Emergency Surgery

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Contents

F. Billmann, T. Keck (eds.), *Essentials of Visceral Surgery*,
https://doi.org/10.1007/978-3-662-66735-4_16

16.1 Polytrauma: Abdominal Trauma

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Key Points

- Abdominal trauma (in Europe): ≥80% blunt, ≤20% penetrating; mainly due to traffic and occupational accidents.
- Stabilization according to ATLS (Advanced Trauma Life Support) criteria: Airways, **Breathing**, Circulation, Disability, Exposure (ABCDE).
- FAST (Focused Assessment with Sonography in Trauma) examination: for all patients.
- Contrast-enhanced computed tomography (CT): only for patients hemodynamically stable.
- Circulatory unstable patients with positive FAST: immediate laparotomy.
- Circulatory stable patients without evidence of hollow organ perforation: non-operative approach.

16.1.1 Anatomy of the Abdomen

External Divisions

Anterior Abdominal Wall

- From costal margin to symphysis.
- On both sides from midline to anterior axillary line.

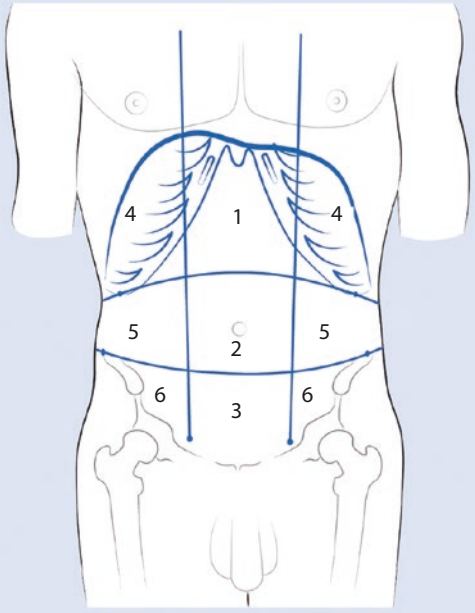
Lateral Abdominal Wall (= Flank)

- Between anterior and posterior axillary line.
- 6. intercostal space to iliac crest.

Abdominal wall = anterior +2 lateral abdominal walls.

Quadrant/Sector Breakdown

- Regional structure of the abdominal wall (◘ Fig. 16.1).
- Sector breakdown:
  - Epigastric region.
  - Umbilical region.
  - Pubic region.
  - Left and right hypochondrium.
  - Left and right lateral abdominal region (= Regio hypogastrica = Flank).
  - Left and right inguinal region (= groin).



1 epigastric region
2 umbilical region
3 pubic region
4 hypochondriac region
5 lateral abdominal region (hypogastric region)
6 inguinal region

◘ **Fig. 16.1** Regional division of the abdominal wall

Internal Divisions

Abdominal Cavity

- Definition: Abdominal cavity = peritoneal cavity + preperitoneal and retroperitoneal cavity.
  - Peritoneal cavity.
  - With liver, pancreas, spleen, small and large intestine, uterus (in pregnant women), filled urinary bladder.
  - Intracostal abdomen: special subdivision of the abdominal cavity, in the rib cage, with diaphragm, liver, pancreas, spleen.

- Retroperitoneal space (= retroperitoneum).
  - With kidneys, ureters, pancreas, aorta and v. cava.
  - Continuity with preperitoneal space and infraperitoneal space.

Pelvis

- Definition: pelvic cavity = true pelvis = peritoneal + infraperitoneal cavity.
- With urinary bladder, urethra, rectum, small intestine, ovaries and uterus.

16.1.2 Injury Mechanisms (Aetiology and Pathophysiology)

Blunt Abdominal Trauma

- Approx. 80% of abdominal injuries in Central Europe.
- Mainly: traffic and work accidents.

Deceleration Trauma

- Shear forces with traction on organs and vascular trunks.
- Injuries: Spleen (40%), liver (35%), small intestine (10%).

Crush Injury

- Between abdominal wall and spine and posterior thoracic wall.
- Especially vulnerable = solid organs (liver, spleen, kidneys).

Compression Injury

- Due to impacts, external compression (e.g. seat belt).
- Abrupt increase in intra-abdominal pressure: rupture of a hollow organ.

Penetrating Abdominal Trauma

- Often criminal/suicidal intent.
- Mostly single cavity, rarely double cavity injuries.

Stab Wound

- Prognostically most favourable form of injury.

Gunshot Wound

- Different injury patterns.
- Impact/Prognosis dependent on:
  - Velocity of projectile.
  - Type of projectile.
  - Firing distance.

Impalement Injury (Due to Accidents)

- Degree of injury depending on shape and penetration depth.
- Combination injuries (impalement + blunt trauma) possible.

16.1.3 Management and Diagnosis

Primary Management

Treatment According to ABCDE Rules (ATLS): Simultaneous Identification + Stabilization of Life-Threatening Injuries

- Airway/cervical spine protections ("**A**irway, with cervical spine protections").
- Ventilation, ensure gas exchange ("**B**reathing").
- Hemodynamic stabilization ("**C**irculation").
- Neurological status ("**D**isability").
- Exposure, complete undressing ("**E**xposure").

Anamnesis

- Rapid investigation of accident mechanism and timing.
- Allergies, medications, etc.

Clinical Presentation

- Inspection (ecchymosis, "seat belt sign", eviscerations, foreign bodies).
- Palpation, percussion, auscultation.
- Rectal examination (prostate protrusion for urethral lesion, evidence for bleeding, sphincter tone for neurological status).

Caution

- Intra-abdominal blood loss and/or small bowel rupture may remain asymptomatic for a prolonged period; development of peritonism takes several hours!
- Overlooking blunt abdominal trauma in unconscious patients.

Laboratory Tests

- Haemoglobin + haematocrit, electrolytes, creatinine and urea levels, blood coagulation, blood gas analysis, glucose, serum amylase, alcohol level, urinalysis, drug screening in urine and pregnancy test if necessary.
- Blood grouping + irregular antibody search; in case of circulatory instability, crossmatch red cell concentrates.

Diagnostic Imaging

X-ray

- Conventional images = low significance in abdominal trauma.

Ultrasound Examination

- FAST examination (Focused Assessment with Sonography for Trauma).
- Detection of free fluid and possible organ rupture (liver, spleen, kidney).
- Poor sensitivity compared to CT (82%).

Computer Tomography

- Whole-body spiral CT "Trauma spiral CT" in polytrauma; abdominal and pelvis CT scan in isolated abdominal trauma.
- No oral contrast agent necessary.
- Sensitivity for intra-abdominal injuries 98%, specificity 99%.

Caution

- Repeated clinical examination of the abdomen in addition to ultrasound + CT in the presence of a significant trauma mechanism.
- CT only if patient is hemodynamically stable.
- Diaphragmatic ruptures, intestinal perforations and pancreatic injuries often not visible at the beginning; in case of suspicion: repeat CT after 36–48 h.

16.1.4 Therapeutic Procedure

Conservative Therapy

Key Points

- Conservative therapy only in stable patients without relevant coagulation disorder.
- Conservative therapy never in case of hollow organ injury!

Blunt Abdominal Trauma

- Continuous (intensive medical) monitoring; also possible in the case of major injuries to parenchymatous organs.
- For liver and spleen injuries:
  - Regular clinical, laboratory and ultrasound control.
  - Conservative approach successful in over 80% of cases.
- Selective arteriography: for liver/spleen/vascular injury.
- Rarely also other interventional measures (drainage, stent placement, etc.)

Penetrating Abdominal Trauma

- Exploration of the wound:
  - Deep fascia intact: Conservative management possible.
  - Deep fascia injured: Diagnostic laparoscopy with evidence/exclusion of penetration of the parietal peritoneum, laparoscopic bowel revision if necessary (only reliable with sufficient overview).

Surgical Therapy

Circulatory Instability

- Patients with positive FAST: emergency laparotomy.
- If FAST not conclusive: If possible, stabilize patient so that CT can be performed…

Circulatory Stability

- Negative FAST and/or CT: Think of other sources of bleeding/shocks.

Major Visceral Trauma/Complex Surgery

- High mortality: due to intraoperative metabolic disturbances.
- Avoid lethal triad at all costs:
  - Coagulopathy.
  - Hypothermia.
  - Metabolic acidosis.
- Strategy:
  - Damage-control laparotomy: find source of bleeding quickly + stop bleeding; stop leakage of bowel contents, close bowel with GIA (blind closure), remove perforated sections.
  - Stabilization in the intensive care unit with the goal of euvolemia + coagulation recovery + warming up of the patient.
  - Planned second-look relaparotomy after 24–48 h for definitive treatment of the abdominal injuries, stoma creation if necessary.

Surgical Procedure

Damage Control Concept (Damage Control Laparotomy)

- Access always via median laparotomy.
- Abdominal packing: Packs inserted in all 4 quadrants, systematic exploration of all 4 quadrants.
- Emergency subdiaphragmatic clamping of the aorta, if necessary.
- Identification of the source of bleeding.
- In case of bleeding from liver parenchyma: possibly Pringle manoeuvre (temporary clamping of the hepatoduodenal ligament), debridement of avital parts of the parenchyma, possibly perihepatic packing (especially in case of coagulation disorder).
- For bleeding from the spleen: splenectomy.
- Bleeding from aorta and iliac vessels: sutures for small lesions, intra-arterial shunt for larger injuries.
- Injury to the vena cava: direct suturing, packing retrohepatic vena cava.
- After hemostasis: look for hollow organ perforations: Direct suture for small perforations; resect larger ones using linear stapler; no anastomosis, no enterostomy! Stomas are only created on the occasion of the second-look laparotomy.
- Leave the abdomen open (= avoid compartment syndrome; ► Sect. 16.3).
- Possibly postoperative angiography for interventional embolisation.

16.1.5 Guidelines

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16.2 Ileus/Obstruction

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16.2.1 Definition—Classification

Definition

- Intestinal ileus = intestinal obstruction = (small/large) bowel obstruction.
- Interruption of the flow of gastrointestinal contents from oral to aboral direction.
- Distinction:
  - Functional ileus = paralysis.
  - Mechanical ileus = mechanical obstruction.

Classification

- By course: Acute vs. subacute vs. chronic.
- By completeness: Complete vs. incomplete.
- According to localization: high vs. deep small bowel ileus vs. large bowel ileus.

16.2.2 Epidemiology

- Common clinical picture.
- Approx. 300,000 laparotomies in USA/year due to small bowel obstruction = approx. 850,000 hospitalization days = 1.3 billion USD per year.

16.2.3 Pathophysiology

"Circulus Vitiosus" of Ileus

- Numerous feedbacks.
- Based on intestinal distention.

Intestinal Distention

- Increase in wall tension → consecutive microcirculatory disturbance: intestinal wall edema + hypoxia of the intestinal wall.
- Consequences of bowel wall edema and bowel wall hypoxia:
  - Transmigratory peritonitis: hypoxia → impaired mucosal barrier and additional stasis → bacterial overgrowth + transmigration.
  - Increase in fluid sequestration into the intestinal lumen, intestinal wall and abdominal cavity.
  - Activation of biogenic amines, kinins and release of interleukins → formation of prostaglandin E with protein degradation, leukocyte increase and fever.
  - Ileus disease = hypovolemic, septic-toxic shock up to multi-organ failure (as final stage).

Normal postoperative normalization of intestinal transit

- Small intestine: 5–24 h.
- Stomach: 1–2 days.
- Colon: 2–3 days (from oral to aboral).

16.2.4 Diagnosis

Key Points

- Previous surgery as a clue of possible adhesions.
- Clinical presentation: palpation + auscultation.
- Abdominal CT scan: increasingly imaging of choice.
- Electrolyte and fluid balance: In the time interval between diagnosis and surgery.

Medical History and Symptoms

- Previous surgery as a clue of possible adhesions (bands).
- Previous changes in bowel habit (more often than complete ileus/obstruction) as the first symptom.
- Pencil thin stool.
- Weight loss.
- Family history.
- Fever.
- Night sweats.
- Loss of appetite, nausea, vomiting.
- Stool and wind retention.
- Acute onset pain: spasmodic/nonspecific.
- Increase in abdominal girth (meteorism).

Clinical Examination: Status Survey

- Fluid status and vital signs.
- Inspection: Surgical scars, hernias.
- Palpation:
  - Abdominal point tenderness.
  - Blumberg's sign/rebound tenderness.
  - Abdominal guarding ("défense musculaire").
  - Resistances.
- Percussion: Meteorism
- Auscultation:
  - Hyperperistalsis in mechanical ileus (high-pitched, metallic bowel sounds).
  - Intestinal sounds sparse or absent (so-called dead silence) in paralytic ileus.
- Digital rectal examination obligatory:
  - Stenoses.
  - Rectal Tumor.
  - Impacted stool.
  - Blood on the glove, if any.

Lab

- Signs of dehydration (hematocrit, creatinine, urea, electrolytes, acid-base balance)
- Infection parameters (CRP, leucocytes).
- Lactate and LDH: sign of vascular ileus (**caution:** normal in 40%).
- Additional parameters to exclude other causes and differential diagnoses: amylase, lipase.

Quantity/Quality of Gastrointestinal Contents Discharged

- Quantity/quality of gastrointestinal contents drained (gastric tube).

Imaging

Ultrasound

- Dilated fluid-filled intestinal loops.
- Intestinal peristalsis.
- Stenoses/strictures.
- Incarcerated hernias.
- Small bowel intussusception.
- intestinal wall thickening.
- Extraluminal free fluid with ineffective peristalsis (in severe ileus) ("to-and-fro" or "whirling" appearance of intra-luminal contents).
- Detection of obstruction: sensitivity 83%, specificity 100%, localization: 70%, etiology: 23%.

Conventional Abdominal Radiograph

- Supine and standing or left lateral position (abdominal overview).
- Obstruction sign:
  - Gas-fluid levels if the study is erect: interface between air/liquid in the intestine.
  - Free air, aerobilia (in 50% with gallstone ileus), shadowing concretions, foreign body.
  - Obstruction localization: eventually possible using gas-fluid level arrangement.
  - Hyperinflation/distention of the caecum in colonic ileus.
- Detection of obstruction: sensitivity 77%, specificity 50%, localization 60%, etiology: 7%.

Contrast-Enhanced (Gastrographin) Conventional X-Ray

- 100 mL gastrographin p.o./gastric tube, abdominal X-ray after 4–6 h.
- Statement possible about the level and extent of ileus.
- ileus resolved if contrast medium in colon in 24 h (sensitivity 97%, specificity 96%).
- Important in "high" proximal small bowel ileus, as no gas-fluid level formation in the normal abdominal radiograph.
- In addition, therapeutic laxative effect.

Caution
Contrast agent can convert a subileus into a complete ileus.

Computer Tomography (CT scan)

- Increasingly imaging modality of choice.
- Pathognomonic ileus signs = local wall thickening due to wall edema and caliber jump (dilated small bowel loops >2.5 cm up from outer wall to outer wall proximal to obstruction and normal caliber or collapsed loops distally) ± evidence of free abdominal fluid.
- Detection of obstruction: Sensitivity 93%, Specificity 100%, Localization: 93%, Etiology: 87%.

16.2.5 Mechanical Ileus (Mechanical Obstruction)

- Important measure: Balancing the electrolyte and fluid balance: In the time interval between diagnosis and surgery.

Etiology and Pathogenesis

Etiology (◘ Table 16.1)

Pathogenesis

- Classification according to the type of obstruction:
  - Luminal obstruction (from inside).
  - Compression (from outside).
  - Strangulation (single band, adhesion, volvulus, abdominal wall hernias).

Table 16.1 Causes of mechanical obstruction

Mechanical small bowel obstruction	
External cause	Adhesions, Single band Internal and external hernias Tumors (pancreas, bile duct, etc.) Volvulus Intraabdominal abscess Intraabdominal hematoma Pancreatic pseudocyst Superior mesenteric artery compression syndrome Intra-abdominal drains Peritoneal carcinomatosis
Intraluminal causes	Neoplasms Gallstones Foreign bodies Bezoar
Intramural causes	Neoplasms Strictures (Crohn's disease, etc.) Hematomas Intussusception Actinic enteritis Regional enteritis
Mechanical large bowel (colon) obstruction	
Common causes	Malignancies Volvulus Diverticulitis Pseudo-obstruction (Ogilvie syndrome) Hernia Anastomotic stricture
Rare causes	Intussusception Stool impaction Strictures Foreign bodies External pressure

- Classification according to localization:
  - Small bowel obstruction (80% of all intestinal peristalsis disorders; high vs. low); bands, malignancies, hernias, adhesions (= 90% of the etiologies).
  - Large bowel (colonic) obstruction (10–15% of all intestinal peristalsis disorders): Stenosing cancer, diverticulitis, volvulus, etc.

Specific Symptoms

High Small Bowel Ileus

- Colicky pain.
- Severe biliary or clear vomiting.
- But: meteorism may be absent.

Low Small Bowel Ileus

- Colicky pain.
- Meteorism.
- Fecal vomiting (Miserere).

Large Bowel Obstruction

- Meteorism.
- Less pain.

Complications

- Ischemia.
  - In case of strangulation with vascular involvement.
  - Especially in the case of mobile small intestine (e.g. in the context of a single band obstruction, in the case of incarcerated hernia, etc.).
- Paralysis

16.2.6 Paralytic Ileus/Functional Obstruction

Etiology and Pathogenesis

Etiology (Table 16.2)

Pathogenesis

- Impairment of the muscular function of the intestinal wall (motility disorder).
- Reaction to various organ diseases, consequences of inflammation, injuries, circulatory or metabolic disorders.
- Reflective:
  - After major abdominal surgery.
  - For intra-abdominal pathology.
  - Response to acute extra-abdominal diseases.
- Special form: Intestinal pseudoobstruction (Ogilvie syndrome) = peristalsis disorder of the colon due to various causes with distension of the caecum.

Table 16.2 Causes of paralytic ileus

Primary forms		
	Myopathic chronic familial pseudoobstruction	
	Neuropathic chronic pseudoobstruction	
Secondary forms		
Intra-abdominal pathologies	Inflammatory	Peritonitis, abscess, colitis
	Mechanical	Operation, Foreign Body
	Chemical	Gastric juice (perforated gastric ulcer), bile, blood
	Autoimmune	Serositis, vasculitis
	Intestinal ischemia	Arterial, venous
Retroperitoneal pathologies		Pancreatitis Haematoma Trauma, e.g. vertebral body fracture Urolithiasis Pyelonephritis, etc.
Extraabdominal disease	Thoracic pathologies	Myocardial infarction Pneumonia Decompensated heart failure Rib fractures
	Metabolic changes	Electrolyte imbalance, e.g. hypokalemia Porphyria Hypothyroidism Hypoparathyroidism Uremia Lead poisoning
	Drugs	Opiates Anticholinergics Antihistamines Catecholamines Antidepressants
	Sepsis	
	Chemotherapy or radiotherapy	
	Trauma	Craniocerebral trauma Thoracic trauma Spinal cord injuries

Specific Symptoms

- Symptoms of the underlying pathology → Symptoms of obstruction → Symptoms of "ileus disease."

16.2.7 Therapy

Key Points

- Always general measures ± antibiotic therapy.
- Absolute indication for surgery:
  - Complete mechanical ileus.
  - High small bowel ileus.
  - Peritonitis with/without paralysis.
  - Strangulation Ileus.
  - Vascular ileus.
  - Gallstone ileus.

Conservative Therapy

General Measures

- Parenteral nutrition therapy (caloric intake).
- Monitoring and compensation of the fluid/electrolyte loss.
- Gastric tube (relief of gastrointestinal tract distension, electrolyte balance, reduction of aspiration risk).
- Regular clinical examination by experienced surgeon.

Antibiotic Therapy

- In the case of bacterial translocation.

Drug Stimulation in Paralytic Ileus

- Sympathicolysis (e.g. peridural catheter).
- Parasympatheticomimetic agents (e.g. neostigmine).
- Prokinetics (e.g. metoclopramide, cisapride).
- Erythromycin: stimulation of gastric peristalsis.
- Hyperosmolar substances.
- If Emergency surgery unlikely; hyperosmolar gastrographin:
  - Reduces bowel wall edema.
  - Promotes peristalsis.

Surgical Therapy

Goals

- Intestinal Decompression.
- Restoration of gastrointestinal patency and blood circulation.

Emergency Indications

- Complete mechanical ileus.
- High small intestine ileus.
- Peritonitis with/without paralysis.
- Strangulation ileus.
- Vascular ileus.
- Gallstone ileus.

Relative Indications

- Chronic recurrent ileus (abdominal adhesions).
- Subileus due to chronic inflammatory bowel disease.
- Peritoneal carcinomatosis.
- Ogilvie's syndrome.

Surgical Procedure

Surgery for Ileus

- Perioperative antibiotic therapy.
- Median laparotomy (extended, if necessary); eventually laparoscopy if single band expected.
- Access to the peritoneal cavity.
- Adhesiolysis: to localize obstruction + to treat obstruction.
- Possibly bowel segment resection (injury, stenosis, tumor, ischemia).
- **Careful** bowel decompression (**caution:** serosal lesions, endotoxin washout, postoperative atony).
- Assessment of blood flow/vitality of the intestine.
- Schedule second-look laparotomy if needed.

16.2.8 Specific Therapy for Certain Types of Ileus

Paralytic Ileus

Conservative Therapy

- Basic therapy as outlined (► Sect. 16.2.7 "Conservative therapy").
- Drug stimulation.
- Elimination of triggering factors.

Surgical/Endoscopic Therapy

- Only in case of severe intestinal distension with consecutive risk of wall ischemia and rupture (especially in the cecal region).
- Endoscopic decompression, decompression tube.
- Surgical creation of a fistula/stoma.

Vascular Ileus

- Revascularization.
  - Embolectomy.
  - Thrombectomy.
  - Aortomesenteric bypass.
- Resection of avital bowel segments.

Strangulation Ileus

- Mechanical ileus with impaired blood circulation.
- Adhesiolysis, single band resection (possibly laparoscopic), hernia repair.
- Resection of avital bowel segments.

Postoperative Ileus

- Wait and see.
- Supportive therapy.

Caution: make the difference between postoperative ileus and ileus due to surgical complications:

- E.g. anastomotic insufficiency, intra-abdominal abscess.
- In case of surgical complications: Revisional surgery/intervention = necessary.

Ogilvie's Syndrome

- Indication for surgery: In case of hyperinflation of the caecum >10 cm
- Interventions:
  - Ileostomy creation.
  - Colon resection.
  - Possibly endoscopic decompression.

Large Bowel Obstruction Due to Colon Cancer

- Procedure dependent on:
  - Localization of the cancer.
  - General condition of the patient.
  - Local intraoperative findings.

Conservative Therapy

- Palliative.
- Colonoscopic stent insertion.

Bridge-to-Surgery

- Goal = staged surgery (tumor operation in a second step).
- Indications:
  - Compensated ileus (= no distension of the small intestine).
  - Obstruction in the left hemicolon/rectum + mild symptoms.
- Methods:
  - Colonoscopic stent insertion.
  - Creation of ileostomy or colostomy.
  - Colonoscopic decompression tube.

Primary Resection

- Indication = Decompensated ileus.
- On the Right side (caecum to mid-transverse): Single-stage resection with simple or extended hemicolectomy and aspiration of the contents of the small intestine.
- On the Left side:
  - Resection with primary anastomosis, possibly insertion of a defunctioning ileostoma.
  - Discontinuity resection (Hartmann's procedure).

Gallstone Ileus

- Removal of stone via enterotomy, cholecystectomy.
- Resection of the cholecystoduodenal fistula controversial (high morbidity).

Volvulus

- Attempt at colonoscopic derotation/decompression.
- If necessary staged surgical colopexy.

16.3 Abdominal Compartment Syndrome

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16.3.1 Definitions

Key Points

- Abdominal compartment syndrome (ACS) = multiorgandysfunction.
- Intra-abdominal hypertension = origin of dysfunction.
- Mostly in critically ill patients.
- Wide range of medical and surgical clinical pictures.

Intra-Abdominal Pressure (IAP)

- Abdomen = polycompartment model.
  - Bounded by rigid, bony (ribs, pelvis, spine) and flexible (abdominal wall, diaphragm) structures.
  - Closed space with physiological pressure = intra-abdominal pressure (IAP).
- IAP:
  - Normally in a "steady state".
  - Changes depending on wall characteristics (external influencing factors)/filling state of the abdominal cavity (internal influencing factors).
- Measurement (gold standard):
  - Indirect measurement: bladder pressure (in mmHg) (► Sect. 16.3.4 Bladder pressure measurement).
  - Normal levels: 5–7 mmHg in a healthy person, up to 10 mmHg in an intensive care patient.

IAP:

- Good correlation between bladder pressure and IAP.
- Aterated level in: Adhesions, pelvic hematoma or fracture, pelvic girth, neurogenic bladder.

Abdominal Perfusion Pressure (APP)

- Calculation: APP = mean arterial pressure (MAP) minus IAP (APP = MAP – IAP).
- Provides conclusions about perfusion of the abdominal organs.
- APP >60 mmHg: correlation with better survival in ACS.

Intra-Abdominal Hypertension (IAH)

- IAP continuous or over a prolonged period ≥12 mmHg.
- Classification:
  - Grade I: IAP = 12–15 mmHg.
  - Grade II: IAP = 16–20 mmHg.
  - Grade III: IAP = 21–25 mmHg.
  - Grade IV: IAP > 25 mmHg.

Abdominal Compartment Syndrome (ACS)

- Prolonged elevation of IAP >20 mmHg + new onset of organ dysfunction/failure.
- Distinction: Acute vs. chronic ACS.
- Destructive course of the disease comparable to ACS of the extremities.

16.3.2 Aetiology

Classification of Abdominal Compartment Syndrome (ACS, ◘ Table 16.3)

- Primary ACS: Due to intra-abdominal injury/disease (abdominal trauma, hematoperitoneum, pancreatitis).
- Secondary ACS: Without initial abdominal focus (hemorrhagic shock, infusion therapy, mesenteric ischemia, and reperfusion).

Pathophysiology

- IAH = Restriction of organ function → ACS.
- At the cellular level: swelling, hypoxia, dysfunction.
- Cardiovascular IAH = diaphragmatic elevation induced:
  - Decreased cardiac output.

Table 16.3 Classification and causes of abdominal compartment syndrome (ACS)

Division	Etiology
Primary ACS (= acute)	Penetrating abdominal trauma Intraperitoneal bleeding Pancreatitis External compression: e.g. after polytrauma, traffic accident, explosion Pelvic fracture Ruptured abdominal aortic aneurysm Perforated gastric ulcer
Secondary ACS (no acute event, subacute fluid accumulation with IAH)	Large volume administration during resuscitation (>3 L) Large-scale combustion (especially third degree combustion) Penetrating/blunt abdominal trauma without visible injury Postoperative After packing and fascial closure Sepsis
Chronic ACS	Peritoneal dialysis Morbid obesity/ extreme adiposity Cirrhosis Meigs' syndrome Intraabdominal mass

IAH intra-abdominal hypertension

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  - V. cava compression (reduced backflow, blood pooling to the pelvis and lower extremities).
  - Decreased compliance and contractility due to direct compression.
- Pulmonary IAH = decreased thoracic volume induced:
  - Atelectasis + oedema (reduced oxygen diffusion).
  - Increased ventilation pressures needed (parenchymal lesion).
  - Ventilation-perfusion mismatch (increased intrapulmonary shunt fraction, increased alveolar dead space volume).
- Renal IAH = venous compression induced:
  - Decreased venous outflow.
  - Arterial vasoconstriction (activation of the renin-angiotensin-aldosterone system).
  - Decrease in glomerular perfusion and diuresis.
- Gastrointestinal IAH = decreased mesenteric blood flow induced:
  - Decreased mucosal perfusion.
  - Compression of the mesenteric veins.
  - Wall Edema.
  - "Circulus vitiosus" with end result of intestinal ischemia, lactic acidosis and possible bacterial translocation, sepsis, multiple organ failure.
- Liver IAH = decreased portal return induced by compression:
  - Decreased degradation of lactate.
- Cerebrovascular IAH = increased intracranial pressure:
  - Critical cerebral perfusion → cerebral ischemia.

IAH:
- Increased risk of thrombosis due to blood pooling in the lower extremities.
- Visceral "Circulus vitiosus": End result = intestinal ischemia.

16.3.3 Clinical presentation

Key Points

- Key role: compliance of the abdominal wall + intra-abdominal volume.
- Mostly lack of communication with severely ill patients.

Risk Factors

- Key role: compliance of the abdominal wall + intra-abdominal volume.
- "Deadly triad": acidosis + hypothermia + coagulopathy.
- Massive fluid substitution with crystalloids (hemorrhage, sepsis, burns).

- Anthropomorphology:
  - Male (intra-abdominal fat distribution).
  - Higher age.
  - Obesity.
  - Small size.
- Comorbidities and/or increased intrabdominal volume:
  - Ascites.
  - Fluid-filled intestinal loops (ileus, mesenteric ischemia).
  - Hepato–/splenomegaly.
  - Pancreatitis.
- Increased tension of the abdominal wall/diaphragm.
  - Fascia closure after damage-control laparotomy.
  - Active muscle contraction (pain).
  - "Body Builder".
  - Anasarca.
  - Abdominal wall hematoma (especially rectus sheath hematoma).
  - COPD (chronic obstructive pulmonary disease), mechanical ventilation (PEEP, "positive end-expiratory pressure"), pneumonia.
  - Burns.

Symptoms

- Distended abdomen.
- Oliguria to anuria.
- Hypercapnia and hypoxia (increase in ventilation pressure).
- Circulatory insufficiency.
- Decreased organ perfusion to lactic acidosis.
- Decreased cerebral and limb perfusion.
- Hypotension, tachycardia, increased jugular venous pressure, peripheral oedema, diffuse abdominal pain.

If ACS is imminent = usually lack of communication in a seriously ill patient.

16.3.4 Diagnosis

Key Points

- Bladder pressure = gold standard.
- Mortality in abdominal compartment syndrome = 40–100%.

Bladder Pressure Measurement (Gold Standard)

- Measurement in mmHg, at the end of expiration, in supine position.
- Reference point = mean axillary line.
- Instillation of 25 ml saline solution into an empty bladder.
- Regular measurement in intensive care patients with risk factors for ACS (4 to 6 h).

Clinical Presentation

- Poor predictor (► Sect. 16.3.3).

Imaging

- Not helpful for diagnosis; mostly CT.
- Signs on imaging: Visible IAH.
  - Diaphragmatic Protrusion.
  - Vein compression (especially inferior vena cava).
  - Abdominal distension.
  - Renal compression.
  - Bilateral inguinal hernias.

Caution

- Clinical diagnosis (detection) should be made before the formation of ACS at the stage of IAH.
- Mortality of ACS = 40–100%.

16.3.5 Therapy

Key Points

- Supportive measures + surgical pressure relief.
- Temporary → definitive abdominal closure.

General Principles of Therapy

WSACS Guidelines

- WSACS = "World Society of the Abdominal Compartment Syndrome": Recommendation of a Therapy Algorithm, Update 2013 (► Sect. 16.3.6).

Supportive Measures

- To lower the IAP ± surgical abdominal decompression.

Goals

- Improvement of abdominal wall compliance.
- Reduction of intra-abdominal volume.

Conservative Measures

Improvement of Abdominal Wall Compliance

- Analgesia.
- Sedation.
- Relaxation + anxiolytic therapy, respiratory support.
- Neuromuscular blockade.

Decrease in Intra-Abdominal Volume

- Balanced fluid management, fluid restriction if possible.
- Gastric/colonic compression:
  - Gastric tube/rectal tube (intestinal tube).
  - Enemas.
  - Prokinetics (metoclopramide, erythromycin, neostigmine).
  - Endoscopy: decompression of hollow organs.
- Drains:
  - Percutaneous drainage for obvious intraperitoneal fluid accumulation (ascites, hematoperitoneum).
  - Interventional drainage of an intra/retroperitoneal collection (abscess, pseudocyst).

Surgical Measures

- Surgical decompression = definitive therapy.
- Relevant complications of surgical therapy.
- Mortality up to 50% (depending on the etiology of ACS).

Decompressive Laparotomy

- Standard procedure.
- Median laparotomy (opening the linea alba + abdomen).

New Alternatives

- Minimally invasive percutaneous endoscopic component separation technique.
- Subcutaneous linea alba fasciotomy (SLAF).

Temporary Abdominal Closure

- Initially: leaving open (the fascia) of the abdomen.
- Abdominal wound dressing with bridging of the fascial ends (= prevention of evisceration/heat loss).
  - Vicryl mesh.
  - "Bogota bag" (sterile plastic bag).
  - "Packing" with wet cloths.
  - Vacuum dressing (= gold standard).

Caution

- Operative measures for all strategies: risk = small bowel fistula.

Definitive Abdominal Closure

- Recommendations:
  - Early.
  - If possible in the same hospitalization (usually within 5–7 days).
- For long-term open abdomen:
  - Often "loss of domain" of the abdominal organs (= covering with granulation tissue).

- Needed closure with "mesh graft": transplantation or cutaneous displacement flap.
- Subsequent fascial closure: only aim for after 9–12 months after.

16.3.6 Guidelines

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16.4 Intestinal Ischemia

B. Weixler and H. Hoffmann

16.4.1 General

- Intestinal ischemia = mesenteric ischemia.
- Classification of intestinal ischemia:
  - Due to the speed of formation/evolution.
  - Due to the severity of intestinal circulatory disorders.
- Two types of intestinal ischemia:
  - Acute intestinal ischemia (= acute mesenteric ischemia).
  - Chronic intestinal ischemia (= chronic mesenteric ischemia).

16.4.2 Acute Intestinal Ischaemia

Key Points

- Causes of acute mesenteric ischemia (AMI): embolus (50%), arterial thrombosis (25%), nonocclusive (20%), venous thrombosis (5%).
- Causes of ischemic colitis: Nonocclusive (95%).
- Clinical presentation: extent of subjective abdominal pain often in contrast to indolent abdominal examination.
- Three-phase clinical course: Acute pain interval (0–6 h); pain-free interval with intestinal paralysis, so-called "rotten peace" (7–12 h); renewed pain interval with peritonitis and septic shock with intestinal necrosis (12–24 h).
- Diagnosis: abdominal CT angiography with contrast medium.
- **Caution:** Normal lactate does not exclude intestinal ischemia!
- Therapy: Rapid start with broad-spectrum antibiotics; always surgical embolectomy and/or arterial thrombectomy, resection of avital intestinal segments if necessary.

Epidemiology

- Prevalence: approx. 1% of all patients with acute abdomen.
- In >70-year-olds: Prevalence = 10%.
- 60–70% of all intestinal ischemias = acute mesenterial ischemia (AMI).

Etiology and Pathogenesis

- Mesenteric artery embolism (50%):
- Cardiac embolus (most common).
- Ruptured plaque of the proximal aorta.
- Mesenteric artery thrombosis (25%):
  - Atherosclerosis (most common cause).
  - Arteritis.
  - Dehydration.
- Nonocclusive Mesenterial Ischemia (NOMI; 20%): Hypoperfusion and/or vasoconstriction in the splanchnic area (in heart failure, sepsis, cardiac or abdominal surgery, use of vasopressors, ergotamines, cocaine).
- Mesenteric vein thrombosis (5%):
  - Genetic hypercoagulability (>75%).
  - Paraneoplastic.
  - Cirrhosis of the liver.
  - Pancreatitis.
  - Pregnancy.

Ischemic colitis = most common form of intestinal ischemia: 95% of ischemic colitis due to NOMI in the area of the vascular anastomoses of the colonic arteries (left flexure and rectosigmoidal junction).

Clinical Presentation

Non-Specific Clinical Presentation

- Sudden onset of periumbilical abdominal pain.
  - Often in discrepancy with the inconspicuous abdominal examination.
  - Often accompanied by nausea, vomiting, diarrhea.
- Localized pain over affected bowel segment.
- In ischemic colitis, hematochezia/bloody diarrhea (typically only after 24 h).

Caution

- The first clinical examination of the abdomen may be completely normal!
- Compared to acute mesenteric ischemia, the pain associated with colonic ischemia is often not as severe.

3-Phase Clinical Course (Rarely Detectable)

- Acute pain interval: colicky pain + vomiting, diarrhoea, shock (after 0–6 h).
- Pain-free interval: intestinal paralysis, acidosis, "rotten peace" (7–12 h).
- New pain interval: peritonitis + septic shock with intestinal necrosis (12–24 h).

Diagnosis

Anamnesis

- Previous thromboembolic events.
- Postprandial abdominal pain (= "abdominal angina").

Laboratory Tests

- Lab levels unreliable; suggest AMI, but can **never** rule it out!
- Marked leukocytosis (>15,000/µl in 75% of patients).
- Elevation of lactate, LDH, CK or amylase = indicators for extent of tissue damage.
- Lactate increase = late; a lactate that remains constant during the course must suggest other diagnoses (sensitivity 90–96%, specificity 60–87%).

Important: If AMI is suspected (with normal laboratory) = no delay due to further diagnosis.

Caution

Lactate levels:

- Only increased lactate = indicative.
- Normal lactate = no exclusion of mesenterial ischemia!
- Explanation for normal levels: complete circulatory arrest in the ischemic area = no drainage of accumulated lactate = no lactate in the peripheral circulation.

Diagnostic Imaging

- CT angiography of the abdomen with contrast medium:
  - Imaging of vessel occlusion (= Contrast Medium stop).
  - Exclusion of tissue necrosis (intramural gas in the GI tract-pneumatosis intestinalis, portal venous gas: gas in the V. portae-hepatis branches).
  - Exclusion of bowel perforation (abdominal free air).

Therapy

Caution
As a principle: If there are clinical signs of intestinal necrosis = generous indication for exploratory laparotomy!

Stabilization

- Always intensive medical monitoring/treatment.
- Oxygen administration + circulation stabilization + fluid balancing.
- Broad-spectrum antibiotic therapy (after only a few hours of ischemia = disintegration of the mucosal barrier = bacterial translocation).

Acute Mesenteric Artery Embolism

- Emergency laparotomy (see "Operative procedure" below).
- Embolectomy.

Acute Mesenteric Artery Thrombosis

- Emergency laparotomy (see "Operative procedure" below).
- Surgical thrombectomy.
- Alternative option = angioplasty within 8 h after symptom onset in stable patients without peritonism.

Acute Mesenteric Vein Thrombosis

- Emergency laparotomy: if evidence of bowel necrosis.
- Conservative therapy: Only in the absence of intestinal necrosis:
  - Heparin bolus 80 U/kg BW, not exceeding 5000 U, then infusion at 18 U/kg BW/h.
  - ICU-Monitoring.

Ischemic Colitis and Nonocclusive Mesenteric Ischemia (NOMI)

- Intensive care monitoring in the absence of gangrene or perforation: surgery necessary in only about 20% of patients (gangrene/perforation):
  - Improvement in heart function.
  - Correction of hypovolemia and metabolic acidosis.
  - Stop vasopressors.
  - Anticoagulation = no evidence.
- Emergency laparotomy if signs of intestinal necrosis (see "Operative procedure" below).

Surgical Procedure
Acute Mesenteric Artery Embolism

- Access via median laparotomy.
- Exposure of the superior mesenteric artery (SMA) in the mesenteric root: At the inferior border of the pancreas (lesser sac – bursa omentalis)/through inframesocolic access (transverse colon).
- Vascular incision proximal to the embolus + embolectomy (Fogarty catheter size 3/4).
- Examination of intestinal vitality (peristalsis and colour); examination of blood flow using vascular Doppler probe.
- If embolectomy not satisfactory: mesenteric vascular bypass if necessary.
- Resection of necrotic bowel + creation of primary anastomosis, ileostomy if necessary.
- In case of inconclusive exploration/findings: laparostoma (leaving the abdomen open; e.g. insertion of an abdominal vacuum dressing + second-look laparotomy within 24–48 h).

Surgical Procedure
Acute Mesenteric Artery Thrombosis

- Access via median laparotomy.
- Exposure of the superior mesenteric artery (SMA) in the mesenteric root: at the inferior border of the pancreas (lesser sac – bursa omentalis)/through inframesocolic access (transverse colon).
- Identification of the affected arteries and intestinal segments (inspection, Doppler).
- Thrombectomy + arterial reconstruction; aortomesenteric bypass if necessary.

- Checking the vitality of the intestine/if necessary, segment resections with primary anastomosis.
- In case of inconclusive exploration/findings: Stapler closure of the bowel ends + laparostoma, second-look laparotomy within 24–48 h.

16.4.3 Chronic Mesenteric Ischaemia (CMI)

- Chronic mesenteric ischemia = chronic intestinal ischemia.

Etiology and Pathogenesis

Etiology

- Atherosclerosis of the mesenteric vessels (>95% of CMI).

Median Arcuate Ligament Syndrome (= MALS = Dunbar Syndrome)

- Controversially discussed clinical entity.
- Chronic intestinal ischemia = possible clinical presentation.
- Pathogenesis: Compression of the coeliac trunk by the median arcuate ligament (diaphragm).
- Rare: Diagnosis of exclusion, sometimes posture or respiration dependant pain.

Pathogenesis (= Atherosclerosis)

- Lack of increase in blood flow while increased demand.
- Reduced inflow.

Clinical Presentation

- Postprandial pain (typical "abdominal angina").
  - 10 min-3 h after food intake.
  - Mostly epigastric or periumbilical.
- Food aversion.
- Weight loss.

Diagnosis

- Diagnostic imaging.
  - Angiography = gold standard.
  - CT angiography (sensitivity 96%, specificity 94%): Additional information (e.g., topographic relationships).
  - MRI angiography (often only 25% of the course of IMA can be visualized).
  - Duplex ultrasound (SMA can be visualized in 90%, coeliac trunk in 80%).

Therapy

- Elective surgical vascular reconstruction.
- Percutaneous transluminal angioplasty (PTA).
- "Acute-on-chronic mesenteric ischemia = treat as an emergency.

16.4.4 Guidelines

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