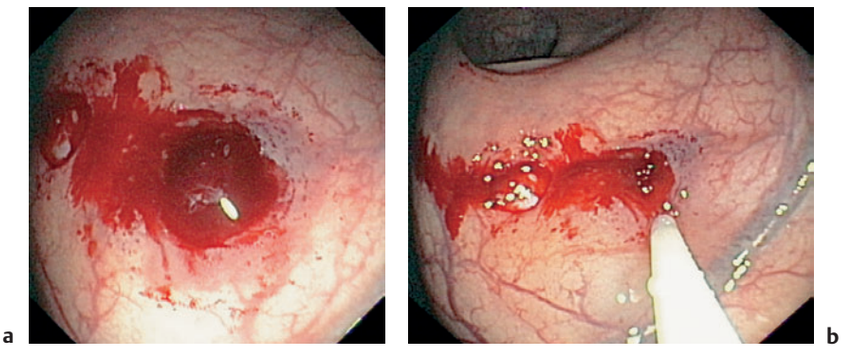


Fig. 20.**19** **Rebleeding following excisional biopsy.**
a Bleeding at the biopsy site.
b One ml of epinephrine (1 : 10 000) is injected underneath the biopsy site. The injected fluid spreads around the injection site, slightly elevating the mucosa.

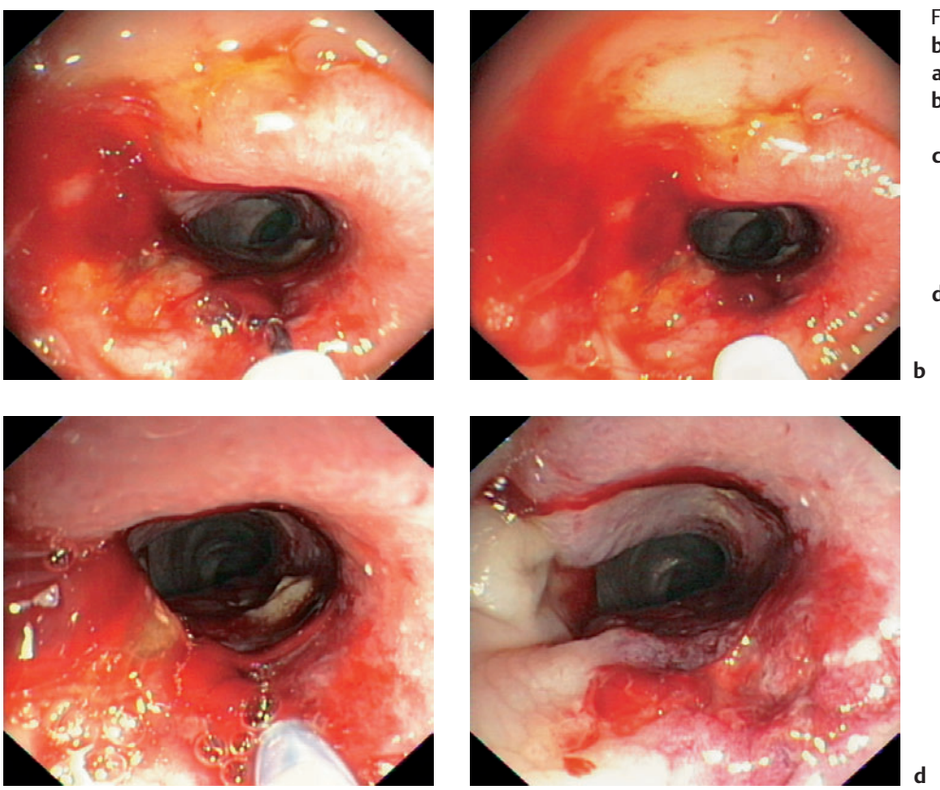
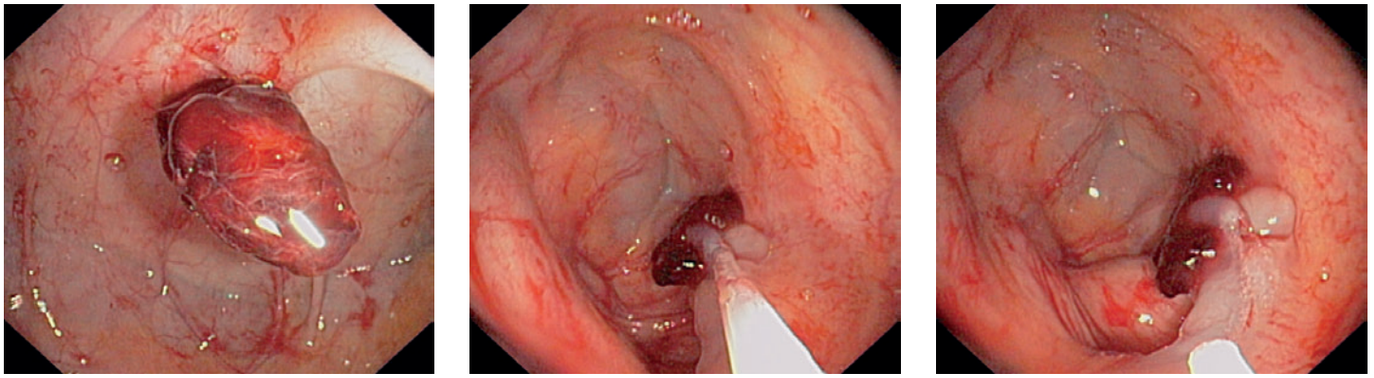


Fig. 20.**18** **Epinephrine injection of a bleeding rectal carcinoma.**
a The needle is extended.
b The extended injection needle is inserted into the bleeding tissue.
c Epinephrine is injected underneath the bleeding point. It is advisable to use individual doses of 1–2 mL of epinephrine and if necessary to make several injections at various points.
d Cessation of bleeding. The tissue has a glassy, white–pale appearance.

Procedure

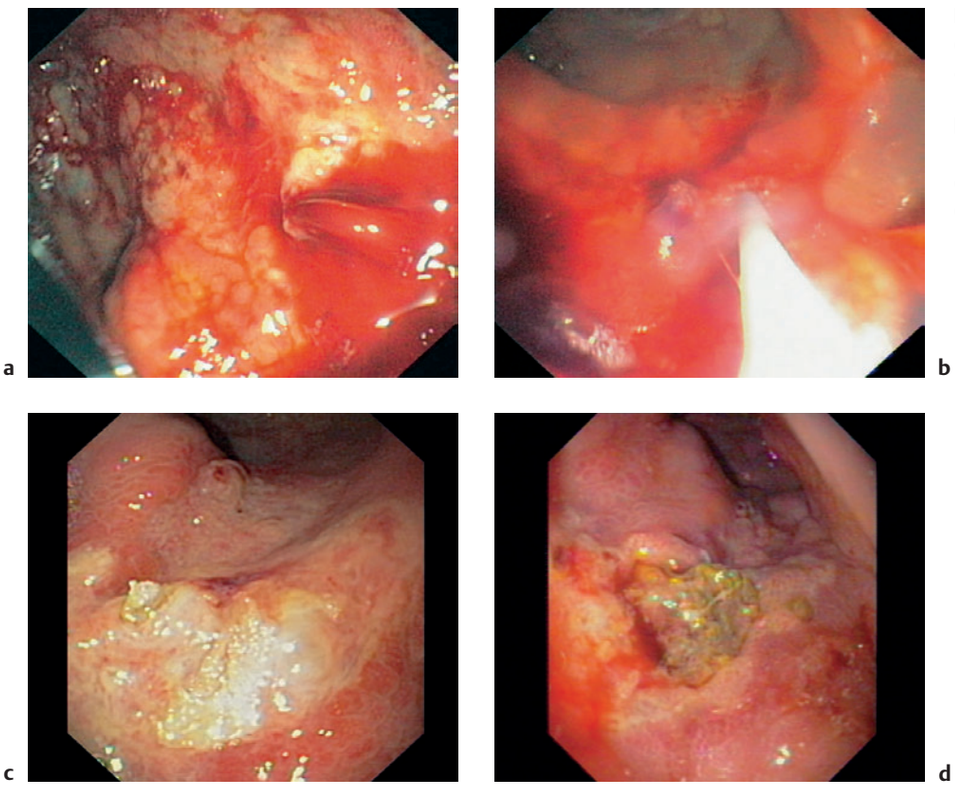
The injection needle is advanced through the endoscope's instrument channel. Epinephrine (synonyms: Suprarenin, adrenaline) injection (dilution 1 : 10 000 to 1 : 100 000) is used for achieving hemostasis (Fig. 20.**17**). Injection of bleeding vessels is made at three to four points at 1–2 mm from each other around the bleeding point. The injection achieves hemostasis by both a vasoconstriction effect and also compression of the vessel resulting from a "cushion" effect from the injected fluid. Individual injection dose should be as low as possible (1–2 mL, 1 : 10 000 dilution) as the absorption of catecholamine has systemic effects. Tachycardia, arrhythmia, and hypertensive emergency have been reported following submucosal epinephrine injection (28). As a rule, a total of 4–16 mL (1 : 10 000 dilution) can be injected in upper gastrointestinal bleeding (23); most of the epinephrine is metabolized in first pass hepatic metabolism. Adoption of these recommendations for lower gastrointestinal bleeding appears sensible.

Injection therapy with diluted epinephrine can basically be used to achieve hemostasis of any bleeding lesion in the colon, i. e., diverticular bleeding, tumor bleeding (Fig. 20.**18**), and rebleeding following polypectomy or biopsy (Fig. 20.**19**). In many cases, it is a good idea to first inject epinephrine to stop bleeding and improve visualization. For adherent clots (Fig. 20.**20**), it is advisable to first inject



a b c

Fig. 20.**20** **Rebleeding after APC therapy of an angiodysplasia in the ascending colon.**
a Adherent clot on the lesion.
b Epinephrine (1 : 10 000) is injected underneath the clot.
c Following injection the mucosa is elevated and whitish in color. Afterward a hemoclip is applied to the lesion (not shown here). There was no further rebleeding.



a b c d

Fig. 20.**21** **Acrylic glue (Histacryl) injection of a bleeding rectal varix.**
a Spurting bleeding from a rectal varix in a patient with liver cirrhosis.
b The varix is injected with acrylic glue (Histacryl).
c Cessation of bleeding after injection.
d Ulcer at the injection site found during repeated endoscopy a few days later.

epinephrine underneath the clot in order to better remove it without risking immediate hemorrhage. In a second step, coagulation or mechanical hemostasis methods can be applied if necessary.

Injection of other agents. In addition to epinephrine, other agents, such as absolute alcohol, have also been used in injection therapy. The effect of these agents, however, is not superior to epinephrine and, moreover, they can cause mucosal ulcers and thus perforation. Sclerosing agents have been used for obliteration of angiodysplasias. Marwick and Kerlin (21) used 1.5% Sodium tetradecyl sulfate on angiodysplasias in the upper gastrointestinal tract. Bemvenuti and Julich reported on sclerotherapy of angiodysplasias with ethanolamine (2). Polidocanol (1%) could also conceivably be used as a sclerosing agent. Injection therapy using sclerosing agents is less predictable than thermocoagulation making the latter preferable in our opinion.

Bleeding from rectal varices. Bleeding from rectal varices can often only be stopped by injection of a cyanoacrylate tissue glue (Histacryl, Glubran), similar to gastric fundus varices and esophageal varices (Fig. 20.**21**). Mucosal ulcers caused by extravascular injection can be problematic (Fig. 20.**21 d**) and can result in rebleeding and other problems. The equipment used is the same as for esophageal varices (Fig. 20.**22**). Only the use of Glubran is approved for this indication at least in Germany, though our own use of Histacryl for more than a decade mainly in the treatment of esophageal varices and gastric fundus var-

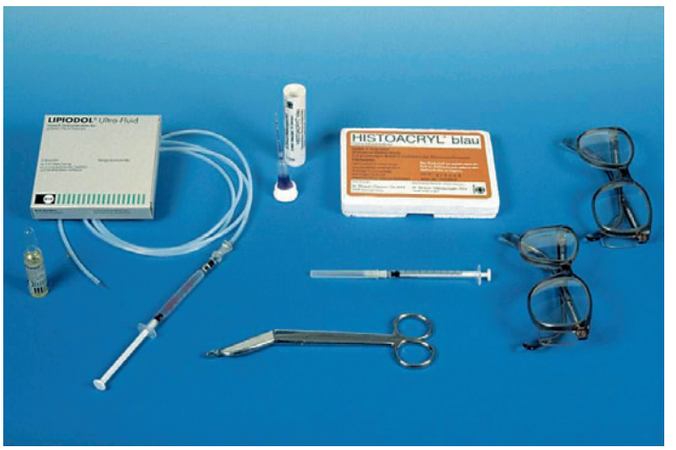


Fig. 20.**22** **Accessories for acrylic glue (Histacryl) therapy.** Acrylic glue (Histacryl) vials, Lipiodol solution, an injection needle, and a disposable needle are required. It is important that all examiners and assistants wear protective glasses to prevent injury in case acrylic glue (Histacryl) accidentally spurts out of the injection needle. Lipiodol should be injected through the needle before and after the injection of acrylic glue (Histacryl).

Fig. 20.**23** **Hemoclip loaded on application device** (Olympus) (image provided courtesy of Olympus).

ices has proved itself. Although it has been recommended that the injection needle be flushed with distilled water prior to Histacryl, in our own experience we found the use of Lipiodol more appropriate. Generally, 0.5 mL of Lipiodol is required to fill the injection needle. Lipiodol is then mixed 1:1 with Histacryl and this mixture is injected, in several portions if necessary. Afterward, the injection needle is again filled with Lipidiol, in the previously determined amount, and injected, thereby transporting any remaining Histacryl into the vessel (varix).

Mechanical Methods

Hemoclips

There are a number of reasons which make metal clips an attractive alternative to the more common methods of hemostasis. First, clips allow definitive and secure closure of bleeding vessels (3) and the endoscopist can immediately recognize whether a vessel has been occluded. Another important aspect is that no complications have been reported so far. In a comparative study by Chung et al. (5) on patients with Dieulafoy lesions, some of which were in the colon, the initial hemostasis effect of hemoclipping was clearly superior to injection therapy and rebleeding was less frequent. Hemoclips are manufactured by Olympus and are comprised of stainless steel ribbons. The clips do not interfere with magnetic resonance imaging (MRI). The delivery catheter (Fig. 20.**23**) is available in various lengths (165, 195, and 230 cm) and diameters (2.8 and 3.2 mm) allowing placement in the working channels of most flexible endoscopes. The catheter can also be rotated which allows optimal orientation of the clip delivery position. Clips are available in different versions with various angles of the jaws (90°/135°) and lengths (4, 6, and 8 mm).

Procedure
The clips are loaded onto the clip application device and retracted into the device sheath before it is placed in the endoscope. The sheath is then advanced through the working channel. When the tip of the application device is protruding from the end of the endoscope, the hemoclip can be advanced and opened. The clip should be pressed gently against the bleeding source and then closed (Fig. 20.**24**, ▣ 20.**2**). Afterward, it is released from the application device. Procedures for clipping a vessel can vary (Fig. 20.**25**). It may be possible to stop bleeding, i. e., to occlude a visible vessel with a single hemoclip, (Figs. 20.**26**, 20.**27**). However, it may also be necessary to use several clips to achieve optimal hemostasis (Fig. 20.**28**). Important requirements for hemoclip application are good visibility of the bleeding source and accuracy. Tangential application is also possible.

The hemoclip usually falls off after some time. This does not cause tissue damage or ulceration. The only disadvantage is that hemoclips that have fallen off and are suctioned with an endoscope can clog the working channel of the instrument and lead to costly repairs.

Band Ligation

Ligation of hemorrhoids is a proved, simple, and inexpensive treatment method (Fig. 20.**29**).

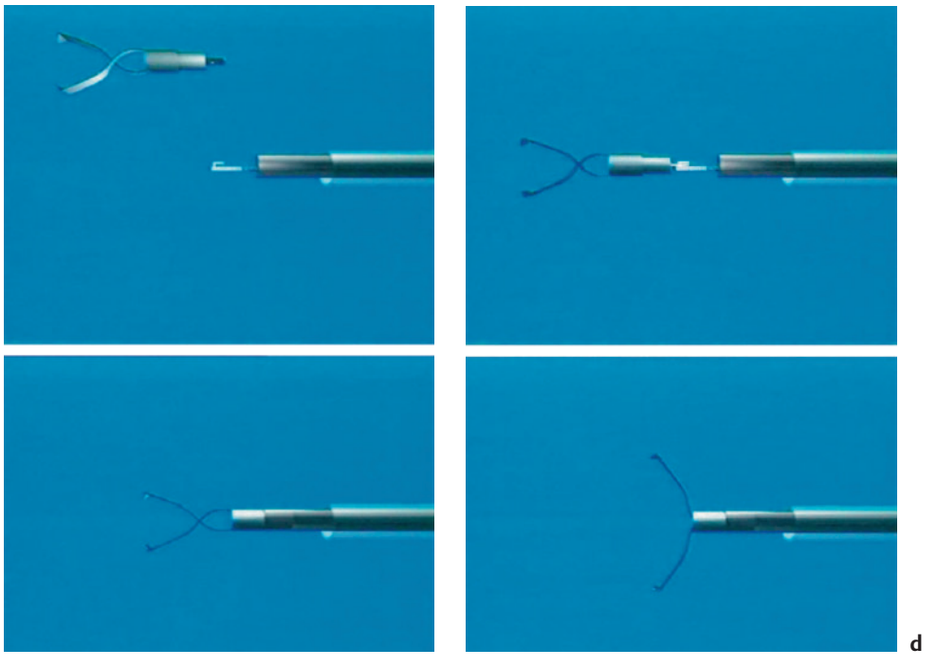


Fig. 20.**24** **Loading and deploying a hemoclip** (Olympus).

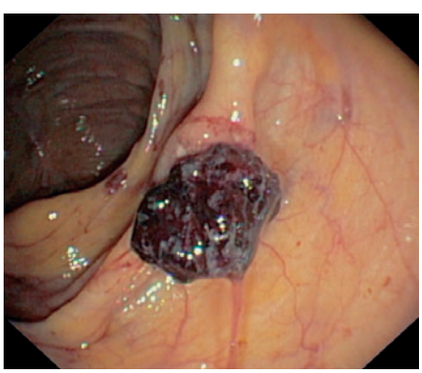
a The clip is hung on the applicator's extended hook.

b Next the clip is drawn into the applicator sheath, thereby closing the clip. The applicator is then advanced through the working channel of the endoscope.

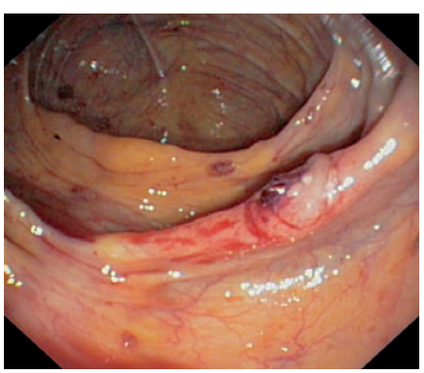
c When the tip of the applicator is protruding from the endoscope the clip is advanced. The clip opens on leaving the applicator sheath.

d The clip is then opened at the instrument handle. Preloaded disposable clip applicators are also available (images provided courtesy of Olympus).

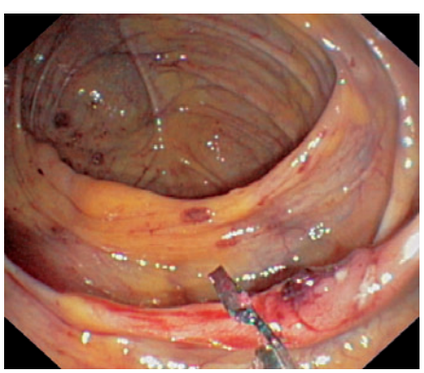
20.2 Endoscopic hemoclip application



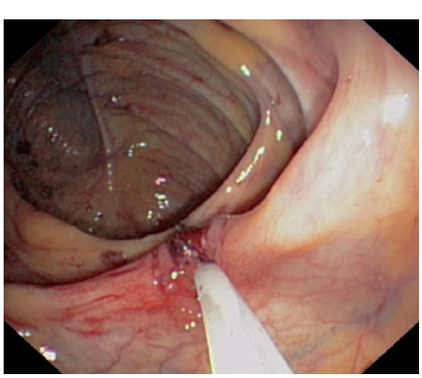
a Blood clot at a polypectomy site in the ascending colon.



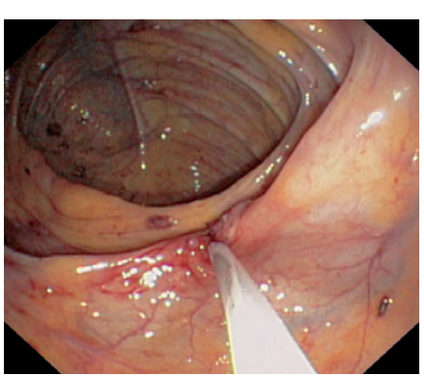
b Visible vessel after washing off the clot.



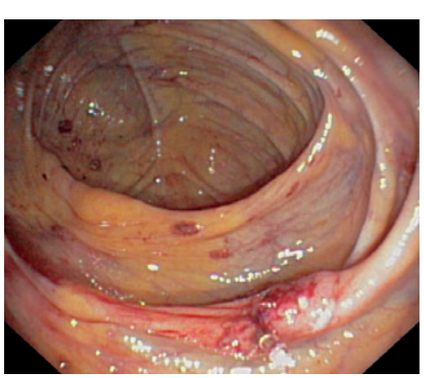
c The opened hemoclip (Olympus) is advanced to the polypectomy site.



d The opened hemoclip is gently placed on the visible vessel.



e The clip is closed on the visible vessel.



f The clip has been closed and is released from the applicator. The hemoclip seals the visible vessel at the polypectomy site.

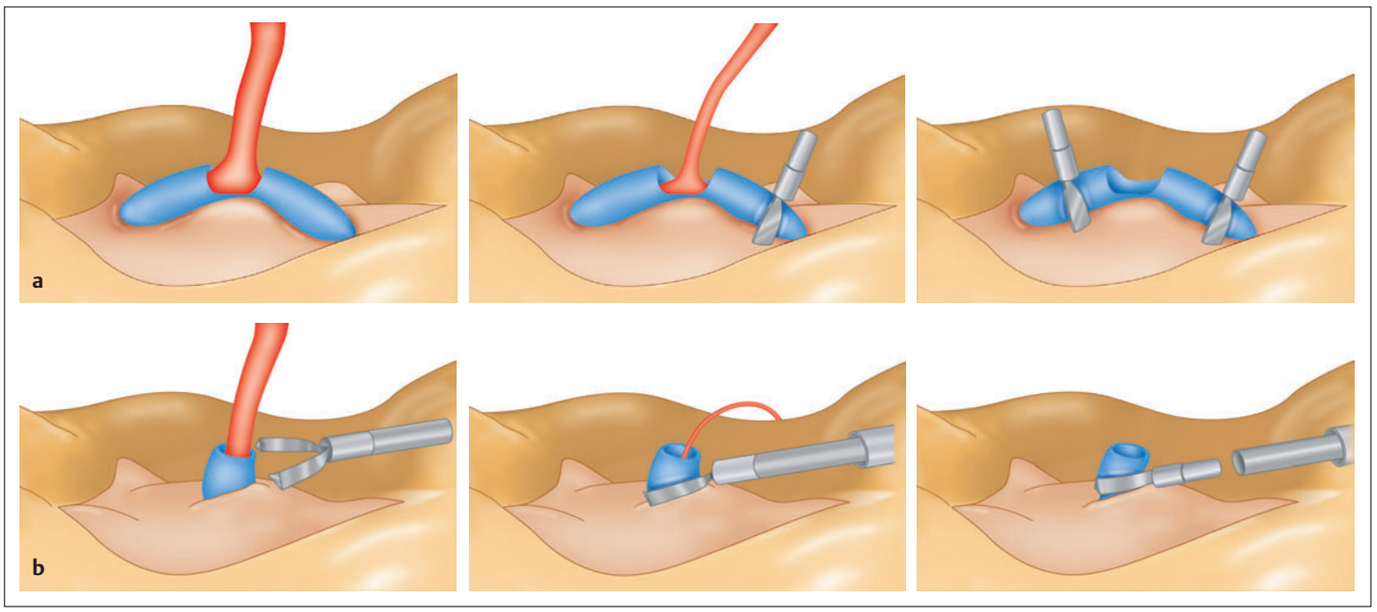


Fig. 20.**25** **Possible clip applications for spurting vascular hemorrhage.**

a The vessel demonstrates lengthwise erosion. A clip is used to close the vessel on either side of the bleeding point.

b Bleeding from the open end of a visible vessel. The vessel is occluded underneath the bleeding point with a clip (illustrations based on images provided courtesy of Olympus).

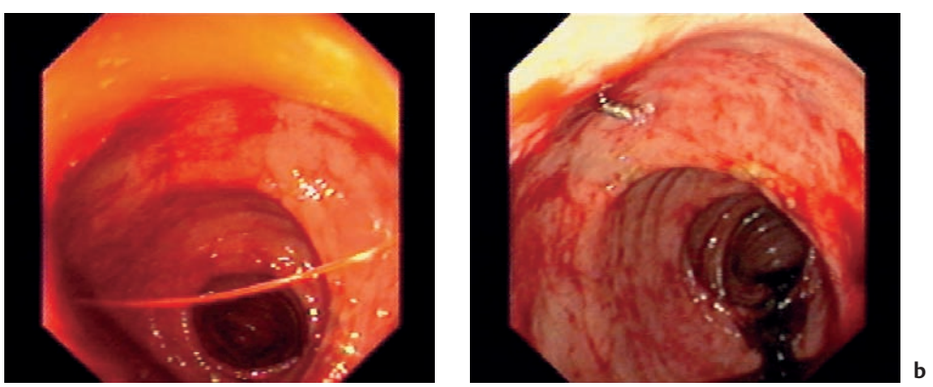


Fig. 20.**26** **Bleeding from a rectal ulcer.**

a Spurting bleeding.

b Bleeding source occluded with application of a single clip (Hemoclip, Olympus).

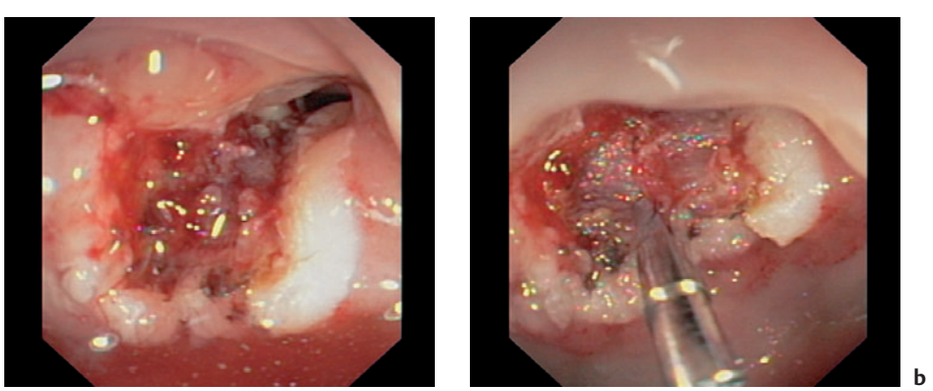


Fig. 20.**27** **Visible vessel.**

a Polypectomy site with a protruding visible vessel.

b Prophylactic application of a clip closes the potential bleeding source.

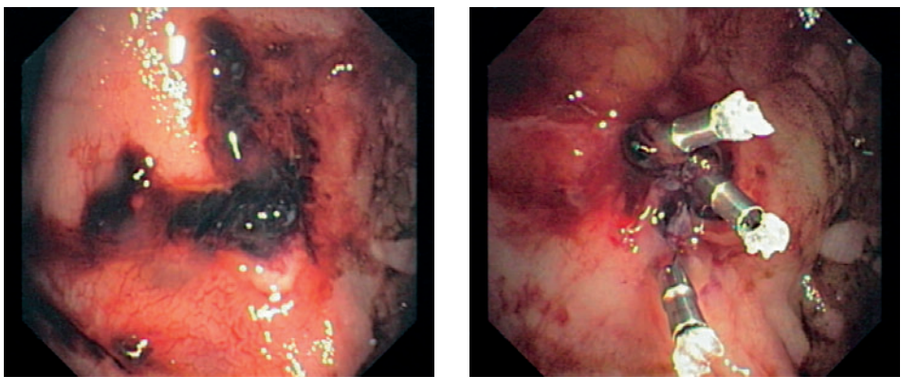


Fig. 20.**28** **Several visible vessels.**
a Several visible vessels protruding from a polypectomy site.
b Application of three clips definitively seals the bleeding sources.

Procedure for hemorrhoid ligation
Under visualization with a proctoscope, suction is applied using a special applicator (Fig. 20.**30**) to an internal hemorrhoid localized above the dentate line. A rubber band is then placed around the base of the suctioned hemorrhoid pile, ligating it (Figs. 20.**29**, 20.**31**). The band falls off after a few days leaving a small ulceration. Complications include pain, rebleeding, or, in rare cases, abscess formation. After about three weeks, the ablation site has healed and the next session can proceed.

Other bleeding sources. Bleeding sources in the colon can also be treated using technology and instrumentation commonly used for treating esophageal varices. The ligation device consists of a transparent cap loaded with the rubber bands and mounted on the endoscope tip, a trip wire allowing the deployment of ligatures, and a deployment mechanism that is connected the accessory attachment of the working channel. For using a commercially available ligation device (Sixshooter, Boston Scientific) with a colonoscope, the trip wire must be lengthened (33). The bleeding source and mucosa are suctioned and the ligating band deployed. After a few days, the necrotic area falls off, leaving a flat ulceration. Use of band ligation has been reported in angiectasias in the upper gastrointestinal tract (34), diverticular bleeding (32), and postpolypectomy bleeding (1, 26, 27). However, it is not clear whether simpler endoscopic alternatives could have been used in these cases. One has to remember that loading the device requires withdrawing from the colon and then advancing the instrument again in the colon, this time under decreased visibility due to the cap. After the necrotic tissue has been sloughed off, the ulcers underneath may bleed.

Endoloop

A detachable nylon snare (Endoloop) is available from Olympus. During endoscopy, the tissue is ensnared and ligated by the Endoloop, which then detaches from the instrument. The chief indication is prophylactic, before the removal of larger polyps (Fig. 20.**32**), in order to prevent rebleeding. The snare can also be used to ensnare and ligate a bleeding polyp stalk (see also Chapter 18).

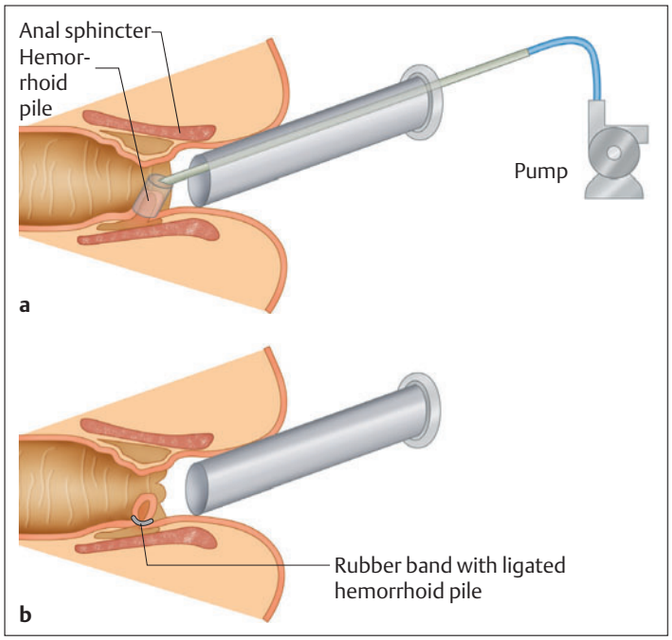


Fig. 20.**29** **Schematic illustration of ligation of internal hemorrhoids.**
a Suction is applied to the internal hemorrhoid and a rubber band is placed over the hemorrhoid.
b Hemorrhoid ligated with rubber band around its base.

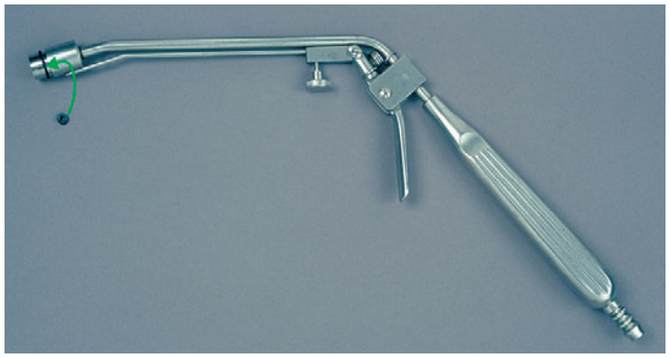
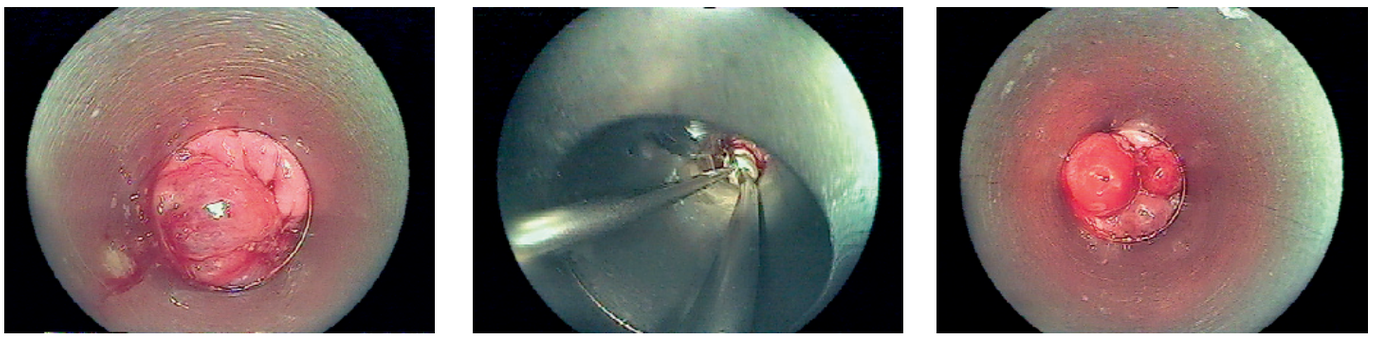


Fig. 20.**30** **Application device for rubber band ligation of hemorrhoids (Paul Drach).** The tip of the applicator contains an inner metal ring inside an outer metal ring. The rubber band is placed on the inner metal ring that is in front of the outer ring. The applicator handle has an attachment for a tube connecting it to a suction pump.



a b c

Fig. 20.**31** **Ligation of internal hemorrhoids.**
a Proctoscopic view of the hemorrhoid pile.
b The application device (Fig. 20.**30**) is advanced through the proctoscope. Suction is applied to the hemorrhoid pile. The forward motion of the outer metal ring loops the rubber band over the suctioned hemorrhoid pile.
c View of the ligated hemorrhoid pile.

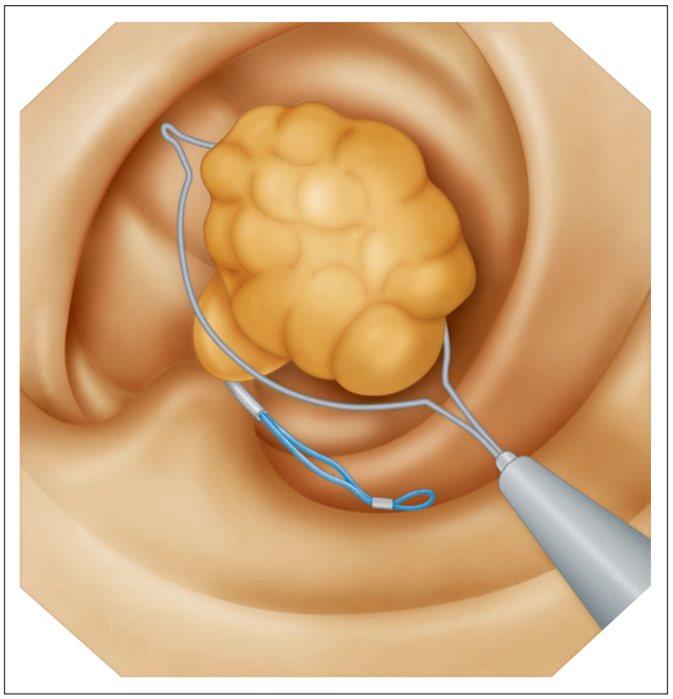


Fig. 20.**32** **Endoloop (Olympus).** Detachable snares are usually used prophylactically. The Endoloop is lassoed around the polyp stalk and closed to ligate the stalk. The Endoloop can also be used for postpolypectomy ligation of a bleeding stalk remnant (image based on illustration provided courtesy of Olympus).

Procedure for Endoloop application
First, the round or elliptical nylon snare is grasped with a small hook and retracted into the delivery system. The delivery system is then advanced in the working channel of a flexible endoscope. At the application site, the snare is advanced out of the shaft and unfolds. The snare is lassoed around the polyp stalk and then closed. It is important that the snare not be closed too tightly as this may cut through the stalk. The snare is then detached from the delivery device.

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21 Management of Benign Strictures

R. Scheubel

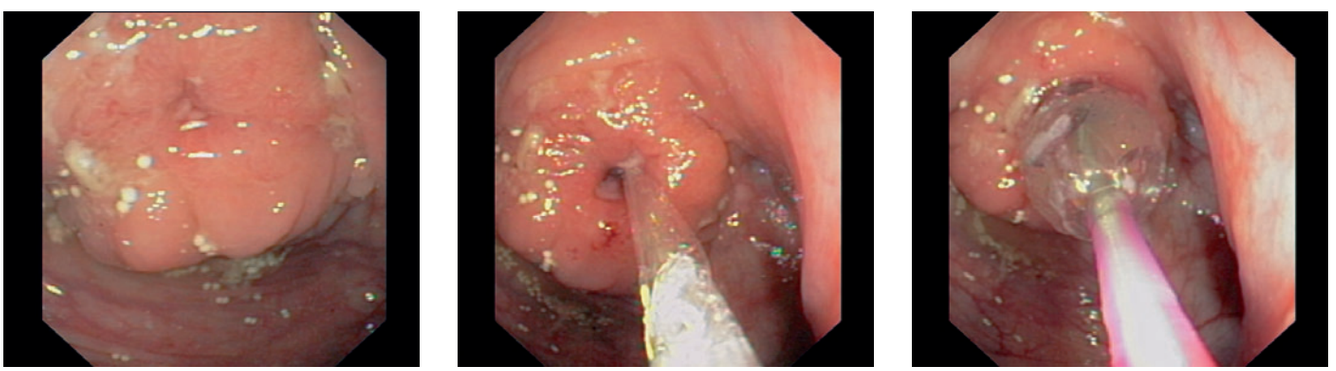
Definition and Causes

Benign stenoses can be caused either by inflammatory healing processes involving scarring (mainly associated with Crohn disease, but also ischemic colitis and NSAID [nonsteroidal anti-inflammatory drug] colitis) or they may occur as postoperative stricturing.

Colitis. Stenosis is particularly common in fibrostenotic Crohn disease. Depending on the pattern of colon involvement, strictures can involve the terminal ileum, the Bauhin valve, or other colon segments. Strictures may appear with scarring and a smooth mucosa or they may also have variously deep ulcers if occurring in a phase of florid inflammation. Stricture formation has also been associated with rapid healing processes following intensive anti-inflammatory therapy (e.g., anti-TNF antibodies). Typically, stenosis formation has a predilection for the anastomosed region around ileocolostomies (▣ 21.**1 a**, **h**). Stenosis in a confirmed diagnosis of ulcerative colitis, however, always raises the index of suspicion for malignancy and must be operated.

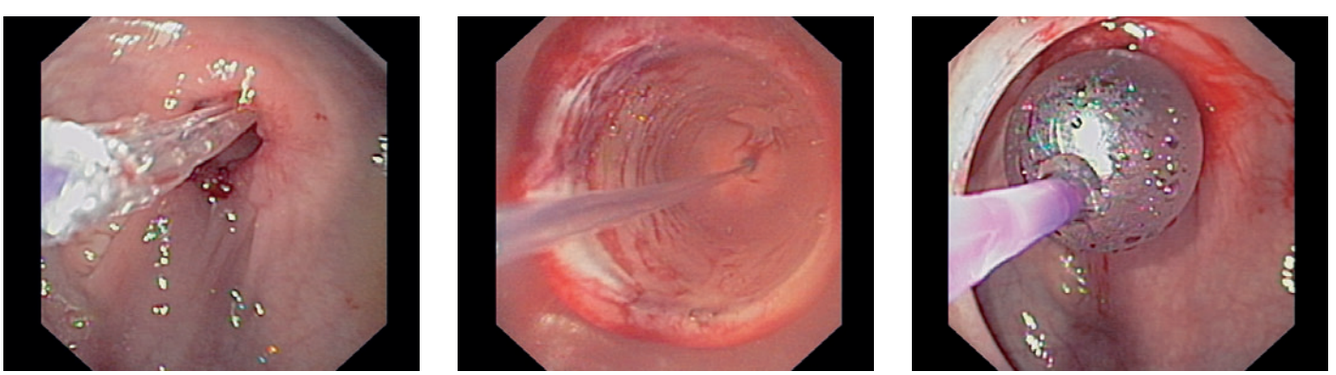
The occurrence of strictures following ischemic colitis or NSAID colitis varies and the degree of severity is unpredictable. Chronic courses, in particular, can lead to symptomatic manifestation. Later stages of radiation colitis are often associated with strictures affecting longer segments.

▣ **21.1 Balloon dilation of benign strictures**



a **b** **c**

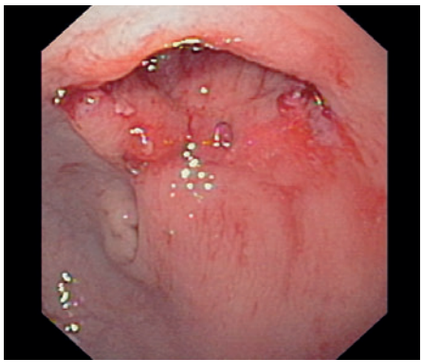
a–c Crohn disease. Inflammation and stenosis at ileoascendostomy (**a**). Advancement of a TTS balloon through the stenosis (**b**). Inflation of the balloon in the stenosis (**c**).



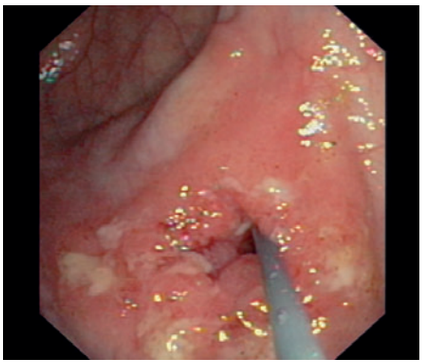
d **e** **f**

d–g Stenosis at anastomosis following sigmoid resection due to diverticulitis. Stenosis not passable. Advancement of the TTS balloon (**d**). Inflated balloon in the stenosis, filled with water and contrast agent. One can look through the balloon to the stenosis wall and to a certain degree see it dilating (**e**). Correct positioning of the balloon in the stenosis is controlled endoscopically during the entire procedure (**f**). In many cases, radiological surveillance is thus unnecessary.

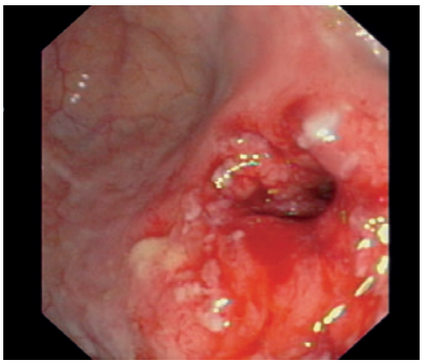
21.1 cont.



g Final image after three sessions of balloon dilation. The stenosis is now sufficiently opened and endoscopic passage is possible. A few staples are visible at the edge.



h



i

h, i Stenosis and inflammation of ileocolonic anastomosis in Crohn disease. "Laying out" the guidewire (**h**). After balloon dilation the stenosis is dilated and has bloody lacerations (**i**).

Postoperative strictures. The frequency of postoperative strictures is highest after resection in diverticulitis, followed by association with complicated healing processes such as anastomotic leakage (▣ 21.1**d**).

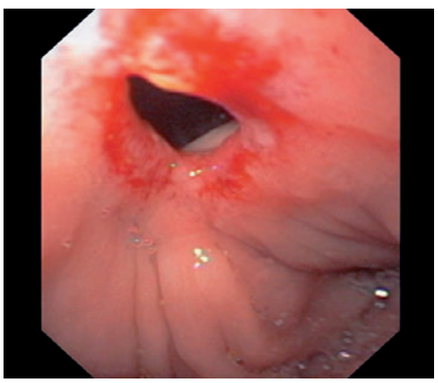


Fig. 21.1 **Anastomotic stenosis in upper rectum,** covered with blood after bougienage.

Diagnosis and Therapy

The role of endoscopy is primarily diagnostic, evaluating the nature of the stricture by examining mucosal appearances (still inflamed? residual tumor?). Though radiology is often superior for determining the length of stenosis, endoscopically one can often already determine whether a stenosis is concentric (usually following anastomosis), a postoperative angulation of the bowel, or a longer narrowing.

The concentric ones are best suited for endoscopic intervention, whilst a kinked, narrowed stricture often becomes symptomatic again. Stenoses longer than 5–8 cm in Crohn disease are often unresponsive to endoscopic treatment.

Appropriateness of endoscopic therapy

- suitable stenosis: short, "ringlike" stricture, concentric,
- unsuitable stenosis: long (> 5 cm), eccentric, sharply angulated.

Principles of endoscopic therapy

- Bougienage: entails a risk of eccentric and undesirable expansile force over longer bowel segments (Fig. 21.**1**).
- Balloon dilation: placed either over a guidewire using radiographic control (limitations in the right colon) or through the endoscope using a balloon which is advanced through the working channel and placed under direct visualization (TTS = through the scope balloon dilation). Balloon dilation has the advantage of direct visualization through the balloon (filled with a mixture of contrast agent and water) into the stenosis (▣ 21.**1e**) during the procedure; expansile force of dilation is evenly distributed radially (▣ 21.**1**). Discrete lacerations involving slight or moderate bleeding on the stenosis ends immediately following dilation are considered normal (Figs. 21.**2**–21.**4**).
- Electroincision using high frequency current: either using argon plasma coagulation or electrical needleknives. The use of papillotomy devices with pure cutting current has also been reported for benign strictures, but we avoid their use. We prefer incision with an argon beamer, in two sessions if necessary. This method is particularly suitable for short anastomotic "ring" stenoses (Fig. 21.**5**).
- There is little confirmed evidence and only a few small and retrospective case series on the occasional use of corticosteroid injection in such stenoses to prevent restenosis.

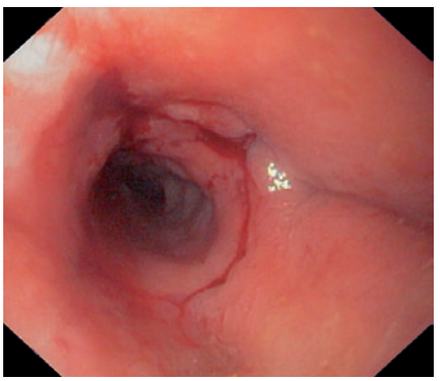


Fig. 21.2 **Anastomotic stenosis in middle rectum,** light bleeding, considered normal, at the stenosis ends following balloon dilation.

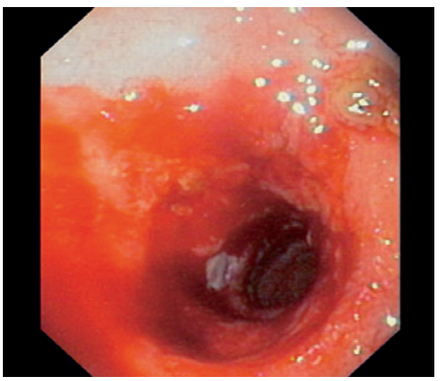


Fig. 21.3 **Anastomotic stenosis in middle rectum,** moderate bleeding immediately after dilation. The patient comes every two to three years for balloon dilation and is without complaints.

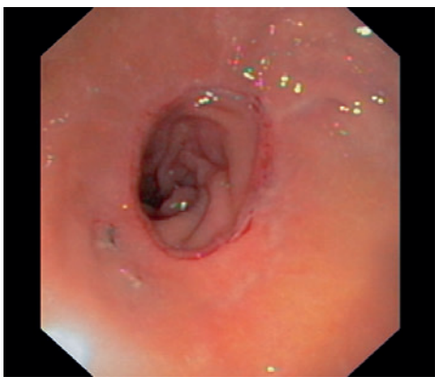
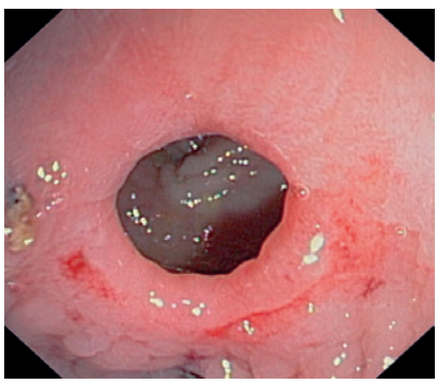
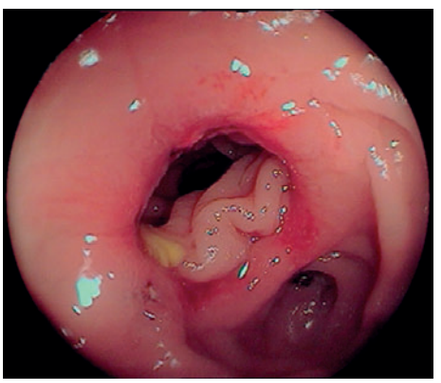
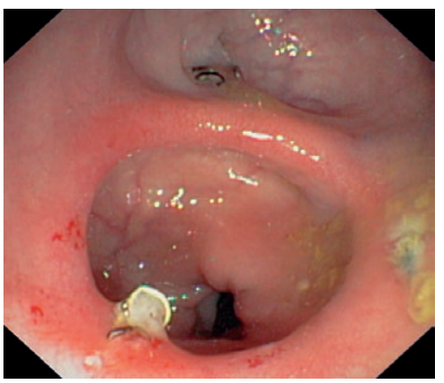


Fig. 21.4 **Discrete laceration of the stenosis ends** after balloon dilation of a rectosigmoidostomy, within normal range.







a **b** **c**

Fig. 21.5 **Short concentric stenosis.**

a Stenosis after rectal resection with stapler, not passable, prior to treatment.

b After the first laser incision with APC.

c The concentric stenosis is opened after two sessions of APC laser incision. A few staples can be seen on the ends.

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22 Fistulas and Postoperative Leakages

W. Schmidbaur

The formation of fistulas or anastomotic leakages can be associated with inflammatory diseases (especially Crohn disease) or therapeutic interventions, which lead to disruption in the continuity of the walls of hollow organs (Tab. 22.**1**).

This chapter addresses relevant aspects of these diseases for the colonoscopist. Enterocutaneous fistulas (like the perianal fistula in Fig. 22.**1**) will not be specifically discussed as they are often complex proctological or surgical problems that would be beyond the scope of this book.

Table 22.**1** Definitions

Fistulas
▶ Present at birth or acquired tubelike connections between two cavities or one cavity and the skin surface
Anastomotic leakage
▶ Any extraluminal extravasation from the region around an anastomosis is an anastomotic leak: defined as a complete bowel wall defect in the region around a surgical suture leading to communication between intraluminal and extraluminal spaces (7)

Fistulas

Colovesical fistulas. Colovesical fistulas occur in up to 2% of patients with diverticulitis (references in 2). Other causes include Crohn disease, malignancy, radiation therapy, and trauma. The rate of spontaneous healing is very low (2%). Thus, surgical intervention is usually the therapy of choice, though endoscopic therapy can also be attempted. Fistulas can also occur involving the urethra (Fig. 22.**2**).

Rectovaginal fistulas. Rectovaginal fistulas (Figs. 22.**3**–22.**5**) are also usually due to therapy-related or inflammatory causes. If endoscopic therapy is attempted, it should be performed together with a gynecologist if possible.

Enteroenteric and enterocutaneous fistulas. There is normally no endoscopic therapy approach for enteroenteric and enterocutaneous fistulas that appear, for example, in Crohn disease (Figs. 22.**6**, 22.**7**). Therapy mainly consists of medication and, if necessary, surgical intervention.

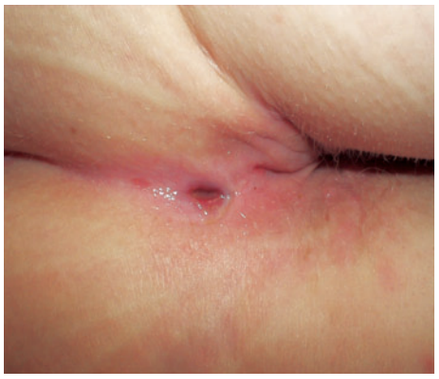


Fig. 22.**1** **Perianal fistula opening** in Crohn disease.

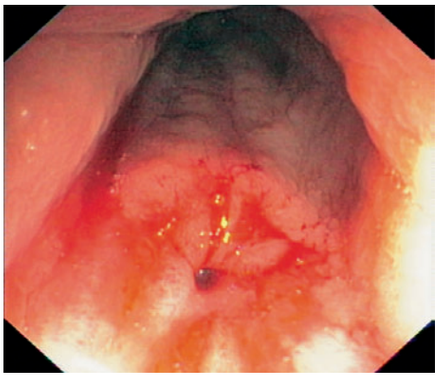


Fig. 22.**2** **Radiation-induced fistula including the urethra** (prior rectal carcinoma).

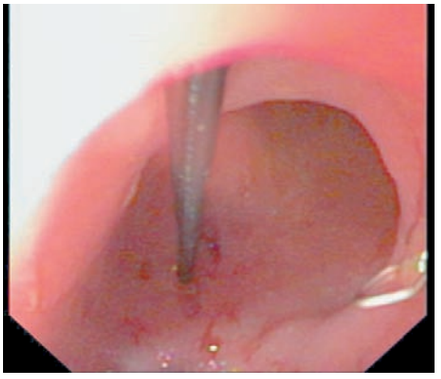


Fig. 22.**3** **Rectovaginal fistula.** The small opening of this fistula was hidden behind a rectal fold and was thus not visible under endoscopy. It was identified at transvaginal cannulation.

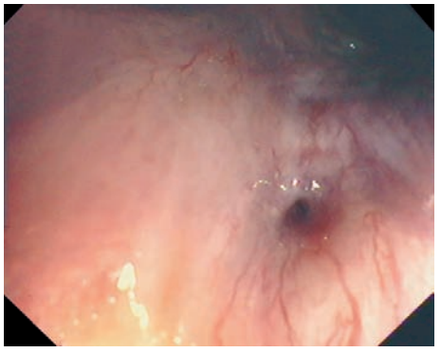


Fig. 22.**4** **Radiation-induced fistula in vagina** (prior rectal carcinoma).

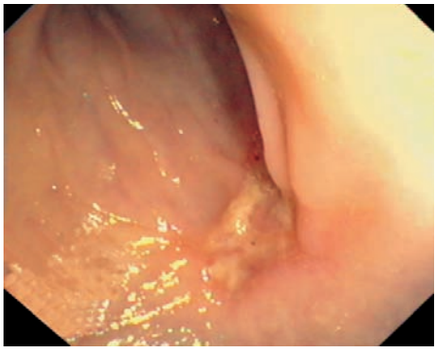
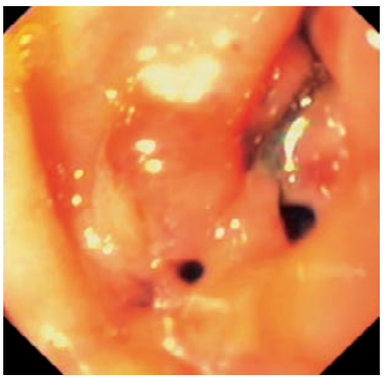


Fig. 22.**5** **Fistula not immediately visible during endoscopy.** The fistula was found after administration of a contrast dye in the ulcerlike defect.



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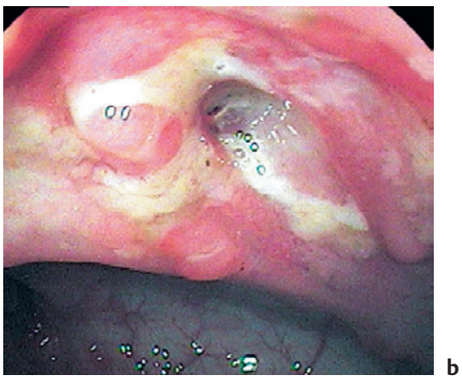

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Fig. 22.6 **Fistulizing Crohn disease.**
a Severe initial episode of Crohn disease. Fistulas were first discovered in the duodenum.
b The other end of the fistulous tract with several openings in the colon; widespread inflammation and ulcers next to the tract.

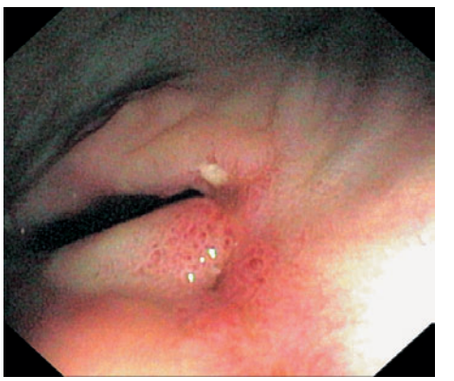
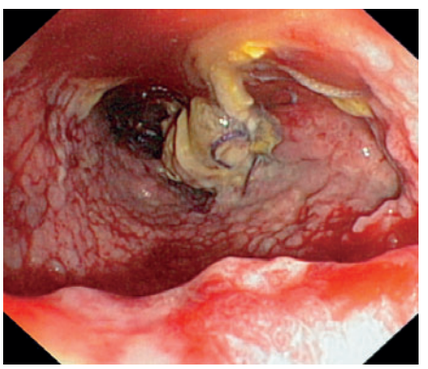


Fig. 22.7 **Tiny fistula opening in an indentation next to the Bauhin valve.** The fistula ended after a few centimeters in the terminal ileum, which had massive inflammatory changes.

22.1 Varying aspects of anastomotic leakage



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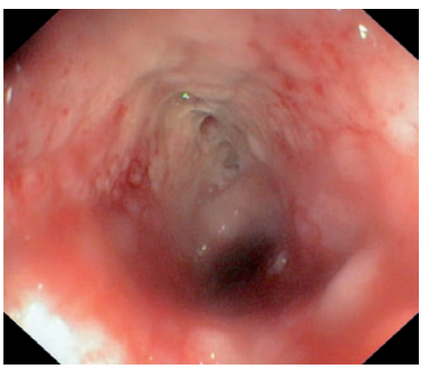

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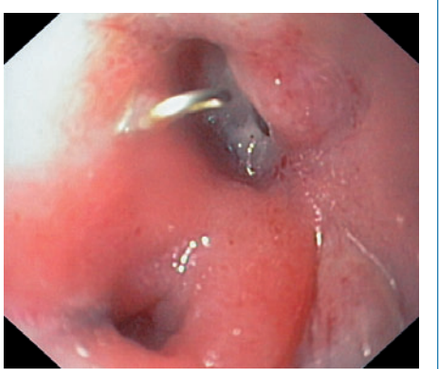
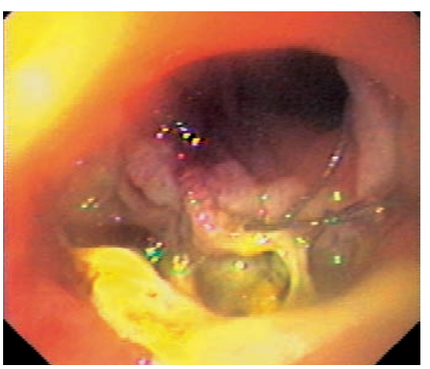
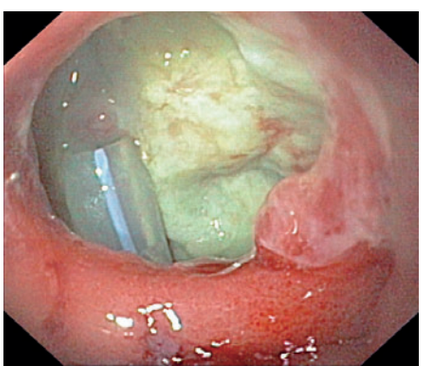
a, b Development of an anastomotic leak. Circular, covered 7-cm long anastomotic leakage after sigmoid resection with unremarkable clinical signs (**a**). After four weeks it appears to be healing well (**b**).



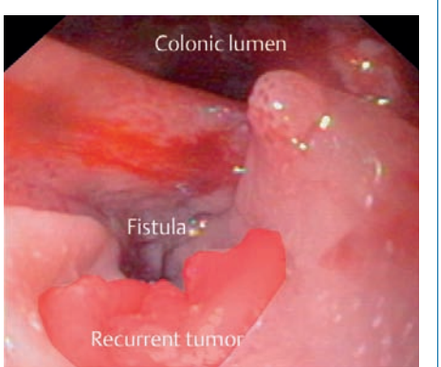
c Anastomotic leak with visible staple near the edge of the fistula; at the lower left the anastomosed region.



d Narrow suture dehiscence; remaining sutures.



e Dehiscence, local drainage is visible, sliding intermittently into the bowel lumen.



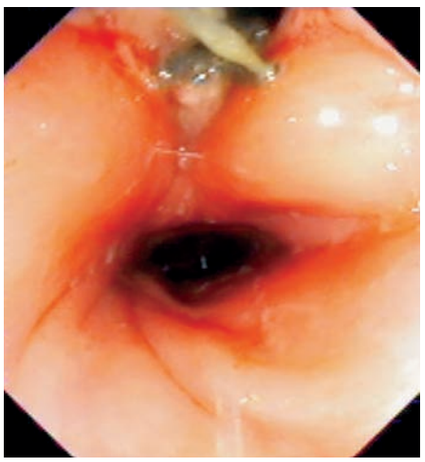
f Refractory fistula five months after operation; a recurrent tumor is now visible.

Anastomotic Leakage

Definition and prevalence. An anastomotic leakage is any extraluminal extravasation from the region near the anastomosis. It is defined as a complete defect in the bowel wall near the surgical line resulting in communication between intraluminal and extraluminal spaces (7) (▣ 2.**1**, 22.**2**, Figs. 22.**8**, 22.**9**).

Prevalence figures on anastomotic leakage vary according to localization of the anastomosis and reporting author. According to the literature, leakage rates have decreased considerably in

22.2 Anastomotic leakage with necrotic cavities



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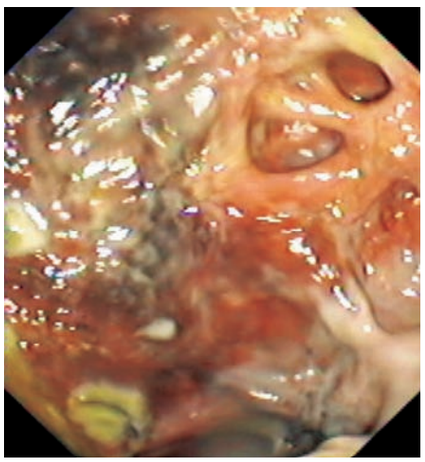

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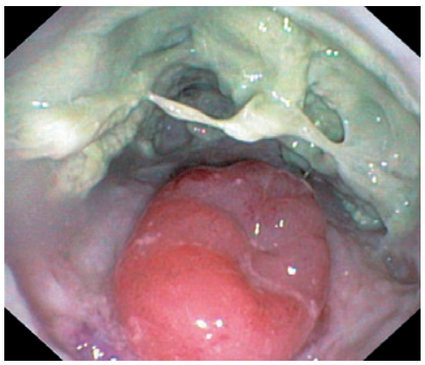
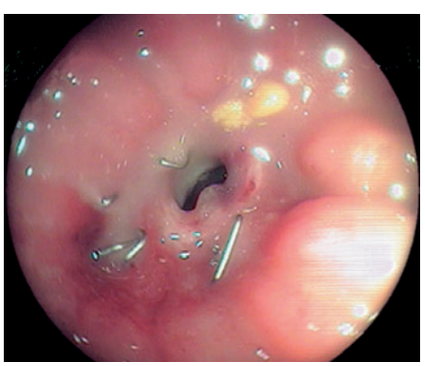


c Large horseshoe-shaped dehiscence with sloughed cavity.

a, b Necrotic cavity with small opening. At the top of the image a relatively small opening to a necrotic cavity (**a**). Base of the necrotic cavity (**b**).



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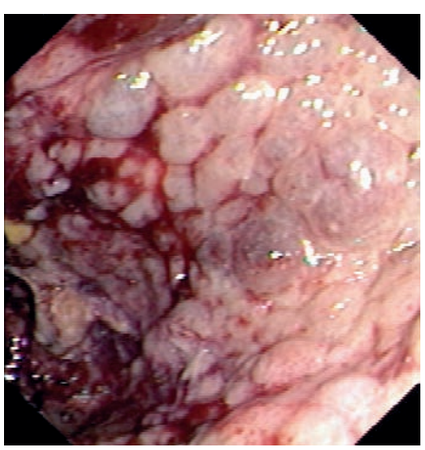

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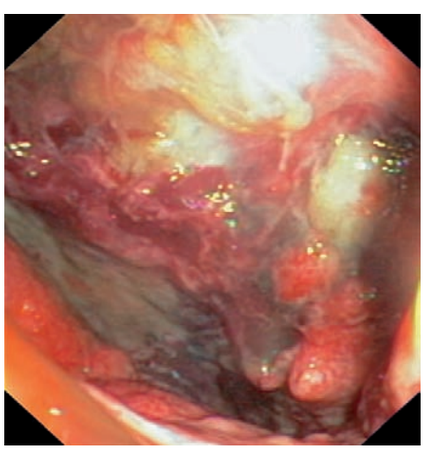
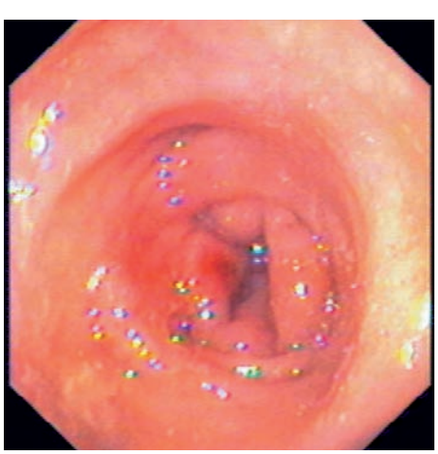


f Large dehiscence, recurrent bleeding from the base of the necrotic cavity.

d, e Stenosis distal to necrotic cavity. Strictured anastomosis after low anterior rectal resection (**d**). After dilation, the instrument enters a necrotic cavity. The continuation of the lumen is not visible (**e**).



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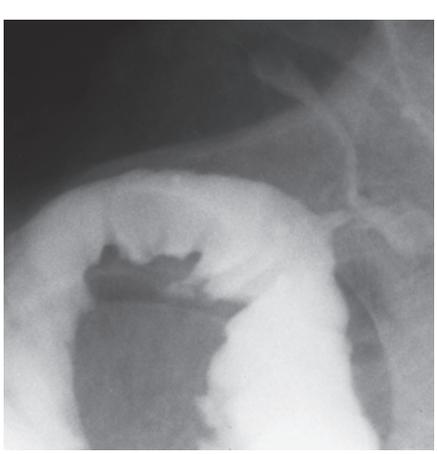


b

Fig. 22.8 **Endoscopic and radiologic views of a fistula.**

a Endoscopic view: small fistula opening to a narrow, branching fistula following low anterior rectal resection.

b Using radiologic contrast imaging a Y-shaped fistula tract is visible on the cranial side of the bowel with a small clubshaped retention cavity.

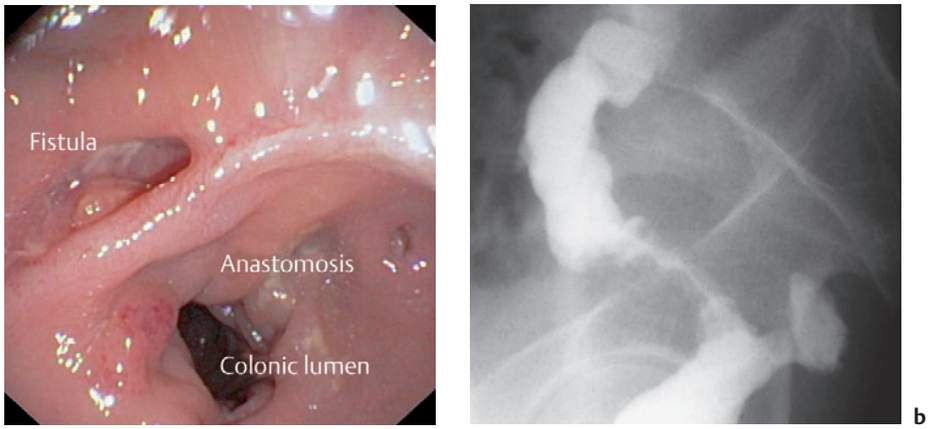


Fig. 22.9 **Endoscopic and radiologic views of an anastomotic leak.**
a Endoscopic view: ovalshaped anastomotic leak.
b Using radiographic contrast imaging, a small cavity next to a vessel is visible.

the past 10–20 years. Leakage rates following low anterior rectal resection are reported in the surgical literature at 6–14%.

Causes. Anastomotic leakages can be caused by local and/or systemic factors. Local factors include suture technique, necrosis, contamination, tissue ischemia, and tissue tension at the site of the anastomosis. Systemic factors include malnutrition, older age, and concomitant diseases associated with delayed healing or tissue hypoxia. Due to the considerable rate of leakage, many surgeons prefer to attach a protective stoma, especially after low anterior rectal resection.

Changes in the appearance of secretion are one clinical sign of an anastomotic leak, especially in situations where local drainage appears to be functioning well. General clinical symptoms are not always present. Nonetheless, postoperative clinical worsening of the patient's condition should always raise suspicion of an anastomotic leak. Radiologic tests are generally used for primary confirmation of suspected diagnosis (Figs. 22.**8**, 22.**9**).

Intra-abdominal anastomotic leaks require surgical management to prevent development of diffuse peritonitis. However, for extraperitoneal low rectal anastomoses, conservative therapy or endoscopic measures are appropriate.

Diagnosis

Confirmation of clinical suspicion is initially with radiologic testing, usually by means of conventional contrast radiograph or computed tomography (CT).

Fistulas. Even fistulas that are clearly visible in radiologic tests can often be difficult to identify endoscopically in the colon due to their frequently small openings. Additional procedures are needed to assist in identifying fistulas. For rectovaginal fistulas, for example, cannulation over a wire from the vagina can be helpful (Fig. 22.**3**). For colovesical fistulas contrast material (e.g., methylene blue) may be useful and can be administered via the installed bladder catheter. The urinary catheter is also absolutely essential for minimizing intravesical pressure to maintain the success of endoscopic fistula closure.

Anastomoses. Anastomoses can already be endoscopically examined in the first few days following operation with an acceptable level of risk and without danger of additional iatrogenic injury. The potential for simultaneous radiographic diagnosis is also an advantage as it can precisely demonstrate any leak or fistula (including cavity) and assist in evaluation before or during endoscopic therapy. It should be noted that unsuccessful endoscopic primary intervention does not compromise later surgical intervention.

Endoscopic Interventions

The spontaneous course of an anastomotic leakage is difficult to predict. According to the literature, 50–80% leakages ultimately heal spontaneously with sufficient drainage, systemic antibiotic therapy, parenteral nutrition, and local irrigation. Median length of time required is six weeks, after which the rate of spontaneous healing drops markedly. The aim of endoscopic treatment is to reduce follow-up treatment by ensuring more rapid closure of the fistula.

Based on experience, some fistula types offer little chance of successful endoscopic closure. These are malignant fistulas, "stomalike" fistulas, fistulas in a florid inflammatory stage of Crohn disease, and narrow fistulas with a distant cavity without sufficient drainage (pooling discharge, abscess formation). We do not advise endoscopic closure in such situations. The healing process can be supported with regularly occurring targeted irrigation (Fig. 22.**10**). For larger leakages following low anterior rectal resection, a stoma must often be attached prior to endoscopic therapy (if a protective stoma has not already been attached).

Management of fistulas or leakages is most often done using fibrin sealant or mechanical clips. Table 22.**2** gives an overview of current treatment methods.

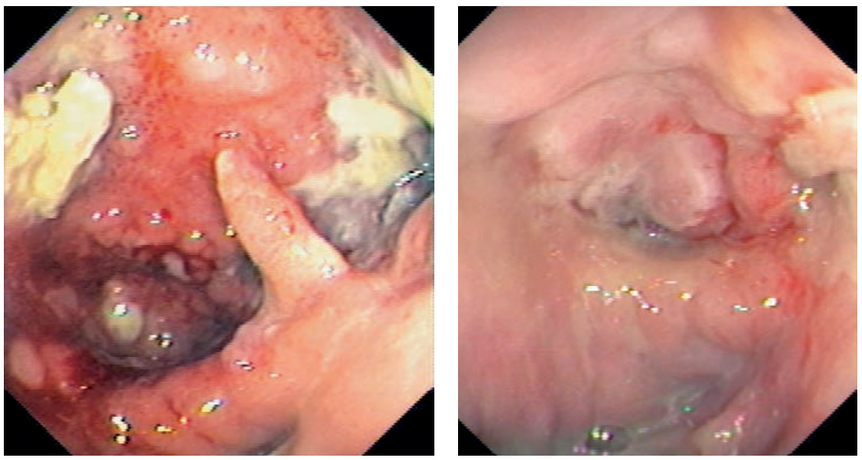


Fig. 22.**10** **Irrigating an anastomotic leakage.**
a Large anastomotic leak; irrigation is the only therapy.
b Three weeks later: cleaned base of wound, noticeably flatter cavity lined with granulation tissue.

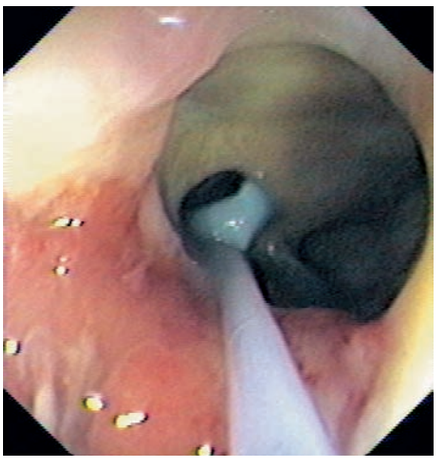


Fig. 22.**11** **Fibrin gluing of a longer fistula.**

Fistula Closure and Management of Dehiscence Using Fibrin Sealant

Endoscopic debridement. If closure is the aim of therapy, the fistulous tract or wound dehiscence must first be cleaned to ensure longer-lasting closure. Depending on size and extent, "endoscopic debridement" can be performed with an irrigation probe (under radiographic control if possible) or even mechanically for larger necrotic cavities, using various instruments (e.g., dormia basket). Therapy should be repeated in frequent intervals (e.g., every three days) until its efficacy is clearly seen. Only for very recent leakages that are apparently clean can closure therapy begin in the first session, immediately after initial irrigation.

Preparing the fistula tract or wound edges. Before each treatment (for both fibrin sealant and clips) the fistula tract or wound edges must be prepared. Fistulas tend to rapidly build up a "covering" of almost normal-appearing tissue allowing the fistula to persist underneath. Therefore, mere closure is often insufficient for long-term success. Nowadays it is common procedure to abrade the mucosa so that easier and longer-lasting contact forms between the fistula borders. This can be achieved with a firm cytology brush and thorough mechanical abradement. The much simpler, and in our view, more effective method uses argon plasma coagulation (APC) (9). For debridement of a fistula using APC, we use a low power setting (30–40 W) and brief pulses to coagulate the surface in a ring around the edges of the entire fistula.

Method
Before fistula closure (whether with fibrin sealant or clips) we first examine the fistulous tract using radiography. A standard ERCP (endoscopic retrograde cholangiopancreatography) catheter is very useful for cannulation of a longer fistulous tract. A guide wire (e.g., Terumo) may also be used occasionally for longer tracts to achieve complete cannulation or imaging. Deep cannulation is especially important when such fistulas are to be glued. A dual lumen catheter can be advanced as far as possible over the inserted wire into the fistula, which is then debrided and instilled with fibrin sealant. Administration of sealant begins as far away from the fistula opening as possible with the objective of first filling the distant fistula regions thus minimizing pooling of secretion. Per session 1–4 mL of fibrin sealant are applied (Fig. 22.**11**).

The use of a dual lumen catheter is preferable as otherwise irrigation of the lumen between application of fibrin sealant and thrombin causes too much fluid to be introduced in the fistulous channel and a portion of the glue is washed out into the colonic lumen. Endoscopic suction should not be applied after application of the fibrin sealant.

Table 22.**2** Therapeutic procedure for fistulas or leakages

- Irrigation/debridement
- Fibrin closure
- Clipping
- Stenting
- Suturing
- EndoVAC

Success rates. Like debridement, the application of sealant has to be repeated in intervals of several days until either closure has been achieved or until it is determined that treatment is ineffective and must be discontinued. If treatment is unsuccessful (five or six sessions) and there is lacking potential for operative intervention we usually revert to supportive therapy with irrigation/debridement which is performed on an outpatient basis and at larger time intervals (e.g., once or twice per week). Most patients can thus be spared surgical revision, which frequently involves extirpation. In the literature, 70–80 % of all fistulas can be healed, though the duration (median 33, range 4–365 days) (8) and number of sessions vary greatly. In

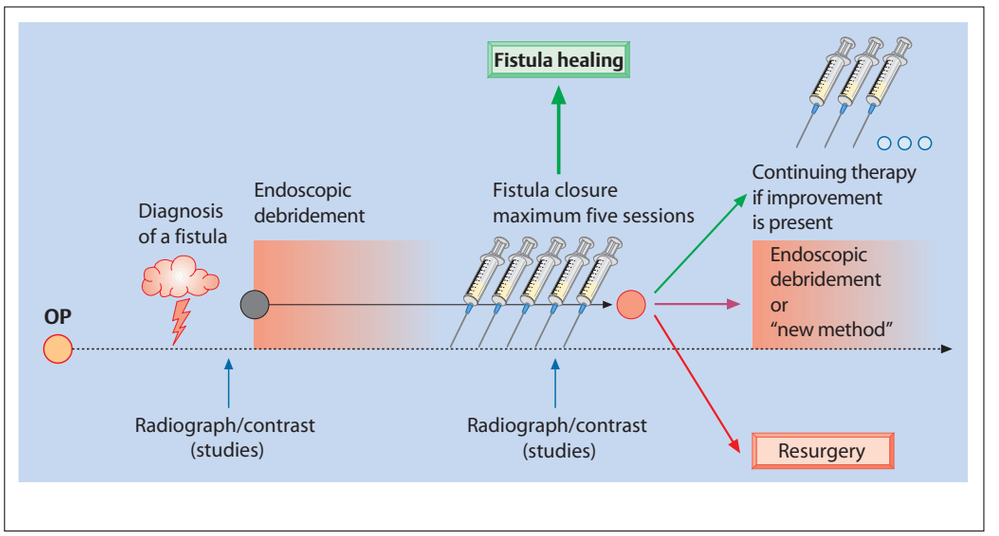


Fig. 22.**12** **Flowchart showing management of postoperative fistulas in the colon.** Diagnosis of a fistula is followed by radiological imaging. Endoscopic examination follows with the aim of evaluating the endoscopic situation or beginning therapy. Fistula debridement varies in length of time required. After this the fistula is closed using fibrin sealant (and possibly also clips). If the fistula has not closed after five to six sessions, the situation must be re-evaluated. If there are signs of healing, therapy is continued. If there is no potential for improvement, then the fistula is merely supportively irrigated or new procedures considered (e.g., stenting, endoscopic suturing, etc.). Reoperation is only considered as an ultima ratio.

the lower gastrointestinal tract, an average of 10 sessions (15 for larger leaks) can be reasonably expected (literature overview in 3).

Figure 22.**12** illustrates fistula closure schematically.

Fistula Closure and Management of Dehiscence Using Clips

Method

The application of an endoclip follows several relatively simple steps. First, the stainless steel clip is loaded onto the clipping device and retracted into the protective Teflon sheath. The loaded application device is then advanced through the working channel of a standard endoscope. As soon as the Teflon sheath comes into view, the clip can be advanced from the sheath. Using traction on the clip applicator, the prongs of the clip can be opened gradually. As the clip opens, the prongs click into place at each width. While the orientation of the open clip is rather by chance, the rotation of the clip can be controlled. However, rotation ends up being much less precise than was hoped for, given the distance and friction working against clip mechanics. Tension can sometimes be reduced by closing the clip a notch after it has been fully opened, making the clip easier to rotate.

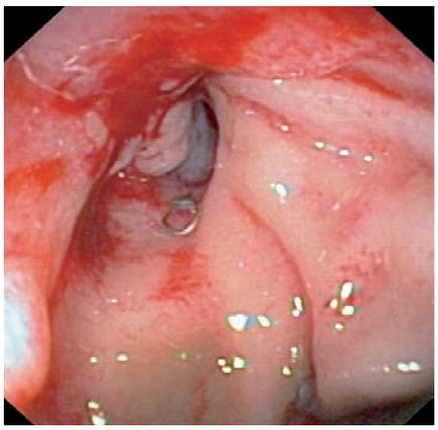
As soon as the opened prongs (12 mm) have been positioned at the targeted position, the clip can be closed by firmly pulling on the deployment mechanism. After closing the prongs the clip automatically disconnects from the holder at a predetermined breaking point.

For using clips in the closure of fistulas and dehiscence, grasping the fistula borders as far laterally as possible and placing several clips next to each other can be a useful approach. The clips can also be placed alternating on either side. By grasping healthy tissue and clipping both sides, closure can be achieved step-by-step. Using this "zipper" principle, closure is possible of even openings with an initial diameter much larger than the breadth of the clips. Figures 22.**13**–22.**15** give an impression of dehiscence management using clips.

Based on our own experience, it can be quite helpful with larger leakages to combine clipping and fibrin sealant and after successful clip application to carefully inject a fibrin sealant using a dual lumen injection needle between the clips (Fig. 22.**16**). Controlled studies are, however, not available.

Managing small perforations with clips. An attractive aspect of using endoclips is the potential for immediate therapy of small perforations following endoscopic interventions such as polypectomy and mucosectomy. While this procedure has been relatively frequently described in the literature on the upper gastrointestinal tract, data on procedures in colonoscopy are sparse (11). From a purely technical standpoint and concerning primary success of closure, there should be no difference between the upper and lower gastrointestinal tract. However, the clearly higher probability of bacterial contamination in the lower gastrointestinal tract makes this procedure risky. If bacterial load can be reduced by thorough preparation, and if closure of the leakage follows immediately, in selected cases such a nonsurgical procedure can be attempted, but only in close consultation with the abdominal surgeon, and if the patient is placed on a parenteral nutrition, systemic antibiotic therapy, and under close clinical supervision.

The use of clips for fistula closure or to seal leakages was first described by Rodella in 1998 (6).



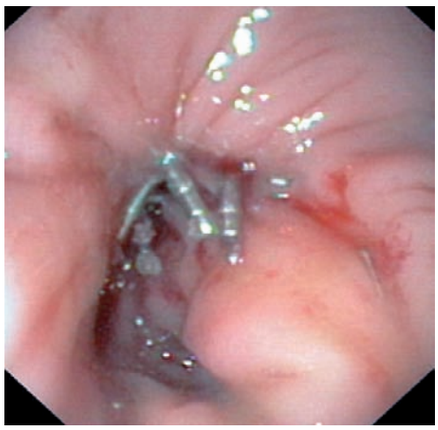
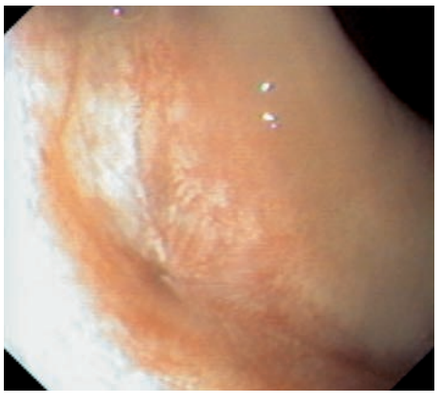


Fig. 22.**13** **Management with clips.**
a Visible clip on an anastomotic leakage.
b After clipping.



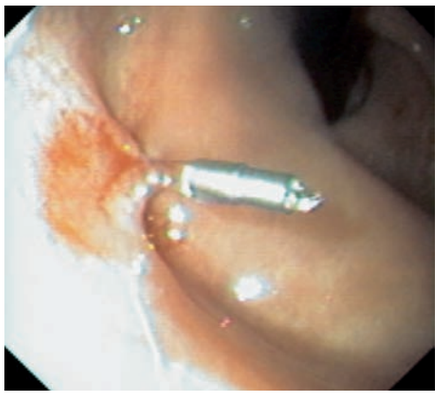
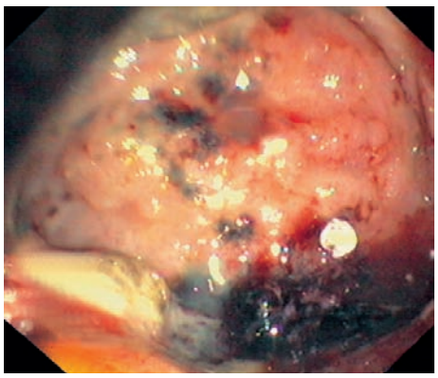


Fig. 22.**14** **Clipping of a fistula.**
a Tiny rectovaginal fistula that is only visible as an indentation.
b Fistula closure with a clip.



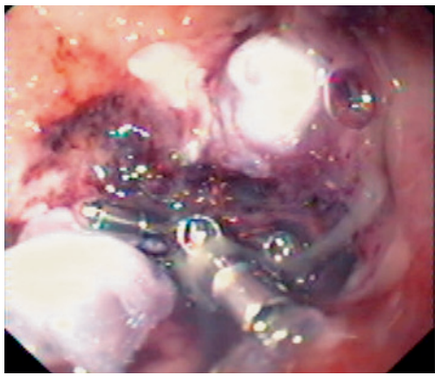
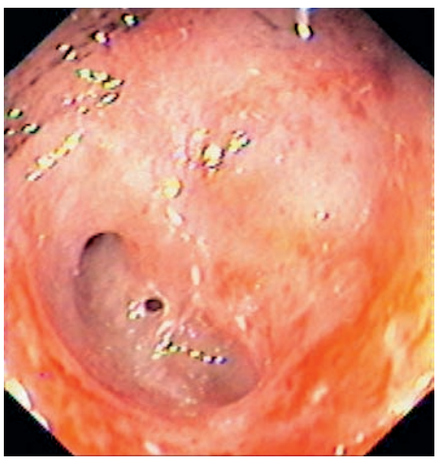


Fig. 22.**15** **Management of bleeding with a clip.**
a Large cavity in anastomotic dehiscence; endoscopic closure not possible. Rebleeding from the vessels at the base of the cavity.
b Management of bleeding using a clip. Afterward, irrigation continued.



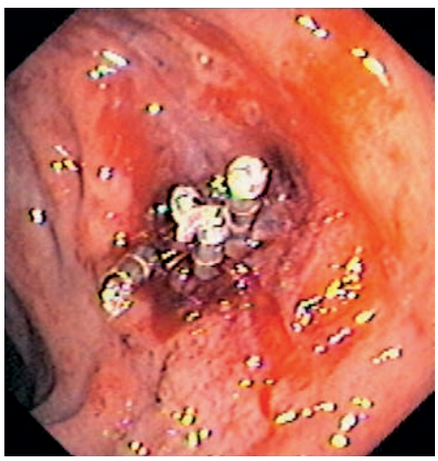


Fig. 22.**16** **Fistula management with clips and fibrin sealant.**
a Patient with prior sigmoid resection and a fistulous opening in a pseudodiverticulum at the anastomosis.
b Successful closure with clips and fibrin sealant injection between the clips.

Differential Therapy Using Fibrin Sealant/Clips

Clip application and fibrin glue therapy are two procedures that can certainly complement each other and can even be used simultaneously (Fig. 22.**16**, ▣ 22.**4 d–i**). The decision on one procedure vs. the other is highly individual and can hardly be regulated. The following considerations are thus intended to be merely suggestions.

Clips are more appropriate for fresh wound edges that are not subject to very high tissue tension. Too much tension on the wound edges prevents primary closure or leads to rapid dislodgment of the clip. In such cases, fibrin sealant is the better alternative. A small fistula opening, however, can sometimes be closed in a single session using one or more clips, after necessary debridement. For complex fistulous tracts, the use of fibrin sealant again is more suitable. ▣ 22.**3** and 22.**4** show therapy courses of fistulas and leakages, in some of which a combination of fibrin sealant and clips was used.

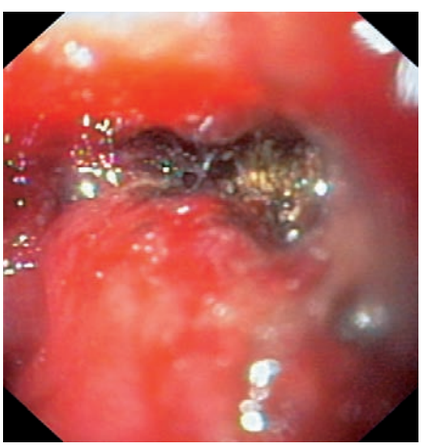
There is one point that must be considered in any occluding therapy. In particular, when treating leaks in the colon, attention must be paid to increased risk of bacterial contamination. Closure thus often requires drainage of the fistulous tract. Mostly an intraoperative drainage already in place is sufficient, as long as a connection is present. Otherwise, additional (external) drainage may well be necessary, e.g., under CT control to prevent pooling of secretion or abscess formation.

Stents

In the upper gastrointestinal (GI) tract, covered wire mesh stents are well suited to traversing fistulas and dehiscences. In the colon, however, this type of stenting is still uncommon, and is reserved for special cases, e.g., traversing complex fistulas caused by malignancy (2) (Fig. 22.**17**). It should also be noted that enteral stents that can be placed endoscopically cannot be used because they are noncovered. Ultimately, an esophageal stent (e.g., Ultraflex) was used, though it is not officially approved for this indication. During endoscopic placement of a guidewire under radiographic control, the target position of the stent ends is marked using either metal markings on the skin or mucosal injection of iodized oil (Lipiodol). After the instrument has been withdrawn, the stent is advanced over the guidewire and deployed under radiographic control.

In addition to the established methods that have been mentioned, two recent methods have been developed which may become available.

▣ **22.3 Management of fistulas and anastomotic leaks**



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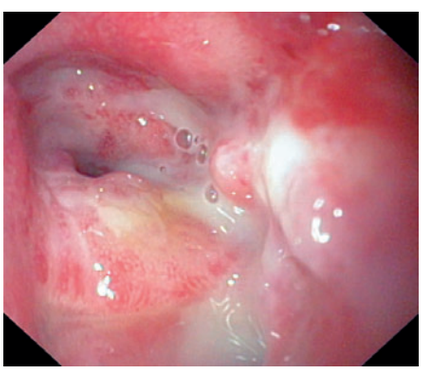

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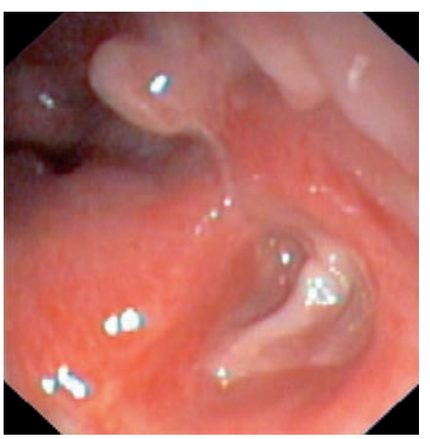
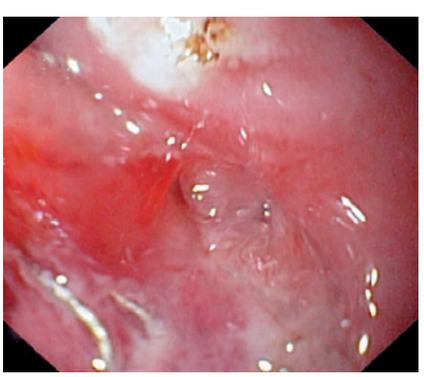


c

a–c Repeated fistula therapy, debridement with APC, heavier secretion in the interim. Fistula residuum.



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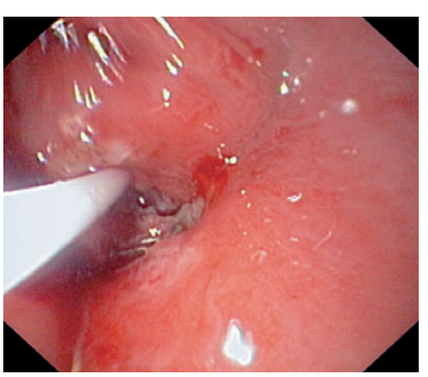

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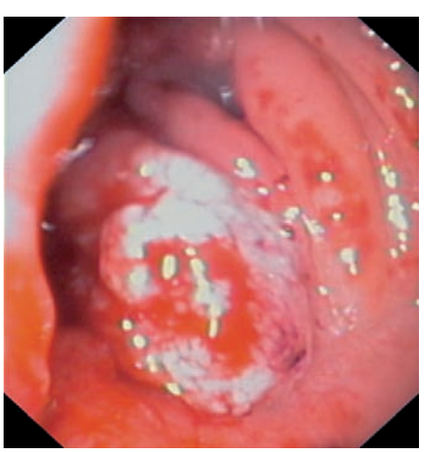
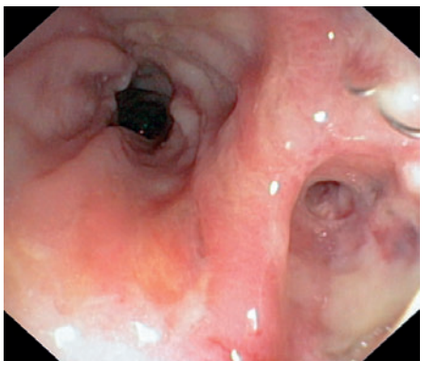


f

d–f Fistula closure after therapy, repeated therapy when it reopened. Recurrent tumor three months later.

22.3 cont.



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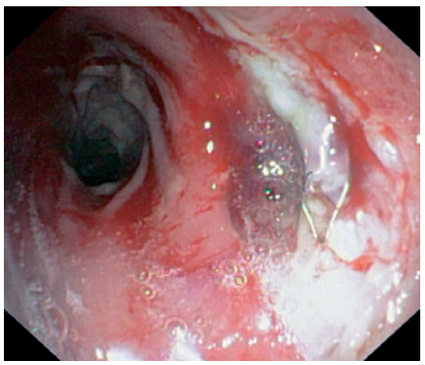

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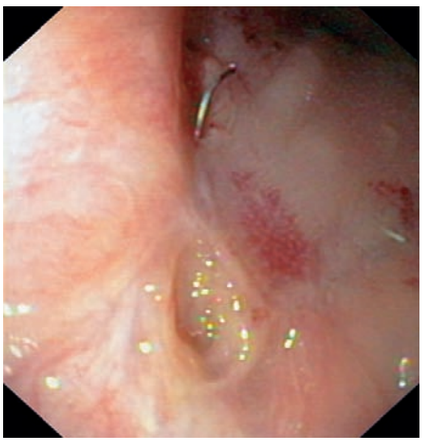
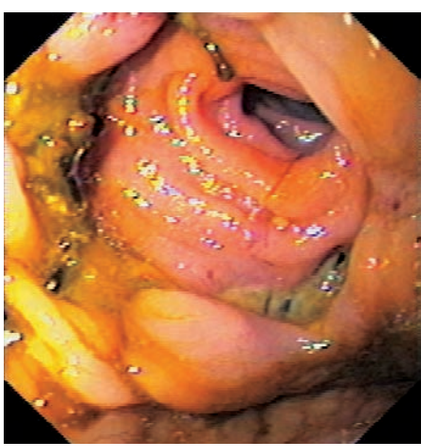


i

g–i Leak after irrigation, repeated fibrin glue therapy, healing.

22.4 Management of anastomotic leakages (clips and in some cases also fibrin glue)



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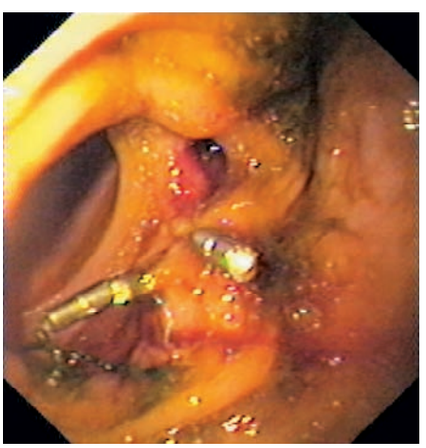

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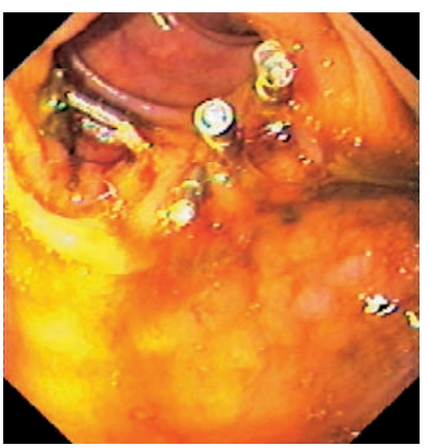
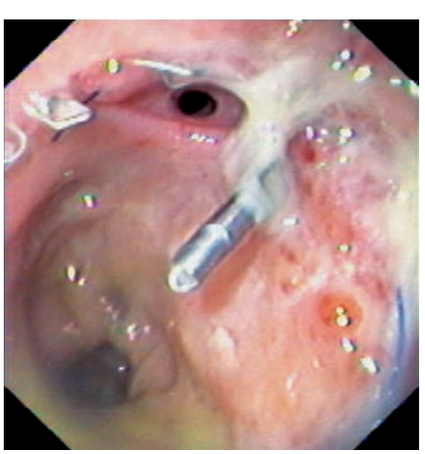


c

a–c Narrow anastomotic leak over half the circumference, easily grasped; application of several clips.



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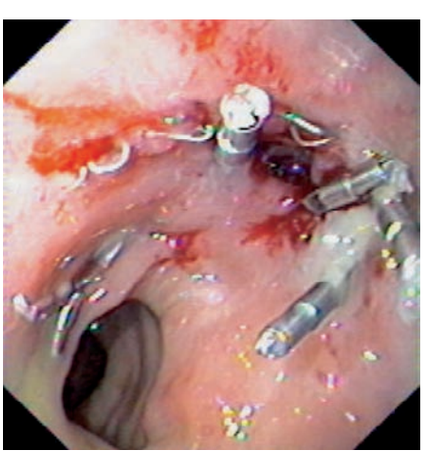

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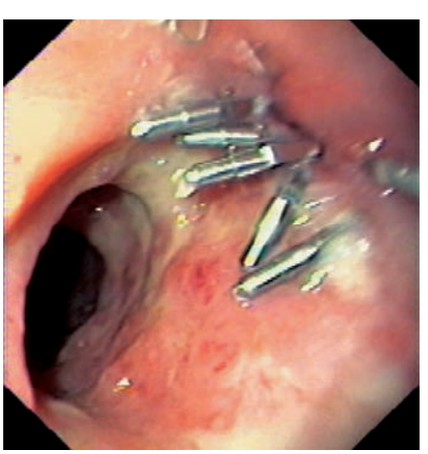
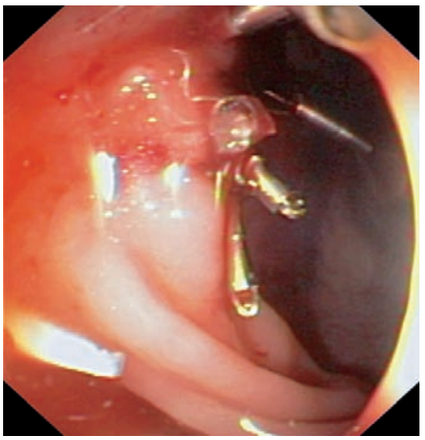


f

d–f Anastomotic leak with long fistulous channel; several fibrin sealant injections and clipping. In the upper part in **f** the small, persistent fistula.

22.4 cont.



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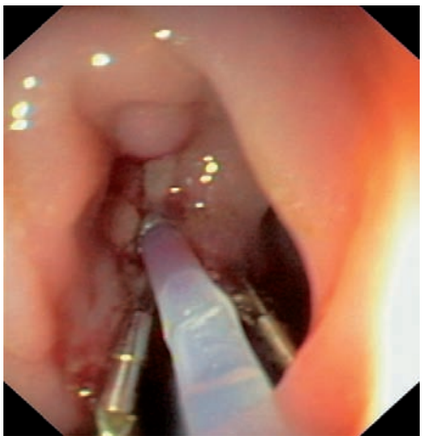

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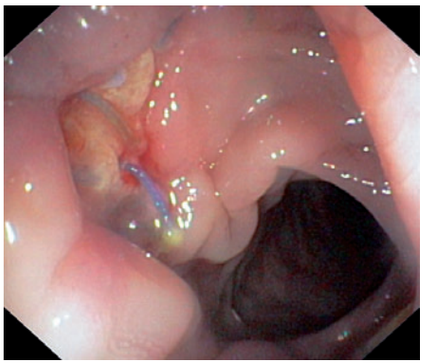


i

g–i Repeated therapy with fibrin sealant and clipping. Healed dehiscence (granulation tissue).

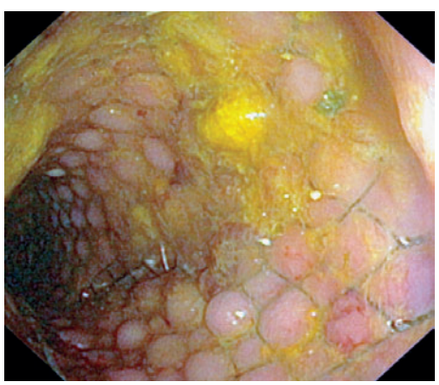


Fig. 22.**17** **Prior stenting of a complex fistulous tract** (postoperative following carcinoma). Distal stent end–uncovered part of stent.

Endoscopic Suturing

Endoscopic suturing systems have primarily been used for treatment of gastroesophageal reflux disease. In principle, the same instrumentation can be used for closure of fistulas or leakages, though data from the literature on their use is sparse. We will report here on our initial experience with these indications.

In the system that we use (Cook), an additional external accessory channel is attached to the end of the endoscope for the suturing system. The ESD (endoscopic suturing device) kit comprises two components: a suture delivery device and a mechanism that installs a titanium crimp (which acts like a knot) and cuts off the suture tails (Fig. 22.**18**). In the colon, the use of suturing techniques should be restricted to problems near the anal canal, as the rigidity of the instrument makes application distal from sharply angling or curving bowel segments difficult, or even impossible (▣ 22.**5**).

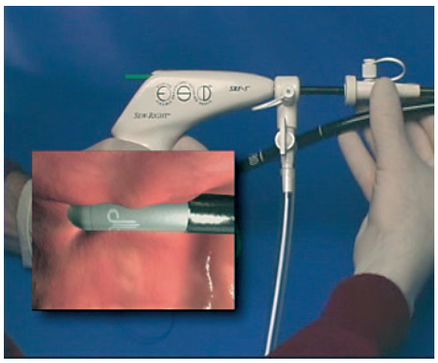
EndoVAC

Another new method for treating large dehiscences after anterior rectal resection has been introduced by Weidenhagen (10). This technique is based on the idea that between endoscopic treatments, the close proximity of the anastomosis and the sphincter can cause recurring blockages of septic secretion and gas in the intestinal lumen, which, following the path of least resistance, eventually leads to additional pressure in the wound cavity. Dehiscence in turn worsens, causing persistent interference with the healing process. This can make the application of basic principles of wound treatment impossible and is thus a considerable drawback in previous endoscopic treatment procedures.

The EndoVAC system uses a new endoscopically supported application system to place an openpored polyurethane sponge in the dehiscence cavity. The sponge is then connected by a tube via the sphincter to a vacuum. The system is changed under endoscopic support three times per week. The EndoVAC technique allows continual and effective drainage of the perianastomotic abscess by removing the accumulated secretion. The contact between the openpored sponge and the abscess wall results in significant debridement. Additionally, the negative pressure automatically leads to a reduction in the size of the wound cavity and supports the growth of granulation tissue in conjunction with the polyurethane sponge.

The EndoVAC applicator is made up of two coaxially placed tubes. The outer tube is used as the intake tube for the sponge system and the inner, somewhat less flexible tube, is used to advance and position the sponge system (Fig. 22.**19**).

Present data indicate that the wound cavity heals faster as a result of this technique, while the functionality of the sphincter apparatus is maintained (▣ 22.**6**).



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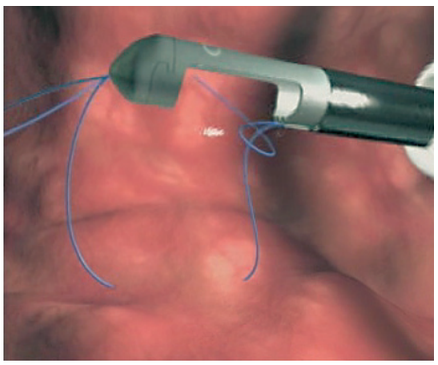

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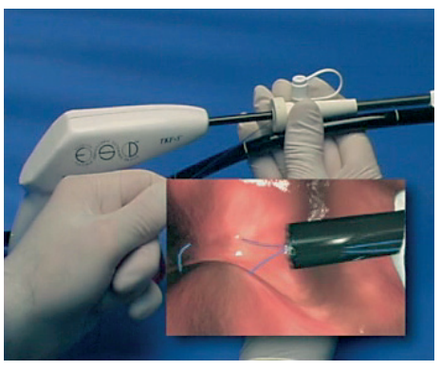

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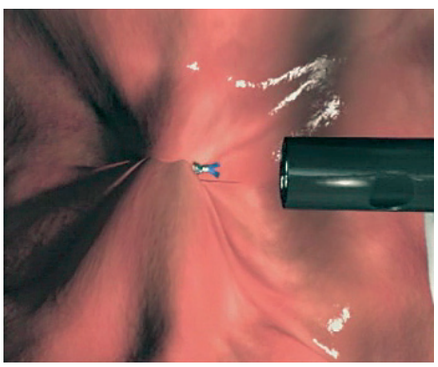
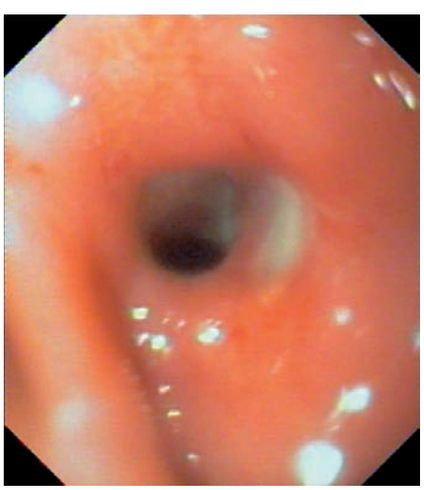


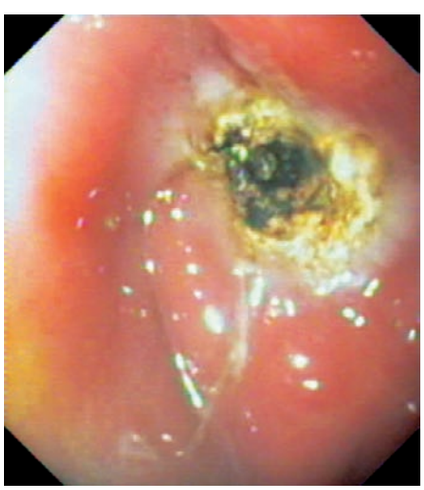
d

Fig. 22.18 **Suturing technique for fistulas.**
a Suturing device handle.
b Placing the suture.
c Knot application device.
d Installing the titanium crimp.

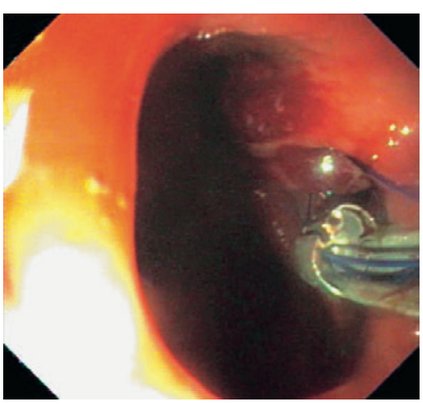
22.5 Suturing of a fistula



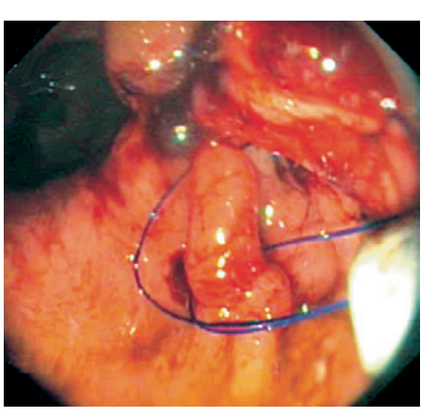
a Refractory fistula.



b Debridement of the fistula with APC.



c Following placement of the first suture.



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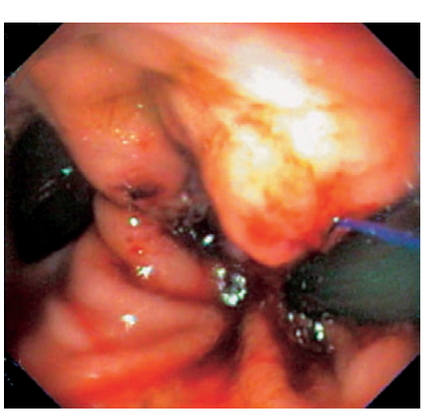


e

d Both sutures in place; tightening the loops.
e Placing the titanium crimp; tightened suture.

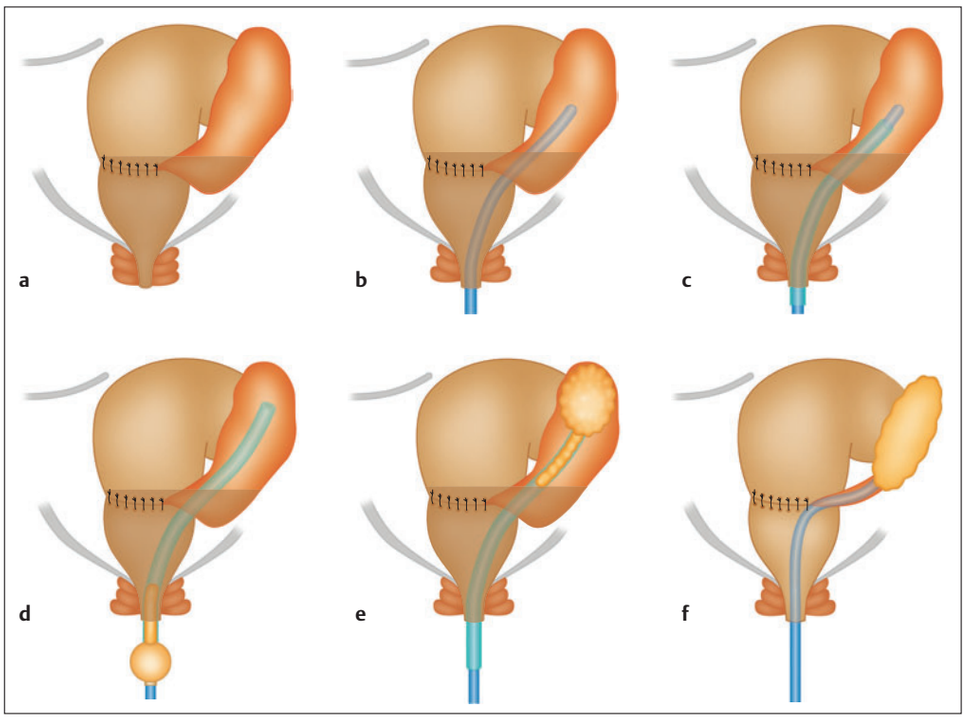
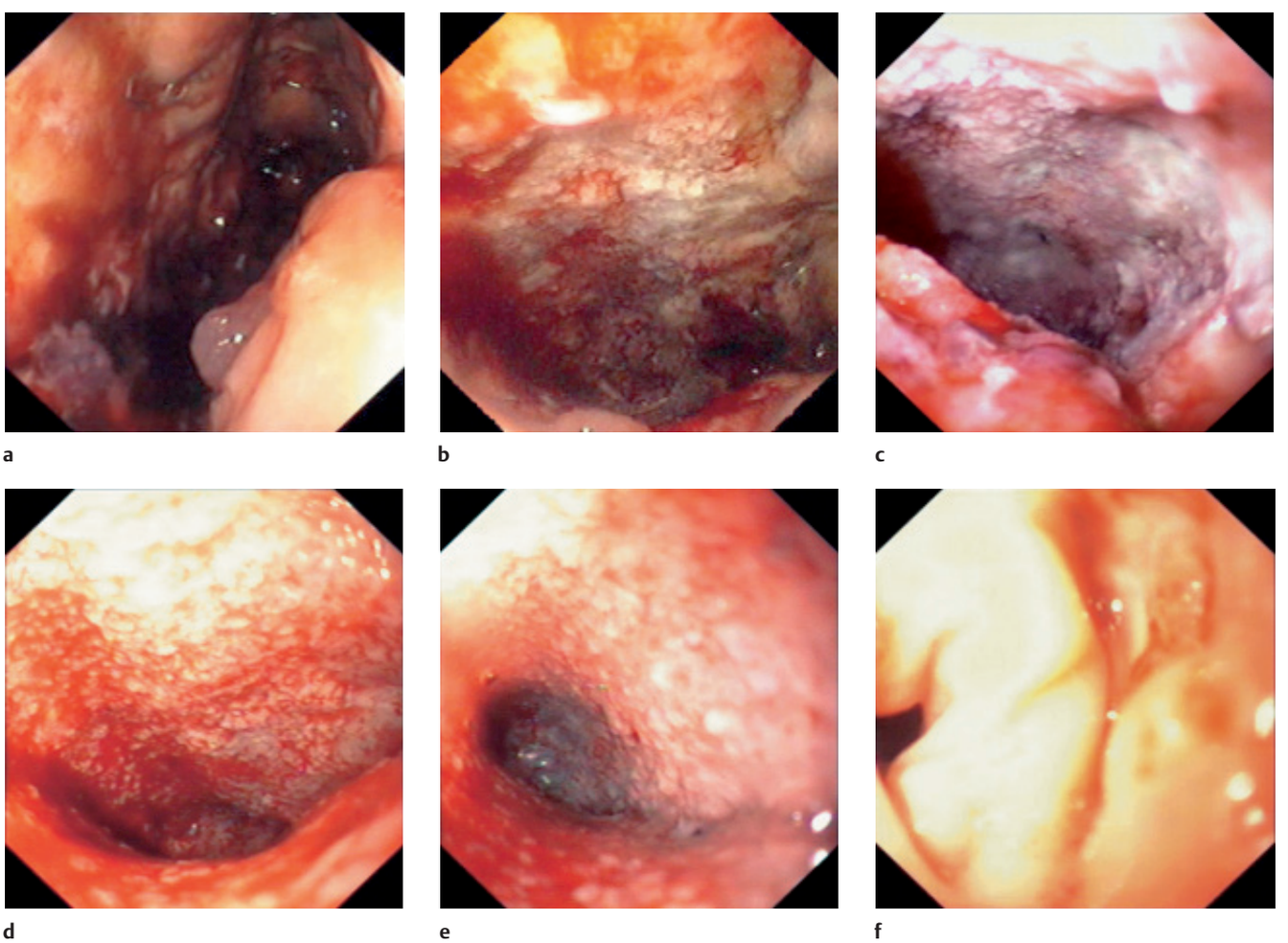


Fig. 22.19 **Principle of EndoVAC system** (R. Weidenhagen)
Advance of a thin-caliber flexible endoscope into the wound cavity. The insertion tube of the EndoVAC system is guided under endoscopic visualization to the end of the cavity. The insertion tube is left there and the endoscope is withdrawn. The compressed sponge system is then introduced into the insertion tube. Deployment of the sponge system over the placement tube occurs on withdrawal of the insertion tube. Positioning is controlled endoscopically. A suction device is connected at the end of the EndoVAC system, which is protruding from the patient's anus, leads to collapse of the cavity, whereby the sponge ideally has contact with the entire cavity wall.

22.6 EndoVAC therapy



a–f Anastomotic leak at 7 cm from the anus with dehiscence over the entire circumference. The endoscope can be advanced 15 cm into the cavity. Healing progression: images from use of EndoVAC therapy on days 1, 4, 11, 18, 21, and 28 (images: R. Weidenhagen).

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23 Removal of Foreign Bodies

W. Schmidbaur

Foreign bodies usually enter the digestive tract through the mouth or the anus. Occasionally they enter transmurally (penetration) or were originally implanted for therapeutic purposes. There seems to be no ingestible object that has not been swallowed at some time and no object that fits in the rectum that has not been placed there (Tab. 23.**1**).

Principles of Endoscopic Foreign Body Removal

The majority of ingested foreign bodies manage to pass the bowel in the short or long term (a number of hours to several days) without any problem and sometimes even leave the body unnoticed and spontaneously. However, foreign bodies can also persist and can cause obstruction—due to the disproportion between their size and the width of the intestinal lumen, especially at sites of predilection—or injure the bowel wall due to their shape (Tab. 23.**2**). Therapy indications are influenced by type, size, and localization of the foreign body. Possible procedures include rigid or flexible endoscopy, surgical measures, or a combination of methods. Removal of foreign bodies is the oldest endoscopic intervention procedure in the gastrointestinal tract: not long after Johannes von Miculicz invented the esophagoscope in 1881, Morell Mackenzie removed a piece of bone from the esophagus.

Pre-examination. Only a third of patients explicitly seeks medical attention for an irremovable foreign body in the rectum and in most cases, manipulation is concealed. Patients tend instead to elusively complain of "pain in the anus." In nearly half of such cases, the foreign body can be detected by digital rectal examination. The remainder of cases requires radiologic evaluation. Indications for the necessity of radiologic studies for diagnosis include atypical sexual behavior, a lax anal sphincter, and blood or mucosal discharge.

Prior to endoscopy, radiographic studies should attempt to precisely localize the foreign body and exclude perforation. Endoscopic removal of foreign bodies is often very time consuming and sometimes risky. Moreover, patient cooperation cannot always be expected. Anesthesiological support during examination can be prudent.

Table 23.**1** Definition

Foreign bodies
▶ Foreign bodies in the gastrointestinal tract are all inorganic objects in the bowel lumen, in rare cases also indigestible food masses
▶ Foreign bodies are usually ingested or introduced per rectum. Dislodged therapeutic devices (e.g., stents) are uncommon

Table 23.**2** Sites of predilection for persistence of foreign bodies in the colon

- Ileocecal valve
- Appendix
- Hepatic and splenic flexures
- Diverticula
- Rectosigmoid junction
- Anal canal
- Existing stenoses, anastomoses

Removal of Foreign Bodies in the Colon

Method

The set of instruments for colonoscopic recovery of foreign bodies corresponds to these used in the upper gastrointestinal (GI) tract. Various polypectomy snares, special forceps with hooked jaws, multiple-pronged grasping forceps, and baskets are used. For mobilization, a balloon can also be used, inflated distal to the foreign body. The selection of instruments is based on shape, material, and size of the foreign body; very often a range of instruments must be tried out (Figs. 23.**1**, 23.**2**).

- Snares are especially suited for removal of longer objects. The closed snare should be slid past the foreign body and opened. Ensnarement should be around the end of the object toward the proximal bowel, due to better leverage when withdrawing it toward the endoscope. Passage of curves or flexures is simpler when the foreign body is as close as possible to the endoscope during withdrawal.
- Alligator forceps are most appropriate for "digging into" to soft foreign bodies improving grasp. The same is true for multiple-pronged grasping forceps, which also have proved useful for grasping flat objects such as coins or blister packs.
- Small baskets can be used universally and are especially good for small, round objects that may also be very hard. Foreign body size is a limitation for all flexible endoscopic devices.
- The availability of radiographic equipment can often be very helpful for the procedure.
- For mobilization of a foreign body in the colon, additional external compression/massage of the abdomen performed by an experienced endoscopic assistant or a second examiner can be helpful. Occasionally, a combined approach with laparoscopic mobilization of a foreign body that has migrated to the colon followed by transanal removal is necessary (6).
- An indication for operative endoscopic or surgical intervention to extract a foreign body is uncommon. In most cases a foreign body will spontaneously pass the colon.

Ingested Foreign Bodies

Ingested foreign bodies that pass the pylorus are often harmless, especially in adults, and usually manage to pass the colon, embedded in stool, without causing any problems. Exceptions are bizarre objects with sharp edges, such as chicken bones, which can become wedged transversely in the colon and impact the wall (3), perforating the bowel.

Batteries. Batteries are problematic because they contain toxic and/or caustic metallic salts (silver oxide, zinc oxide, mercury oxide, or lithium oxide) or alkalis (sodium hydroxide or potassium hydroxide). In particular, small button batteries common nowadays in electronic devices are very easily ingested. They are also not that easy to manage endoscopically, given their shape. Unlike with most other ingested objects, if the battery is already out of reach and not primarily removable with esophagogastroduodenoscopy, its further passage through the GI tract must be followed radiologically under close clinical surveillance. Button batteries are especially vulnerable to corrosion within a few hours and can leak. If further passage is not rapid, intervention is indicated. If the foreign body is within reach of colonoscopy after passage, endoscopic intervention can be reattempted. If not, surgical recovery using laparotomy is necessary. Snares, dormia baskets, and Roth Nets (with a trap device connected to the snare) are suitable for extraction.

Foreign bodies in the appendix. Small, ingested foreign bodies such as dentures can sometimes be localized in the appendix (Fig. 23.**3**). In most cases, appendicitis will necessitate endoscopic extraction or appendectomy. Endoscopy may be attempted, though foreign bodies located in the appendix are often out of reach of the endoscope unless a portion happens to be protruding into the cecum. The "wait-and-see" approach is recommended for blunt foreign objects with unremarkable clinical signs (5).

Iatrogenic Foreign Bodies

The second most common category of foreign bodies comprises those of iatrogenic origin, such as probes, balloons or metal/plastic prostheses.

Biliary prostheses. Plastic biliary prostheses usually pass the bowel spontaneously. However, they can also result in GI tract obstruction. If the prosthesis can be reached, it can be extracted with a forceps or polypectomy snare.

Metal stents. Dislodged metal stents such as duodenal enteral stents or colonic stents are problematic because of their size as well as their sharp edges and ends. They can easily cause obstruction as well as injury of the bowel wall with resulting bleeding or perforation. Retrieval is relatively difficult, especially if the stent is located above a stenosis. Endoscopic removal should be attempted (7), though surgical intervention may prove necessary.

For removing a dislodged metallic mesh stent, a similar technique is attempted as that used, for example, with esophageal stent migration in the stomach. The aim is to slide a snare over the stent, positioning it as close to the middle as possible, and closing it there. This causes the stent to collapse in a V-shape in the middle and the stent ends to fold in, away from the direction of pull, preventing the sharp or pointed edges from

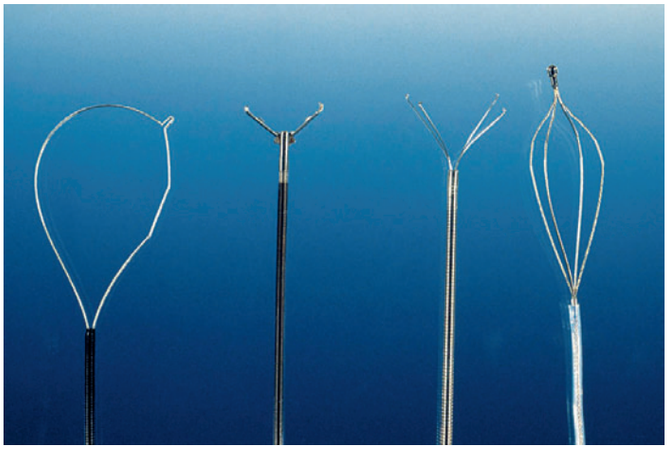


Fig. 23.**1** **Instruments for removal of foreign bodies in flexible endoscopy.** Foreign body retrieval snare, alligator forceps, four-pronged grasping forceps, and basket.

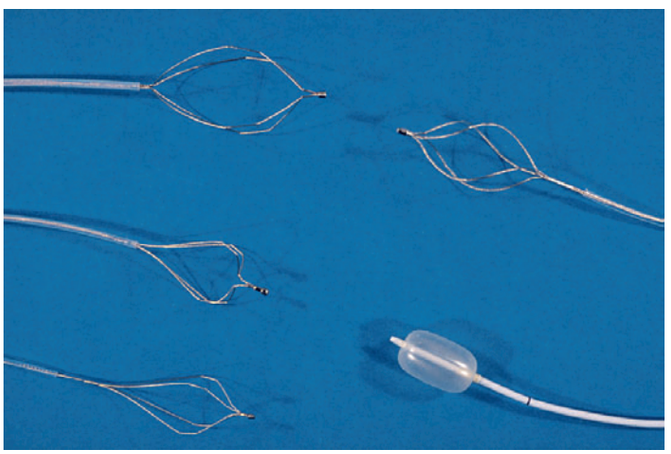


Fig. 23.**2** **Instrumentation for retrieval of smaller foreign bodies in flexible endoscopy.** Dormia baskets made of monofilament and wire; also a balloon for extraction.

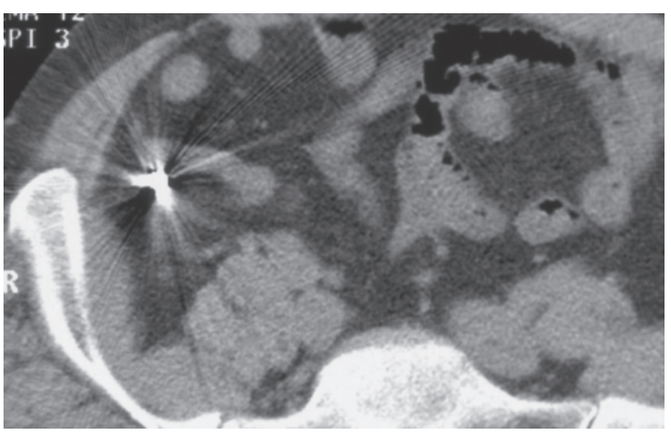


Fig. 23.**3** **CT image of a solid metal foreign body and artifacts in the appendix region.** This turned out to be a swallowed gold tooth.

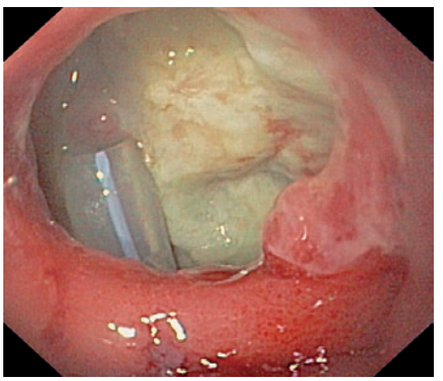


Fig. 23.4 **Iatrogenic foreign body;** surgically placed local drainage visible through an anastomotic leak after low anterior rectal resection.

causing injury on withdrawal. One case study reported an interesting technique for stent retrieval of a stent that was wedged in the rectum so that a snare could not be used (4). A wire with a flexible tip was advanced through the stent mesh, creating a loop in the rectum. After sliding out the endoscope, leaving the wire, the instrument was advanced again and the end of the wire lying in the rectum was ensnared and withdrawn. Afterward the stent was withdrawn through the anus by pulling on the loop.

Gastric balloons. Gastric balloons (e.g., BioEnterics) that remain in the stomach for several months can migrate in the GI tract after spontaneous emptying of water contents, causing symptoms of partial bowel obstruction or ileus (1). Before extraction, care must be taken that the balloon is completely empty. If not, it can be perforated in several places with a thick injection needle and the fluid suctioned. The balloon wall is very smooth and firm which makes normal snares or grasping devices slide off. The optimal instrument is a specially developed hooked grasping forceps (BioEnterics); if one is not available, our own experience shows that a sharp alligator forceps can be used to poke a hole in the balloon after which the forceps can be anchored in the hole for extraction.

Drainage. Figure 23.**4** shows a surgically placed local drainage that was protruding into the colon lumen from a wound dehiscence, preventing closure of the dehiscence. The local drainage was removed and the dehiscence wound closed with repeated sessions of irrigation and fibrin application.

Clips. Adherent hemoclips in the colon—as used for achieving hemostasis or fistula closure—are considered harmless foreign bodies (Fig. 23.**5**). There is thus no urgent need to remove them. Our own experience with one patient who "suddenly" recovered after retrieval of a hemoclip used for hemostasis in the sigmoid colon following polypectomy, should certainly be considered merely anecdotal evidence.

Foreign Bodies Introduced Per Rectum

Elongated foreign bodies inserted into the rectum are especially prone to migration during anal or rectal manipulation of the colon, sometimes surprisingly far, into the splenic or even hepatic flexures (8).

Colonoscopic removal is usually possible (e.g., removal of a bag of heroin in Fig. 23.**6**). Although this case of a "bodypacker" involved only minimal amounts of drugs, removal of a suspicious object should nonetheless be attempted very carefully. Tearing the bag open would release the drugs, potentially causing life-threatening intoxication.

Removal of Foreign Bodies in the Rectum

The spectrum of objects inserted transanally is extremely varied and a list of possible objects is provided in Tab. 23.**3**. Motives are mostly sexual or criminal in nature and the type and size of the objects often exceeds the bounds of anatomical and physiological imagination. Transanally inserted foreign bodies often involve the added complication of serious injury to the rectosigmoid and also the sphincter muscles, which often require complex proctological surgical reconstruction. The situation is often made worse by laymen's attempts at removal using unsuitable

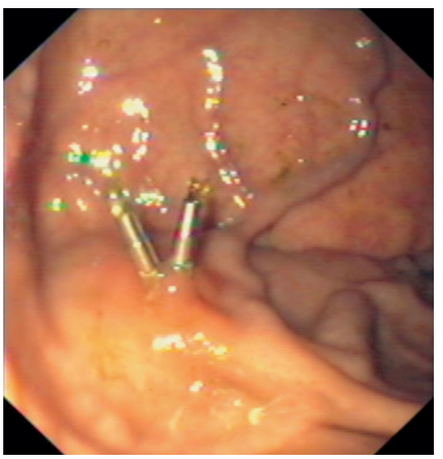
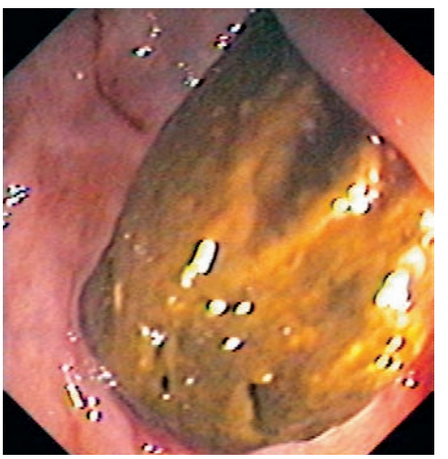


Fig. 23.5 **Iatrogenic foreign body;** a result of prior fistula management with adherent Endoclips.



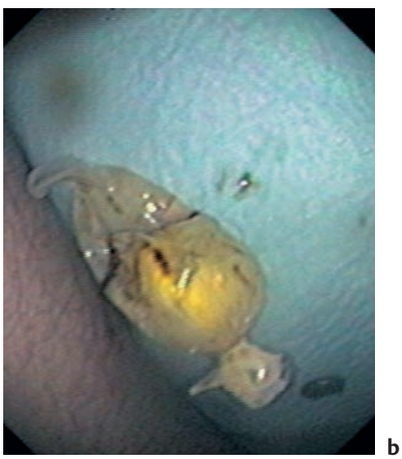


Fig. 23.6 **Drug pouches.**
a Endoscopic aspect of a foreign body that slipped upward into the sigmoid colon.
b The same foreign body after retrieval. A one-day supply of heroin contained in a condom and concealed in the rectum.

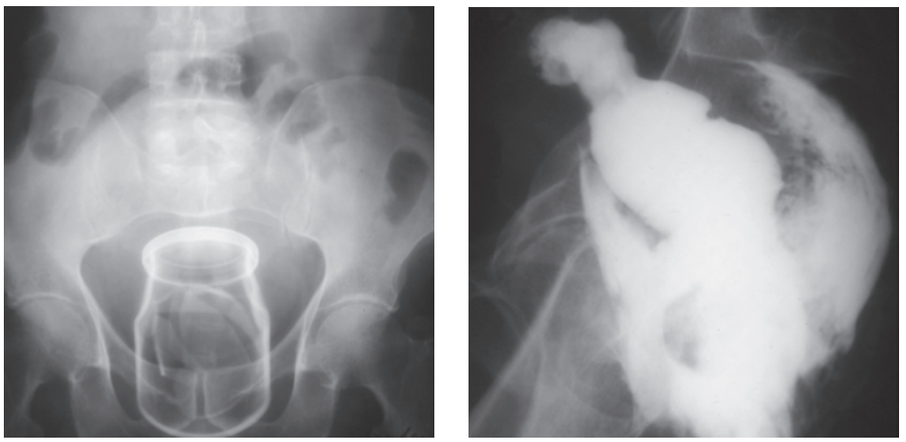


Fig. 23.7 **Perforation from a foreign body.**
a Radiographic image of a glass jam jar with lid, inserted rectally.
b After transanal surgical extraction of the jar, contrast enema revealed rectal perforation.

methods or by the patients delaying seeking medical attention for several days. In most cases, manipulation is not admitted.

Method

- Anorectal foreign body retrieval belongs to the domain of rigid endoscopy and a vast arsenal of instruments, ranging from wide forceps and balloon catheters to an obstetric forceps.
- General anesthesia is usually necessary due to the pain involved in intervention and to achieve better relaxation of the sphincter muscles.
- Foreign body removal in the rectum can be exceptionally difficult and requires an experienced surgeon; therapy must often be varied according to the individual situation. Laparotomy is necessary in ca. 10% of patients.
- Perforation must always be excluded after removal of a foreign body, and the patient must be adequately observed.

Figures 23.**7**–23.**9** show examples of successfully removed foreign bodies. Containers open at the top (glass jars, bottles) can complicate removal in that they may suck themselves firmly to the rectal wall. Figure 23.**10** shows a patient who presented with prior anal bleeding. After cessation, an elongated defect was found in the rectum. Patient history revealed insertion of a plastic bottle.

Table 23.**3** Examples of foreign bodies introduced into the rectum (based on 2)

- Razor blades, screws, screwdrivers, can openers, hairpins
- Fish bones, bone splinters
- Drug pouches
- Chair leg, spade handle, vacuum cleaner attachment
- Cola bottle, champagne bottle, jam jar
- Spray can, light bulb, candle
- Ping-pong ball, boccia ball
- Bullet
- Cucumber, carrot, corncob, banana
- Rolled-up newspaper
- Massage stick, vibrator, hard rubber tubing, rubber phallus

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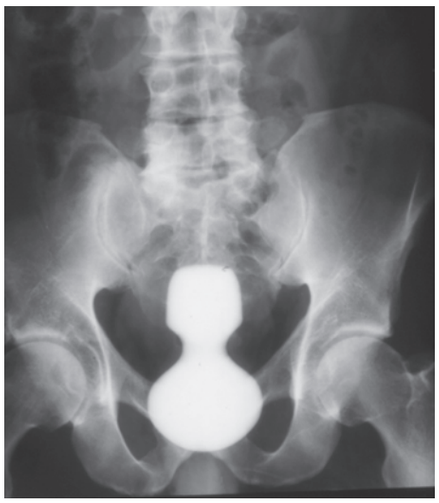

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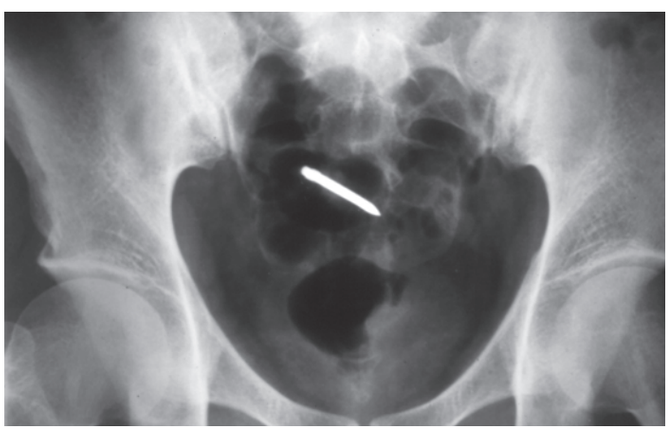
Fig. 23.**8** **Glass stopper.**
a Foreign body inserted per rectum prior to retrieval (in abdominal radiograph).
b Rectally inserted foreign body after removal.



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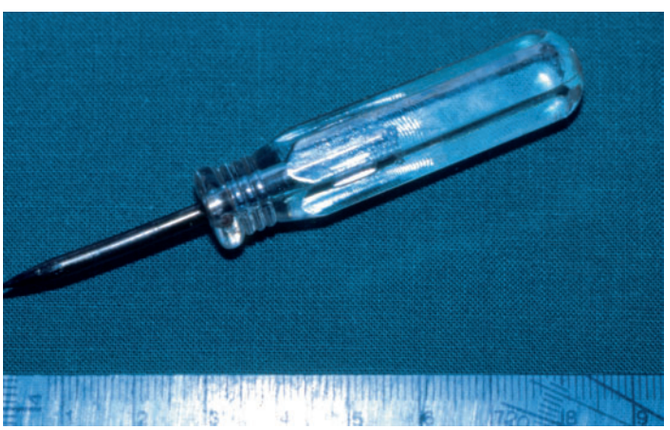

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Fig. 23.**9** **Screwdriver.**
a Solid metal foreign body in the colon, which had to be removed with operative intervention.
b The object seen on radiograph turned out to be a screwdriver.

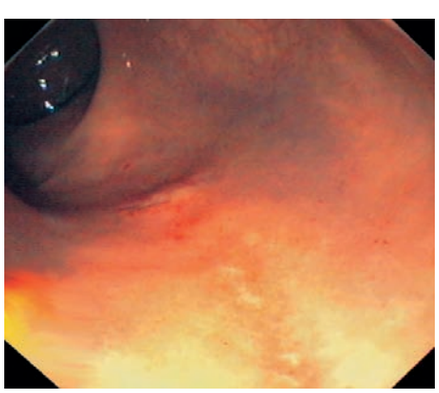


Fig. 23.**10** **Prior lower GI bleeding (self-limiting).** Mechanical lesion in the rectum after insertion of a plastic bottle.

24 Decompression Tube Placement

J. Barnert

Definitions

Distention of the colonic lumen has various causes. Two forms of distension are basically distinguished: prevented passage due to mechanical obstruction leading to dilation of proximally located bowel segments and dilation of the colon without mechanical obstruction, i. e., pseudo-obstruction of the colon.

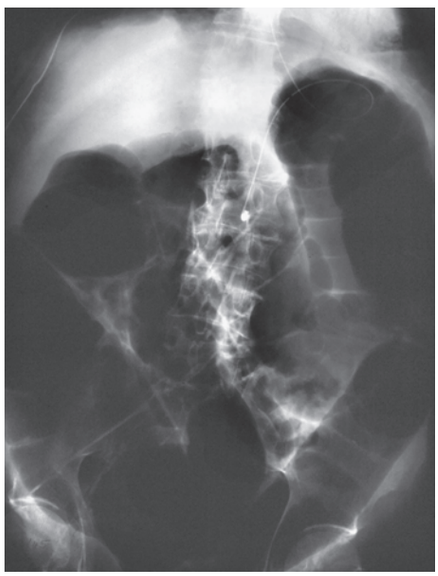


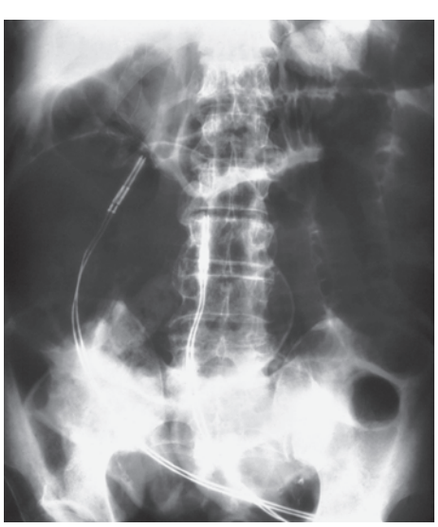
Fig. 24.**1** **Pseudo-obstruction of the colon.** Abdominal radiograph in a patient with sepsis syndrome on artificial respiration and with massive distention of the entire colon, especially the cecum (image provided courtesy of Dr. V. Remplik, Institute for Diagnostic Radiology and Neuroradiology, Augsburg Clinic).

Acute Pseudo-obstruction of the Colon

Definition and Pathogenesis

In 1948 Leithauser and Ogilvie simultaneously reported acute dilation of the colon without mechanical obstruction (15, 20). This entity, nowadays referred to as acute pseudo-obstruction of the colon (synonym: Ogilvie syndrome) is uncommon, but can lead to massive problems. Radiographs demonstrate distention of the colon, as a rule affecting the cecum, ascending colon, and transverse colon; in rare cases the descending colon is also affected. The decisive factor in diagnosis is distention of the diameter of the cecum to more than 9 cm (Figs. 24.**1**, 24.**2**). The cecum in a healthy individual is 3.5–8.5 cm in diameter. Colon haustration is maintained.

Pathogenesis remains somewhat unclear. It is not certain whether primary abnormal motility is in the right or left hemicolon. One possibility is that relaxation of the smooth musculature in the right hemicolon leads to an accumulation of gas and enteral contents. However, it is also possible that abnormal motility in the left hemicolon causes functional obstruction of passage and thus distention of the colon segments proximal to this region. The causes of acute pseudo-obstruction are varied (Tab. 24.**1**). According to compiled studies (30) pseudo-obstruction occurred in 23% of patients following an operation and in 11% following trauma; in 17% it was associated with cardiopulmonary disease and in 15% with systemic disorders, such as metabolic imbalance, intoxication, and infection.



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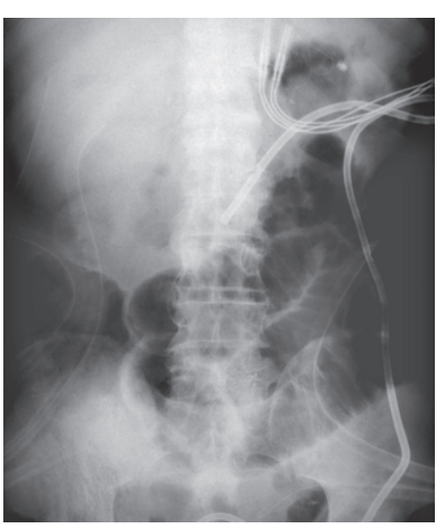

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Fig. 24.**2** **Acute pseudo-obstruction of the colon** in an intensive care patient who had to undergo complicated cardiac surgery.
a Abdominal radiograph with distended colon (ECG lead on the abdomen).
b Radiographic image after endoscopic decompression of the colon and placement of a decompression tube (Wilson–Cook), the tip of which is in the transverse colon (images provided courtesy of Dr. V. Remplik, Institute for Diagnostic Radiology and Neuroradiology, Augsburg Clinic).

Table 24.**1** Diseases associated with acute pseudo-obstruction of the colon

Postoperative conditions
▶ Laparotomy
▶ Orthopedic operations (hip, knee)
▶ Gynecological interventions (cesarean section, hysterectomy)
▶ Urological interventions
▶ Neurosurgical operations (laminectomy)
Traumatology
▶ Retroperitoneal injuries
▶ Spinal cord injuries
▶ Burns
Systemic and neurological diseases
▶ Infections (sepsis, viral infections)
▶ Diseases of the central and peripheral nervous system (Parkinson disease, multiple sclerosis, Guillain–Barré syndrome)
▶ Cardiopulmonary diseases (heart failure, pneumonia, artificial respiration)
▶ Endocrine and metabolic diseases (hypothyroidism, diabetes, electrolyte imbalances)
▶ Renal insufficiency
▶ Carcinomas (paraneoplasias, nerve infiltrations—lumbar and retroperitoneal)
▶ Gastrointestinal and hepatological diseases (pancreatitis, liver failure)
Medication/Drugs
▶ Narcotics (morphine, benzodiazepine)
▶ Psychopharmaceuticals (tricyclic antidepressants, phenothiazine)
▶ Anticholinergic drugs
▶ Clonidine
▶ Cytostatic drugs (Vincristine)
▶ Calcium antagonists
▶ Anti-Parkinson drugs
▶ Digitalis drugs
▶ Alcohol

Clinical Picture and Course

The chief symptom is abdominal distention. The abdomen remains soft, however, and does not demonstrate any localized guarding. Around 80% of patients complain of abdominal pain (29), though it is generally not severe. One study reported only 10% of patients complaining of pain symptoms (11). Severe abdominal pain is a sign of complications such as ischemia and perforation. The patient is usually constipated, but may also present with diarrhea or watery stool. Patients complain of nausea and vomiting in slightly more than half of cases.

Perforation and bowel ischemia. In 3% (24) of patients overdistention can lead to perforation of the cecum as the cecum has the thinnest bowel wall of all colon segments. The risk of perforation appears to begin occurring at a cecal diameter > 12 cm; at > 14 cm it rises to 23% (29). Duration of distention of the cecum also appears to be correlated with perforation risk. If perforation occurs, mortality increases to 43–46% (18, 25). In 7–10% of patients with acute pseudo-obstruction of the colon (11, 27, 29), pseudo-obstruction is associated with bowel ischemia, probably as a result of increased transmural pressure. Mortality associated with acute pseudo-obstruction of the colon is difficult to estimate, as most seriously ill patients die of grave underlying causes and not as a result of colon distention. Mortality in conservative therapy is reported at nearly 10% (overview in 30).

Therapy

Conservative therapy. The first step in therapy of acute pseudo-obstruction is the correction of factors supporting it. This includes correcting electrolyte imbalance and other metabolic imbalances. A nasogastric tube should be attached to relieve the upper gastrointestinal tract and the patient should remain on a liquid diet. Forced mobilization and body positioning (frequent changing of patient position, lying on the abdomen with raised pelvis) can help empty the large bowel of gas. The only scientifically confirmed method in pharmacological therapy is the use of the cholinesterase inhibitor neostigmine. Ponec et al. (22) showed that in 10 out of 11 patients in the group receiving neostigmine, intravenous administration of 2 mg of neostigmine led to a decrease in the diameter of the cecum, stool, and wind; among the 10 patients in the placebo group, there was no effect. However, two of the 10 patients who initially responded to neostigmine suffered a relapse.

Endoscopic therapy. In 1977 Kukora and Dent (14) became the first the show that colonoscopic decompression of the distended bowel in acute pseudo-obstruction is both possible and effective. However, there is a problem in that in 20% of patients successful decompression is followed by recurrence (30). Taking individual reports together, decompression would achieve relief of acute pseudo-obstruction in the colon in 80% of patients (30), although in individual studies such as one by Harig et al. (13) the failure rate is much higher at 50% as is the recurrence rate at 44%. Bernton et al. (4) optimized this method of endoscopic therapy by placing a decompression tube in the colon after suctioning the air during colonoscopy and then leaving the tube in the patient for a period of time (Figs. 24.**2**, 24.**3**). In the studies compiled by Wegener and Boersch (30) the initial success rate with additional placement of a decompression tube was 86%; therapy was ultimately successful in 79% of patients. The rate of complications related to colonoscopic decompression methods is low at 0–4% (8). Perforations are the most serious complication. Opinions diverge on whether or not bowel ischemia, occurring in 7–10% of patients with acute pseudo-obstruction, is a contraindication. Various authors have reported successful decompression despite existing bowel ischemia (10, 17).

It takes an experienced examiner to perform colonoscopic decompression. Preparation of the patient in terms of bowel cleansing is not possible. Additionally, morphine should be avoided, in order to retain as much bowel motility as possible. Visualization during colonoscopy is generally poor, because of stool and massive bowel distention. However, stool in acute pseudo-obstruction of the colon—not including the sigmoid and rectum—is usually watery and can be suctioned (Fig. 24.**4**).

Procedure

Given the presence of stool, the selected colonoscope should have the widest working channel possible. Air insufflation should be kept to a minimum during examination. Examination under such conditions requires a great deal of time and should be planned accordingly. Especially in the right hemicolon, attention should be paid to bowel wall appearances in order not to miss potentially existing ischemia. As already mentioned, bowel ischemia in the colon is not an absolute indication for discontinuing examination. In any event the examiner should attempt to reach the cecum.

The decompression tube should be placed after reaching the cecal pole. Various techniques have been described in the literature. Some authors insert the tube in "piggyback" fashion on the colonoscope, either grasping the tip of a catheter with a snare (26) or grasping a thread fixed to the tip of the catheter with a forceps (4, 12). This technique may be useful when there is no potential for radiographic control.

Most authors, including ourselves, prefer catheter placement using Seldinger technique (Fig. 24.**5**). In this method, after reaching the right hemicolon—best done after intubating the cecum—a firm, 480 cm long and 0.035 inch wide wire is advanced in the lumen. The colonoscope is carefully withdrawn under intermittent radiographic control and at the same time the wire is advanced so that the tip remains in position (in the cecum or transverse colon). At the same time, as much air is suctioned from the colon as feasible. After the endoscope has been removed from the colon, the actual decompression tube (polyethylene), which has several large holes on the sides of its proximal end and a guiding catheter inside, is advanced over the guidewire toward the cecum under continual radiographic control. Problems can arise if the wire begins "looping" in the colonic loops. It is thus important that the guiding catheter containing the wire always be held taut without allowing the wire to slip out. Slight rotation of the guiding catheter during passage of angulated or looping colon segments is sometimes helpful. When the decompression tube has reached the desired location, the wire is first removed from the guiding catheter and then the guiding catheter is removed from the decompression tube. For this method to work smoothly, the wire and guiding catheter should be lubricated with e.g., MCT oil. The decompression tube is then fixed on the patient's buttocks or the inner thigh with adhesive tape and can remain for several days without a problem. Regular cleaning of the tube several times daily prevents clogging and ensures drainage. In addition to this coaxial tube, there are also others where first a wire is introduced and then the decompression tube is advanced over this. The number and placement of holes on the side of the decompression tube can vary (Fig. 24.**6**). Specially made, anatomically adapted tubes have also been developed (9) (Fig. 24.**7**).

There are different opinions concerning optimal position of the tip of the decompression tube. It appears, however, to be sufficient that the tip be placed at least in the transverse colon (Fig. 24.**2**); it is not absolutely essential that it be placed at the cecal pole (11). In order to solve the problem of difficult passage of the sigmoid and avoid looping of the catheter, it has been suggested that a sigmoid stiffener be used, through which colonoscope and catheter are advanced (Fig. 24.**8**). Using a sigmoid stiffener, Berger et al. (3) successfully placed decompression tubes in all cases; in the control group using conventional techniques success occurred in only three out of seven patients.

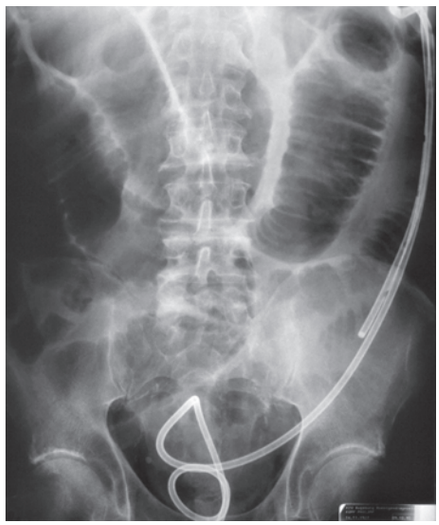


Fig. 24.**3** **Decompression tube.** Abdominal radiograph after endoscopic decompression in a patient with acute pseudo-obstruction of the colon. The tip of the tube is inverted in the descending colon, resulting in malpositioning of the decompression tube.

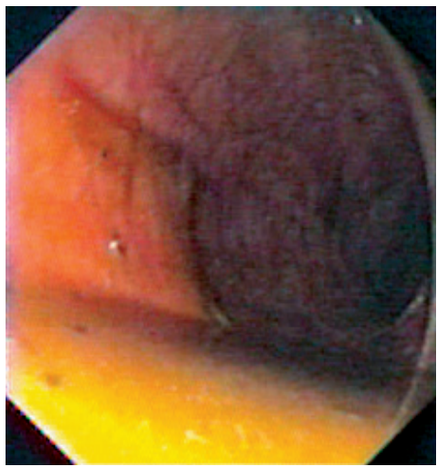
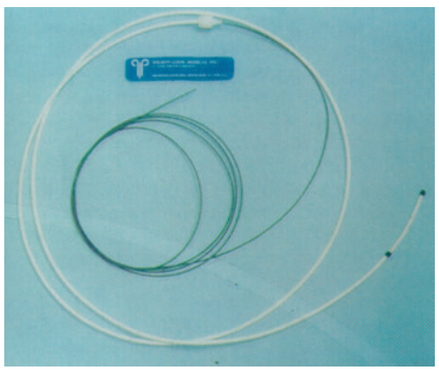


Fig. 24.**4** **Endoscopic aspect in acute pseudo-obstruction of the colon.** The lumen is dilated and filled with watery stool.

Surgical intervention. Surgical intervention is a last resort for acute pseudo-obstruction as it is associated with a mortality rate of 35 % (30). An alternative to the usual cecostomy is catheter drainage of the cecum, an intervention that can be performed under local anesthesia. Laparoscopic attachment of a cecostomy has also been reported (5). Prior to performing this minimally invasive technique, however, it is necessary to exclude perforation and bowel ischemia. Figure 24.**9** provides an overview of the therapeutic algorithm for acute pseudo-obstruction.



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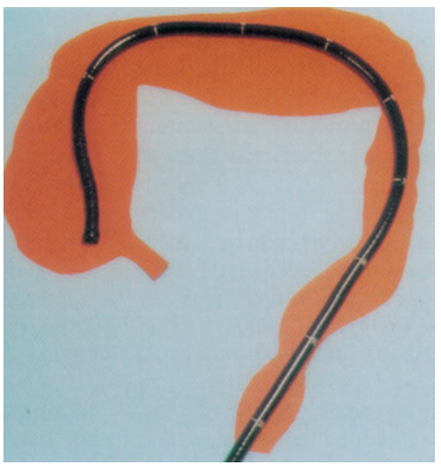

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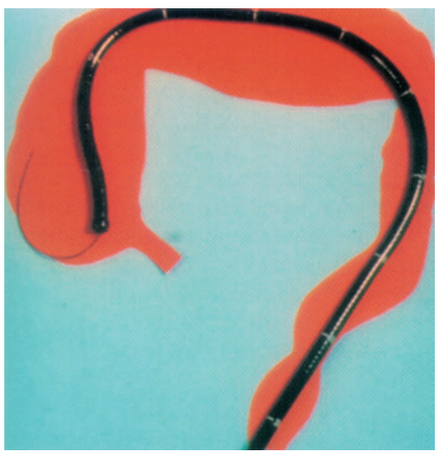

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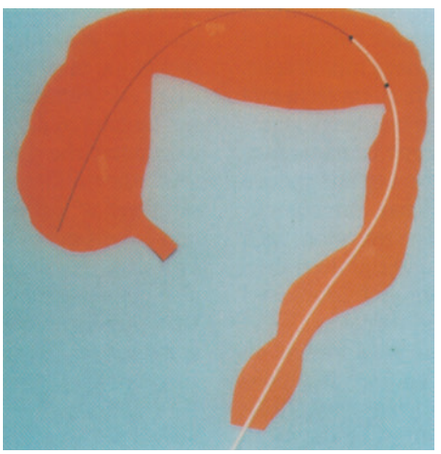

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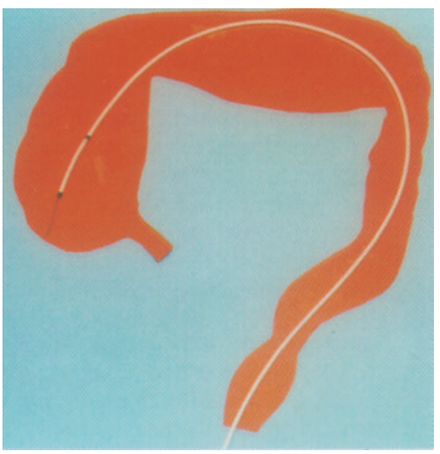

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Fig. 24.**5** **Endoscopic placement of a decompression tube in the colon based on Seldinger technique** (from 21).

a Commercially available coaxial decompression tube from Wilson–Cook with guidewire.

b Decompression tube with inner pilot wire to stiffen the tube.

c Endoscopic intubation of the colon to the cecum.

d Advancing the wire through the endoscope. Careful withdrawal of the instrument (under radiographic control), leaving the tip of the wire in the cecum.

e The decompression tube with the inner guiding (stiffening) catheter wire is advanced (also under radiographic control) over the guidewire already placed in the colon lumen. The wire is held firm with slight traction.

f The tip of the decompression tube has reached the cecum. The inner pilot wire has thereafter to be carefully withdrawn (also under radiographic control).

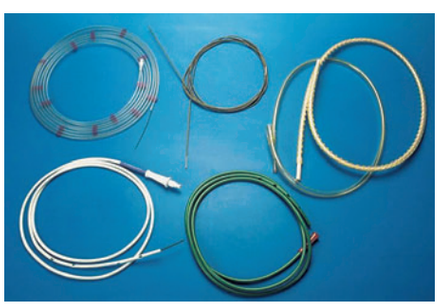


Fig. 24.**6** **Various models of decompression tubes** from various manufacturers.

Mechanical Obstruction

Volvulus

Pathogenesis. Colonic volvulus is a rotation of the colon around an often elongated mesocolon (Fig. 24.**10**). The sigmoid colon is most often affected (> 70%); the cecum and transverse colon are markedly less often affected. Colonic volvulus is a common cause of colonic obstruction in developing countries; elongation of the sigmoid due to the fiber-rich diet is thought to play a role etiopathogenically. In the western world, colonic volvulus is often associated with neurological diseases (Parkinson disease, multiple sclerosis, diseases of the spinal cord) and psychiatric disorders. In psychiatric disorders, it is the administration of psychopharmaceuticals with negative side effects on colonic motility that is blamed. Patients with severe constipation are also at risk.

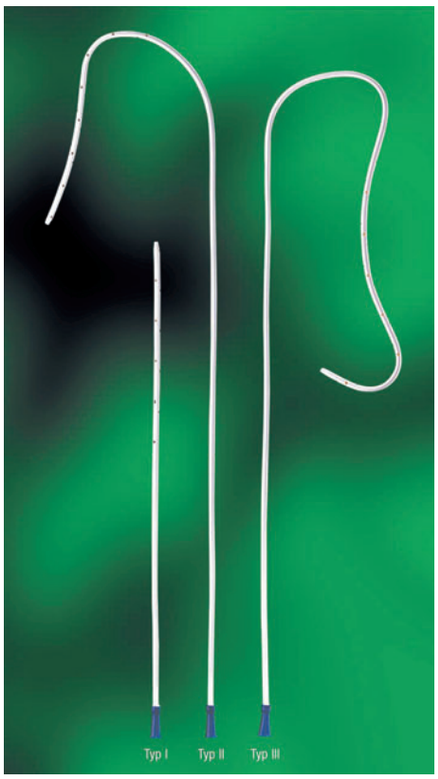


Fig. 24.7 **Anatomically adapted decompression tubes** (based on 9) available from Bard for decompression of the descending colon (left), the transverse colon (center), and the ascending colon (right).

Fig. 24.8 **Overtube (Olympus) serving as stiffener.**

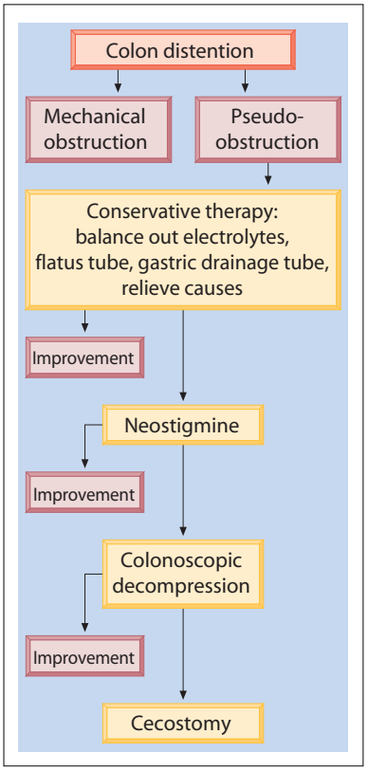


Fig. 24.9 **Algorithm for therapeutic approach in pseudo-obstruction of the colon.**

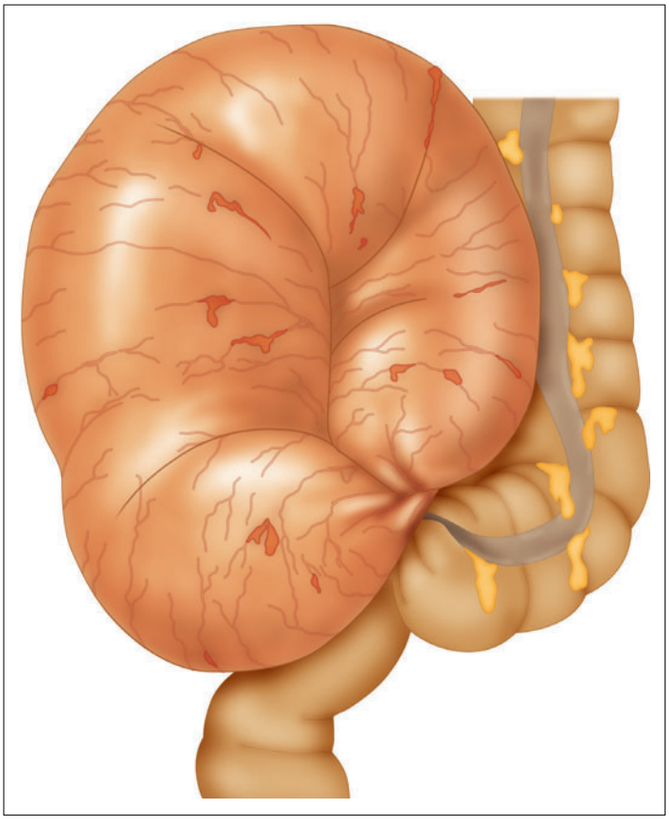


Fig. 24.10 **Volvulus in the sigmoid colon** (based on 19). The sigmoid is twisted around its mesocolon, resulting in ischemia of the torsioned sigmoid colon.

Clinical picture. Patients present with abdominal pain, constipation, and gas. More than 50% of patients report similar prior attacks which spontaneously improved. Torsion of the affected colon segment and its mesentery results in obstructed passage and ischemia of the affected bowel. In a typical case radiologic diagnosis is not difficult. Abdominal radiography reveals a dis-

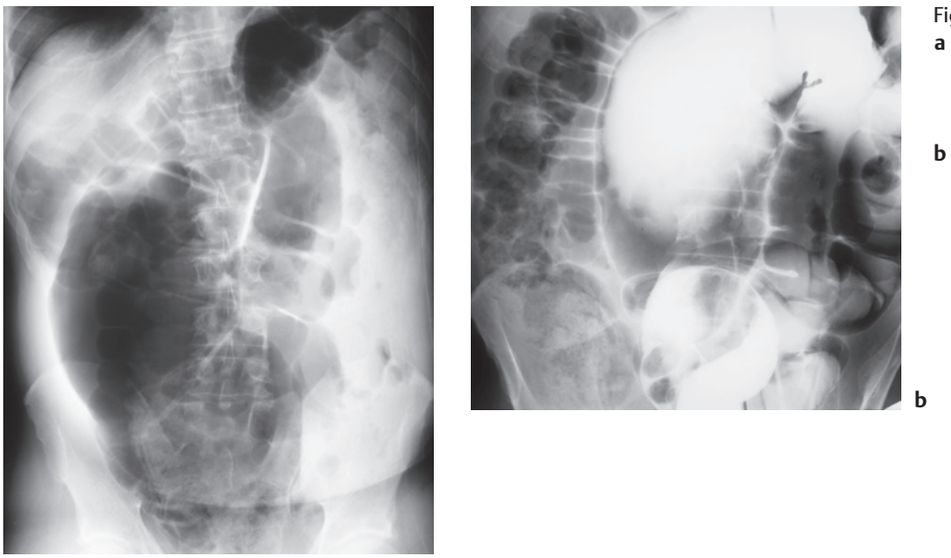


Fig. 24.**11** **Volvulus in the sigmoid colon.**
a Radiographic image without contrast agent: overdistended sigmoid colon demonstrating typical "coffee bean" appearance.
b Radiographic image after rectal administration of a contrast dye for better visualization of the torsioned sigmoid (images provided courtesy of Dr. V. Remplik, Institute for Diagnostic Radiology and Neuroradiology, Augsburg Clinic).

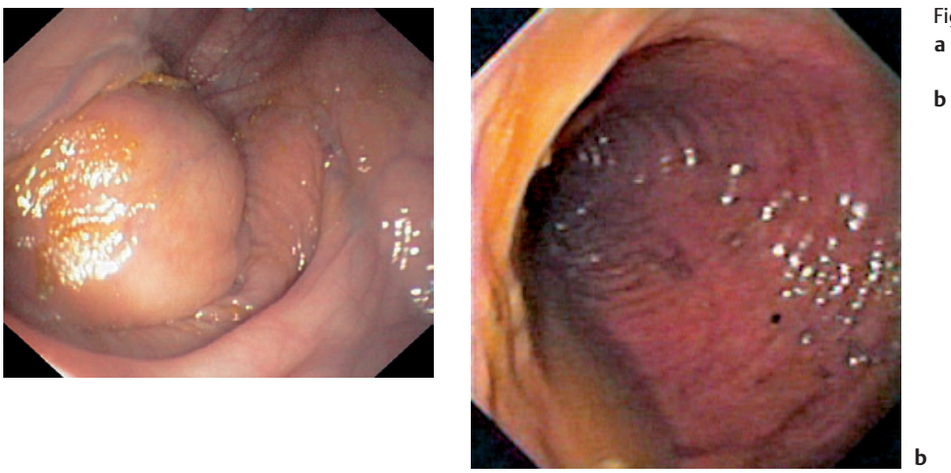
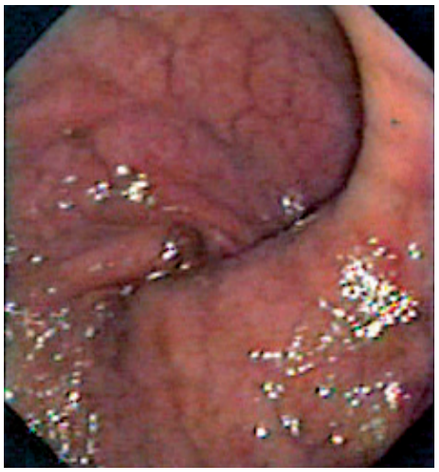


Fig. 24.**12** **Sigmoid volvulus.**
a Endoscopic appearance of torsion with spiraling mucosa.
b Dilated sigmoid lumen near the volvulus. Mucosa shows no signs of ischemia.

tended sigmoid colon loop (Fig. 24.**11**), often with the typical appearance of a coffee bean, upside-down "U" or omega. Interpretation of a radiograph without use of a contrast agent is sometimes difficult.

Endoscopic therapy. Urgent surgical intervention is associated with a mortality rate of 50%; thus, an alternative to surgery is desirable. Endoscopic reversal of torsion and decompression of a sigmoid volvulus has a success rate of 58–100% (6, 16, 23). Figures for volvulus in other colon segments are lower. Unfortunately, reversal of torsion is accompanied by a high rate of recurrence (30–90%). Leaving a decompression tube in the colon for two to three days following successful therapy appears to be an important factor in success.

Procedure
The rectum is generally empty at endoscopy and should be carefully insufflated with air. At the rectosigmoid junction or just beyond it there is a point where the lumen is obstructed by torsion of the sigmoid colon (Figs. 24.**12**, 24.**13**). The endoscopic aspect is one of a spiraling mucosa. The endoscopist should attempt to carefully pass this point, though this often means passing along the mucosa without direct vision. If the mucosa is ischemic, the scope should never be advanced without visualization (Figs. 24.**13 b**, 24.**14**). Air should be intermittently insufflated to assist in the reversal of the torsion. If the distended and torsioned sigmoid colon can be reached (Figs. 24.**12 b**, 24.**13 b**) usually a considerable amount of air and watery stool can be suctioned, which in turn usually untwists the torsion. Attention should be paid at this point (as before) to possible bowel ischemia (Figs. 24.**13 b**, 24.**14**) or even necrosis. The mucosa should be washed off and if it appears healthy, the scope can be advanced until reaching the colon segment before the



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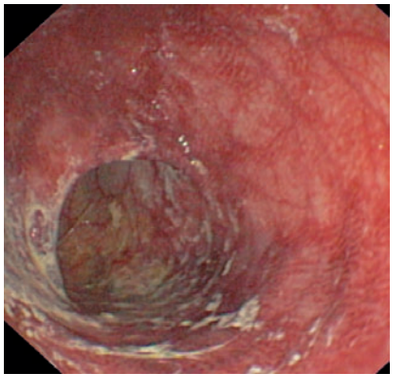


b

Fig. 24.**13** **Sigmoid volvulus.**
a Endoscopic aspect of torsioned and closed-off sigmoid lumen.
b Dilated lumen near the volvulus with ischemic (nontransmural) mucosa.

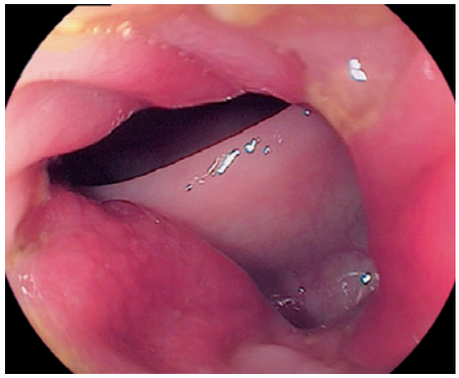


Fig. 24.**14** **Sigmoid volvulus.** Reddened and apparently ischemic mucosa.

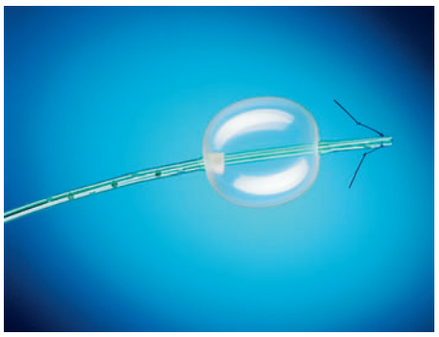
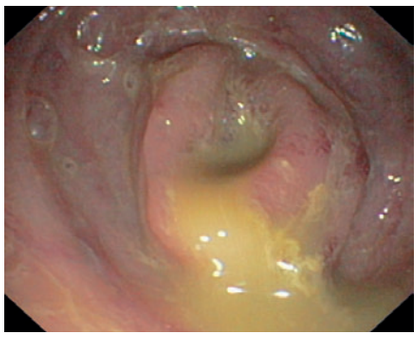


Fig. 24.**15** **Decompression tube with inflatable balloon at its tip** for better anchoring (preventing dislodgement) in the stenosis (Wilson–Cook).



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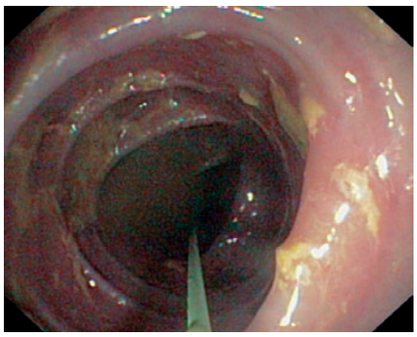

b

Fig. 24.**16** **Sigmoid carcinoma causing stenosis.**
a Endoscopic appearance of the carcinoma.
b Dilated colon above the stenosis with wire already in place. Using Seldinger technique a decompression tube was placed over the wire for lasting decompression.

volvulus where a decompression tube is placed using the technique described for acute pseudo-obstruction (Fig. 24.**5**).

Definitive surgical therapy is desirable due to the high rate of recurrence after endoscopic reversal of torsion. The mortality rate in elective intervention (after prior reversal of the volvulus) is reported at 6% (2). In younger patients without any underlying diseases, surgical intervention in the form of resection of the elongated colon segment is generally unproblematic (1).

Malignant and Benign Stenoses

Therapy of malignant and benign stenoses is also described in Chapters 19 and 21.

The results are rather poor for patients in whom colon carcinoma has led to complete bowel obstruction (Fig. 24.**16**). Thus, colostomy is attempted to relieve the large bowel. If possible, endoscopic decompression of the distended colon proximal to the stenosis should first be performed and then a one-time intervention with resection of the tumor. Placement of a metal stent is one possibility for decompression and is successful in 80–90% of patients. A drawback to this procedure is the relatively high cost of the stent. Additionally, stent placement is not successful in every patient due to anatomical reasons, for example, sharp lumen angulation caused by the malignancy. Placement of a decompression tube—which is also much less costly—may he useful in this situation (Fig. 24.**15**).

Procedure
The technique for placing a decompression tube is performed in much the same way as for acute pseudo-obstruction, though the occluding tumor is often impossible to pass. In such cases, cannulation and passage of the malignant stenosis with a Terumo wire should be attempted (Fig. 24.**16 b**). After replacing the soft wire with a more rigid one, one can often advance the decompression tube through

the stenosis. If this is not successful, careful dilation of the stenosis with a balloon can still be attempted. After placing the probe, the dilated colon segment should be thoroughly cleaned with fluid irrigation and prepared for operation.

Eguchi et al. (7) report that the placement of a decompression tube in malignant obstruction was clinically successful i. e., distention was relieved, in 44 %. The patients who benefited most were those in whom the onset of ileus was less than three days previously. Tanaka et al. (28) had an even higher clinical success rate at 62 %; endoscopic placement of the tube was successful in 34 of 36 patients without any problems.

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