This chapter describes, in macroscopic terms, the anatomy and embryology of the intraperitoneal organs and the peritoneum that wraps around them. The chapter lays the anatomical foundation for a detailed presentation of each organ that appears later in the book. It offers a basic orientation to the abdomen and introduces the most common anatomical terms: peritoneum, fascia, omentum, mesentery, ligament, and so forth.

This chapter is also aimed at helping the reader to develop a three-dimensional picture of the viscera in the peritoneum and in the body as a whole. Since detailed anatomical descriptions are not always easy to read, we suggest that the reader go back and forth to the chapters on the individual organs.

Developmental steps from the formation of the gut tube to the differentiation into individual viscera are well documented here, as is how the abdominal and thoracic cavities are created. Understanding embryological movements and changes helps to visualize the arrangement of the viscera in the abdomen. Embryological development is twofold: an organ must find its place and develop its form. The positional change in development is the main subject of this chapter. On the other hand, the process of morphogenetic growth, developing individual form, creates the intrinsic architecture of the organ and is the basis of intravisceral movement. This will be presented in Chapter 5 on motility, and in greater detail for each individual organ in Chapters 13–21.

3.1 EMBRYOLOGY

Embryonic growth is subject to biodynamic laws and genetic influences. It requires proper nutritional support, removal of waste products, and adequate space. If these factors are insufficient, growth is slowed down or impeded with respect to intensity (nourishment) and direction (space). Initially, growth is made possible through special metabolic activities within the tissues and between the different types of tissue. Once the vessels emerge, three abdominal arteries (celiac trunk, superior mesenteric, and inferior mesenteric) take on the main nutritional support of the growing peritoneal organs.

Note: Since this chapter deals with development in the womb, the term embryonic in this context also includes fetal development.

3.1.1 Development of the Digestive Tube

Until the end of the second gestational week, the embryonic disk (blastodisk) consists of two germ layers, the endoderm and ectoderm, separated from each other by a basal membrane. Both germ layers participate in forming the third layer, the mesoderm, which develops between the two. During the course of the third week, the mesoderm starts separating endoderm from ectoderm. They stay in contact only in the areas of the buccopharyngeal and cloacal membranes.

The blastodisk, which at this point is trilaminar, folds along a sagittal (cranio-caudal) and a horizontal (lateral) plane (Fig. 3.1a and b). The lateral and cranio-caudal folding of the embryo leads to the development of an endodermal tube. Initially, this tube consists only of a cranial and a caudal part since the middle part of the embryo opens into the yolk sac, which degenerates at a later point during embryonic development.

The pressure difference between the amniotic and chorionic cavities is the main catalyst for the lateral folding motion. The amniotic fluid increases in volume, which stimulates surface growth of the amnion and directs this growth into an anterior, lateral expansion towards the chorionic cavity (extraembryonic coelom).

The lateral folding movement occurs simultaneously with the formation of somites (primitive segments). Paraxial mesoderm becomes organized and is segmented into somites, while the lateral plate mesoderm splits into somatopleuric and splanchnopleuric mesoderm. Somatopleuric mesoderm later forms the parietal serous lining of the body cavities (parietal peritoneum) while splanchnopleuric mesoderm forms the serous membrane ensheathing visceral organs (visceral peritoneum). They do not participate in segmentation. Later, the mesoderm of the somites migrates to the somatopleuric layer, leading to a secondary segmentation, which then precipitates the innervation of the parietal peritoneum. Cranio-caudal folding of the embryo is mainly caused by the rapid cranial growth of the neural
tube. As this tube grows, it runs against the body wall and aorta, causing it to bend.

Initially, the digestive tube arising through all these processes is embedded in mesenchymal tissue and runs sagittally (Figs. 3.2a and b). The double-layered mesentery, which later develops from the mesenchyme, connects the visceral and parietal mesoderm layers.

3.1.2 Development of the Coelomic Cavity

Lateral folding of the mesoderm leads to the formation of a cavity, the intraembryonic coelom, which will later separate into a peritoneal and a pericardial cavity. This coelom is located between the two split layers of lateral mesoderm. The splanchnopleuric mesoderm lies directly adjacent to the endoderm of the digestive tube, whereas the somatopleuric mesoderm follows the lateral and ventral body wall of the embryo. The cavity that has arisen between the two mesoderm layers is the peritoneal cavity.

As the ventral abdominal wall is closed off, the intraembryonic coelom is separated from the extraembryonic coelom, resulting in a closed body cavity. The growing organs gain in volume and take up more space, which causes this (peritoneal and pericardial) cavity to increase in size and the visceral and parietal serous membranes to grow closer together.

(For further information on the growth of the intraperitoneal organs, see sections 3.1.3–3.1.5; more detailed information on the embryonic development of the individual organs can be found in the appropriate chapters.)

3.1.3 Visceral Descent

Once the digestive tube is formed, it begins its longitudinal growth, which amounts to a kind of visceral descent (Figs. 3.3a–c). For example, the developing stomach at the end of week four is located in the cervical region. As the viscera descend, the stomach finds its final position in week seven when it assumes its place at the height of T11 through L3.
This descent comes about due to the cranio-caudal growth gradient in the digestive tube with the growth difference between the body wall and the visceral content. Growth is more intense in the cranial region than in the caudal region. First, the esophagus quickly grows lengthwise in the caudal direction. During this growth phase, the esophagus grows faster than the remaining tube and its environment. It quintuples in length, while the rest of the body only triples in length during the same period. As the esophagus grows, the stomach and liver precursors are moved caudally, while the trunk and head move cephalad. Thus, the descent and the straightening of the embryo are interrelated.

At the end of the visceral descent, the stomach and liver have found their final positions. Subsequently, these organs just change their individual shapes and move in relation to one another.

### 3.1.4 Separation of the Cavities

The intraembryonic coelom begins as one continuous cavity, that is, the peritoneal, pericardial, and pleural cavities are connected to each other via pleuroperitoneal canals (Figs. 3.4a and b). Once the diaphragm develops, it splits off the peritoneal cavity.

The visceral descent entails a descent of the septum transverse from the cervical region, which, along with other mesodermal structures, forms the diaphragm between heart and liver (Fig. 3.5a-c; Chapter 6). At this point, the lateral pleuroperitoneal canals are still open, giving the lungs, which arise from the foregut endoderm, space to grow.

The pleuropericardial membranes fold and participate in the development of the diaphragm, thus closing...
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the coelom. The peritoneal cavity is now closed cranially; the parietal peritoneum adheres to the undersurface of the diaphragm, anchoring the peritoneum upward.

3.1.5 First Fusion and Spatial Growth

- First fusion: top and bottom poles

Once visceral descent is completed, the gut and other intraperitoneal organs grow further and change their positions in the abdominal cavity (Figs. 3.6a and b). The stomach moves toward the left, while the duodenum rotates to the right and then attaches to the posterior parietal peritoneum. The attachment of the descending duodenum

Fig. 3.5a–c Descent of the diaphragm

Fig. 3.6a Spatial growth of the stomach and duodenum
to the posterior parietal peritoneum via Treitz’s fascia is the most secure attachment in the peritoneum (Fig. 3.7).

The duodenum grows and moves to the left, where the duodenojejunal junction attaches via the suspensory muscle of the duodenum (muscle of Treitz) to the diaphragm (see Fig. 3.32c below). This constitutes the top pole (Hinrichsen 1993) of the midgut rotation. At the distal end of the sagittal tube (hindgut), the bottom pole arises from a fixation of a structure, which later develops into the descending colon. This structure attaches to the posterior parietal peritoneum while rotating and ascending laterally and to the left. The visceral peritoneum, the left mesentery, and the posterior parietal peritoneum fuse to form the left Toldt’s fascia (Fig. 3.8a). The top and bottom poles become fixed points for the subsequent rotational growth of the midgut (Fig. 3.8b). The sigmoid colon also grows between two fixed points, that is, the original medial mesenteric root and the secondarily attached Toldt’s fascia of the descending colon.

- **Growth and rotation of the midgut**

The midgut, which is supplied by the superior mesenteric artery, rapidly grows in length and rotates counterclockwise by 270° (Figs. 3.9a and b show 90° of rotation). The small intestine grows between the top and bottom poles. While the duodenum, pancreas, and superior mesenteric artery attach to the posterior parietal peritoneum (via the fascia of Treitz), the descending colon grows cephalad and to the left. This causes the small intestine to rotate counterclockwise by 90° (first rotation). The intense longitudinal growth of the midgut causes a physiological umbilical hernia, that is, the gut migrates into the umbilical cord (Figs. 3.9c and

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**Fig. 3.6b** Rotation of the ventral pancreatic bud and subsequent rotation of the duodenum to the right

**Fig. 3.7** Attachment of the duodenum to the parietal peritoneum
shape during expiration. This not only stimulates the elasticity of the wall tissues, but also the intrinsic neurological and vascular structures. As a result, the organ wall is able to maintain its normal tone, while the glands located in the wall are induced to produce secretions. The whole process has a decongestive and also a stimulating effect on the wall tissues. Thus, all the processes of phase C help the organs to maintain their normal elasticity, their intrinsic motility, and consequently their inherent autonomy. The activities of the diaphragm and abdominal wall during quiet breathing likewise help the peritoneal content to maintain its elasticity. During all the physiological processes in the body, this activity is controlled by the metabolic needs of the person.

6.5 MOVEMENT OF THE DIAPHRAGM AND THE THORAX DURING DEEP BREATHING: MOBILIZATION OF THE PERITONEUM AND ITS CONTENT

This section describes a theoretical model that is based on a normal physiological process. During deep breathing, the diaphragm moves with increased force. As breathing depth increases, it also changes the direction of its movement and its shape (Fig. 6.11).

The movement of the diaphragm and the thorax can be categorized into three different sequential phases, with three different directions (mobilization phases 1-3).

6.5.1 Mobilization Phase 1

- **Diaphragm**
  
  The diaphragm moves further down during deep inhalation than during quiet breathing. It compresses the peritoneal content from top to bottom. The mediolateral parts of the hemidiaphragms move down the furthest. The diaphragm expands laterally and anteriorly, and it flattens out on the transverse plane. Thus, the movement of the diaphragm is not exactly like a piston in a cylinder because the thorax (cylinder) widens toward the bottom (inspiration) and the diaphragm (piston) flattens out. Simultaneously, the central tendon shifts anteriorly (Fig. 6.12). The diaphragm rotates anteriorly around its frontal axis, causing the posterolateral muscle fibers to develop a pre-stress. The organs below the domes of the hemidiaphragms are induced to rotate anteriorly also.

- **Thorax**
  
  During mobilization phase 1, the thorax widens in all directions. The diaphragm and the accessory respiratory muscles support this shape change.

  In the lower third of the thorax, the ribs move laterally like the handles on a bucket (Fig. 6.13). The frontal thorax diameter enlarges, while the zone of apposition becomes smaller. The thoracic wall pulls apart the increasingly stressed lung parenchyma via the pleural membranes. The diaphragm and thoracic wall move apart around the zone of apposition, where the

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**Fig. 6.11a–c**  MR imaging (from Cluzel P, et al. [2000]: Diaphragm and chest wall: Assessment of the inspiratory pump with MR imaging. Radiology 215: 574-83)
costodiaphragmatic recess is located. Similar to a bellows that is opened, more air flows into the lungs. Simultaneously, the thoracoabdominal pressure gradient increases, accompanied by an increased hemodynamic effect of respiration.

- **Peritoneum and lumbar spine**

  Fig. 6.14 shows the transition from phase C to mobilization phase 1. The peritoneum sinks more anteriorly than posteriorly. It moves anteroinferiorly along with its contents, the anterior parietal peritoneum (APP) more so than the posterior parietal peritoneum (PP). The volume within the cavity stays almost the same, but the pressure increases. The lumbar spine has a tendency to increase its curvature (lordosis) since it is pulled forward by the contraction of the transversus abdominis muscle. The lumbar spine is able to follow the respiratory movement because of its inherent elasticity. On the level of the pelvis, however, the opposite dynamic is at work. The sacrum rotates anteriorly, the pelvis posteriorly.

- **Movement of the peritoneal content and general explanation**

  The organ chapters contain more detailed information about all of the individual structures. At this point, we will just summarize a few of the important facts.

  **Fixed and Mobile**

  The attachment of the organs inside the peritoneum to the parietal system (peritoneum/diaphragm) varies in strength depending on the organ:
  - Secondarily retroperitoneal structures, for example, the descending duodenum, ascending and descend-
ing colons, and head of the pancreas are strongly fixated. They do not move in relation to the parietal peritoneum (PP).

- Ileum, jejunum, transverse colon, sigmoid colon, and cecum are very mobile structures. These organs are fixated posteriorly (not so much superiorly) via their mesenteries and possibly via reinforcing ligaments. They move quite a bit with respect to the PP.

- Other structures are not directly attached to the PP, but are closely connected to it via an anterior and posterior mesentery. Among these structures are the liver (which is also fused with the diaphragm), stomach and spleen (which are also linked to the diaphragm via ligaments), tail of the pancreas, and ascending duodenum (which is attached to the PP via the muscle of Treitz). These structures can move, but they do not move around freely (see Chapter 3).

As a general statement, we can say that the structures above the transverse mesocolon (T for top) are more fixated, and that the structures below it (B for bottom) are more mobile.

**Spatial Movement**

During mobilization phase 1, the diaphragm moves the organs. This means that they move in relation to their supply structures, that is, the nerves, vessels, vascular trunks, and mesentery, and also in relation to adjacent organs. This movement is referred to as spatial mobility.

**Intravisceral Movement**

The movement of each organ in response to the action of the diaphragm depends on its positional relationship with the diaphragm. Depending on where a portion of the organ is located in the body cavity, it has a different spatial relationship with the diaphragm. The following list shows a few of the different positional relationships between portions of an organ and the diaphragm:

- The right lobe of the liver is located below the right hemidiaphragm; the left lobe is located below the left hemidiaphragm.
- The upper portion of the stomach is located directly posteroinferomedially to the left hemidiaphragm, whereas the lower portion is located further away from the diaphragm and sits on the right side.
- The jejunum is located on the left side, closer to the diaphragm than the ileum, which is located on the right. The jejunum is also situated more horizontally than the ileum, which lies more vertically.

It makes sense that diaphragmatic movement has different effects on the different portions of an organ.

When the moving diaphragm causes an organ to move within the body cavity, it will also induce an internal, intravisceral movement (torsion and bending) within the organ. This is almost as if the organ is “wring out.” This will lead to increased compression in some places and to traction in others. The compression supports venous decongestion in the organ and also increases its content pressure. The traction stimulates the different tissues of the organ wall. This activity stretches the wall musculature, internal neurovascular structures, and the intravisceral connective tissue, such as the gland cells in the wall.

Spatial mobility is greater in B (bottom) than in T (top), since T is more fixated and B is more mobile.

**Note:** Mobility causes intravisceral movements. We might also label this intrinsic mobility, but since the driving force behind this movement is outside the organ, it is not truly intrinsic. Functionally, intravisceral torsion through thoracic respiration stimulates the intrinsic mechanisms, although the force is extrinsic in nature.
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Duality

Most viscera and organs can be subdivided into two anatomical portions, A and B, which function differently from each other. The body uses this duality with respect to an organ’s mobility. The movement of the diaphragm has different effects on the intrinsic mobility of portions A and B, even causing them to move in two different directions.

Directional Elasticity

Elasticity of an organ has a specific direction depending on the organ’s embryological growth. The body uses this directional elasticity to maintain or restore the local autonomy of the organ (see Chapter 5).

If diaphragmatic mobilization compresses an organ in the direction opposite its elasticity, it will react by expanding, similar to an elastic sponge that is squeezed, and will resume its original form once compression ceases. This internal expansive counter-movement corresponds to an organ’s intrinsic motility toward inspir (see Chapter 5).

Accumulation of Different Movements

Different movements can add up from phase to phase. For example, when an organ is mobilized beyond mobilization phase 1 into phase 2, movements of intrinsic and spatial mobility accumulate. The position of maximal inspiration of phase 1 constitutes the starting point for movements from phase 2. In other words, at the beginning of phase 2, the structure is already compressed and stretched internally and has also moved downward in the body cavity. Another movement of intrinsic mobility, that is, compression and traction, is added to the intrinsic mobility from phase 1.

Example: When you compress a sponge from the top, you can add pressure to it by squeezing from the sides, too.

Similarly, the starting point for spatial mobility in phase 2 is the position where inspiration from phase 1 ended. Thus, the structure at the beginning of phase 2 lies deeper than at the beginning of phase 1. The same is true for the transition from phase 2 to phase 3. Step by step, the organ moves further downward.

Spatial Mobility

Fig. 6.15 shows the principal movements of the organs during phase 1. The structures move in the body cavity in the following manner:

- The diaphragm sinks and expands anteriorly, pushing the organs below the diaphragm downward, and causing them to rotate anteriorly. During phase 1, the intraperitoneal structures are moved caudally.

There is a greater amplitude of movement in T (top).

- The liver sinks and rotates anteromedially. The amplitude of the movement predominates anteriorly since the organ is most mobile in this area. Posteriorly, the liver is attached to the diaphragm.

- The upper part of the stomach, which is located posteromedially below the diaphragmatic dome, sinks and is moved anteromedially by the diaphragm. The bottom part of the stomach sinks less than the upper part.

- The descending duodenum, which developed into a curved structure during embryonic growth, decreases its lordosis. The more mobile ascending duodenum moves anteriorly, causing its middle horizontal portion to straighten out on a transverse plane.

- The small intestine is shaped like a fan, which closes caudally, starting with the jejunum.

- The transverse colon, which is attached to the 9th and 10th ribs via its lateral ligaments, is stretched owing to the bucket handle movement of the ribs. This is similar to a lose rope, which is tightened because it is pulled at both ends. Simultaneously, the transverse colon moves anteroinferiorly.

- The loop of the sigmoid colon also closes caudally,
starting with its proximal portion.
- The cecum sinks, while its C shape is straightened.
- The ascending and descending colons act like the descending duodenum, albeit to a lesser degree.

**Intravisceral Movements**

During mobilization phase 1, the internal effect of compression phase C increases. All peritoneal organs twist internally in a direction opposite their “directional elasticity.” The amplitude of this torsion is greater than the amplitude during intrinsic motility; the tissue spring is compressed even further. This increases the decongesting effect on the tissues and the stimulatory effect on the organ wall. The pivot point of this internal torsion lies at the level of the pacemaker cells in the gut, as is the case with intrinsic motility. Mobility causes intravisceral movement to stimulate the intrinsic mechanism.

**Nerves and vessels**

As the organs move in the body cavity (spatial mobility), they also change their position relative to their vascular trunks. They lose their normal physiological resting position, which creates mechanical stress in the vessels. First the distal arterial structures closer to the organ are subjected to traction. As described in Chapter 5, the body reacts by lifting the organ into its original physiological position, inducing extrinsic motility in inspir.

Intrinsic mobility brings about an even stronger compression in mobilization phase 1 than in phase C. This compression is stronger in T (top) than B (bottom), because the organs are more securely attached superiorly.

More than a slight compression of the organs may result. If the compression is so strong that the organs are twisted, the intrahepatic pressure rises. This creates an increased portocaval pressure gradient, which causes more portal blood to move into the caval system. If there is an elasticity problem, which causes a venous congestion in the wall of the intestine or in the tissue of the glandular organs (e.g., the liver), this effect can reduce the congestion. Simultaneously, this twisting greatly stimulates intrinsic myogenic and neurological structures in the organs. The tone of the organ wall increases (possibly as a result of the stimulation), and the relationship of content pressure and wall tension normalizes, causing elasticity to normalize also.

**Ligaments**

During mobilization phase 1, the ligaments move along with and coordinate these movements. The inertia of the mobilized organ causes the ligaments to become tight. The principle of this concept is similar to two spheres in a pendulum (Fig. 6.16). As you lift up one sphere and then let it go, it will hit the other sphere and, in so doing, transmit kinetic energy to it. If the two spheres are connected to a spring, the spring will be compressed as the first sphere moves toward the second and stretched after it hits it.

### 6.5.2 Mobilization Phase 2

**Diaphragm**

During this second phase of deep inspiration, the diaphragm sinks even lower. It uses the peritoneal content as an abutment and rotates outward along with the ribs (Fig. 6.17).

The muscle fibers of the diaphragm, which were subjected to pre-stress during mobilization phase 1, contract and induce an external rotation in both hemi-diaphragms. Diaphragm and ribs form a mobile unit, where the ribs move along with the diaphragm (Loring and Konno 1982).

**Thorax**

The accessory respiratory muscles (intercostals, serratus posterior, rhomboids) and the diaphragm control the movement of the thorax during this phase, which consists of an anteromedial rotation of the ribs and an anterior movement of the vertebral bodies (Fig. 6.18). As inspiratory volume increases, the ribs build up a
progressive resistance to the movement of the thorax.

- **Peritoneum and lumbar spine**

Both sides of the peritoneum rotate externally and the entire peritoneal cavity continues to move anteroinferiorly. The lumbar column elastically follows this movement and increases its curvature (lordosis). The elastic tension of the abdominal wall increases (Fig. 6.19).

- **Peritoneal content**

Mobilization phase 2 adds further movements to the spatial and intrinsic mobility of the organs from mobilization phase 1. The structures in the peritoneal cavity follow the diaphragm as it rotates externally and inferiorly. This movement is stronger the closer to the diaphragm and the more external in the cavity these structures are located (Fig. 6.20).
13.4.4 Wall Structure

Along the curvatures, there are mainly longitudinal muscle fibers (Fig. 13.14). The fibers of the middle layer are arranged vertically to the longitudinal fibers. They encircle the entire stomach and become denser the closer they get to the pylorus (Fig. 13.15). The innermost oblique layer is mostly located at the anterior and posterior borders of the stomach and becomes sparse to nonexistent at the curvatures.

The antrum, pylorus, and superior duodenum form a functional motor unit with the pylorus as the most constricted area (Fig. 13.16). The antrum is responsible for emptying the stomach and the superior duodenum prevents reflux. Narrowing of the pylorus is achieved by a change of the fiber arrangement in this area and a longitudinal stretching of the pylorus. This is similar to the functioning of the lower esophageal sphincter.

13.4.5 Vascular Supply

Arterial supply

Both curvatures contain vascular arcades, which are located inside the adjacent mesentery. The left gastric artery exits from the celiac trunk and anastomoses with the right gastric artery, which arises from the hepatic artery proper (Fig. 13.17). The arcade formed by the anastomosis is located in the lesser omentum, at the entry to the omental bursa (see Fig. 13.22).

The left gastro-omental artery originating from the lateral end of the splenic artery connects with the right gastro-omental artery, which comes from the gastro-duodenal artery. The arcade formed by these arteries is bigger. It runs along the edge of the lesser omentum.

In addition to these arcades, there is a network of shorter arteries, such as the short gastric artery ascending from the lateral end of the splenic artery, the anterior cardiotuberosity artery (a branch of the left gastric artery), and the posterior cardiotuberosity artery (a branch of the splenic artery), which supply the fundus and the cardia. The splenic artery passes through the posterior wall of the omental bursa, inside the posterior gastric mesentery. The short vessels exiting the splenic artery pass along the bursal wall before entering the stomach. The arteries enter the stomach wall and form a dense, large vascular network in the gastric submucosa. The vessels in this network have many anastomoses. This submucosal plexus, except for the area of the lesser curvature, is well supplied through the internal vascular network.
Venous supply
The veins are arranged similarly to the arteries, that is, there are intrinsic plexuses and extrinsic arcades. The lesser arcade drains directly to the portal vein, just distal to where the portal and azygos systems anastomose. The greater venous arcade drains into the splenic vein on the left and into the superior mesenteric vein on the right.

13.4.6 Autonomic Innervation

- Intrinsic nervous system
  - Meissner’s plexus
  - Auerbach’s plexus

- Extrinsic nervous system

  - Parasympathetic nervous system
As described earlier (section 13.1), the two vagus nerves rotate to the right along with the esophagus (Fig. 13.1). Due to this rotation, the left vagus nerve passes along the anterior side of the stomach, while the right passes along its posterior side. The nerve fibers thus do not accompany the vessels to the stomach.

  Once they pierce the diaphragm at the esophageal hiatus, the left vagus nerve gives off the hepatic branch, while the right vagus nerve gives off the thick celiac branch, which continues on to the celiac ganglion where it synapses. Both nerves continue vertically along the lesser curvature, the left anteriorly and the right posteriorly. In their course, they give off four to six branches at different levels, the nerves of Latarjet. These branches innervate the gastric muscles and mucosa at different segments.

  The distal portion of the stomach (antrum/pylorus), which is located in a more horizontal plane, is innervated by the pyloric nerve. This nerve always originates from the left vagus nerve. Sometimes it branches off of its hepatic branch. The gastroepiploic nerve runs along the greater curvature of the stomach. Its exact origin is unknown (Fig.13.18).

  - Sympathetic nervous system
The sympathetic fibers exit the spinal cords at segments T6–9 at the level of the diaphragmatic crura and continue to the celiac ganglion as greater splanchnic nerves. They synapse at the ganglion and continue on to the stomach, along with the vessels. These vessels mainly receive sympathetic innervation. Only few sympathetic fibers innervate the muscular wall of the stomach. The innervation becomes denser in the area around the pylorus. Spinal cord segment T6 supplies the cardia, whereas segment T9 mainly supplies the pylorus.
Sympathetic and parasympathetic fibers innervating the stomach do not run in parallel. Sympathetic fibers follow the course of the arteries after synapsing at the celiac ganglion, whereas the two vagus nerves pass along the esophageal wall and enter the stomach directly.

### 13.4.7 Attachments

Early on in embryogenesis, the ends of the stomach attach to the diaphragm and the duodenum, respectively (Figs. 13.19 and 13.20).

- **Posteromedial attachment:** The gastrophrenic ligament connecting the stomach with the diaphragm runs posteromedially (Fig. 13.24). Thickenings from the anterior and posterior mesentery extend into this ligament.
- **Left attachment:** The gastrolienal ligament, which constitutes a thickening of the posterior gastric mesentery, attaches the stomach to the spleen.
- **Anterolateral attachment:** The gastrocolic ligament connects the stomach to the transverse colon.
- **Inferior left attachment:** A peritoneal attachment runs from below the spleen to the lateral diaphragm.
- **Right attachment:** The stomach is connected to the liver via the lesser omentum, which also develops
Fig. 13.21 Different portions of the lesser omentum (according to Bouchet and Cuilleret)

Fig. 13.22 The bottom left portion of the omental bursa grows and expands. The entry to the omental bursa is formed by the vascular arcade of the stomach. The bottom part of the Fig. depicts the greater omentum, which is continuous with the omental bursa.

Fig. 13.23 Entryway to the inferior recess of the omental bursa (viewed from lateral right). The inferior vena cava is located posterior to the entry, the portal vein anterior (according to Perlemuter and Waligora).

Fig. 13.24 The bursa continues behind the stomach, terminating in the gastrophrenic ligament (at the top left; according to Perlemuter and Waligora).
thickenings. These thickenings form the hepato-gastric ligament.

Lesser omentum

The lesser omentum can be subdivided into three portions with three different content matters and qualities. These present the body with several possible ways to compensate for dysfunction (Figs. 13.21 and 13.23).

- *Pars vasculosa* includes the portal vein, hepatic artery proper, and the bile duct.
- *Pars flaccida* includes the vascular arcade of the lesser curvature.
- *Pars condensa* is a denser tissue structure that is continuous with the esophagus.

“Directional elasticity” of the lesser omentum

The coelomic fissure between body wall and liver starts expanding once the liver starts growing at an enormous rate. This causes the anterior mesentery to grow from front to back. The direction of the growth determines the direction of the tissue’s elasticity.

13.5 INTRINSIC ACTIVITY AND ELASTICITY

Depending on the digestive state of the GI tract, that is, its state during the interdigestive phase and the postprandial phase, there are many diverse factors that influence its elasticity. Mechanical, metabolic, and neurohormonal factors in the gut are responsible for maintaining the elasticity and for keeping its local autonomy.

Beyond its myogenic structure and its basic tone, the functioning of the lower esophageal sphincter is also influenced by neuroendocrine and metabolic factors. Substances such as fat, alcohol, chocolate, peppermint, cholecystokinin, and progesterone lower the tone of the lower esophageal sphincter, while gastrin, motilin, and substance P elevate it.

13.5.1 Interdigestive Phase

Wall tension

The entire GI tract possesses a basic myogenic tone, which is part of the intrinsic quality of its smooth muscle cells. It is built up via overlapping action potentials and the formation of a syncytium through gap junctions. This tone is maintained even when there is no innervation of the structure or when the intramural neurons are blocked due to the intake of medical drugs.

The interstitial cells of Cajal, which are located at the junction between the distal and proximal portion of the stomach, create an excitatory plateau, the so-called slow waves. These in themselves do not trigger action potentials. Instead, they create a starting point for subsequent action potentials triggered by many different stimuli. In contrast to the remainder of the gut, the slow waves become more frequent toward the distal end of the stomach. This could attest to the stomach’s function of mixing chyme.

The migrating motor complex, a myogenic cyclical activity in the gastrointestinal smooth muscle, develops in the interdigestive phase. Each MMC cycle is subdivided into three phases:

- **Phase 1**: This is the longest phase of the MMC. It does not, however, display any motor activity. Slow waves propagate toward the antrum, and the proximal stomach maintains a medium tone.
- **Phase 2**: Here, there are contractions, whose frequency increases over time.
- **Phase 3**: The contractions become more pronounced and increase in frequency. This phase lasts 10 to 20 minutes.

The MMC, which also develops in the small intestine (though at a later time during the interdigestive phase), progresses in coordinated fashion. As the proximal stomach contracts, the distal stomach relaxes, whereas the contraction of the distal stomach has an inhibiting effect on the duodenum and a maximal relaxing effect on the pyloric region. The result is a peristaltic propulsion of the gastric content, that is, of secretions and indigestible food. The cells of Cajal are responsible for MMC activity, which only ceases at the time of the next food intake. It is possible that MMC activity continues even during food intake and that it is merely covered up by postprandial motor activities.

During MMC activity, motilin concentration in the plasma increases, pointing to the possibility that motilin may also be involved in influencing MMC activity. The hormone gastrin, which has a paracrine effect, may support the muscle tone in the area around the gastric antrum.

Content pressure

In the interdigestive phase, the body releases a basic amount of secretions and mucus, whose production is locally controlled by paracrine hormones and Meissner’s plexuses. Additionally, saliva is produced and transported to the stomach. MMC activity controls the release of substances into the stomach.

13.5.2 Postprandial Phase

During the postprandial phase, local control is supplanted by higher structures (e.g., the parasympathetic
nervous system) and by endocrine hormones. Thus, the stomach is not really autonomous during this phase. However, the normal physiology of this phase helps the stomach to keep its autonomy.

**Wall tension and content pressure**

Once food is consumed and enters the stomach, its proximal portion reacts with a “stress relaxation.” A vagovagal reflex, controlled via afferent nerve fibers at the upper pharynx and esophagus, causes a progressive adjustment of the stomach wall to the increased stomach contents (mechanical stress). This mechanism allows the stomach to adjust to an increase in volume (filling) while the pressure of its contents increases only slightly during this process.

*Note:* The general biomechanical rule regarding the gut tube is that mechanical stress (filling, stretching, etc.) produces an active counter-reaction—a contraction, not a relaxation of the wall. The most normal and most physiological stimulus is an increase in intraluminal pressure, that is, filling in the process of eating and peristaltic transport. The luminal filling produces a pressure increase that induces a stretch on the gut wall muscles. Normally, the response to stretching is an elastic rebound and a contraction of wall muscles, but in the stomach (and to some extent in the cecum and rectum), there is a physiological stress relaxation. Since stomach volume changes physiologically, a complete visceral diagnosis would include the volume of the organ (see section 18.3).

The proximal portion of the stomach controls the reaction to fluids, while the distal portion controls the reaction to solid foods. Simultaneous contractions with low amplitude develop in the fundus. They increase the intragastric pressure, causing a pressure gradient between the stomach and duodenum.

**Example:** Envision the stomach as an open, upside-down wineskin. The pylorus is never closed so that the wine (stomach contents) can be freely squeezed out of it.

Generally, the stomach does not store fluids (see “Fluid Tests” in section 13.9.3). When solid food particles enter the stomach, it reacts with intense peristaltic waves that are generated by pacemaker cells (Figs. 13.25–13.27). Contrasctions of the circular muscles, adapted to the slow-wave pattern, “catapult” the stomach contents toward the pylorus. The amplitude of the contractions is determined by the consistency of the chyme and by the neurohumoral environment.

Antrum and pylorus contract simultaneously, macerating bigger food particles and “catapulting” them back toward the proximal stomach. The resulting shear stress assists in the maceration process. Only particles smaller than 1mm can pass through the pylorus.

Hormones are also actively involved in peristalsis. The most important hormone is gastrin, which is released as a result of the presence of amino acids and peptides and due to the activity of the vagus nerve (possibly via the neurotransmitter GRP, gastrin-releasing peptide). Gastrin increases the frequency of contractions around the antrum and elevates the tone of the lower esophageal sphincter. Its main function consists of stimulating parietal cells to produce hydrochloric acid during gastric secretion.

Emptying of the stomach is controlled via meta-
bolic, mechanical, and neurohormonal processes. Sour chyme in the duodenum inhibits emptying and so does the presence of fat and protein in the duodenum (see Chapters 15 and 16).

Mechanical control of gastric emptying is done through modulation of the tone of the pylorus and its opening. Hormonal control is exerted when the presence of cholecystokinin inhibits emptying of the stomach. In addition, the pressure gradient between the stomach and duodenum plays a role in gastric emptying.

13.6 OSTEOPathIC MOTILITY: INTRINSIC AND SPATIAL

Embryological growth movements in the body cavity determine the relationship between the supply vessels and the organs, and thus they also determine extrinsic motility. Intrinsic growth movements determine the form of the organ, the “directional elasticity” of the wall, and therefore the structure’s intrinsic motility. Intrinsic motility is a movement inside of the structure, whereas extrinsic motility refers to a spatial movement in the body cavity.

■ Intrinsic motility

Intrinsic motility derives from the morphogenetic growth patterns of an organ. Usually, each structure is internally twisted, and this torsion, set into motion, helps the structure maintain its own autonomy. The intrinsic movement caused by the torsion in the stomach stimulates both pacemaker cells and mechano-sensitive enteric nerve cells that work to keep the tone of the stomach wall constant. During the inspir phase of intrinsic motility (following the morphogenetic growth pattern), the entire stomach expands according to the directional elasticity of its tissue: the proximal portion expands posterolaterally and to the left, while the distal portion expands to the bottom, right, and front. The upper part turns left while the lower part turns right, introducing torsion in the stomach. Looking at the stomach from the front, there is also a sidebend visible: the greater curvature is growing more than the lesser one, putting the stomach into a sidebend to the right.

Palpating intrinsic motility is only possible in a bimanual palpation, where our hands are in contact with the inner architecture of the organ, with one hand following the proximal stomach while the other follows the distal stomach.

■ Extrinsic/spatial motility

When the stomach loses its position and becomes ptotic, it will pull on its vessels. They will react by lifting the stomach back to its normal position. If the stomach loses its elasticity, and therefore its position, it causes a movement where the stomach rotates to the right and tilts into the horizontal plane. Simultaneously, the stomach is tightened, so that the spatial movement also creates an intrinsic movement corresponding to the intrinsic motility toward inspir (see Chapter 5).

13.7 MOBILITY: SPATIAL AND INTRAVISCERAL MOVEMENTS DUE TO THORACIC RESPIRATION

The movement of an organ through thoracic respiration (mobility) has an effect on both its place in the abdominal cavity and its form. The downward motion of the diaphragm will at some point move the viscera downward in the abdominal cavity. This effect is spatial in the sense that the organ changes its position; the viscera move in space in relationship to the body wall (e.g., belly muscles and spine). Besides the spatial effect, there is another that impacts the form of the organ. Since the organ is compressed in mobility, there is a change in volume as well. With deeper inhalation, there is also intravisceral movement: there is torsion because the proximal and distal parts of the stomach rotate in different directions; there is side-bending since the lesser curvature shortens while the greater curvature lengthens; and there is flexion/extension as the upper and lower parts of the stomach move anteriorly or posteriorly.

This subchapter on stomach mobility develops the spatial and intravisceral movements at great length. The same principles and forces apply to all the viscera, although the next chapters do not offer the same in-depth discussion.

■ Compression phase

The resting activity of the diaphragm assists the stomach in keeping its normal elasticity and helps it maintain its autonomy (phase C).

Postprandially, the stomach’s content pressure may increase more or less depending on the type and quantity of food. Elasticity may also increase as the stomach pushes outward in all directions. The diaphragm tries to avoid that from above by increasing its compression. This elevated compression supports the stomach in restoring its normal elasticity. When the food intake is either too large or difficult to digest, this causes a primary trauma to the stomach due to mechanical reasons. There are also primary traumas due to metabolic and neurohormonal reasons.

During the other mobility phases, the stomach starts moving in the body cavity. As the movement becomes progressively lower throughout the phases,
the starting point for inhalation moves further downward. This stimulates central neurovascular structures. Simultaneously, the stomach gets twisted differently in each phase, entailing a stimulation of the stomach wall. Compression of the stomach supports venous decongestion of the stomach wall.

### Mobilization phase 1

During this phase, descent and anterior rotation of the proximal stomach are the most dominant movements (Fig. 13.28).

- **Spatial movement:** The diaphragm moves antero-inferiorly, and pushes the proximal stomach in the same direction; the distal stomach moves less, or not at all. During this movement, the fundus moves anteromedially.

- **Intravisceral movement:** The stomach is compressed because its proximal portion is moved inferiorly while the distal part stays in place. The stomach is also twisted because of the anteromedial rotation of the proximal portion. Since the proximal stomach moves anteriorly and more to the midline, there is also flexion and sidebending in the organ.

#### Example

When you squeeze a sponge against the direction of its elasticity, a counterforce will develop in the sponge, making it expand in order to regain its original shape.

### Mobilization phase 2

- **Spatial movement:** The proximal portion of the stomach now rotates externally. The distal portion slides along the transverse colon, causing it to come into a more horizontal plane. All of these movements stimulate adjacent neurovascular structures (e.g., vascular arcades, nerves of Latarjet) and the nerves and vessels passing through the gastric mesenteries.

- **Intravisceral movement:** The axis, around which the proximal and distal portions are twisted, has moved out of the vertical plane (Fig. 13.29). Compression becomes progressively stronger toward the stomach’s distal end.

### Mobilization phase 3

- **Spatial movement:** The proximal stomach rotates posteriorly and inferiorly, while the distal portion increasingly sinks along with the posterior parietal peritoneum and rotates to the right (Fig. 13.29). The ensuing traction causes a pull on the vascular pedicle of the celiac trunk that informs it of the stomach’s movement and stimulates it at the same time.

- **Intravisceral movement:** An anterior/posterior compression ensues along with a torsion of the distal and proximal stomach. The distal stomach is twisted toward the right front and the proximal stomach to the back (Fig. 13.29). The axis of torsion has shifted toward a vertical plane, with the upper stomach moving back and the organ moving into extension.

### 13.7.1 Relational Mobility of the Stomach

#### Continuity between esophagus and stomach

The diaphragm plays an important role in the closure of the lower esophageal sphincter. Pressure in this sphincter (high pressure zone) increases in proportion to the abdominal pressure so that the pressure inside the stomach always stays lower than the pressure in the lower sphincter. This prevents reflux of the gastric contents into the esophagus. Two other factors that prevent reflux are an active contraction in the lower esophageal sphincter (controlled by the sympathetic and parasympathetic nervous systems), and an increased tone of the diaphragmatic crura. When the
pulled cranially, the body uses the parietal system to compensate for the stomach. Go back and check the findings from the parietal test (see Chapter 7). What do you find?

When you do an inhibition test, use the right hand to palpate and slightly lift up the stomach. Simultaneously, use the left hand to check whether the increased myogenic tone in the related area (thorax/diaphragm/quadratus lumborum/legs) goes back to normal.

- **Pylorus test**
  
  Same as “Fluid Tests” in section 13.9.3.

### 13.9.8 Relational Diagnosis

If the stomach has lost its autonomy, another organ can compensate for it from the outside. If its elasticity is increased, it may be supporting another visceral structure. While performing this diagnostic test, have the patient breathe quietly.

- **Relational elasticity**
  
  - With your right hand, palpate the stomach and gently lift it up. With your left hand, check whether this causes the increased elasticity of a neighboring structure to go back to normal.
  
  - Possible structures to check are the liver, descending duodenum, transverse colon, left kidney, small intestine, and all the levels of Glenard’s apron system (Figs. 13.47–13.51).

- **Relational motility**
  
  - With your right hand, palpate the stomach and gently lift it up. With your left hand, check whether this causes the increased motility of another structure to disappear.
  
  - Possible structures that could have increased motility are the three aprons of Glenard, including the liver and the left kidney.

- **Relational mobility**
  
  - Does the stomach move in tandem with another structure? If it does, is there a compensatory relationship between the two structures?
  
  - Possible structures that could compensate are the transverse colon, liver, duodenum, descending colon, sigmoid colon, and left kidney.

### 13.10 Treatment Techniques for the Stomach

If the body uses a cyclical compensation method, you should fix one phase while letting the other phase go unhindered. Thus, you increase the body’s compensatory activity during the fixed phase. This causes increased decongestion, boosts intrinsic/extrinsic stimulation, and helps the stomach to regain its normal
13.10.1 Hyper-resistance of the Stomach: Metabolic, Mechanical, or Psycho-relational Trauma

The stomach is hyperelastic due to primary trauma and needs to be decongested. In order for this to happen, you need to boost compression and increase the torsion applied to the stomach wall during inhalation. Use both hands and follow the movement of the stomach during exhalation and inhibit its movement during inhalation (see Chapter 6). It is very important to respect the tissue elasticity in a hyperactive organ. One might also detect an increased volume and the need for expansion accompanied by hyperelasticity. In this case, our palpation allows for a lot of volumetric expansion in the visceras. We do not limit the space of the organ through our manual compression on the organ, but rather give space, separate the viscera from its neighbors, and allow the inner forces of the body to do the job of decongestion.

13.10.2 Compensation via Mobility

- Mobilization phases 1–3

Have the patient assume a supine position and palpate the stomach with both hands (see Chapter 6).

**Indirect Technique**

This technique boosts the extrinsic effect of mobilization phase X (1, 2, or 3).

**Step 1**

- Have the patient breathe in, and follow the stomach in during mobilization phase X.
- As the stomach moves in the body cavity, extrinsic neurovascular structures are stimulated to a different degree depending on the mobilization depth (see section 13.7).
- Simultaneously, the stomach becomes increasingly more compressed and intrinsically stimulated.

**Step 2**

- As the patient starts breathing out, keep the stomach in its inspiratory position so that it stays down even though the diaphragm goes up.
- This increases the mechanical traction on the extrinsic neurovascular structures. This time the pull comes from the diaphragm.
- The increased traction causes a broader stimulation and reaction of the stomach than would have been possible without therapeutic help.

**Step 3**

- Repeat steps 1 and 2 until you feel a normal elasticity.
- During inhalation, the proximal stomach is pulled up and the stomach may start developing motility instead of mobility.

**Direct Technique**

Use this technique to increase the intrinsic effect of mobilization phase X, that is, you will increase venous decongestion and stimulation of the wall structures.

**Step 1**

- Have the patient breathe out and follow the stomach.

**Step 2**

- When the patient starts breathing in, hold the stomach in its expiratory position. This increases the compression and intrinsic stimulation of the stomach by the diaphragm.
Step 3

- Repeat steps 1 and 2 until compression to the stomach is evenly applied from all sides, as during the compression phase. This concludes your treatment.
- If the body changes its mobilization phase from X to X-1 (e.g., from mobilization phase 3 to phase 2), this also concludes your treatment.

Reinforced indirect technique

This technique is similar to the indirect technique except that the therapist increases the direction of the stomach (in mobilization phase X) at the end of inhalation. Pull the stomach slightly more in the direction of the ptosis.

Note: This stretches and stimulates the external neurovascular structures even more than the indirect technique.

Mobilization phase 2

- Have the patient assume the right side-lying position. This will cause the stomach to sink further.
- Stand next to the pelvis. At this position, you can apply the maximum amount of weight to push the stomach cranially.
- Place the left hand below the umbilicus. With the ulnar edge, push obliquely past the abdominal wall into the abdomen. Shape the hand like a bowl to receive the stomach.
- As you face the patient, place the right hand on the side of the thorax.

Phase 1

- Have the patient take a deep in-breath. This helps you to contact the stomach.
- With the left hand, form a bowl to receive the stomach (Fig. 13.52).
- Follow the stomach during exhalation and keep the contact with the stomach intact.
- Follow the stomach in both directions as the patient returns to quiet breathing, and check to see whether there is mobility during quiet breathing.

Phase 2

- Have the patient inhale normally and inhibit the sinking of the stomach. As he exhales, follow the stomach cranialward.
- This treatment may take several cycles before the stomach maintains good contact with the diaphragm.

Phase 3

- With your right hand, feel for the lateral lifting (bucket handle movement) of the costal arch.
- As the patient inhales, follow this movement of the ribs. As the patient exhales, use your right hand to keep the thorax in this elevated position (Fig. 13.53).

Phase 4

- At the conclusion of the treatment, the costal arch stops moving.
- Use both hands to accompany the stomach and thorax in their normal mobility.
The normal cecum will feel like any other organ, not palpable as a distinct structure, but integrated in its visceral environment. There is, within physiological parameters, a normal fluctuating of expansion/contraction, but neither one is dominant and the fluctuation is probably not palpable. The functional ability of the cecum is expansion; this is to say that the compensatory reservoir of the cecum lies in its expanding qualities. A cecum in expansion is more likely to be in compensation, while a jejunum in expansion is more likely to be a loss of compensation, as is a contracted cecum. In our diagnostic approach, compensation shows the strength of an organ to react to challenges, while the loss of compensation is something we have to take very seriously in our treatment approach.

### 18.3.1 Vascularization

The last branch of the superior mesenteric artery on the right is the ileocolic artery, which anastomoses with the ileal artery, the terminal branch of the superior mesenteric artery (Figs. 18.9.a and b, previous page). These two branches form the anterior and posterior cecal arteries, which run around the cecum in the shape of a fork.

Venous drainage of this area is done via the superior mesenteric vein, which runs through the mesentery, just like the superior mesenteric artery (Fig. 18.10). The superior mesenteric vein connects with other veins of the digestive tract posterior to the pancreas in the retroperitoneal space. All these veins then drain into the portal vein.
Lymphatic drainage of this digestive portion is an interesting topic in light of the immune function of the vermiform appendix. The appendix may have been involved in the metabolic functioning of the intestines early on in embryonic development, but “retired” from this function later in its development. Only the large number of lymph follicles gives this structure a functional place in the immune system. The lymphatic vessels from these lymph follicles run to the mesentery, similar to the local veins.

**18.3.2 Innervation**

The nerve pathways follow the vessels: The mixed sympathetic/parasympathetic nerves run from the autonomic plexuses of the organ wall through the mesentery into the superior mesenteric plexus, which is located at the trunk of the superior mesenteric artery (L1). Here, the sympathetic and parasympathetic nerve fibers separate.

The sympathetic fibers form the minor splanchnic nerve, which runs through the diaphragmatic crura to the sympathetic trunk, where it enters the spinal cord and runs cephalad to the spinal segments T11-L1 (colon) and T11/12 (cecum). The parasympathetic fibers form the right vagus nerve, which runs to the celiac plexus and further cephalad (Table 18.1).

Pain sensation is referred to the following dermatomes:

- Cecum-vermiform appendix and ascending colon: right T9-L1 and C3-4
- Cecum-vermiform appendix: right T10-12 (diagnostic point: McBurney’s point)

**18.4 MOTILITY**

- Intrinsic motility: In the inspir phase, the cecum expands into its C-shape (Fig. 18.11a).
- Extrinsic motility: In the inspir phase, the cecum rotates (part of the midgut rotation), ascends, and rotates counterclockwise around its anteroposterior axis (Fig. 18.11b).
18.5 MOBILITY
See Fig. 18.12 a and b.

- Compression phase
The cecum is compressed evenly from all sides. Its intravisceral elasticity is stimulated and the drainage of fluids out of the organ is enhanced.

- Mobilization phase 1
The cecum sinks, moves slightly anteriorly, and straightens its C-shape.

- Mobilization phase 2
The cecum sinks and rotates externally.

- Mobilization phase 3
The cecum sinks and rotates posteriorly.

18.6 DIAGNOSIS

18.6.1 Topography
Normally, the cecum is located three fingers’ width medially from the right ASIS on a line drawn between the umbilicus to the ASIS (Fig. 18.13).

18.6.2 Inspection
- Is the volume of the cecum visible?
- Relationship between cecum and right iliac fossa: position of the right ilium (inflare or outflare, distance from ASIS, umbilicus, anterior/posterior ilium)
- Presence of organ mobility during quiet breathing
- Pelvic and leg position: Is there an outward rotation of right leg?
- Trophic state of the abdominal wall musculature
• Dermal activity in a spinal segment
• Presence of scars, for example, from appendectomy, hysterectomy, C-section, inguinal hernia

18.6.3 Jarricot Test
Test dermatome T10, at the junction between the jejunum and the ascending colon.

18.6.4 Percussion
The normal sound is sonorous with a low pitch if there is presence of air and the cecal wall is relaxed.

Note: If there is an invagination of the small intestine, percussion will produce a dull sound. The same is true when the cecum is filled with chyme.

18.6.5 Palpation

Palpation of the cecum
• Have the patient assume the supine position. Stand to the right of the patient.
• With the fingers of both hands, palpate from lateral to medial (Fig. 18.14) to find the cecum. Is it palpable? Is it sensitive to pressure?

Interpretation of Findings
• Hyper-resistance (E = +), hypersonorous sound, high pitch:
  These are typical findings in a cecum that either reacts with increased content pressure or has to cope with increased content, for example, in the case of reflux due to a jammed hepatic flexure (possible liver dysfunction). The cecum is excessively bloated (expansion of its luminal volume) and has shifted laterally.

• Sensitivity:
  The cecum is reactive and sensitive to touch. A ptotic, atonic cecum is no longer reactive, that is, it is often not sensitive to touch. It is also located more medially.