APPENDIX AND CECUM

Embryology, Anatomy, and Surgical Applications

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DEVELOPMENT OF THE CECUM AND THE VERMIFORM APPENDIX

The development of the cecum and of the vermiform appendix (Fig. 1) is closely related to the development of the midgut. Four structures are considered as intestinal derivations of the midgut: the (1) small intestine (without the upper duodenal part), (2) cecum and vermiform appendix, (3) ascending colon, and (4) right half of the transverse colon. All parts of the midgut are supplied by the superior mesenteric artery.

During the early stages of development, the midgut is attached to the dorsal wall of the body by means of a short dorsal mesenterium. A ventral mesenterium is not present. A broad connection with the yolk sac exists ventrally and becomes strongly narrowed by the developing bending of the embryo. Consequently, the omphaloenteric (i.e., vitelline) duct is formed, which vanishes during the sixth week of gestation. If this reduction is disturbed, one of several types of Meckel's diverticulum may develop, depending on the type and mode of persistence of the duct. For a short time after the elimination of the omphaloenteric duct, a small bulge remains where the duct has inserted at the intestine. This remnant completely disappears during subsequent development. Because the midgut grows considerably faster than does the rest of the embryonal body, it experiences various regular movements and rotations, which can be divided into three phases: the (1) physiologic umbilical hernia (6th gestational week; phase 1), (2) closure of the physiologic umbilical hernia (10th gestational week; phase 2), and (3) peritoneal fixation of the midgut (12th gestational week; phase 3).

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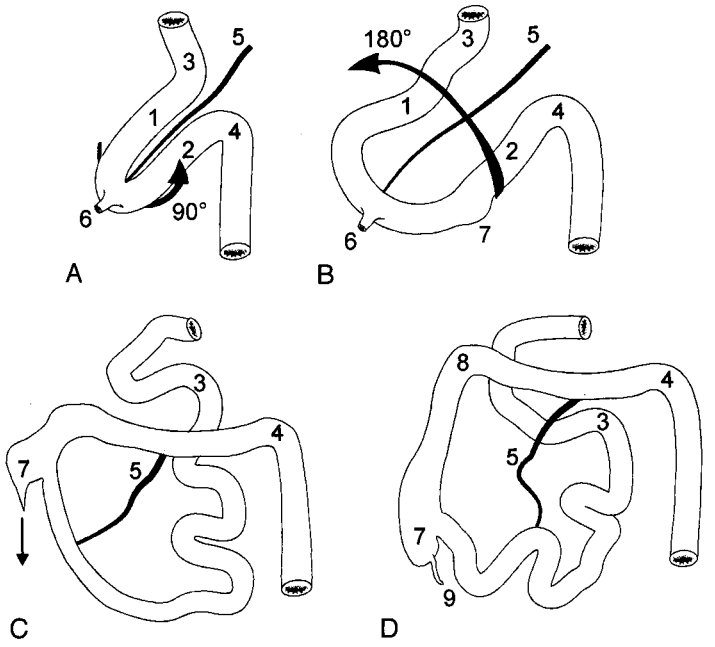

Figure 1. Normal development and rotation of the midgut. *A*, The midgut forms a loop with a superior or prearterial (1) and an inferior or postarterial (2) segment. Where these segments meet the dorsal wall, the duodenojejunal flexure (3) and the left colic flexure (4) are formed. The loop is located in the base of the umbilical cord (physiologic herniation) with the superior mesenteric artery (5) as an axis. The vitelline duct (6) inserts at the apex of the loop. *B*, The loop undergoes a counterclockwise twist, first of 90° and then of 180° (total: 270°). The cecal pouch (7) becomes visible. *C*, The cecal pouch is located directly below the liver, and grows out caudally. Descensus of the cecum *(arrow). D*, At last, the cecum (7) with the appendix (9) is positioned in the right iliacal fossa and the right flexure (8) is formed.

Phase 1

During phase 1, the midgut experiences considerable elongation, resulting in a hairpin-shaped loop. Cranially and caudally, this loop joins the intestinal sections located at the dorsal wall of the body (i.e., later duodenum and later descending colon) in a sharp bend ("cranial" and "caudal" basepoints, according to Hinrichsen[26]). The cranial basepoint corresponds to the later duodenojejunal flexure, and the caudal basepoint, to the left colic flexure.

As in the embryonal cavity, because of the enormous size of the liver and the kidneys, room to accommodate the intestinal loop is unavailable, and the loop extends into the extraembryonal coelom of the umbilical cord. This configuration, first noticed by Meckel[39] in 1817, is considered the physiologic umbilical hernia and develops during the sixth week of gestation (length of the embryo, 10 mm). The intestinal loop is subdivided into proximal (prearterial) and distal (postarterial) segments. The vitelline duct inserts at the vertex of the loop and marks the dividing point of the two segments.

The proximal segment grows strongly in length and forms six primary

intestinal loops, whereas the cecum starts from the distal loop like a bud. This early structure of the cecum simultaneously indicates the border between the later colon and the later ileum. Accordingly, the later ileum is located cranially, and the later colon, caudally. Within the intestinal loop, as an axis, lies the superior mesenteric artery, around which the intestinal loop rotates 90° counterclockwise. As a result of this rotation, the proximal segment of the loop assumes a right-hand position, and the caudal segment, a left-hand position.

Phase 2

During the 10th week of gestation (length of the embryo, 4–5 cm), the intestinal convolute starts to relocate into the embryonal body cavity. This process is completed fairly quickly[21, 22, 35] and is difficult to observe. The forces responsible for the back-positioning of the intestinal convolute are unknown. The decrease in size of the mesonephric bodies, the general enlargement of the embryonal cavity, and the relative decrease in size of the liver are probably contributing factors. Because the relocation is accomplished comparatively fast, tensional forces resulting from the rapid enlargement of the liver are assumed. According to Broman,[7] the caudal border of the enlarging liver presses the umbilical loop caudally and produces a force that pulls the intestinal loop into the body cavity. The problems of the formation of the umbilical loop and of the repositioning of the physiologic umbilical hernia are discussed in detail by Estrada[14] and Kiesselbach.[31]

In the course of the repositioning of the umbilical hernia, an additional counterclockwise rotation of the midgut by 180° occurs, resulting in a total rotation of 270°. Following this additional rotation, the budlike complex of the early cecum is now located on the right-hand side of the upper abdominal cavity, directly below the liver and at the level of the iliac crest. The beginning elongation of the transverse colon pushes the cecum toward the right wall of the body. As the colon continues to grow, it must slide in the caudal direction ("descensus"). In the course of this process, the right colic flexure is formed. Subsequent growth completes this descensus, and the cecum finally lies in the right iliac fossa. During this stage of development, the entire intestine still features a mesenterium.

Phase 3

After the intestine has reached its final length, the mesenterium of the ascending and descending colon is pushed against the dorsal body wall and fuses completely with the parietal peritoneum. The resulting layer of connective tissue between the gut and the dorsal wall lacks blood vessels and is known as *Toldt's fascia*. Toldt's fascia can be used for the detachment of the colon without bleeding. By the process just described, the ascending and the descending colon assume a secondary retroperitoneal position.

Development of the Appendix and Ileocecal Valve

The initial structures of the cecum and vermiform appendix become visible as the so-called "bud of the cecum." In this stage, the conic bud is located in the distal segment of the umbilical loop, directly in the vicinity of the apex of

the loop. Stretching and elongation of the colon result in the descensus of the cecum and of the appendix. As the complex of the vermiform appendix is pushed ahead of the cecum, it might assume various positions. The retrocecal, retrocolic, or pelvine position of the appendix must be considered to have occurred by chance, not based on immutable laws of development.

Postpartum, the cecum exhibits increased growth laterally, resulting in the dislocation of the vermiform appendix in the medial direction. According to Broman,[8] the increasing accumulation of meconium within the colon is the cause of the increase in diameter of this section of the intestine. Because of a mucosal fold, the distal cecum cannot be filled completely with meconium, so that growth is not stimulated there and the vermiform appendix borders against the cecum as a thin structure.

In addition to these general details concerning the development of the intestine, the development of the ileocecal valve of Bauhin merits attention. During the third month of gestation (length of the embryo, 5–6 cm), the initial cecal complex bends sharply against the colon, so that the terminal part of the ileum is pressed together in the craniocaudal direction and acquires a flattened, wedge-shaped configuration.[56] In the next step, the terminal part of the ileum becomes invaginated into the colon, during which, cranially and caudally, the walls of the colon and terminal part of the ileum are pressed together and fuse completely, so the two lips of the ileocecal valve are composed of two complete intestinal walls and are not simply mucosal folds.

The vermiform appendix becomes visible in the eighth week of gestation (length of the fetus 10–12 cm), and the first accumulations of lymphatic tissue develop during the 14th and 15th weeks of gestation.[32] The first minute accumulations of lymphatic cells are located directly below the epithelium and influence the later development of these epithelial cells. Some lymphocytes penetrate into the epithelial layer of the vermiform appendix, which distinctly contains fewer goblet cells than the other colic mucosa. The vermiform appendix, tonsils, and Peyer's patches possess no lymphatic vessels draining to their lymphatic apparatus and are different from lymphatic nodes.

SURGICAL ANATOMY OF THE CECUM–APPENDIX COMPLEX

Cecum

The cecum, with the vermiform appendix, forms the first section of the colon (Figs. 2–4). The pouchlike cecum is approximately 6 cm in length and approximately 8 cm in width and is variable in shape. The cecum shows all distinctive features of the colon, such as teniae, haustra, and fatty epiploic appendices. It is located below the point where the ileum joins the colon. Two incisures, one on the ventral side and one on the dorsal side, are called the *ventral* and *dorsal cecocolic incisures*. These incisures delineate the cecum against the ascending colon. Inside of the cecum, these structures correspond to the frenula of the ileocecal valve of Bauhin.

The dome-shaped haustrum, located at the lowest position of the cecum in the standing position, forms the bottom of the cecum, the cecal fundus. The teniae join each other at the base of the appendix and form a continuous longitudinal muscular layer on the outside of the appendix (the free tenia leads to the hidden appendix and functions as an appendix pointer). In the range of the orifice of the vermiform appendix, lateral trains of muscle fibers branch from

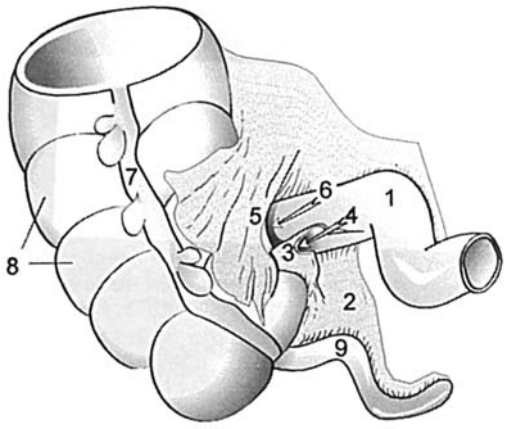

Figure 2. Anterior view of the ileocecal complex. Pars terminalis ilealis (1). Mesenteriolum (2). Plica ileocecalis inferior (bloodless fold of Treves) (3). Recessus ileocecalis inferior (4). Plica ileocecalis superior (5). Recessus ileocecalis superior (6). Taenia libera (7). Haustra coli (8). Appendix vermiformis (9).

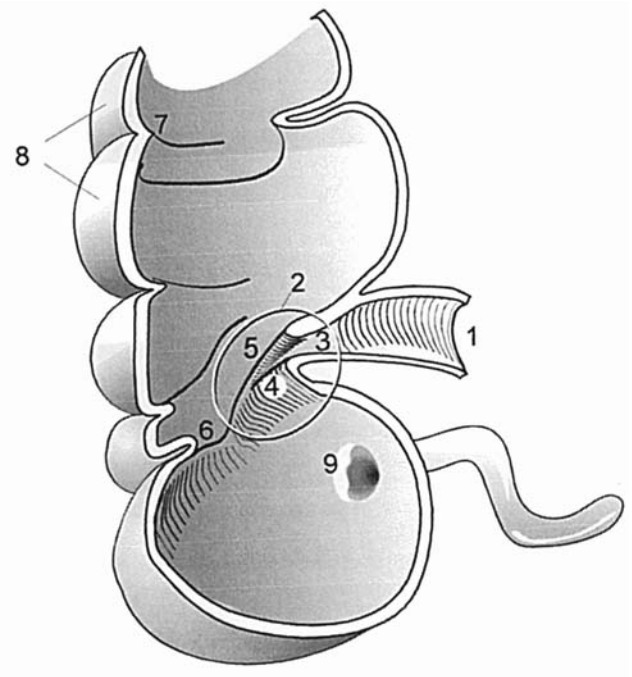

Figure 3. Internal structure of the ileocecal complex. Pars terminalis ilealis (1). Papilla ilealis (2). Ostium ileale (3). Labrum ileocecale (sive inferius) (4). Labrum ileocolicum (sive superius) (5). Frenulum ostii ilealis (6). Plica semilunaris (7). Haustra coli (8). Ostium appendicis vermiformis with Gerlach's valve (9).

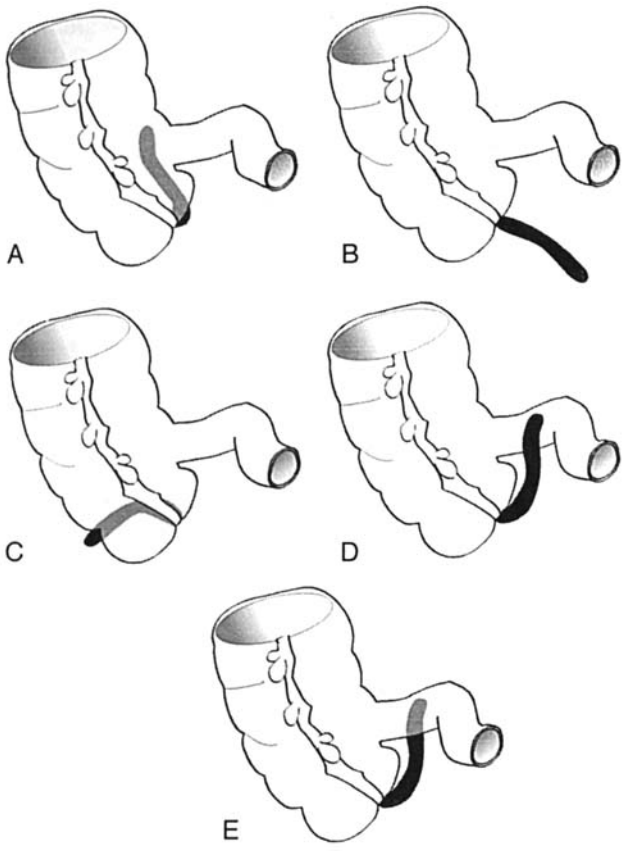

Figure 4. Positions of the vermiform appendix. *A*, Retrocecal (65%). *B*, Descending, pelvine (31%). *C*, Transverse, retrocecal (2.5%). *D*, Ascending, paracecal, preileal (1%). *E*, Ascending, paracecal, postileal (0.5%). *(Data from* Wakeley CPG: Position of the vermiform appendix as ascertained by analysis of 10,000 cases. J Anat 67:277, 1933.)

each tenia and mesh intensively with the inner annular muscle layer. Such a meshing of muscle fibers is also present at the apex of the appendix.[6]

The cecocolic junction of various mammals presents a distinctly developed sphincter system. Such a sphincter is pronounced in herbivores but also present in carnivores. According to endoscopic, radiologic, and anatomic observations,[43] such a cecocolic sphincter system is also established in humans.

Within the lumen of the cecum, the ileal orifice is located at the ileal papilla. The ileal orifice is lined by two lips, called the *ileocolic* (superior) and *ileocecal* (inferior) *lips*. From these lips, the frenulum of the ileal orifice arises and is a prominent ridge extending to the left border of the cecum. In the 1998 issue of *Terminologia Anatomica*,[17] the common expression *ileocecal valve* is no longer listed because, according to Rosenberg and DiDio,[47] this part does not constitute a valve and the closing mechanism is supposed to be located in the terminal ileum.

In the right fossa, the cecum rests on the iliac muscle, sometimes extending onto the psoas major muscle or hanging over the linea terminalis into the pelvis.

These varieties in location are caused by the variable means of attachment of the cecum. Different degrees and forms of the cecal attachment are responsible for these various positions. The main configurations of the cecal attachment can be classified as fixed cecum, mobile cecum with secondary fixation, and mobile cecum.[23] Lateral to the psoas major muscle, a flat depression, termed the *cecal fossa*, can be seen by lifting up a mobile cecum. This distinct fossa is delineated laterally by the cecal fold and can communicate continuously with the retrocecal recess. The tight-filled cecum contacts the anterior wall of the body, but, if emptied, it collapses so that the small intestinal loops can interponate between the cecum and anterior body wall.

According to general opinion, the ileal orifice projects on McBurney's point (denoting the right third point of the right spinoumbilical line, also called *Monro's line*), whereas the origin of the appendix projects either on McBurney's point, Lanz's point (right third point of the interspinal line), or Kümmell's point (right side and somewhat below the umbilicus). The position of the cecum varies and depends on the cecal filling and on the filling stage of the adjacent organs. The position of the vermiform appendix also varies and depends on many other factors. McBurney's point, Lanz's point, and Kümmell's point cannot be considered to be more than preliminary points of orientation.

In the region of the ileocecal complex, structures move from an intraperitoneal to an extraperitoneal position. Sometimes, typical peritoneal dislocations occur, resulting in characteristic peritoneal folds and recesses. In the angle between the terminal part of the ileum and the cecum, the superior ileocecal fold extends on the ventral side of the cecum. This fold contains the anterior cecal artery and typically does not reach the vermiform appendix. The superior ileocecal recess, with its caudally directed entry, lies below this fold. The inferior ileocecal fold extends from the antimesenterial border of the terminal part of the ileum to the mesenteriolum of the vermiform appendix and spreads on its ventral area. This fold contains neither adipose tissue nor blood vessels, but some individual fibers of smooth muscles may be present. Because the vessels in this peritoneal fold are lacking, it is commonly called the "bloodless fold of Treves." Behind the inferior ileocecal fold, the minor and inconspicuous inferior ileocecal recess is hidden, with the entry to this recess directed caudally.

The entire ileocecal complex is vascularized by the ileocolic artery, which branches from the superior mesenteric artery. The ileocolic artery reaches the cecum in the corner between the cecum and the terminal part of the ileum and divides into five branches: (1) several ileal rami, which supply the terminal part of the ileum; (2) the anterior cecal artery, which supplies the anterior circumference of the cecum; (3) the posterior cecal artery, which supplies the posterior circumference of the cecum; (4) the colic ramus (also called the *ascending ramus*), which ascends at the medial border of the cecum, supplying this area and the beginning of the ascending colon; and (5) the appendicular artery, which runs dorsally (rarely, ventrally) to the terminal part of the ileum into the mesenteriolum of the vermiform appendix.

According to the literature,[33] the appendicular artery originates from the iliac ramus in 35% of cases, from the division of the ileocolic artery in 28% of cases, from the anterior cecal artery in 20% of cases, from the posterior cecal artery in 12% of cases, from the ileocecal artery in 3% of cases, and from the ascending colic ramus in 2% of cases (Fig. 5). The veins accompany the arteries. Drainage of the lymph is provided by the ileocolic lymph nodes, located along the superior mesenteric artery, and by celiac nodes into the cisterna chyli (Pecquet's reservoir). Near the ileocolic valve, some minor lymphatic nodes, called the *prececal* and *retrocecal nodes*, lie directly under the serous membrane of the cecum.

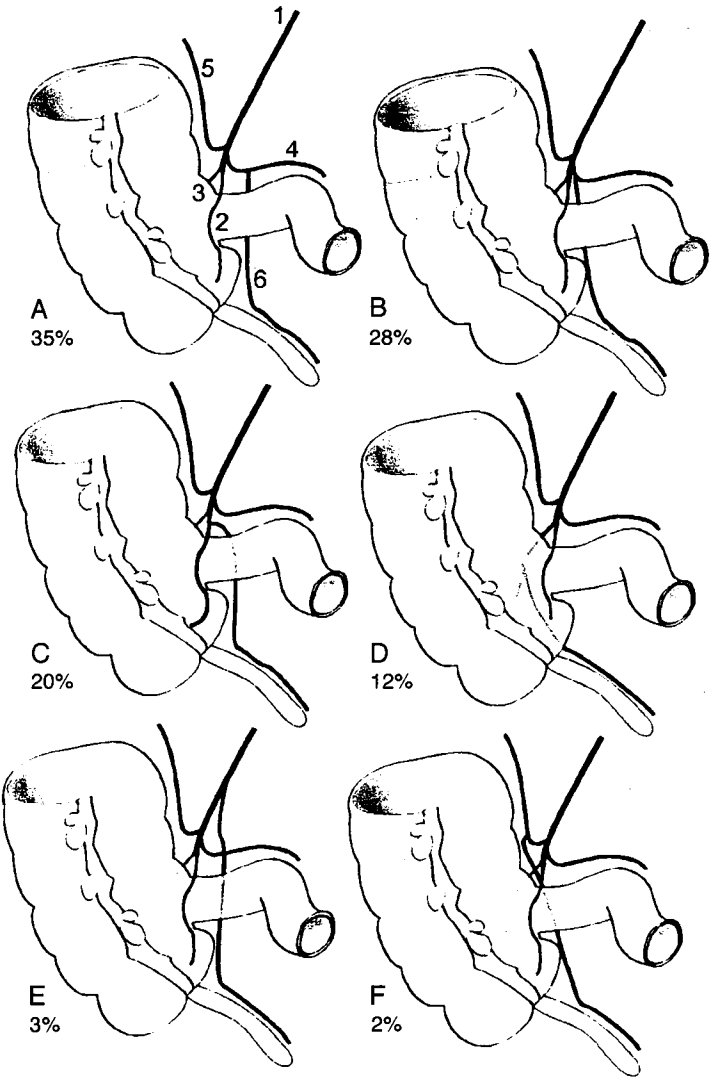

Figure 5. Variations of the origin of the appendicular artery. A. ileocolica (1). A. cecalis anterior (2). A. cecalis posterior (3). R. ilealis (4). R. colicus (5). A. appendicularis (6). A = artery; R = ramus. (*Data from* Lippert H, Papst R: Arterial variations in man. München, Bergmann JF, 1995.)

Malformations of the Cecum

Malformations of the cecum are primarily malpositions resulting from a disturbed gut rotation. The many manifestations of this entity can be classified as follows.

Nonrotation

Nonrotation develops if the umbilical loop does not fullfill the last step of the gut rotation of 180°, so that the inferior segment of the umbilical loop is relocated into the abdominal cavity first during the repositioning of the physiologic umbilical hernia. Consequently, the entire colon remains double-foldedly positioned in the left part of the abdominal cavity (a so-called "left colon"), without any retroperitoneal fixation. The entire small intestine lies in the right part of the abdominal cavity. Many of these malformations are symptom free, but volvulus commonly accompanies nonrotation.

Malrotation

Malrotation occurs if the umbilical loop does not complete the last 90° of the rotation. As a result, the cecum remains below the pylorus, becoming attached by Ladd's ligaments at the dorsal body wall. These ligaments cross the duodenum and are able to compress this structure, with the result being duodenal stenosis. They can also cause a strangulation ileus.

Subhepatic Cecum

In approximately 6% of cases, the elongation of the proximal colon in the third phase of the gut rotation does not occur, omitting the descensus of the cecum. In these cases, the cecum–appendix complex remains directly below the liver. Many transitional forms exist between the normal position in the right iliac fossa and the subhepatic position.

Mobile Cecum

Mobile cecum develops if the beginning of the ascending colon does not achieve retroperitoneal fixation. In an extreme variant of this embryologic abnormality, the colon lacks retroperitoneal fixation to the extent that the whole gut (i.e., small intestines, ascending colon, transverse colon, and descending colon) possesses a collective mesenterium, called the *commune mesenterium.* The mobile cecum and common mesenterium predispose to a volvulus or to malposition of the appendix.

Hyper-rotation

Hyper-rotation, a rare malformation, is a gut rotation of 450° causing the cecum to lie directly at the left colic flexure. Another hypothesis explains this anomaly with an unlimited descensus of the cecum, which pushes the cecum at first into the pelvis and subsequently cranially to the dorsal abdominal wall. A typical case was reported by Low and Hildermann.[34]

Inverse Cecum

Inverse cecum, a rare occurrence, is characterized by an early subhepatic fixation of the cecum below the liver directly after a normal gut rotation. During the elongation of the transverse colon, the cecum bends upwards (Fig. 6).

Retroperitoneal Cecum

The cecum–appendix complex and ascending colon become enclosed by a peritoneal membrane (i.e., Jackson's paracolic membrane). This membrane devel-

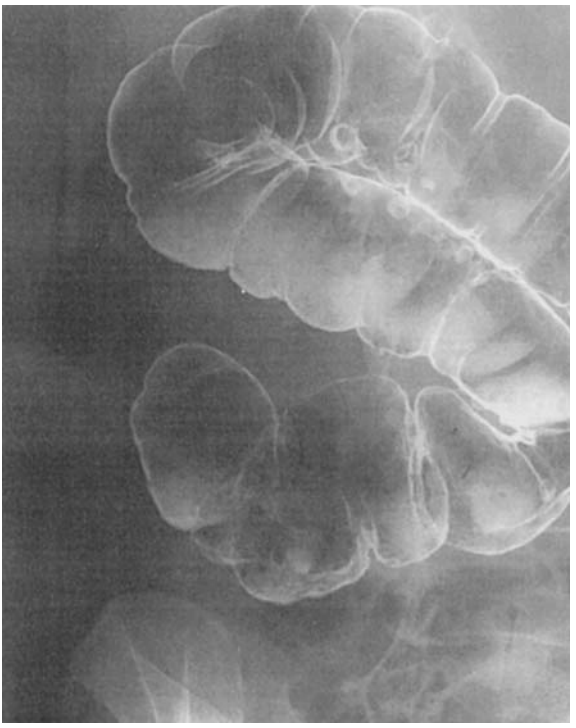

Figure 6. Incidental finding of a cecum inversum in a patient with uncharacteristic abdominal symptoms. Note the inverse cecum and the enlarged ascending colon.

ops if the cecum is pushed under the laterodorsal peritoneal fixation. Jackson's membrane contains parallel blood vessels that allow for the differentiation from bloodless peritoneal adhesions.

Internal Hernias

In the paracecal region, internal hernias may develop. According to a review by Schumpelick and Klinge,[49] they are the second most common group after paraduodenal hernias. Most paracecal hernias enter at the recesses of the left paracecal region and are located within the retrocecal recess (also called *Rieux's hernias*). A special type of paracecal hernia is Short's hernia, with an entry on the right side of the cecum.[29]

SURGICAL ANATOMY OF THE VERMIFORM APPENDIX

The appendices of adults are pencil-shaped structures with a length of approximately 9 cm. Short forms (5 cm) and long forms (35 cm) exist.[52] According to anatomy textbooks, the appendix originates at the posteromedial border of the cecum, on average 1.7 to 2.5 cm below the terminal part of the

ileum.[41, 51] Beside this typical origin, several variations occur that can be classified, according to Treves,[58] into four types: type 1, the appendix is of a fetal type, with a funnel-shaped origin of the appendix; type 2, the appendix originates from the cecal fundus; type 3, the appendix originates dorsomedially out of the cecum (most common type); type 4, the appendix originates directly beside the ileal orifice.

If the lumen of the appendix narrows abruptly after its cecal origin, this type is known as the *cylindric* type of appendix. Seldom (2–3% of adults), the funnel-shaped type (type 1, according to Treves[58]) is present. This type is also characterized by a continuous narrowing of the cecum toward the appendix. The funnel-shaped type is typically found in anthropoid apes and can be explained as persistence of a fetal condition.

The appendix possesses a small mesenteriolum, which contains the appendicular artery within its free border. Because arterial arcades are missing in the mesenteriolum, the appendicular artery must be a terminal artery.[53] The result of this anatomic condition is that perfusion with blood, such as in the situation of an inflammation, cannot be increased, so that ischemic damage may develop.[52, 53] The anatomic situation of the blood vessels is beside the architecture of the appendiceal wall, an important causal factor of appendicitis.

The base of the appendix is also supplied by small blood vessels arising from the anterior and posterior cecal arteries. These vessels may cause severe bleeding into the colic lumen if the appendiceal stump is ligated insufficiently.[52] The venous blood is drained through the ileocolic veins and the right colic vein into the portal vein. The lymphatic vessels drain into the ileocolic lymph nodes located along the superior mesenteric artery. Finally, they reach the celiac nodes and the cisterna chyli (Pecquet's reservoir). This passage has anastomoses to the retroperitoneal lymph nodes. Small lymph nodes are sometimes present within the mesenteriolum.

The position of the appendix varies among individuals. According to an investigation of 10,000 cases,[59] five positions (see Fig. 4) can be identified: (1) ascending appendix in the retrocecal recess in 65.0% of cases (most common type), (2) descending appendix in the iliac fossa in 31.0% of cases, (3) transverse appendix in the retrocecal recess in 2.5% of cases, (4) paracecal and preilial ascending appendix in 1.0% of cases, and (5) paracecal and postilial ascending appendix in 0.5% of cases. Non-fixed (appendix libera) and fixed (appendix fixa) appendices must be distinguished. The position of a nonfixed appendix is changed permanently. The descending appendix (i.e., in the pelvine position) is, in most cases, a nonfixed appendix. The appendiceal orifice can be partially covered by an inconstant and variable mucosal fold, known as Gerlach's valve.

Malformations of the Appendix

Malformations of the appendix are rare. According to the exhaustive investigation of 50,000 cases by Collins,[11] only eight malformations were detected. Four cases were agenesis, and four, partial or complete duplication. The agenesis of the vermiform appendix is a rare occurrence, and causal reports are often unreliable because of confusion with the hypoplasias and intramural positions. In a critical review,[10] 57 proved cases of agenesis of the appendix were found. The author found three additional cases. Duplication is also rare; less than 60 cases were reported in the literature through 1987.[5] According to morphology, duplication can be classified into five different types[46]:

Type A—Distal duplication with a common basis (i.e., bifid appendix)

Type B1—Two appendices originate from a common cecum on both sides of the ileocecal valve
Type B2—A normal appendix with a normal origin combined with a rudimentary form originating from one of the cecal teniae
Type B3—One normal-shaped and normal-positioned appendix is combined with another one originating from the right colic flexure[4]
Type C—Double ceca, each with its own appendix

The classification by Waugh[60] differentiates only three types:

Type 1—Appendix with two separate luminae and a common appendiceal muscular wall
Type 2—Two completely separated appendices originating from the cecum
Type 3—A normal-shaped and normal-positioned appendix combined with a hypoplastic one, with possible atypical origin

Excluding cases that can be registered according to these classifications, rare types may occur, including triplication,[55] accessory appendix without communication to the cecum,[5] helical appendix (i.e., spiral-shaped appendix),[40] intramural appendix,[1] and appendix–umbilicus fistula.[30] Only single cases of some of these rare forms have been reported.

Appendiceal Diverticula

Another rare occurrence is the diverticulum of the appendix.[15] These diverticula can be explained as rudimentary duplications. They must be differentiated from false appendicular diverticula (i.e., internal and external pseudodiverticula), which are characterized by a mucosal prolapse through a hiatus within the muscular layer of the appendiceal wall, perhaps around the entrance of blood vessels. These pseudodiverticula are acquired changes,[12, 19] with an estimated prevalence of 0.3% to 2.2%.[54] They are usually small, with a diameter of 3 to 5 mm, and may occur multiple.

SURGICAL APPLICATIONS

Appendicitis

The diagnosis of acute appendicitis as a surgical entity was first made by Fitz[18] in 1886. He described the course of the acute inflammation of the appendix and the development of peritonitis. He also recommended appendectomy. A few years later, McBurney[37] described the clinical manifestation of acute appendicitis and proposed the localization of skin incision in the right lower abdomen for surgical approach to the appendix.[38]

Acute appendicitis is the most common surgical emergency disease of the abdomen. It occurs most commonly in young adults, with a peak in the second and third decades of life (Fig. 7).[57] The amount of lymphoid tissue in the appendix seems to have a role in the development of acute inflammation of the appendix because the peak is in the second and third decades of life. Children younger than 2 years of age rarely have appendicitis, probably because, at this age, the configuration of the appendix (i.e., like an inverted pyramid) makes obstruction of the lumen unlikely.

Obstruction of the lumen seems to be the most dominant factor in the

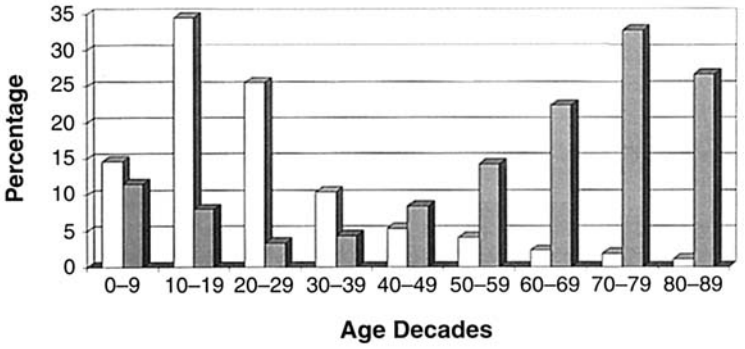

Figure 7. Percentage of appendectomies *(open* bars, $n=2631$) and percentage of perforation (*shaded bars*, $n=214$) in different age decades of 2631 patients with appendicitis at the Surgical University Clinic of Aachen from 1986 to 1995. The frequency peak for appendicitis is in the second and third decades of life. There are two frequency peaks for perforated appendicitis, in early childhood and older age.

development of acute appendicitis and is usually caused by fecaliths (Fig. 8), but vegetable or fruit seeds, intestinal worms (particularly ascarid), and inspissated barium are also known to cause appendiceal obstruction, as does hypertrophy of lymphoid tissue with compression of the appendiceal lumen. Fecaliths are found in approximately 40% of cases of acute appendicitis, 65% of cases of gangrenous appendicitis, and approximately 90% of cases of perforated appendicitis.

The appendix vermiformis is a rudimentary organ with a special myoarchitecture.[53] The wall of the cecum is able to stretch because of a diagonal, rhomboidlike mesh of collagen fibers, but the horizontal collagen fibers of the appendix wall allow for only a minimal passive expansion of the lumen. Also, the foldless mucosa does not have a sufficient reserve in cases of swelling.

The appendix secretes 2 to 3 mL of mucus daily. After occlusion of the appendiceal lumen, normal secretion of the appendiceal mucosa persists. Pseudomyxoma peritonei, caused by rupture of a mucocele of the appendix, impressively proves the ability of the appendix to secrete mucus. The appendix is able to continue secretion until gangrene and perforation occur, which rapidly produce distension because the average luminal capacity of the vermiform appendix is only approximately 1 mL. According to the law of Laplace, this leads to a rapid increase of intraluminar pressure because of the restricted ability of the appendiceal wall to stretch[13] (Fig. 9). Secretion of as little as 0.5 mL leads to an increase of pressure of approximately 45 mm Hg.[3] This phenomenon also explains the possibility of perforation of the appendix within a few hours in cases of acute inflammation, so all patients with suspected acute appendicitis need hospitalization and close clinical monitoring if appendectomy is not performed immediately.

Distension stimulates nerve endings of visceral afferent pain fibers and leads to dull, diffuse pain in the midabdomen. Distension continues from rapid multiplication of the bacterial flora of the appendix. Increasing pressure leads to occlusion of the capillaries and venules, but the arteriolar inflow continues. This results in vascular congestion. The increasing distension leads to reflex nausea and vomiting and an increase of diffuse abdominal pain. When the inflammatory

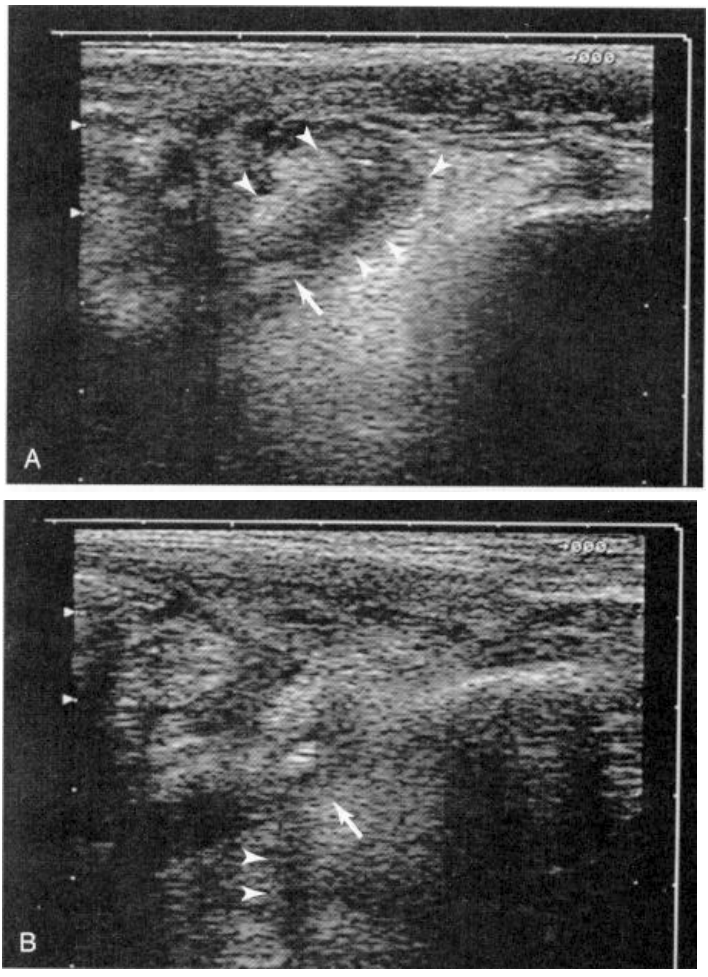

Figure 8. Ultrasonography of acute appendicitis caused by fecalith *(arrows,* longitudinal *[A]* and cross-section *[B]*). Note the fluid-filled appendix *(arrowheads [A])* and the typical sound shadow caused by the fecalith comparable with the typical finding in gallstones *(arrowheads [B])*.

process has involved the serosa of the appendix and the parietal peritoneum in the region, the characteristic shift in pain to the right lower abdominal quadrant is produced.

The impaired blood supply caused by vascular congestion leads to a compromise of the appendiceal mucosa, allowing for bacterial invasion of the deeper coats. Absorption of bacterial toxins and necrotic tissue products causes fever, tachycardia, and leukocytosis. The increasing pressure starts to exceed the arteriolar pressure, and in the area with the poorest blood supply, the antimesenteric border, ellipsoidal infarcts develop. Finally, perforation occurs, usually through the infarcted areas. This sequence is usually inevitable, although pathologic

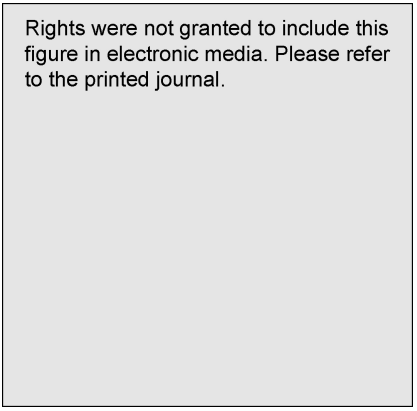

Figure 9. Volume of the appendix. According to the law of Laplace (T = P*r/2: T = wall tension; P = intraluminal pressure; r = radius), obstruction of the lumen leads to direct increase of intraluminal pressure because the ability of the appendiceal wall to swell is low (P = T*2/r). Because the daily mucus production of the appendix is approximately 2 to 3 mL, a regular appendix with a volume of approximately 0.6 mL is filled within hours—which may explain the sometimes rapid clinical course in patients with acute appendicitis. (Modified from Dreuw B, Truong S, Riesener KP, et al: The value of sonography in the diagnosis of appendicitis: A prospective study of 100 patients [in German]. Chirurg 61:880, 1990; with permission.)

investigation often reveals scarring of the appendix, suggesting resolved acute appendicitis. Lumen obstruction caused by lymphoid hypertrophy may spontaneously resolve.

Although in some patients with acute inflammation of the appendix, the disease may spontaneously resolve, in which patients this will occur cannot be predicted. Untreated, obstruction of the lumen leads to gangrene and perforation. Investigators report that 15% to 30% of patients have ruptured appendicitis on hospital admission, with an even higher percentage (up to 50%) in the pediatric age group and up to 90% in geriatric patients.[25, 57]

When the diagnosis of acute appendicitis has been established, immediate appendectomy is indicated after short preparation concerning general anesthesiologic principles. In cases of acute appendicitis without perforation or perforated appendicitis without peritonitis, the incision should be made in the right lower abdomen, below McBurney's point. The aponeurosis of the external oblique muscle is dissected in the course of the fibers. The internal oblique muscle, located below, is bluntly separated in the direction of the muscle fibers. The peritoneum is incised in the craniocaudal direction. Then the cecum is identified, captured, and rotated up and out until the base of the appendix can be seen. The vessels of the mesoappendix are transected, with particular attention paid to the appendicular artery. A crushing clamp is applied to the base of the appendix to form a groove for ligation and destruction of the mucosal layer. Finally, the stump of the appendix is inverted, and a purse-string suture is placed through the seromuscular layers of the cecum. One study[2] has demonstrated that invagination of the stump significantly reduces the incidence of bacterial contamination of the peritoneal cavity. Perforated appendicitis with generalized peritonitis requires a midline incision because of the need for abdominal lavage and drainage in all abdominal quadrants.

Anomalies of the Location of the Appendix

The appendix is typically located in the right lower abdomen. Unusual locations in cases of malrotation or situs inversus have been reported to be anywhere in the abdomen—even in the thoracic cavity.[16] But even in cases of location in the right lower abdomen, unusual locations may cause physicians difficulty in finding the appendix. Location within the cecal wall, necessitating an incision of the cecal wall to remove the appendix, has been reported.[1] This and other findings of bizarre locations of the appendix underline that a knowledge of the abnormalities of the appendix and a careful intraoperative search are necessary to find the appendix in such cases. In cases of acute abdominal pain without obvious cause, an abnormal location of the appendix must be considered, and an exploratory laparotomy must be performed.

Appendiceal Diverticulosis

Diverticular formation of the vermiform appendix is often missed. Clinical symptoms may include those of acute appendicitis in cases of inflammation or

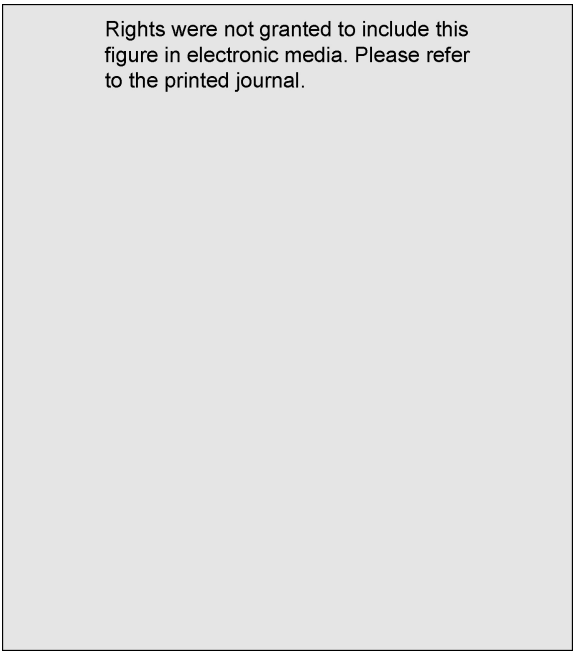

Figure 10. Surgical specimen of an appendiceal diverticulum (cross-sections in different levels). Fecalith (A). Small false diverticulum (B). Yellow lining caused by purulent peritonitis (C). Peridiverticulitis with small abscess (D). Lumen of the appendix (E). Mucosa in the lumen of the diverticulum (F). Note that the diverticulum is covered completely by inflamed tissue, which makes it impossible to identify it during surgery. (*From* Franke J, Tons C, Tietze L, et al: Perforated diverticulum of the vermiform appendix [in German]. Chirurg 69:574, 1998; with permission.)

chronic right lower abdominal pain in patients with histologically unaltered appendices, but the clinical course in cases of acute appendiceal diverticulitis is more prolonged and subacute than that of typical appendicitis.[19] This underlines the need for surgical exploration in any case of acute abdominal pain of unclear cause. The diagnosis is usually made by a pathologist (Fig. 10). Appendectomy is advised when diagnosis of diverticulosis of the vermiform appendix is made because the perforation rate of inflamed appendices with diverticulosis is higher (27.0%) than is the rate of those without diverticulosis (6.6%).[61]

Congenital Defects of the Appendix

Congenital defects of the appendix are rare and of little clinical importance, but if they are present, they may be the cause of confusing clinical symptoms and may make a differential diagnosis difficult.[19] Surgeons must consider the possibility of an appendix duplex or triplex when a patient presents with typical symptoms of appendicitis but a previous history of appendectomy, but one of the spectrum of other differential diagnoses, including other diseases of the appendix or cecum, is much more likely. These include:

Surgical diseases of the appendix
- Appendicitis
- Congenital anomalies of the appendix and related diseases
  - Diverticula
  - Duplication
  - Appendix triplex
  - Agenesis
  - Malrotation
  - Volvulus
- Tumors
  - Lipoma
  - Pseudomyxoma
  - Carcinoid
  - Carcinoma

Surgical diseases of the cecum
- Diverticular disease
  - Cecal diverticulitis
  - Diverticular disease with hemorrhage
  - Diverticulum of the right colon
- Angiodysplasia
- Malrotation
  - Mobile cecum
  - Cecal volvulus
- Endometriosis
- Neoplasms
  - Lipoma
  - Polyps
  - Carcinoma
  - Carcinoid
  - Leiomyosarcoma
  - Lymphoma

Inflammatory bowel disease
  Ulcerative colitis
  Crohn's disease
  Ischemic colitis
  Radiation enterocolitis
  Pseudomembranous colitis
  Amebic colitis
  Campylobacter enterocolitis
  Actinomycosis
Megacolon
  Acquired
  Congenital

Intraoperative diagnosis of agenesis of the appendix is difficult and is definite only after median laparotomy and careful search for anomalies of the location of the appendix. Detailed postoperative diagnostics may be necessary to find the cause of symptoms. For example, in a patient seen by the authors, Crohn's disease was finally diagnosed.[45]

Volvulus of the Appendix

Isolated volvulus of the appendix is rare.[42] The clinical symptoms are the same as those of acute appendicitis, and immediate appendectomy is indicated. The initial sonographic finding of a tumor of the ileocecal region may lead to unneccessary diagnostic examinations.[42]

Neoplasms of the Appendix

Neoplasms of the appendix include adenocarcinoma, carcinoids, and mucocele. They are usually diagnosed at operation or autopsy. Malignant diseases of the appendix are treated with respect to the general principles of colon surgery for malignancy. For curative intention, a right hemicolectomy, including the mesocolon, is advisable. Various benign tumors of the appendix are of no clinical significance except that, in rare circumstances, they may produce acute appendicitis because of obstruction of the lumen.

Lipoma

Benign lipomas are commonly present in the colon, most often localized near the ileocecal valve. Patients with benign lipomas may present with symptoms of bleeding, incomplete obstruction, or intussusception. Small lesions may be removed endoscopically; large ones require segmental colon resections or open colotomy and removal of the tumor.

Mucocele of the Appendix

The appendicular mucocele, a benign tumor of the appendix filled with mucus, was first described by Rokitanski in 1842.[28, 57] The pathogenesis of this entity is not generally accepted. It is probably a retention cyst caused by occlusion of the appendicular lumen resulting from inflammatory strictures or benign tumors, such as carcinoids or polyps. Mucus production continues and increases

the endoluminal pressure. Because pyogen microbes are absent, the condition does not lead to acute appendicitis. This theory is supported by experimental induction of appendicular mucocele by ligation of the appendicular base,[9, 48] but some investigators believe that the mucocele is a true mucus-producing benign or malignant tumor.[20]

Myxoglobulosis of the Appendix

The myxoglobulosis is a special form of appendicular mucocele. The prevalence has been estimated to be 1 in 47,000 population.[27] It is characterized by small (i.e., 0.1–1.0 cm), pearl-like, globular formations occupying the dilated appendicular cavity. The combination of lumen occlusion with chronic inflammation seems to be fundamental in the development of myxoglobulosis. The rupture of a mucocele or myxoglobulosis leads to pseudomyxoma peritonei ex appendice, so surgery for the treatment of these entities must avoid injury of the removed specimen and contamination of the peritoneum with the mucus.

Diverticular Disease

Cecal Diverticulitis

Potier[44] first described diverticulitis of the cecum in 1912. This so-called *cecal diverticulitis* is caused by a solitary true diverticulum that is considered to be congenital in origin. This lesion is uncommon and found in younger individuals. It is relatively common among the native Hawaiian population.[24] In contrast to diverticula of diverticulosis coli, these diverticula contain all layers of the bowel wall and are found only in the cecum and ascending colon. The pathogenesis of infection occurs in the same manner as in diverticulitis coli. The ostium of the diverticulum is obstructed by feces. Consequently, inflammation, caused by continued secretion and infection by resident bacteria, develops. The symptoms are the same as for acute appendicitis, so the correct diagnosis is made preoperatively only in approximately 5% of cases by contrast medium enema. In approximately 80% of cases, this condition is misdiagnosed as acute appendicitis.

Surgical treatment depends on the intraoperative findings. In cases of a limited inflammatory process restricted to the diverticulum, a diverticulectomy and inversion of the stump, as for appendicitis, may be done. A ruptured diverticulum with abscess necessitates drainage of the abscess and removal of the diverticulum. If diverticulectomy cannot safely be performed, it must be carried out later.

When inflammation involves the cecum, resection of the diverticulum and the adjacent cecum should be done if the resection margins are out of inflamed tissue and the ileocecal valve is not compromised. Otherwise, and in cases of inflammation of the whole cecum, an ileocecal resection with ileoascending anastomosis is mandatory. In cases of general peritonitis, an ileocecal resection with ileostomy and a transverse colon mucus fistula is performed. Other surgical options are Mikulicz double-barrelled enterostomy after resection of the infected bowel, and proximal ileostomy and closure of the distal end as a modified long Hartmann's pouch. Finally, a right colectomy, as is performed for the treatment of patients with cancer, may be necessary if a chronic diverticular disease with productive inflammation and fibrosis is present, which makes differentiation from colon cancer difficult.

Diverticular Disease with Hemorrhage

Most colon diverticula are located in the sigmoid colon, where inflammation is the most likely cause of clinical symptoms, but massive bleeding usually originates from diverticula located in the right colon. Erosion of the peridiverticular arteriole by encrusted stool within the diverticulum causes massive bleeding. It is not rarely life-threatening and usually occurs in elderly patients with multiple accompanying disease. The diagnosis of intestinal bleeding is easy because of a perianal loss of a large amount of blood, but the site of origin of the bleeding may be difficult to determine. A standardized diagnostic procedure is useful in any case of perianal blood loss.

First, upper gastrointestinal bleeding must be ruled out. Placement of a gastric tube gives some hints if the evacuated fluid contains bile but no blood. Only upper gastrointestinal endoscopy is able to exclude a gastroduodenal ulcer as the reason for rectal blood evacuation. Then rectoscopy, proctoscopy, and flexible sigmoidoscopy should exclude bleeding from hemorrhoids, inflammatory bowel disease, or cancer. A full colonoscopy is sometimes possible without previous bowel preparation because the large amount of blood leads to partial cleansing of the bowel. If a patient is stable, the next step is orthograde lavage with 10 L of saline solution applied through a nasogastric tube, followed by a full colonoscopy. In approximately 70% of cases, this allows for localization of the origin of the bleeding if the colon is the source, but the bleeding commonly has stopped spontaneously, in which case colonoscopy cannot localize the origin of the bleeding. If rebleeding occurs, colonoscopy must be redone.

Scanning with technetium 99m–labeled erythrocytes is another study to detect the possible bleeding site. It may provide important hints for the application of selective arteriography. Arteriography is indicated if colonoscopy is impossible or did not find the origin of bleeding but bleeding continues, but a bleeding rate of 0.5 mL/min is necessary for a positive finding on arteriography.[36] When the bleeding vessel has been visualized, selective infusion of vasopressin may be successful to stop bleeding in approximately 80% of cases. Transcatheter embolization should be reserved for selected cases because the development of ischemia is unpredictable. As a last resort, surgical exploration and performance of ileostomy and colostomy are preferable to "blind," subtotal colectomy because the rebleeding rate is approximately 30%. Intraoperative panendoscopy with intraluminal transillumination and guiding of the scope by the surgeon is helpful to find angiectasia of the bowel.

Angiodysplasia

Angiodysplasia lesions are almost always located in the cecum and ascending colon. They consist of dilated submucosal vascular spaces. Mucosal involvement and arterioveneous shunts have also been described. Usually they are only a few milimeters in size. Venous congestion caused by increased intramural muscle tension may lead to dilatation of submucosal veins if degeneration of blood vessel walls has occurred, so the disease is usually found in older patients, although young adults may also be affected. Investigators assume that the association between right-sided diverticular disease and bleeding may be caused by coexisting angiodysplasia.

Most patients with angiodysplasia report a previous history of gastrointestinal bleeding. Elective colonoscopy may reveal a small area of unusual reddening or dilated submucosal vessels. Barium enema is normal without any hint of the

disease. In cases of acute bleeding, management is the same as for patients with acute bleeding from diverticular disease, but because these lesions are usually small, they are difficult to visualize if the bowel is full of blood. In this case, selective angiography is the examination of choice. The site of bleeding may present as a blush of extravasated contrast medium. For definitive treatment, surgical resection of the bowel segment that carries the lesion is necessary.

Malrotation

If the cecal rotation of the bowel is incomplete, the cecum remains high and the duodenum is located to the right of the superior mesenteric artery. The bands that fix the cecum (i.e., Ladd's bands) straddle the duodenum and may obstruct it. The vascular supply of the midgut suspends from a narrow pedicle that may twist. Midgut ischemia and high intestinal obstruction occur in cases of midgut volvulus.

Clinical manifestation is minimal in the beginning, with mild upper distension and tenderness. Later, bilious vomiting and bloody stools occur. Finally, in cases of gangrene, signs of sepsis and peritonitis develop. Abdominal radiographs show only unspecific signs, such as abnormal air–fluid levels. Contrast enema may show a displaced cecum, and an upper gastrointestinal series may show absent duodenal rotation, which leads to a more reliable diagnosis.

Early surgical intervention is mandatory to prevent the intestine from developing ischemic necrosis and to allow for recovery from the ischemic situation. The twisted bowel is untwisted counterclockwise. Then the bands between the cecum and the abdominal wall and between the duodenum and the terminal ileum are cut to splay out the superior mesenteric artery. This results in placing the duodenum to the right and the cecum to the left side of the abdomen. An appendectomy should be included to prevent later complications. This procedure was initially described by Ladd and is still preferred today.

Mobile Cecum and Cecal Volvulus

Approximately 10% to 20% of people have a freely mobile cecum and are possible candidates for the development of a cecal volvulus. The rotation usually is 360° to 720° around the mesenteric pedicle and produces a closed-loop obstruction with impairment of the blood supply. A sigmoid volvulus is much more common and may occur simultaneously. The clinical symptoms of the disease are the same as those of small-bowel obstruction. In cases of gangrene, acute abdominal pain develops. Abdominal radiographs show a dilated, ovoid-shaped, large-bowel loop filled with gas that may be located anywhere in the abdomen. The small bowel is also dilated and may show a small-bowel ileus, but the rest of the large bowel is relatively empty. The diagnosis is confirmed by water-soluble contrast enema.

When the diagnosis has been confirmed, exploratory laparotomy is indicated. Reports of colonoscopic detorsion of cecal volvulus are only sporadic. For definitive treatment, a right hemicolectomy is necessary, but some investigators also recommend simple detorsion with cecopexy or cecostomy if the bowel is viable.

Superior or Inferior Herniae Recessus Ileocecalis

Internal abdominal hernias account for approximately 1% of intestinal obstruction. They occur if a viscus is protruding through an opening formed by a fold of peritoneum or through a defect in a mesentery or the omentum. Hernias through the ileocecal fossae are the second most common type of internal hernia. Symptomatic patients present with acute abdominal pain and signs of intestinal obstruction or strangulation.

Therapy includes a reduction of the herniated loop and closure or enlargement of the hernial ring to prevent recurrence. Aspiration of the proximal loop may be helpful. Caution must be used not to injure the right ileocolic or colic arteries when incising the ring.[50]

SUMMARY

Surgeons should be familiar with surgery of the cecum and appendix because the diseases of this region, especially appendicitis, are the most common indications for surgical exploration. Usually, diagnosis of appendicitis and appendectomy are not difficult, but atypical location of the appendix or other anatomic anomalies can make the diagnosis of appendicitis and appendectomy difficult. In cases of atypical anatomy or diffuse clinical picture, especially in young adults or elderly patients, the spectrum of embryologic and anatomic anomalies must be kept in mind to make the correct treatment decision for individual patients. If doubt persists, explorative laparotomy must be performed to avoid overlooking rare, acute, intra-abdominal abnormalities.

References

1. Abramson DJ: Vermiform appendix located within the cecal wall: Anomalies and bizarre locations. Dis Colon Rectum 26:386, 1983
2. Arnbjornsson E: Invagination of the appendiceal stump for the reduction of peritoneal bacterial contamination. Curr Surg 42:184, 1985
3. Arnbjornsson E, Bengmark S: Obstruction of the appendix lumen in relation to pathogenesis of acute appendicitis. Acta Chir Scand 149:789, 1983
4. Biermann R, Borsky D, Gogora M: [Double appendicitis: A rare pathologic entity.] Die Appendicitis duplex: Eine pathologische Raritat. Chirurg 64:1059, 1993
5. Bluett MK, Halter SA, Salhany KE, et al: Duplication of the appendix mimicking adenocarcinoma of the colon. Arch Surg 122:817, 1987
6. Breuer H: Die Konstruktion der Menschlichen Appendix Vermiformis [thesis]. Hamburg, Med Diss, University of Hamburg, 1972
7. Broman I: Entwicklung des Darmes. *In* Broman I (ed): Normale und Abnorme Entwicklung des Menschen. Wiesbaden, JF Bergmann, 1911
8. Broman I: Entwicklung der Verdauungsorgene. *In* Broman I (ed): Die Entwicklung des menschen vor der Geburt. München, JF Bergmann, 1927
9. Cheng K: An experimental study of mucocele of the appendix and pseudomyxome. The Journal of Pathology and Bacteriology 61:217, 1949
10. Collins DC: Agenesis of the vermiform appendix. Am J Surg 82:689, 1951
11. Collins DC: A study of 50,000 specimens of the human vermiform appendix. Surg Gynecol Obstet 101:437, 1955
12. Cremer H: Diverticulum of the vermiform appendix. Beiträge aur Pathologie 159:94, 1976
13. Dreuw B, Truong S, Riesener KP, et al: [The value of sonography in the diagnosis of appendicitis: A prospective study of 100 patients.] Stellenwert der Sonographie bei

der Diagnostik der Appendizitis: Eine prospektive Studie an 100 Patienten. Chirurg 61:880, 1990
14. Estrada RL: Anomalies of Intestinal Rotation and Fixation. Springfield, IL, Charles C Thomas, 1958
15. Favara BE: Multiple congenital diverticula of the vermiform appendix. Am J Clin Pathol 49:60, 1968
16. Fawcitt R: Appendix situated within thorax. Br J Radiol 21:523, 1948
17. Federative Committee on Anatomical Terminology: Terminologia Anatomica: International Anatomical Terminology. New York, Thieme, 1998
18. Fitz RH: Perforating inflammation of the vermiform appendix: With special reference to its early diagnosis and treatment. Transactions of the Association of American Physicians 1:107, 1886
19. Franke J, Tons C, Tietz L, et al: [Perforated diverticulum of the vermiform appendix.] Divertikelperforation der Appendix vermiformis. Chirurg 69:574, 1998
20. Gibbs NM: Mucinous cystadenoma and cystadenocarcinoma of the vermiform appendix. J Clin Pathol 26:413, 1973
21. Gray SW, Skandalakis JE: The colon and rectum. *In* Gray SW, Skandalakis JE (eds): Embryology for Surgeons: The Embryological Basis for the Treatment of Congenital Defects. Philadelphia, WB Saunders, 1972
22. Gray SW, Skandalakis JE: The small intestines. *In* Gray SW, Skandalakis JE (eds): Embryology for Surgeons. The Embryological Basis for the Treatment of Congenital Defects. Philadelphia, WB Saunders, 1972
23. Hafferl A: Colon. *In* Hafferl A (ed): Lehrbuch der Topographischen Anatomie. Berlin, Springer, 1969
24. Harada RN, Whelan TJ Jr: Surgical management of cecal diverticulitis. Am J Surg 166:666, 1993
25. Häcker FM, Berger D, Kirschner HJ, et al: Perforierte Appendicitis im Kindesalter: Welche chirurgischen Maßnahmen sind wirklich erforderlich? Zeutralblatt Kinderchirurgie 8:5, 1999
26. Hinrichsen KV: Intestinaltrakt. *In* Hinrichsen KV (ed): Humanembryologie. Berlin, Springer, 1990, p 516
27. Hollström E: Myxoglobulosis appendicis. Acta Chir Scand 85:347, 1941
28. Hulek M, Vagner Z: [Large mucocele of the appendix imitating an ovarian cyst.] Grosse Mukozele der Appendix imitiert eine Ovarialzyste. Zentralbl Gynakol 100:186, 1978
29. Jamart J, Lassalle C, Boissel P, et al: Right external paracolic hernia or Short's hernia. Acta Chir Scand 149:105, 1983
30. Kadzombe E, Currie AB: Neonatal fistula from the appendix to the umbilicus. J Pediatr Surg 23:1059, 1988
31. Kiesselbach A: Der physiologische Nabelbruch. Ergebrisse Anatomie 34:83, 1952
32. Kyriazis AA, Esterly JR: Development of lymphoid tissues in the human embryo and early fetus. Arch Pathol 90:348, 1970
33. Lippert H, Papst R: Arterial Variations in Man. München, JF Bergmann, 1995
34. Low FN, Hildermann WC: A case of hyperrotation of the colon. Anat Rec 77:27, 1940
35. Mall FP: Über die Entwicklung des menschlichen Darmes und seiner Lage beim Erwachsenen. Arch Anat Entw-Gesch Suppl:403, 1897
36. Margulis AR, Heinbecker P, Bernhard HR: Operative mesenteric arteriography in the search for the site in unexplained gastrointestinal hemorrhage. Surgery 48:534, 1960
37. McBurney C: Experience with early operative interference in cases of disease of the vermiform appendix. N Y State Med J 50:676, 1889
38. McBurney C: The incision made in the abdominal wall in cases of appendicitis. Ann Surg 20:38, 1894
39. Meckel JF: Bildungsgeschichte des Darmkanals der Säugethiere und namentlich des menschen. Deutscher Archiv für die Physiologie 3:1, 1817
40. Mikat DM, Mikat KW: Appendix helicus: A unique anomaly of the vermiform appendix. Gastroenterology 71:303, 1976
41. Otto HF: Appendix vermiformis. *In* Doerr W, Seifert G, Ühlinger E (eds): Spezielle Pathologische Anatomie. Berlin, Springer, 1976

42. Paya K, Saadi S, Hörman M, et al: Isolierter Appendixvolvulus: Ein diagnostischer Fallstrick. Zeutralblatt Kinderchirurgie 8:34, 1999
43. Pellegrini MSF, Manneschi LI, Manneschi L: The caecocolonic junction in humans has a sphincteric anatomy and function. Gut 37:493, 1995
44. Potier F: Diverticueit et appendicite. Bull Mem Soc Anat Paris 37:29, 1912
45. Rau M, Treutner KH, Schumpelick V: Die Agenesie der appendix vermiformis. Leber Magen Darm, in press
46. Remmele W: Appendix vermiformis. *In* Remmele W (ed): Pathologie, ed 2. Berlin, Springer, 1996
47. Rosenberg JC, DiDio LJ: In vivo appearance and function of the termination of the ileum as observed directly through a cecostomy. Am J Gastroenterol 52:411, 1969
48. Rubnitz AS, Hermann HT: Experimental appendical mucocele, myxoglobulosis and peritoneal pseudomyxoma. Arch Pathol 36:297, 1943
49. Schumpelick V, Klinge U: Innere Hernien. *In* Schumpelick V (ed): Hernien, ed 3. Stuttgart, Enke, 1996, p 293
50. Skandalakis JE, Gray SW, Mansberger AR, et al: Hernia: Surgical Anatomy and Technique. New York, McGraw-Hill, 1989
51. Skandalakis JE, Skandalakis PN, Skandalakis LJ, et al: Surgical Anatomy and Technique: A Pocket Manual. New York, Springer, 1995
52. Stelzner F: Die Appendizitis: Handbuch der Inneren Medizin. Berlin, Springer, 1982
53. Stelzner F, Lierse W: [Etiology of appendicitis.] Uber die Ursache der Appendicitis. Langenbecks Arch Chir 330:273, 1972
54. Stout AP: A study of diverticulum formation in the appendix. Arch Surg 6:793–829, 1923
55. Tinckler LF: Triple appendix vermiformis: A unique case. Br J Surg 55:79, 1968
56. Toldt C: Die Formbildung des menschlichen Blinddarmes und die Valvula coli. Sitzungsb Akad Wissensch Math-Naturw Cl, Wien 53:41, 1894
57. Treutner KH, Schumpelick V: [Epidemiology of appendicitis.] Epidemiologie der Appendicitis. Chirurg 68:1, 1997
58. Treves F: Lectures on the anatomy of the intestinal canal and peritoneum in man. BMJ 1:415, 527, 580, 1885
59. Wakeley CPG: Position of the vermiform appendix as ascertained by analysis of 10,000 cases. J Anat 67:277, 1933
60. Waugh TR: Appendix vermiformis duplex. Arch Surg 42:311, 1941
61. Wetzig NR: Diverticulosis of the vermiform appendix. Med J Aust 145:464, 1986

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