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Perioperative gastrointestinal problems in the ICU

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Abstract

Gastrointestinal (GI) problems after surgery are common and are not limited to patients undergoing abdominal surgery. GI function is complicated to monitor and is not included in organ dysfunction scores widely used in the ICUs. In most cases, it recovers after surgery, if systemic and local inflammation and perfusion improve, gut oedema resolves, and analgosedation is reduced. However, perioperative GI problems may have severe consequences and increase the risk of death if not recognized and managed in a timely manner. Careful risk evaluation followed by a complex structured assessment and appropriate management of GI symptoms should minimize the potentially severe consequences and thereby possibly improve outcome.

In the current review, we summarize common non-specific perioperative GI problems and some specific surgery-related abdominal problems, address identification of patients at risk of GI problems, and give suggestions for perioperative GI management.

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Key words: perioperative, postoperative, abdominal surgery, gastrointestinal function, gastrointestinal failure, acute gastrointestinal injury, abdominal problems, critical illness

More than half of the mixed ICU population present different gastrointestinal (GI) symptoms during their ICU stay [1, 2]. Perioperative GI dysfunction is not limited to patients undergoing abdominal surgery [3]. In many cases, GI function recovers after surgery, if systemic and local inflammation and perfusion improve, gut oedema resolves, and analgosedation is reduced. However, if not addressed properly, GI dysfunction may lead to severe consequences and impair the postoperative course [4]. Association of GI dysfunction with adverse outcome has been repeatedly demonstrated [3, 5–7]. Nevertheless GI dysfunction is not uniformly defined.

The current review aims to describe common non-specific GI problems and some specific surgery-related abdominal problems, address possibilities of identification of patients at risk, and give suggestions for perioperative GI management.

METHODS

A MEDLINE and PubMed search was performed using the search terms 'perioperative', 'postoperative', 'abdominal surgery', 'gastrointestinal function', 'gastrointestinal failure', 'gastrointestinal dysfunction', 'intestinal failure', 'acute gastrointestinal injury', 'abdominal problems', 'gastrointestinal

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symptoms', and ('critically ill' OR 'intensive care' OR 'critical care' OR 'critical illness'). The reference lists of identified papers were screened to identify other relevant papers.

RESULTS AND DISCUSSION

Below, we first describe different perioperative problems emphasizing important aspects of assessment and management. Subsequently, we focus on risk assessment and propose a management approach for perioperative GI problems.

1. PERIOPERATIVE GI SYMPTOMS AND SYNDROMES IN THE ICU

Most of the perioperative GI problems are non-specific and may occur with or without primary abdominal/GI pathology, whereas some others are more specific and related to abdominal surgery. A clear and unique classification is not available; we propose assessment as presented below and in Table 1.

1.1. NONSPECIFIC SYMPTOMS AND SYNDROMES

NAUSEA AND VOMITING

Vomiting (emesis) in the ICU is defined as the occurrence of any visible regurgitation of gastric content irrespective of the amount [8], whereas nausea is usually not detectable in sedated and ventilated patients.

Postoperative nausea and vomiting (PONV) has a complex aetiology including patient-, anaesthesia-, surgery- and inflammation-related factors and involving multiple pathways.

Receptors involved include: serotonin type 3 (5-hydroxytryptamine 3, 5-HT3); dopamine type 2; histamine type 1; muscarinic; μ opioid; and tachykinin type 1 (neurokinin, NK1) receptors [9].

Several medications have been reported to alleviate symptoms: 5-HT(3) antagonists, dopamine antagonists, sedatives, corticosteroids, antihistaminics, and anticholinergics [10]. Also, prophylactically administered i.v. acetaminophen reduced PONV, mainly mediated through superior pain control [11]. None of the drugs is clearly superior to another [12], but currently, the most widely used and studied group is 5-HT3 antagonists. In case one drug is ineffective, adding a drug from another group is recommended to block another possible mechanism. For the same reason, multiple drugs with different mechanism of action have been suggested for prophylaxis in high-risk patients [13].

PONV is a known reason for delayed discharge after day surgery [14]. Increased risk of aspiration due to vomiting is the main concern in the ICU. About one-third of surgical patients receiving general anesthesia, nearly half after craniotomy [15] and up to three-quarters of patients presenting with several risk factors [16, 17] may experience PONV. A simple Apfel score may help to identify the patients at risk of PONV, but specific surgery- and anaesthesia-related factors need to be considered additionally [13].

If nausea and vomiting are present preoperatively, maximum emptying of the stomach needs to be achieved before anaesthesia induction and rapid sequence intubation is preferred technique for airway management. A point of care ultrasound may be helpful in assessment of contents in stomach [18].

ABSENT PERISTALSIS

The absence of bowel sounds at careful auscultation is a subjective sign [8], which itself does not carry the risk of severe consequences and should instead be seen as a warning sign to screen for other symptoms of GI dysfunction. The absence of peristaltic movements can be partial (obstruction, ileus) or complete. Complete lack of bowel sounds is abnormal and associated with adverse outcome as shown in several studies [1, 5]. Assessment of this sign can be included in the clinical evaluation of GI function, but no management decisions should be based solely on absence or presence of bowel sounds. Ultrasound-assessment can further help to evaluate peristalsis. However, peristalsis can be present but ineffective so that on ultrasound peristaltic movement can be seen, but propulsion of intestinal content is absent (pendel-peristalsis).

GASTROPARESIS

Outside of the ICU, gastroparesis is defined as the presence of symptoms of gastric retention with objective evidence of delayed gastric emptying in the absence of mechanical obstruction [19]. In the ICU, diagnosis of gastroparesis is usually based on large gastric residual volumes (GRV) or vomiting. A single gastric residual volume exceeding 200 mL, and a total gastric residual volume above 1,000 mL $24h^{-1}$ could be considered as being increased [8]. However, these thresholds are arbitrary. Although GRV is neither specific nor sensitive for gastric emptying, we suggest that GRV above 500 mL should be considered as a possible sign of gastroparesis, needing specific treatment and delay of enteral nutrition [20].

A recent study in mechanically ventilated patients found that not measuring GRV vs. routine GRV monitoring was not associated with increased prevalence of ventilator-associated pneumonia [21]. This study led to recommendations to abandon measuring of GRV [22, 23]. However, mentioned study was performed in mechanically ventilated, mostly non-surgical patients with already established enteral nutrition (EN), whereas GI surgery patients were excluded [21] and results can therefore not be extrapolated to the general nor to the surgical ICU population. Moreover, a significant proportion of patients experienced vomiting in this study

Table 1. Assessment and treatment of different perioperative GI symptoms and syndromes

Problem	Definition/ symptoms	Assessment	Management
Postoperative nausea and vomiting (PONV)	Signs: Nausea Vomiting Severe consequences: Aspiration	Clinical evaluation PONV occurs in up to 1/3 after general anaesthesia, ½ after craniotomy, and ¾ in patients with several risk factors	Prophylaxis for high-risk patients, treatment after (preferably non-opioid) pain control: 1) 5-HT3 antagonist (e.g. ondansetron 4 mg *i.v.* both for prophylaxis (25% reduction) or treatment 2) Dopamine receptor antagonist (e.g. metoclopramide 10 mg *i.v.* or droperidol ≤ 1 mg *i.v.*) 3) low-dose sedatives (e.g. 10 mg of propofol *i.v.*) 5) corticosteroid, mainly for prophylaxis (e.g. dexamethasone 4 mg *i.v.*) 6) NK1-antagonist for prophylaxis (aprepitant 40 mg *p.o.*)
Absent bowel sounds	No bowel sounds heard at cautious auscultation Severe consequence: none	Cautious auscultation. Bowel sounds are absent in about 40% of ICU patients, but absence may indicate bowel paralysis	No specific management. Decisions on EN should not be based on presence of bowel sounds. However, complete absence of bowel sounds is abnormal and may indicate bowel paralysis
Gastroparesis	Signs: Vomiting Gastric residual volume (GRV) > 500 mL Severe consequences: Aspiration	Clinical evaluation. In case of repeated vomiting, gastric volume should be assessed using: 1) GRV or 2) gastric ultrasound Regurgitation/vomiting occurs in 20–40% of ICU patients, GRV > 500 mL in 25%	If GRV > 500 mL $6h^{-1}$ ≥ delay oral or enteral nutrition. Exclude obstruction. Start prokinetics: erythromycin 100–250 mg × 2–3; metoclopramide 10 mg × 3 (1–2 if renal insufficiency), limit for 3 days. Minimize factors impairing gastric emptying and increasing risk of aspiration (opioids, flat head-of-bed *etc.*). Consider laxatives. If no effect within 24h, consider jejunal EN. If GRV < 500 mL $6h^{-1}$ and no other contraindications, start EN at slow rate. If GRV persistently >200 mL $6h^{-1}$ consider prokinetics
Bowel paralysis	No passage for > 3 days Severe consequence: bowel distension	Clinical evaluation	If the bowel is minimally or not distended, GRV < 500 mL $6h^{-1}$ and IAP < 20 mm Hg ≥ start EN 10 mL h^{-1} via gastric tube. Correct electrolyte levels. Increase EN cautiously (e.g. by 10 mL h^{-1} in 12–24h). Add prokinetics as above if GRV is persistently > 200 mL $6h^{-1}$
Bowel dilatation	Small bowel diameter > 3 cm, large bowel > 6 cm, caecum > 9 cm Severe consequence: bowel distension	Assessment of GI symptoms, no specific diagnostics unless symptomatic	Consider/exclude obstruction. Correct electrolyte levels. Consider nasogastric suctioning and imaging if symptomatic. Consider laxation if increased large bowel diameter without obstruction. Monitor GI symptoms
Bowel distension	Abdominal distension, commonly elevated IAP (> 12 mm Hg) and abnormal bowel sounds Severe consequences: Ogilvie's syndrome, ACS, bowel perforation, abdominal sepsis	Imaging indicated if prominent abdominal distension and/or increasing IAP	Nasogastric suctioning and imaging in case of persistent small bowel distention. Consult the surgeon. Neostigmine i/v infusion 0.4–0.8 mg h^{-1} (dose up to 2 mg) if caecum diameter reaches 8–10 cm Consider endoscopic decompression if not resolved after 48h. Rarely, if bowel distension leads to ACS or perforation, surgery becomes necessary
Ogilvie's syndrome	Severe colonic distension, tenderness over caecum and right colon Severe consequences: ACS, bowel perforation	Plain abdominal x-ray is usually sufficient to confirm the diagnosis and estimate the caecal diameter	See 'bowel distension'
Bowel edema	Small bowel wall > 3 mm, colonic wall > 2 mm if distended and > 5 mm if contracted	Visual assessment during surgery, imaging usually not indicated	No specific management. Avoidance of fluid overload, aiming negative fluid balance as soon as safe and tolerated. Cautious initiation of EN
Diarrhoea	≥ 3 liquid stools in total > 250g day^{-1} Severe consequences: Hypovolemia, hypokalemia, hypomagnesemia, malabsorption	Early postoperative diarrhoea is rare ≥ consider bowel ischemia (often blood-stained) Exclusion of Clostridium difficile colitis Differential diagnosis of non-infectious diarrhoea, if infectious excluded Diarrhoea occurs in ~20% of ICU patients	Try to identify the cause. Stop laxatives. Continue EN Specific management for Clostridium difficile colitis including systemic metronidazole and enteral vancomycin Substitute pancreatic enzymes or give hydrolyzed EN for pancreatic exocrine insufficiency Consider cholestyramin for bile acid malabsorption
Intra-abdominal hypertension	Intra-abdominal hypertension (IAH) = intra-abdominal pressure (IAP) ≥ 12 mm Hg Abdominal compartment syndrome (ACS) = IAP > 20 mm Hg along with a new or worsening organ failure	Regular measurements of IAP IAH occurs in 30–40% of ICU patients. ACS is rare	Avoid fluid overload, aim at negative fluid balance as soon as safe and tolerated. Provide adequate analgesia. If IAP increases and the risk of ACS is high, consider gastric decompression through opening/suctioning the gastric tube, colonoscopic decompression if bowel is distended, drainage of intra-abdominal/retroperitoneal fluid collections if present, and deep sedation. If ACS, consider deep sedation, temporary neuromuscular blockade and surgical decompression

ACS — abdominal compartment syndrome; EN — enteral nutrition; GRV — gastric residual volume; IAH — intra-abdominal hypertension; IAP — intra-abdominal pressure; PONV — postoperative nausea and vomiting

(42 vs. 27% in no-GRV vs. GRV-group, $P = 0.002$) [21], hence carrying the potential risk of aspiration (Table 2). Therefore, current evidence is insufficient to abandon GRV measurements in all ICU patients unless routine gastric ultrasound is used instead to monitor gastric filling.

Avoidance of stomach distension is especially vital after upper GI surgery and in spontaneously breathing patients with impaired protective reflexes against aspiration.

The introduction of a nasogastric tube should be seriously considered in patients receiving non-invasive ventilation (NIV) in the early postoperative period, and pros and cons should be weighed for the individual patient. The opening of the tube allows gastric emptying during NIV. However, the tube in place may facilitate regurgitation due to the opening of oesophageal sphincters and impair tightening of the mask.

Importantly, each patient receiving sedatives and opiate analgesics is exposed to increased risk of aspiration. Factors associated with aspiration are presented in Table 2.

We suggest that if a gastric tube is in place, GRV measurements should be performed at least in the early postoperative phase when full EN is not yet established. If GRV is > 500 mL $6h^{-1}$ [20], EN should be delayed, and measures should be considered to promote gastric emptying (see Table 1). Severe consequences of gastroparesis are the aspiration of gastric contents and very seldom, abdominal compartment syndrome due to gastric overdistension [24]. These effects need to be avoided by minimizing the risks and assuring adequate monitoring and management.

If gastroparesis is suspected preoperatively, emptying of the stomach with nasogastric tube should be considered before induction of anaesthesia and all precautions taken to avoid aspiration during induction of anaesthesia. The presence of a nasogastric tube during induction and intubation may, on the one hand, allow evacuation of gastric fluid contents, but may, on the other hand, facilitate regurgitation due to an open lower oesophageal sphincter.

Table 2. Factors associated with increased risk of aspiration

Documented previous aspiration
Decreased level of consciousness (including due to sedation)
Impaired swallowing and coughing reflex:
After prolonged intubation
Neuromuscular disease
Advanced age
Gastroesophageal reflux disease
Delayed gastric emptying, paralytic/obstruction ileus
Persistently high gastric residual volumes
Vomiting
Presence of naso-enteric tube
Abdominal or thoracic surgery or trauma
Any intervention/manipulation on upper airways
Upper airway or oesophageal surgery
Tracheostomy
Supine positioning (flat head-of-bed)
Transportation

BOWEL PARALYSIS

Postoperative bowel paralysis *seu* postoperative ileus is a GI motility disorder characterized by nausea, vomiting, abdominal distension and/or delayed passage of stool after surgery which may be either self-limiting or have severe consequences such as bowel distension [25]. Main mechanisms include agonist action at gut opioid receptors, modulation of gut hormone activity, inflammation, electrolyte balance and autonomic dysfunction [25] accompanied by gut hypoperfusion and edema (see below). 'Simple' postoperative bowel paralysis can be diagnosed only after exclusion of mechanical obstruction and acute severe abdominal/GI pathology, which primarily need intervention. 'Postoperative ileus' occurs relatively often after abdominal surgery, especially in presence of peritonitis and sepsis or shock. Bowel paralysis may also be a sign of new/unresolved acute pathology in the abdomen (e.g. peritonitis, ischemia), which should always be suspected, assessed and treated immediately.

Fluid management is difficult in these patients because both hypoperfusion (due to hypovolemia or vasoconstriction) and large volume fluid resuscitation may lead to paralytic ileus due to bowel edema [26]. Hypovolemia will hamper perfusion impeding oxygen transport to the tissues while hypervolemia with interstitial and bowel edema will as well impair oxygen diffusion, both mechanisms resulting in tissue hypoxia at the top of the intestinal villi.

Postoperative ileus can affect both small and large bowel but is more often limited to large intestine, sometimes called as critical illness-related colonic ileus [27].

In ICU patients, a state without passage of stools can persist for many days without necessarily leading to marked colonic distension [28, 29]. However, avoidance of bowel distension is essential, and therefore colonic ileus requires a pro-active strategy including administration of early oral laxatives (polyethylene glycol or lactulose) [29] and enemas. In case of abdominal distension as a result of bowel paralysis, administration of neostigmine as a slow infusion should be considered [27] after mechanical obstruction has been excluded. With such proactive strategy, endoscopic or surgical decompression is only needed in the most severe cases not responding to conservative treatment. Colonic ileus is no contraindication to EN, unless peritoneal signs or severe intra-abdominal hypertension is present.

A severe consequence of bowel paralysis is bowel distension.

BOWEL DILATATION AND DISTENSION

Bowel dilatation is defined as bowel diameter above 3 cm for the small bowel, above 6 cm for the colon and 9 cm for the caecum [8]. The terms dilatation and distension are often used interchangeably. However, dilatation refers to solely an enlargement (with an increase of the diameter), not necessarily leading to any symptoms. Bowel distension, in contrast, relates to expansion through increased intraluminal pressure, which manifests in clinical signs such as bloating or pain. Pain can be severe and associated with vasovagal reactions. Distension may be complicated by bacterial translocation and subsequent systemic inflammation. The most serious consequence is bowel perforation. Distension can occur proximal to mechanical obstruction, but also without obstruction, caused by impaired bowel motility and excessive gas production by gut microflora. Bowel dilatation/distension can be diagnosed by plain x-ray or CT scan, or during abdominal surgery. Dynamic evaluation is commonly limited to bedside assessment of abdominal pain and distension and patient's general condition. A typical finding is hyper tympanic percussion. Imaging should immediately be undertaken if symptoms are severe and risk of perforation is considered high. Importantly, the thin intestinal wall as a result of severe or prolonged bowel distension may significantly hinder the safe creation of bowel anastomosis in the acute setting. Specific details of the operation with potential postoperative consequences need to be communicated to the intensivist.

Severely isolated distension of the colon is seen as a specific entity, called colonic pseudo-obstruction or Ogilvie's syndrome. This syndrome manifests in gross abdominal distension and tenderness to severe abdominal pain. Distension is commonly most pronounced in the caecum. The absence of bowel sounds is not always noticed and gastroparesis is not necessarily present. The risk of perforation increases exponentially if caecal diameter exceeds 12 cm [30].

If severe bowel distension is present preoperatively, both intraluminal and intra-abdominal pressure are probably increased, leading to increased risk of aspiration. Additionally, intra-abdominal hypertension leads to increased risk of atelectasis, shortening the time for a safe apnoea period during induction.

BOWEL EDEMA

Bowel edema is not uniformly defined, but some agreement exists that small bowel wall thickness above 3 mm, and colonic wall thickness above 2 mm if distended and 5 mm if contracted are indicative for edema [31]. In critically ill patients, underlying mechanisms include: fluid extravasation due to capillary leak; decreased lymph flow due to impaired GI motility, and increased intra-abdominal and/or intra-thoracic pressures; intra-abdominal hypertension (IAH) leading to mesenteric venous compression and venous congestion, venous hypertension, and splanchnic hypoperfusion; and right heart failure.

Bowel edema as a consequence of fluid overload increases the risk of anastomotic leakage [32]. Both fluid overload and hypovolemia should be avoided. Speculatively, a too conservative fluid strategy in initial phase may lead to more bowel edema, because capillary leak increases during prolonged hypovolemia and more fluids will be needed to achieve euvolemia. Bowel edema, especially with concomitant hypoperfusion later on, may contribute to cellular hypoxia in the intestinal wall, and may be associated with malabsorption.

DIARRHOEA

Diarrhoea is defined as the occurrence of liquid stools 3 or more times per day with stool weight equal or above 250 g day^{-1} [33].

Diarrhoea is not frequent in the early postoperative phase unless gut ischemia is present, but may become a problem during prolonged ICU stay. Differential diagnosis with respective management is essential but is covered elsewhere [33]. Importantly, patients with severe diarrhoea may lose substantial amounts of fluid, electrolytes and trace elements (see also 1.2.5) and malnourishment may occur due to maldigestion or malabsorption of nutrients. Monitoring and supplementation of these losses may be challenging and sometimes require a prolonged stay in the ICU. Importantly, diarrhoea may be a sign of severe colitis (e.g. *Clostridium difficile* or chemotherapy-induced), which may result in toxic megacolon or abdominal compartment syndrome (ACS) [34].

INTRA-ABDOMINAL HYPERTENSION

Definitions, risk factors and management options regarding intra-abdominal hypertension (IAH) are summarized in Table 3.

Prevalence of IAH in critically ill patients is high, reaching 30–40%, whereas most of the cases experience mild IAH (intra-abdominal pressure (IAP 12–15 mm Hg) [36, 39, 40]. Increased risk of IAH has been demonstrated in conditions where abdominal wall compliance is decreased [41, 42], abdominal or intraluminal content is increased, or after massive fluid resuscitation. IAH may hamper organ perfusion and thereby directly contribute to organ dysfunction. In addition, IAH may reduce preload and increase afterload to the heart and cause hemodynamic instability, atelectasis and cause translocation of bacterial products from the gut, which may lead to systemic inflammation. Many of risk factors (laparotomy, hemoretroperitoneum, etc) are related to surgery, which underlines the importance of IAP measurements in this population. IAP should be considered as a valuable information dynamically reflecting processes in

Table 3. Definitions, risk factors and management of intra-abdominal hypertension

Definitions on intra-abdominal hypertension in adults [35]	
Intra-abdominal hypertension (IAH)	Sustained intra-abdominal pressure (IAP) ≥ 12 mm Hg
IAH grade I	IAP 12–15 mm Hg
IAP grade II	IAP 16–20 mm Hg
IAP grade III	IAP 21–25 mm Hg
IAP grade IV	IAP > 25 mm Hg
Abdominal compartment syndrome (ACS)	IAP > 20 mm Hg along with new or worsening organ failure
Risk factors of IAH (identified as independent risk factors in studies) [36–38]	
Demographic	Obesity [36–38]
Diagnosis category/syndrome/symptom	Abdominal surgery [37, 38] Sepsis [37] Pancreatitis [38] Hepatic failure/cirrhosis with ascites [38] Gastrointestinal bleeding [38] Respiratory failure (PaO_2/FiO_2 < 300) [38] Ileus [37] Abdominal distension [36] Hemoperitoneum/pneumoperitoneum/intra-peritoneal fluid collection [36]
Disease severity/respectively applied treatment	Abbreviated Sequential Organ Failure Assessment score > 4 points [36] Lactate > 1.4 mmol L^{-1} [36] High positive end-expiratory pressure (PEEP >10) [38] Use of vasopressors/inotropes [38]
Management suggestions [35]	
To avoid development or progression of IAH	— Avoid fluid overload — Aim negative fluid balance as soon as safe and tolerated — Provide adequate analgesia
If IAH is present and IAP increasing and/or the risk of ACS high	Consider the following: — Gastric decompression through opening/suctioning the gastric tube — Prokinetics and rectal enemas — Colonoscopy with bowel decompression in case of colonic distension — Drainage of intra-abdominal or retroperitoneal fluid collections if present — Deepening of sedation
If ACS is present	— Deep sedation — Temporary neuromuscular blockade — Surgical decompression

the abdominal compartment. Therefore, we suggest using IAP monitoring in all patients after complicated abdominal surgery, whereas the role of decreased abdominal compliance should be recognized.

Abdominal compartment syndrome is a severe consequence of increasing IAP, resulting in an immediately life-threatening condition where prompt measures to reduce IAP are needed, including the immediate opening of the nasogastric tube.

Reduction of enteral nutrition (EN) can be considered in case of IAH if IAP levels are increasing under EN, whereas EN should not be given to patients with ACS [20].

The presence of IAH preoperatively is associated with increased risk of aspiration and atelectasis formation, but also hemodynamic instability (due to preload reduction under high ventilatory pressures) during induction of anaesthesia.

1.2. SPECIFIC SURGERY-RELATED PROBLEMS

DIRECT INJURY TO GI TRACT

Major abdominal surgery or direct injury to the GI tract may be associated with all previously described abdominal problems postoperatively. Bowel resection and re-anastomosis leads to disturbed motility also due to changes in entero-enteral signalling. A detailed description of the aspect of the intestines and peritoneal cavity during surgery needs to be communicated to the intensivist along with details on surgical intervention.

INTRA-ABDOMINAL OR RETROPERITONEAL BLEEDING

Nowadays, intra-abdominal or retroperitoneal bleeding is increasingly managed conservatively. This approach requires careful monitoring.

Next to monitoring and optimization of haemoglobin levels and coagulation parameters, monitoring of IAP and regular assessment via ultrasound are essential.

Pre-emptive planning of interventions in case of continuing or recurrent bleeding (angiography, operation) is useful to limit the delay if interventions are indicated.

Intra-abdominal or retroperitoneal bleeding is often accompanied by intestinal hypomotility, especially if blood is not removed, and bowel paralysis usually occurs.

Severe consequences of intra-abdominal bleeding are hemorrhagic shock and ACS.

INTRA-ABDOMINAL INFECTION/ANASTOMOTIC LEAKAGE

Ongoing or unresolved intra-abdominal infection causes peritonitis pain, all previously addressed non-specific symptoms and syndromes, and signs of systemic inflammation. If unrecognized, septic shock may develop. Therefore, if abdominal symptoms or signs are persisting and the general condition is not improving after abdominal surgery, prompt diagnostics and management should be initiated.

BOWEL ISCHEMIA

A definite diagnosis of bowel ischemia can only be made visually during operation or endoscopy. However, there is a wide grey zone between normally perfused and necrotic bowel. Several signs in CT assist to diagnosis, but may sometimes be misleading, e.g. intestinal pneumatosis is by far not always associated with bowel wall necrosis [43].

If the bowel is not necrotic, but ongoing bowel ischemia is suspected during initial laparotomy, second look surgery is usually scheduled whilst leaving the abdomen open in between.

Adequacy of bowel perfusion is challenging to estimate if (re)operation is not indicated/performed. Signs associated with bowel hypoperfusion are non-specific and include clinical deterioration with tense abdomen, increasing lactate and hemodynamic instability.

If ischemic lesions of bowel mucosa are observed during endoscopy, but clinical signs of transmural ischemia are absent, the condition can usually be carefully observed. Trophic feeding could be considered in these cases and possibly even be protective as it may stimulate bowel perfusion and help to restore atrophic mucosa.

A specific condition associated with colonic ischemia is abdominal aortic surgery, where ischemia, usually of the left hemicolon, occurs in 2% after elective abdominal aortic surgery and in 10% after rupture of an aneurysm [44]. Details of the surgical procedure (e.g. level of clamping) and observations during surgery (e.g. large bowel viability) need to reach intensivist to assist in the further interpretation of clinical signs.

HIGH OUTPUT STOMA OR FISTULA

A stoma with output > 2,000 mL day^{-1} or > 1500 mL for two consecutive days is considered a high output stoma [45, 46]. However, also smaller amounts may put the patient at risk of complications. A fistula output > 500 mL is considered high [47]. Losses of enteral secretions may lead to hypovolemia and electrolyte disturbances, and malnutrition may occur in case of malabsorption or short bowel.

If an access distal to the stoma or fistula presenting with high output is available, the collection of the stoma/fistula output and reinfusion into the distal part of the GI tract needs to be considered. Respective methods are described in detail elsewhere [48].

SHORT BOWEL SYNDROME AND ACUTE INTESTINAL FAILURE

Short bowel syndrome is defined as malabsorption caused by a lack of functional small intestine, often occurring when the functional bowel length is below 200 cm [49]. Immediately postoperatively it is usually difficult to predict whether the length of bowel is sufficient for absorption in a longer perspective. The term, acute intestinal failure' is suggested to describe acute situations with reduced functional gut mass, which necessitates intravenous supplementation of fluids and nutrients [50].

From the intensivist's perspective, it is essential to understand the 'detailed' situation after surgery (which parts of the bowel can be expected being functional). Knowledge on the physiology of GI secretions, absorption of nutrients and fluids is required to identify expected maldigestion and malabsorption and to assure respective early supplementation.

Importantly, secretion of digestive enzymes is usually adequate in patients with short bowel syndrome. Therefore (semi)elemental enteral feeding is not necessary.

LIVER SURGERY AND DYSFUNCTION

Liver and biliary surgery includes surgical procedures of a wide range of difficulty form cholecystectomy to liver transplantation. Bleeding or septic complications may occur after all of them, but special attention in perioperative management is required towards: liver dysfunction or failure due to insufficient vital remnant liver tissue or transplant dysfunction; liver hypoperfusion e.g. due to ligation of vessels, packing, but also prolonged global hypoperfusion; inadequate bile delivery into the gut due to e.g. external drainage or internal leakage; bile duct obstruction; and portal vein thrombosis. Typical surgical complications are beyond of the scope of this review and are covered elsewhere [51, 52].

Ischemic hepatitis is characterized by an acute increase of transaminases, which can be seen after transient liver ischemia during surgery or shock. If the residual liver tissue is insufficient, functional impairment develops. Liver enzymes

should be measured through the first postoperative days, whereas hallmarks of postoperative assessment of liver function are monitoring of lactate, coagulation, bilirubin, and ammonia.

Importantly, if hyperammonemia occurs postoperatively, bacterial overgrowth associated with increased production of ammonia in the gut needs to be considered. Management of hyperammonemia includes administration of lactulose, whereas studies on rifaximin are lacking in acute setting [53]. A severe consequence of hyperammonemia is hepatic encephalopathy and eventually coma, although there is no direct relation between the ammonia levels and the depth of hepatic encephalopathy [54, 55].

In case of inadequate bile delivery into the gut fat maldigestion and malabsorption of fat-soluble vitamins should be suspected, assessed and managed. In case of complete external bile drainage options to reinfuse the bile enterally need to be considered.

The dose of drugs cleared by the liver, such as paracetamol (acetaminophen), should be reduced after liver resection and in patients with liver dysfunction to avoid intoxication.

PANCREATIC SURGERY, FISTULA AND EXOCRINE INSUFFICIENCY

Pancreatic insufficiency may occur after pancreatic resection, but also critically ill patients without structural pancreatic pathology may present with exocrine pancreatic insufficiency [56]. Application of hydrolyzed enteral feeding formula or enteral supplementation of pancreatic enzymes should be started if such insufficiency is suspected (e.g. unexplained diarrhoea, increased fecal elastase-1 levels).

A postoperative pancreatic fistula represents a failure of healing/sealing of a pancreatic-enteric anastomosis or a parenchymal leak not directly related to an anastomosis with a drain output of any measurable volume of fluid ≥ 3 days postoperatively with an amylase content greater than 3 times the serum amylase activity [57]. However, the exact value and importance of the drain amylase have been questioned recently.

Management of pancreatic fistula includes administration of octreotide and sometimes discontinuation of enteral nutrition for a limited duration to minimize pancreatic secretion. Severe consequences of the extraluminal, intra-abdominal or retroperitoneal presence of pancreatic secretions are tissue necrosis, erosive bleeding, and bowel perforation.

Acute life-threatening bleeding is a feared complication in case of persistent internal fistula with infection after complicated biliopancreatic surgery. Emergency coiling is the treatment of choice.

In rare occasions, severe hypoperfusion may lead to ischemic pancreatitis, similarly to ischemic hepatitis.

2. IDENTIFICATION OF PATIENTS AT RISK FOR POSTOPERATIVE GI PROBLEMS

To identify a patient at risk for postoperative GI problems is difficult. In contrast with other organ systems, GI problems are heterogeneous and relatively rare among the surgical population not limited to ICU. Common risk assessment models are lacking. For detection of patients at risk both patient-related factors and type of surgery have to be considered.

2.1. PATIENT-RELATED FACTORS

The validated models for high risk surgical patients, such as Physiological and Operative Severity Score for the Enumeration of Mortality and Morbidity (POSSUM score), or American College of Surgeons National Surgical Quality Improvement Program (ASC-NSQUIP), are useful to assess the likelihood of many unfavourable outcomes, including pneumonia and other infectious complications, cardiac problems, renal failure, prolonged length of stay and death [58]. These models, however, have only limited value in prognostication of GI problems specifically.

For risk assessment of postoperative GI problems the following can be outlined:

POSTOPERATIVE NAUSEA AND VOMITING (PONV)

PONV is frequent in non-smoking females. Apfel score helps to identify the patients at risk [13].

GASTROPARESIS AND BOWEL PARALYSIS

The co-morbidities known to be associated with delayed gastric emptying and ileus are diabetes mellitus, trauma, chronic renal failure with uraemia, systemic sclerosis, dermatomyositis, post-vagotomy state. The temporary influence of opioids, beta-adrenergic blockers, alfa-2-adrenergic agonists, hyperglycaemia, low potassium and magnesium, metabolic and respiratory acidosis, are also known to increase the risk for GI motility disorders [59].

GI BLEEDING

Patients taking anticoagulants or/and antiplatelet drugs are at higher risk of gastrointestinal bleeding [60]. The risk of GI bleeding associated with routine postoperative thromboprophylaxis is low and does not overweigh the benefits of anticoagulants in this setting [61].

ANASTOMOTIC LEAK

Patients with poor cardiovascular function and chronic hypoxia, as well as patients with active peritonitis and those on chemotherapy are at increased risk of anastomotic leakage.

2.2. TYPE OF SURGERY

The risk of GI problems is inherently associated with the type of surgery.

ABDOMINAL SURGERY

GI problems are more common and naturally linked to abdominal surgery. Upper GI surgery (oesophageal, gastric) carries the risk of regurgitation and aspiration because of absence or malfunctioning of oesophageal sphincters [62]. Therefore, particular attention should be paid to the positioning of the patient in the immediate postoperative period where the head of bed elevated at least 45° is recommended. Postoperative videofluoroscopy or bedside swallowing test may be helpful in the further assessment of aspiration risk.

In colorectal surgery, the anastomotic leak is one of the most frequent and severe postoperative complications, developing in about 3% of patients. Independent predictors of the colonic anastomotic leak are male sex, intraoperative transfusion and prolonged operative time [63]. Knowing these risk factors might influence the surgical tactics at the end of a procedure, for instance by the formation of a diverting stoma for protection of the anastomosis.

Abdominal vascular surgery is associated with risk of ischaemic colitis. Independent risk factors after abdominal aneurysm repair are length of operation, aneurysm rupture, and renal insufficiency [44]. The importance of hypotension, anatomical details, and retractor trauma has also been stressed indicating the crucial role of good communication with operating surgeon in early recognition of patients at risk of ischaemic colitis.

NON-ABDOMINAL SURGERY

In non-abdominal surgery, the GI problems are infrequent. Nevertheless, their impact is considerable.

After cardiac surgery, GI complications occur only in 0.3% to 3% but are associated with significant morbidity and up to 20-fold increase in mortality [7]. Independent predictors of GI complications are age over 80 years, smoking, inotropic support, NYHA class III–IV, cardiopulmonary bypass time over 150 min, postoperative atrial fibrillation or heart failure, reoperation due to bleeding, and postoperative vascular complication [64, 65]. This list of risk factors indicates that non-occlusive mesenteric ischemia, probably caused by hypoperfusion due to low flow during extracorporeal circulation or low cardiac output, hypotension due to blood loss, and intra-abdominal atheroemboli, has a central role in this subgroup of patients.

3. PERIOPERATIVE MANAGEMENT OF GI SYSTEM

For structured perioperative management, a stepwise approach starting from preoperative risk assessment is recommended (Table 4).

Table 4. Perioperative gastrointestinal management

Preoperative measures
Evaluation and minimization of aspiration risk
Evaluation of the risk of PONV and consideration of prophylaxis
Evaluation of the need and safety of postoperative epidural analgesia
Intraoperative measures
Avoidance of severe hypovolemia and fluid overload
Consideration of intraoperative measurement of intra-abdominal pressure
Planning of postoperative nutrition
Assessment of risk of aspiration in immediate postoperative period
Postoperative measures
Good communication between surgeon-anaesthetist-intensivist
Stabilisation of systemic perfusion, aiming euvolemia and adequate perfusion pressure
Correction of electrolyte and glucose levels
Optimisation of analgesia and sedation considering impact on GI motility
Planning for nutrition, considering the need for nasogastric tube
Planning for monitoring, considering GRV and IAP measurements
Recognition and management of GI problems occurring postoperatively (see Table 1)

3.1. PREOPERATIVE MEASURES

Careful assessment of risks for aspiration and PONV, as well as appropriate prophylaxis belongs to the standards of good anaesthesia care. Epidural analgesia helps to reduce the need for systemic opioids and promotes postoperative recurrence of GI motility. Benefits need to be weighed against the risks of the procedure and delay in surgery.

3.2. INTRAOPERATIVE MEASURES

Intraoperative measures directed specifically to avoidance of postoperative GI problems are limited. The most important aspect is a good communication between the surgeon and anaesthetist throughout the procedure. Anaesthetist has to be aware of specific details of undertaken procedure which may need particular attention in intra- and postoperative period.

Avoidance of severe hypovolemia and fluid overload are equally important. Careful fluid management is required targeted according to physiological and pathological fluid shifts during the operation and baseline cardiac status.

Planning of postoperative nutrition. In some cases, especially in GI surgery, enteral feeding access should be established or directly checked by the surgeon in operation field.

Consideration of intraoperative measurement of IAP. In patients at risk for abdominal compartment syndrome (e.g. ruptured abdominal aortic aneurysm, abdominal sepsis or trauma with continuing need for large fluid resuscitation) intraoperative (during abdominal closure) measurement of IAP should be considered and may help to guide immediate or delayed abdominal closure. Rele-

vant increase in respiratory pressures during abdominal closure needs to be communicated to the surgeon and should trigger measurement of IAP in the operating room.

Assessment of risk of aspiration in immediate postoperative period. A very high risk of aspiration in immediate postoperative period may justify transfer to the ICU with delayed extubation.

3.3. POSTOPERATIVE MEASURES

Communication between the surgeon, anaesthesiologist, and intensivist. Detailed and well-structured handover from operating theatre to ICU is a cornerstone for uncomplicated postoperative care. Good communication is therefore of crucial importance. Details of surgery and possible forthcoming problems need to be discussed. A clear plan regarding the timing and indications for further operations needs to be established, e.g. in damage control surgery. On the other hand, intensivists need to consult the surgeon in a timely manner in case of changing symptoms and signs, and deterioration of the clinical status.

In case of abdominal surgery, the following aspects are important to communicate:

— The exact description of surgical procedure (e.g. location of bowel resections and estimated length of the remaining bowel, blood supply of viscera after abdominal aortic or trauma surgery), including a sketch of current status (incl. drainages) by the surgeon if needed;
— Essential aspects of the surgical field: bowel distention, oedema, viability, suspicion of impaired perfusion (global or regional);
— (Possible) contamination of peritoneal cavity;
— Exact location of anastomoses and stoma(s), the quality of anastomoses (e.g. performed on distended and oedematous bowel, presence of distal or proximal dilatation);
— Risk of (continuing) intraabdominal bleeding;
— Closure of abdomen (e.g. fascial closure, mesh-mediated), presence of tension at closure;
— Drains. The location has to be clarified and marked, defining whether passive or active drainage should be applied, and the contents immediately observed.
— Possible forthcoming GI problems;
— The estimated time to start oral diet/enteral feeding.

In case of any surgery, the following specific aspects related to GI tract need to be clarified:

— Estimated duration of withholding oral intake;
— Risks for introduction of nasogastric tube;
— Specific risks related to surgery. E.g. in neurosurgical patients, estimated risk of development of brain edema needs to be discussed, requiring particular attention when distinguishing from severe PONV.

Further, the following elements of postoperative care should be considered:

Stabilisation of systemic perfusion and aiming euvolemia. Adequate perfusion and oxygenation are crucial to minimize secondary organ damage, including GI system.

Correction of electrolyte and glucose levels. Normal electrolyte and glucose levels are needed for maintenance of homeostasis, but also important for restoration of bowel motility. Although not proven in studies, high-normal levels of potassium and magnesium may appear beneficial for bowel motility in case of paralysis.

Analgesia and sedation. The benefits of opioids and alpha-agonists in pain therapy should be weighed against the risk of decreased GI motility.

Planning of nutrition, together with surgeon, according to nutritional hierarchy (see below).

GI monitoring. Regularly documented clinical assessment should be complemented with instrumental monitoring depending on the severity and nature of the case. If gastric tube is placed, assessment of GRVs is recommended. Assessment of character and measurement of drain, stoma, and/or fistula volumes should be performed daily. In the presence of risk factors of IAH measurements of IAP should be commenced immediately. Blood lactate values should be interpreted in context of possible global and/or splanchnic hypoperfusion.

Management of GI problems. Appearing GI symptoms and syndromes need to be interpreted with caution and specific diagnostics and therapies applied, if required (Table 1).

4. POSTOPERATIVE NUTRITION

For postoperative nutrition we suggest always to consider the following hierarchy of routes: 1) oral diet, 2) gastric feeding, 3) jejunal feeding, 4) parenteral nutrition (PN). In many patients, oral diet can be commenced in few days after surgery and artificial nutrition is not required. If this is not the case, first preference goes to gastric feeding. Importantly, enteral feeding should always be started in a low dose under monitoring of GI symptoms and increased only gradually [20]. If oral or enteral feeding is insufficient or not possible, (supplemental) PN should usually be initiated after four to seven days [66], sometimes earlier, based on the presence and duration of underfeeding before ICU admission and on expected recovery time. Aggressive feeding via any route in the early phase may be harmful and should be avoided [67, 68]. Mechanism of harm may be the risk of overfeeding due to inflammation-related endogenous energy production and refeeding with associated worse outcome [69].

CONCLUSIONS

The risk-assessment and interdisciplinary approach should allow minimization of perioperative GI problems.

Careful clinical assessment of presence and dynamics of GI symptoms, adequate usage of additive tools (such as measurement of gastric residual volumes and intra-abdominal pressure) should allow timely detection of problems. Structured communication and a management plan should help to avoid severe consequences of GI dysfunction in the postoperative setting.

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