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Abdominal signs and symptoms in intensive care patients

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Abstract

Abdominal problems, both as a primary reason for admission or developing as a part of multiple organ dysfunction syndrome during an ICU stay, are common in critically ill patients. The definitions, assessment, incidence and outcome of different abdominal signs, symptoms and syndromes are assessed in the current review. General abdominal signs and symptoms include abdominal pain and distension, as well as other signs assessed during the physical examination (e.g. palpation, percussion). Gastrointestinal (GI) symptoms include vomiting, high gastric residual volumes, diarrhoea, GI bleeding, paralysis of the lower GI tract, bowel dilatation and absent bowel sounds. Although around half of patients suffer from these symptoms, the reported incidences of single symptoms vary within a large range due to variable definitions and case-mix. In a few studies, the total number of coincident GI symptoms was associated with increased mortality. Although acute abdomen is a well-recognized severe syndrome in emergency medicine, its incidence in ICUs is not known. Next to subjective clinical evaluation, intra-abdominal pressure, as a reproducible numerical variable, provides useful assistance in the assessment of the abdominal compartment, whereas intra-abdominal hypertension has been shown to impair the outcome of the critically ill. In conclusion, abdominal symptoms occur in half of patients in ICUs. Clinical evaluation, albeit largely subjective, remains the main bedside tool to detect abdominal problems and to assess GI function in the critically ill. IAP is a useful additional tool in the assessment of abdominal complications in ICUs.

Key words: abdominal signs, abdominal symptoms, abdominal syndromes, abdominal hypertension, vomiting, diarrhoea, bleeding, paralysis, ileus, bowel dilatation, bowel sounds, gastric residual volume

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Abdominal problems are common in critically ill patient. Although they can be the initial condition requiring admission to the intensive care unit (ICU), most often they are a reflection of the severity of the underlying disease. The interactions of a critical illness with abdominal contents are complex and can present themselves in different ways. In order to perform the correct treatment, a complete initial clinical and diagnostic work up for gastro-intestinal (GI) problems, with early assessment of disease severity (looking at aetiological factors and predisposing conditions) is necessary. This should be followed by appropriate monitoring, such as intra-abdominal pressure (IAP) measurements, in order to anticipate complications and to avoid remote organ failure. This global approach should form the basis of patient management.

Within this respect, abdominal signs are an important diagnostic tool to trigger further investigations in emergency-room patients. In ICU patients they are often masked or difficult to assess in the critically ill. Although most of the GI symptoms are not specific, they have been associated

with impaired ICU outcomes [1, 2]. The current review was undertaken to clarify the assessment, incidence and impact of different abdominal signs and symptoms in intensive care patients.

METHODS

A MEDLINE and PubMed search was performed using the search terms 'abdominal', 'gastrointestinal' and 'symptoms'. This search yielded many references, most of which were not relevant to the subject of this paper. The abstracts were screened and selected on the basis of relevance, methodology and number of cases. Full text articles of the selected abstracts were used to supplement the authors' expert opinion and experience. The references were also checked to identify other relevant interesting papers.

RESULTS AND DISCUSSION

There is no clear nomenclature for abdominal signs, symptoms and syndromes. Different terms for diagnoses, symptoms and syndromes are sometimes used interchangeably. Many definitions are not applicable in critically ill patients, because they include the patient's subjective description (e.g. constipation). Definitions for different abdominal symptoms and syndromes specifically in ICU patients were recently proposed by the Working Group on Abdominal Problems (WGAP) of the European Society of Intensive Care Medicine (ESICM) [3].

Various abdominal pathologies are often a primary reason for admission to an ICU (Table 1). On the other hand, abdominal problems may develop as a part of multiple organ dysfunction syndrome during an ICU stay (Table 2). These problems may lead to a certain diagnosis (e.g. Ogilvie's syndrome, bowel ischemia, cholecystitis, peritonitis) or should be assessed as acute gastro-intestinal injury (AGI) [3].

GENERAL ABDOMINAL SIGNS AND SYMPTOMS
ABDOMINAL PAIN

The type, location, severity, chronology and duration of pain give important clues as to the source of the abdominal problem [4]. Diffuse abdominal pain and tenderness is seen in most patients with acute abdominal problems although it can be subtle and masked in the elderly, and by the use of corticosteroids [5]. Abdominal pain is a key symptom of peritonitis. However, as the correct assessment of abdominal pain is not possible in the unconscious or sedated, mechanically ventilated patients, it may be therefore very difficult to reach the diagnosis of peritonitis in such patients. Visceral pain is often poorly localized because of its dull and aching character. It arises from distension or spasm of a hollow organ (e.g. obstruction or cholecystitis). Parietal pain is very well localized and sharp. It arises from peritoneal irritation (e.g. acute appendicitis). Referred pain is perceived to be

Table 1. Overview of acute abdominal diagnoses that may require ICU admission (listed in order of frequency per organ)

Organ	Complications
Esophagus	Boerhaave syndrome
	Malignancy related perforation
	Iatrogenic lesion
	Penetrating trauma
Stomach	Peptic ulcer perforation
	Penetrating trauma
	Malignancy related perforation
	Iatrogenic lesion
Duodenum	Peptic ulcer perforation
	Blunt or penetrating trauma
	Iatrogenic lesion
Small intestines	Ischemic bowel
	Incarcerated hernia
	Penetrating trauma
	Inflammatory bowel disease
	Malignancy related perforation
	Meckel diverticulum
	Iatrogenic lesion
Colon	Appendicitis
	Diverticulitis
	Ischemic bowel
	Inflammatory bowel disease
	Malignancy related perforation
	Blunt and penetrating trauma
	Iatrogenic lesion
	Volvulus
Liver	Blunt and penetrating trauma
	Acute Liver Failure
	Acute toxic or ischemic hepatitis
	Iatrogenic lesion
Gallbladder	Cholecystitis
	Malignancy
	Gallstone perforation
	Choledochus cyst (rare)
	Blunt or penetrating trauma
	Iatrogenic lesion
Pancreas	Acute pancreatitis
	Malignancy
	Trauma
	Iatrogenic lesion
Spleen	Blunt and penetrating trauma
Vascular	Ruptured abdominal aortic aneurysm (AAA)
	Massive rectus sheath haematoma
	Massive retroperitoneal haematoma
Uterus	Extra uterine pregnancy
	Pelvic inflammatory disease
	Malignancy
	Trauma

Table 2. Overview of acute abdominal problems that may occur during ICU stay

Organ	Complications
Esophagus	Erosive esophagitis
	Bile or acid gastro-esophageal reflux
Stomach	Stress related mucosal damage or ulcer
	Stomach necrosis or perforation
	Upper GI bleeding
	Impaired gastric emptying
Small intestines	Stress related mucosal damage or ulcer
	Upper GI bleeding
	Ileus
	Mesenteric ischemia
Colon	Mucosal damage or ulcer
	Lower GI bleeding
	Ileus
	Diarrhoea
	Constipation
	Pseudo-obstruction (Ogilvie syndrome)
	Mesenteric ischemia
Liver	Toxic or ischemic hepatitis
	Impaired liver synthesis function
	Impaired drug metabolism
	Ascites
Gallbladder	Atonic bladder
	Sludge
	Acalculous cholecystitis
Pancreatitis	Toxic or ischemic pancreatitis
	Asymptomatic biochemical pancreatitis
	Acute pancreatitis

near the surface of the body and aching (e.g. basal pneumonia). The location of an abdominal pain with regard to the abdominal quadrants also helps to narrow the differential diagnosis (Fig. 1). The incidence and possible associations with the outcome of abdominal pain in the critically ill are not known.

ABDOMINAL DISTENSION

Abdominal distension is a non-specific sign that may, but does not need to originate from the gastrointestinal tract. It can be defined as a sagittal abdominal diameter (SAD) that is greater than the rib cage or the hip height. Looking sideways at a patient in a supine position this would mean that the shape of the abdomen is like a curved line that extends above the virtual line between the xiphoid and symphysis in a convex way. Studies have shown that obesity and intra-abdominal hypertension (IAH) are associated with

an increased SAD [6, 7]. Causes for abdominal distension are related to an increase in intra-abdominal volume: ascites, bowel edema, hematoma, bowel distension, or ileus.

Abnormal findings on percussion include tympanism in case of high content of air in the abdomen or in the intestines and dullness in case of large amount of fluids (e.g. ascites).

Abdominal discoloration may occur as a sign associated with abdominal wall infection or with pancreatitis (Grey-Turner's or Cullen's signs).

General abdominal signs may be important triggers for further investigations, but are never diagnostic by themselves.

GASTROINTESTINAL SYMPTOMS
ABSENT OR ABNORMAL BOWEL SOUNDS

For an assessment of bowel sounds, auscultation for at least one minute in two quadrants, repeated at least once within a tight time frame, should be performed [5]. Palpation of the abdomen should be performed after auscultations as it may stimulate subsequent bowel sounds [8]. In general medicine, absent or abnormal (for example tinkling) bowel sounds have been considered as an important symptom of acute abdominal pathology. In intensive care, auscultation of bowel sounds – widely used but seldom correctly performed — is often regarded as a method having no particular importance. Diminished bowel peristalsis and the absence of bowel sounds are common in mechanically ventilated patients receiving sedatives, opiates and/or catecholamines. The studies suggest that the bowel sounds may be decreased or absent in half of intensive care patients [1, 9–11]. The lack of uniform definition and subjectivity in assessment are obvious reasons why only few studies have investigated whether the presence of absent peristalsis has at all any impact for intensive care patient. Nevertheless, two studies have demonstrated that absence of bowel sounds is associated with impaired outcome [1, 2].

HYPERPERISTALSIS

Hyperperistalsis is defined as the presence of excessive bowel sounds. Excessive and tinkling bowel sounds are characteristic of bowel obstruction [12]. As bowel dilatation and/or (pseudo)obstruction are potentially life-threatening conditions [12, 13], the occurrence of hyperperistalsis deserves particular attention. The true incidence of this symptom in intensive care patients, as well as the impact on outcomes, however, is not known.

VOMITING (EMESIS)

Vomiting or emesis should be defined in ICU patients as the occurrence of any visible regurgitation of gastric content irrespective of the amount [3]. In spontaneously breathing patients, it is characterized by a contraction of the abdomi-
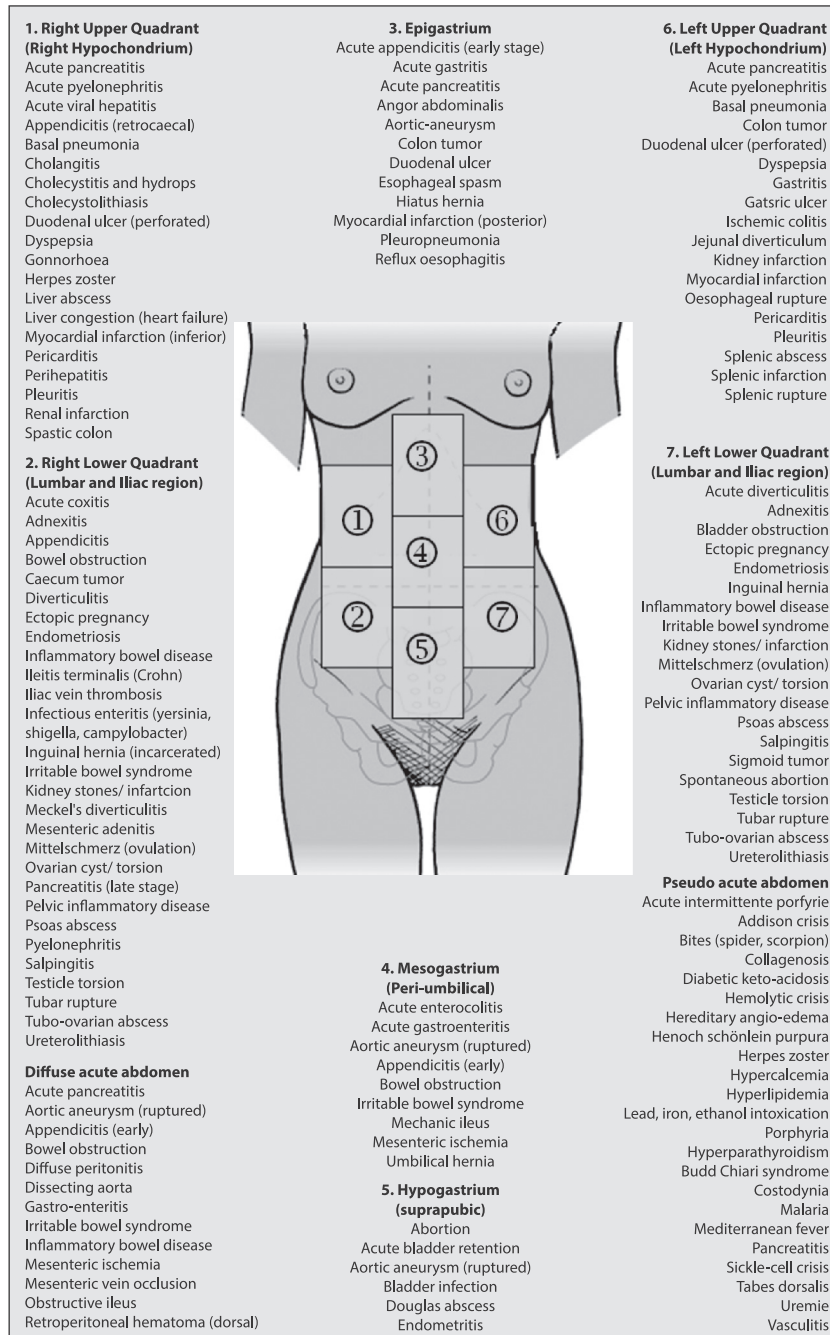
1. Right Upper Quadrant (Right Hypochondrium)
Acute pancreatitis
Acute pyelonephritis
Acute viral hepatitis
Appendicitis (retrocaecal)
Basal pneumonia
Cholangitis
Cholecystitis and hydrops
Cholecystolithiasis
Duodenal ulcer (perforated)
Dyspepsia
Gonnorhoea
Herpes zoster
Liver abscess
Liver congestion (heart failure)
Myocardial infarction (inferior)
Pericarditis
Perihepatitis
Pleuritis
Renal infarction
Spastic colon

2. Right Lower Quadrant (Lumbar and Iliac region)
Acute coxitis
Adnexitis
Appendicitis
Bowel obstruction
Caecum tumor
Diverticulitis
Ectopic pregnancy
Endometriosis
Inflammatory bowel disease
Ileitis terminalis (Crohn)
Iliac vein thrombosis
Infectious enteritis (yersinia, shigella, campylobacter)
Inguinal hernia (incarcerated)
Irritable bowel syndrome
Kidney stones/ infarction
Meckel's diverticulitis
Mesenteric adenitis
Mittelschmerz (ovulation)
Ovarian cyst/ torsion
Pancreatitis (late stage)
Pelvic inflammatory disease
Psoas abscess
Pyelonephritis
Salpingitis
Testicle torsion
Tubar rupture
Tubo-ovarian abscess
Ureterolithiasis

Diffuse acute abdomen
Acute pancreatitis
Aortic aneurysm (ruptured)
Appendicitis (early)
Bowel obstruction
Diffuse peritonitis
Dissecting aorta
Gastro-enteritis
Irritable bowel syndrome
Inflammatory bowel disease
Mesenteric ischemia
Mesenteric vein occlusion
Obstructive ileus
Retroperitoneal hematoma (dorsal)

3. Epigastrium
Acute appendicitis (early stage)
Acute gastritis
Acute pancreatitis
Angor abdominalis
Aortic-aneurysm
Colon tumor
Duodenal ulcer
Esophageal spasm
Hiatus hernia
Myocardial infarction (posterior)
Pleuropneumonia
Reflux oesophagitis

4. Mesogastrium (Peri-umbilical)
Acute enterocolitis
Acute gastroenteritis
Aortic aneurysm (ruptured)
Appendicitis (early)
Bowel obstruction
Irritable bowel syndrome
Mechanic ileus
Mesenteric ischemia
Umbilical hernia

5. Hypogastrium (suprapubic)
Abortion
Acute bladder retention
Aortic aneurysm (ruptured)
Bladder infection
Douglas abscess
Endometritis

6. Left Upper Quadrant (Left Hypochondrium)
Acute pancreatitis
Acute pyelonephritis
Basal pneumonia
Colon tumor
Duodenal ulcer (perforated)
Dyspepsia
Gastritis
Gatsric ulcer
Ischemic colitis
Jejunal diverticulum
Kidney infarction
Myocardial infarction
Oesophageal rupture
Pericarditis
Pleuritis
Splenic abscess
Splenic infarction
Splenic rupture

7. Left Lower Quadrant (Lumbar and Iliac region)
Acute diverticulitis
Adnexitis
Bladder obstruction
Ectopic pregnancy
Endometriosis
Inguinal hernia
Inflammatory bowel disease
Irritable bowel syndrome
Kidney stones/ infarction
Mittelschmerz (ovulation)
Ovarian cyst/ torsion
Pelvic inflammatory disease
Psoas abscess
Salpingitis
Sigmoid tumor
Spontaneous abortion
Testicle torsion
Tubar rupture
Tubo-ovarian abscess
Ureterolithiasis

Pseudo acute abdomen
Acute intermittente porfyrie
Addison crisis
Bites (spider, scorpion)
Collagenosis
Diabetic keto-acidosis
Hemolytic crisis
Hereditary angio-edema
Henoch schönlein purpura
Herpes zoster
Hypercalcemia
Hyperlipidemia
Lead, iron, ethanol intoxication
Porphyria
Hyperparathyroidism
Budd Chiari syndrome
Costodynia
Malaria
Mediterranean fever
Pancreatitis
Sickle-cell crisis
Tabes dorsalis
Uremie
Vasculitis

Figure 1. Differential diagnosis acute abdominal pain (adapted from Malbrain *et al.* [5])

nal muscles, descent of the diaphragm, and opening of the gastric cardia, resulting in a forceful expulsion of stomach contents from the mouth [14]. In mechanically ventilated patients the forcefulness of the act is often undetectable; therefore, vomiting and regurgitation (effortless passage of gastric contents into the mouth) should be assessed together. The incidence of vomiting highly depends on the case mix of an ICU. Concomitant chemotherapy, recent anaesthesia and surgery, as well as abdominal and/or central nervous

system (CNS) diseases are well known predisposing factors for vomiting [15]. Data in the general cohort of critically ill patients are scarce. Many studies investigating nutritional support do not report the incidence of vomiting [16]. In others, incidences ranging from 10 to 20% have been reported [1, 10, 15, 17–20]. Increased IAP may interfere with vomiting. Patients with IAH have been demonstrated to suffer from vomiting/regurgitation in 48.5% of ICU days, in contrast to 28.3% in patients without IAH [21]. Serious complications

such as aspiration pneumonia, dehydration, malnutrition, and disruption of the surgical site are often attributed to vomiting and regurgitation [15, 22]. Whether vomiting has an association with ICU mortality and length of stay is not clear. Mentec *et al*. [10] have shown that vomiting and upper digestive intolerance are associated with nosocomial pneumonia and mortality in nasogastric tube-fed intensive care unit patients. In our previous studies, no difference in mortality between patients with and without vomiting/regurgitation was found [1, 2].

GASTRIC RESIDUAL VOLUME (GRV)

The GRV should be regularly measured according to most of the feeding protocols. The latest study, however, showed no increase in complications, but better achieved caloric goals in patients without GRV monitoring [23]. This finding might change this protocolized approach in the future. The technique for GRV measurement is neither unified nor validated [24–26] and therefore has a wide variation — for example, active aspiration vs. passive outflow of gastric contents, replacement vs. discarding the contents — can be found. Delayed gastric emptying occurs in up to 30–50% of critically ill patients [13] and can result in an increased GRV. Depending on the threshold used for high GRV, it may occur in 5 to 30% of intensive care patients [13, 14, 20]. A GRV above 150 mL has been observed in 28% of medical ICU patients [17]. If 500 mL is taken as the threshold, the incidence is about 5% [2]. The ESICM working group recommends one to consider GRV high if a single volume is > 200 mL [17, 25, 27, 28] or a total gastric aspirate volume >1,000 mL per 24 hours [3]. Although high GRV requires specific attention, it should not automatically result in the discontinuation of enteral feeding. High GRV has been associated with an increased risk of pulmonary aspiration and impaired outcomes in some earlier studies [10]. However, a delay in enteral feeding has similarly been linked to poor results [29]. Therefore, balanced decisions should be made at which levels of GRV enteral feeding has to be withheld. Montejo *et al.* have demonstrated that increasing the cut-off for normal GRV to 500 ml is not associated with increased adverse effects of EN, gastrointestinal complications or in outcome variables [30]. In a prospective study of 377 patients, we did not find a difference in the incidence of high GRV (above 500 mL) between survivors and non-survivors [2].

GI BLEEDING

Gastrointestinal bleeding is defined as any bleeding into the GI tract lumen, confirmed by macroscopic presence of blood in vomited fluids, gastric aspirate or stool [3]. Asymptomatic, endoscopically evident mucosal damage occurs in the majority of ICU patients [11]. Clinically evident GI bleeding, reflecting considerable damage to GI mucosa may be seen in 5–25% of ICU patients [2, 11]. Overt bleeding associated with hemodynamic compromise or the need for blood transfusions, occurs in 1.5–4% of mechanically ventilated patients [11]. GI bleeding itself may increase mortality rates [2], while the risk factors for GI bleeding are mechanical ventilation for > 48 hours and coagulopathy [31, 32]. Several studies have confirmed the benefit of stress ulcer prophylaxis [33–36] while the use of H_2 blocker or proton pump inhibitors is recommended for patients who have risk factors for bleeding [37].

DIARRHOEA

Diarrhoea is defined as having three or more loose or liquid stools per day with a stool weight > 200–250 g per day (or > 250 mL per day) [3]. Due to the lack of a standardized definition, especially in earlier studies, the reported incidences of diarrhoea vary over a very wide range from 2 to 95% [38]. Diarrhoea is, however, a significant problem in intensive care patients which interferes with the adequacy of enteral nutrition [39]. Besides enteral nutrition itself, hypo-albuminemia, intestinal ischaemia, as well as medication use (antibiotics, laxatives) have been demonstrated as risk factors for diarrhoea [39, 40]. The main consequence of diarrhoea is malnutrition, which, if not properly handled, can increase mortality rates [39]. Severe diarrhoea can also result in other serious problems, such as haemodynamic instability due to fluid and electrolyte losses. The severity of the symptoms partly depends on the aetiology of the diarrhoea. *Clostridium difficile* infection is most common nosocomial cause of diarrhoea, and may occur in up to 4% of intensive care patients [41].

Moreover, one-in-five of these patients may develop fulminant colitis with a mortality rate of nearly 60% [42, 43].

PARALYSIS OF THE LOWER GI TRACT (PARALYTIC ILEUS)

Paralysis is the inability of the bowel to pass stool due to impaired peristalsis. Clinical signs include the absence of stool for three or more consecutive days without mechanical obstruction. Bowel sounds may or may not be present. Paralytic ileus is inevitable after abdominal surgery and lasts usually 3 to 5 days [13]. In addition to surgery, other factors such as mechanical ventilation, increased intracranial or intra-abdominal pressure, sedation or sepsis may contribute to development of paralytic ileus [13]. Importantly, in analgo-sedated, mechanically ventilated patients, GI paralysis may be the only sign of ongoing peritonitis. Further risk factors include volume overload [44, 45], hypotension, and use of drugs with known inhibitory effects on GI motility (catecholamines, opioids). In most severe cases, paralytic ileus may lead to severe bowel dilatation, needing immediate interventions. Paralytic ileus is a risk factor of IAH

[46], which in turn is associated with impaired survival. The mortality of Ogilvie's syndrome is reported to be between 10–15% in patients without complications and up to 50% if complications such as ischemia or perforation occur [47, 48].

BOWEL DILATATION

Bowel dilatation is present if the colonic diameter is > 6 cm (> 9 cm for caecum) or small bowel diameter is > 3 cm, diagnosed either on a plain abdominal x-ray or CT scan [3]. Although bowel dilatation is a common sign of obstruction at any level of the GI tract, it may also appear without an obstruction (as in toxic megacolon or acute colonic pseudo-obstruction = Ogilvie's syndrome). The most common predisposing conditions for Ogilvie's syndrome are non-operative abdominal trauma, infections and cardiac diseases [47, 48]. Complex measures including minimal invasive surgery, epidural anaesthesia, restrictive fluid management, early enteral feeding and mobilisation (ERAS protocol) are effective in order to avoid/reduce the duration of postoperative GI paresis and enhance one's recovery after elective abdominal surgery [49].

An increasing number of GI symptoms is associated with impared outcome [1, 2]. Therefore, several GI symptoms occurring together should be considered as a sign of gastrointestinal dysfunction, possibly contributing to impaired outcome.

ABDOMINAL SYNDROMES
ACUTE ABDOMEN

"Acute abdomen" is a syndrome needing immediate intervention. Some of the conditions leading to "acute abdomen" rarely occur in ICU occupants (aortic rupture, ruptured ectopic pregnancy), whereas the others may develop at any stage during the ICU stay (bowel ischemia, ulcer perforation). The symptoms associated with "acute abdomen" are abdominal pain, abdominal distension, nausea, fever, rigidity and/or involuntary contraction of the abdominal muscles in response to palpation [50]. Even if the symptoms that may refer to the presence of "acute abdomen" are the most important to be recognized immediately, data about the incidence of these symptoms in ICUs is scarce [5]. In a patient with abdominal sepsis, an often-overlooked clinical sign is altered mental status. This alteration initially may be so subtle that only close relatives can detect it. In ongoing sepsis, alterations in mental status can range from agitation, anxiety, somnolence, delirium, stupor, epileptic insult to coma. This is called septic or metabolic encephalopathy. Anorexia and nausea are frequent and may precede the emergence of abdominal pain by some time. Vomiting can be caused by hollow viscus obstruction or peritoneal inflammation [51]. Other important elements are: the amount of gastric residuals, the aspect of gastric contents (feculent, bloody,

bilious), and the presence of hematemesis. Diarrhoea is another clinical sign and stool consistency can be changed (or mixed with blood) by alterations in gut mucosal flora or gut hypoperfusion. Fever is clearly the most common manifestation of abdominal sepsis, although it can be masked in immunocompromised patients with neutropenia or under corticosteroid therapy. A patient with severe sepsis can also present one with hypothermia. Some believe that the skin is the mirror of the "inner human being". If the skin is mottled the intestinal mucosa probably is too. One should look at the turgor of the skin and the presence or absence of sudor or sweating, and local or generalised signs of inflammation, the presence of central or peripheral cyanosis (livedo reticularis). One should assess whether the extremities are cold or warm and if the capillary refill is normal (less than two seconds). The outcome of patients with an acute abdomen depends on many issues. The development of GI complications during an ICU stay has been repeatedly shown to increase mortality rates [2, 52–54]. Timely recognition of possible abdominal complications is therefore of utmost importance, independent of whether the patient is admitted because of acute abdomen or develops it as a complication during ICU treatment. Together with a clinical evaluation, diagnostic procedures must always be performed with biochemical and radiological (X-ray, ultrasound or CT) analysis. Cultures should always be taken in case of suspicion of abdominal sepsis. After initial assessments, the clinician should be able to distinguish promptly abdominal problems necessitating immediate surgery. Moreover, when a surgical intervention is planned, it should not be deferred [55].

PSEUDO-ACUTE ABDOMEN

One must always keep in mind other nonabdominal causes of acute abdomen such as inferior myocardial infarction, basal pneumonia, diabetic ketoacidosis, uremia, porphyria, adrenal insufficiency, electrolyte disturbances, lead poisoning, sickle cell crisis, or hemolysis. These conditions are sometimes referred to as "pseudo acute abdomen". Figure 1 summarizes all other conditions that can present as an acute abdomen.

INTRA-ABDOMINAL HYPERTENSION (IAH)

Intra-abdominal hypertension (IAH) is defined as a sustained or repeated IAP , equal or above 12 mm Hg [56, 57]. IAH should also be considered present if the mean of the IAP measurements of the day is 12 mm Hg or higher provided that at least 4 measurements were performed [58]. The most severe manifestation of IAH is abdominal compartment syndrome (ACS), defined as a sustained IAP > 20 mm Hg that is associated with new organ dysfunction / failure [56, 57]. Unlike the symptoms reflecting abdominal compartment, IAH is clearly defined and IAP is a measurable variable. An

increase in IAP influences other organ systems and vice versa [59–66]. At the same time, the exact associations between IAH and gastrointestinal (GI) function/dysfunction are not clear [21]. Although IAP monitoring is strongly supported in mechanically ventilated patients with severe burns, severe trauma, severe acute pancreatitis, liver failure or ruptured aortic aneurysm [67], it should also be considered early in patients with a largely positive fluid balance and other conditions associated with decreased abdominal wall compliance or increased intra-abdominal volume [56]. The ICU patients in whom IAP measurements were not initiated on admission should undergo careful bedside evaluation with a low trigger for starting IAP monitoring [67]. The true incidence of IAH in ICU patients is not known, as no study included all consecutive patients. IAH may occur in 24–38% of patients with an ICU stay >24 hours [46, 68–73], whereas a higher incidence is reported in sub-groups of severe burns (37%) [74], in ruptured abdominal aortic aneurysm (55%) [75], in severe acute pancreatitis (78%) [76] and in severe sepsis (77%) [77]. IAH is reported to occur in 12% of patients after major elective abdominal surgery [5, 78] and in 46% after emergency abdominal surgery needing intensive care [79]. Several studies have reported higher mortality in patients with IAH compared to patients without this syndrome [46, 70, 80], whereas some recent studies do not confirm this finding [2, 69]. Outcomes independently associated with IAH are difficult to determine, as IAH commonly develops in more severe patients [46, 70, 80]. Moreover, there are many interactions with other organ systems and applied treatments. The fact that recent studies do not show independent effects of IAH on mortality might be a reflection of success in the management of IAH during recent years.

CONCLUSIONS

Abdominal symptoms occur in half of the patients in an ICU. Clinical evaluation, albeit largely subjective, remains the main bedside tool to detect abdominal problems and to assess GI function in the critically ill. IAP is a useful additional tool in the assessment of abdominal complications in an ICU. The independent impact of different abdominal symptoms and syndromes remains to be identified. In order to achieve this, a standardized assessment of abdominal signs and symptoms in the critically ill is warranted.

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References:

1. *Reintam A, Parm P, Kitus R, Kern H, Starkopf J:* Gastrointestinal symptoms in intensive care patients. Acta Anaesthesiol Scand 2009; 53: 318–324. doi: 10.1111/j.1399-6576.2008.01860.x.
2. *Reintam Blaser A, Poeze M, Malbrain ML, Bjorck M, Oudemans-van Straaten HM, Starkopf J:* Gastrointestinal symptoms during the first week of intensive care are associated with poor outcome: a prospective multicentre study. Intensive Care Med 2013, 39: 899–909. doi: 10.1007/s00134-013-2831-1.
3. *Reintam Blaser A, Malbrain ML, Starkopf J et al.:* Gastrointestinal function in intensive care patients: terminology, definitions and management. Recommendations of the ESICM Working Group on Abdominal Problems. Intensive Care Med 2012; 38: 384–394.
4. *Erdek MA, Pronovost PJ:* Improving assessment and treatment of pain in the critically ill. Int J Qual Health Care 2004; 16: 59–64.
5. *Malbrain MLNG, De Waele J:* PACT module: abdominal problems. ESICM PACT 2007.
6. *Sugerman H, Windsor A, Bessos M, Kellum J, Reines H, DeMaria E:* Effects of surgically induced weight loss on urinary bladder pressure, sagittal abdominal diameter and obesity co-morbidity. Int J Obes Relat Metab Disord 1998; 22: 230–235.
7. *Sugerman H, Windsor A, Bessos M, Wolfe L:* Intra-abdominal pressure, sagittal abdominal diameter and obesity comorbidity. J Intern Med 1997; 241: 71–79.
8. *Baid H:* A critical review of auscultating bowel sounds. British J Nurs 2009; 18: 1125–1129.
9. *Dark DS, Pingleton SK:* Nonhemorrhagic gastrointestinal complications in acute respiratory failure. Crit Care Med 1989; 17: 755–758.
10. *Mentec H, Dupont H, Bocchetti M, Cani P, Ponche F, Bleichner G:* Upper digestive intolerance during enteral nutrition in critically ill patients: frequency, risk factors, and complications. Crit Care Med 2001; 29: 1955–1961.
11. *Mutlu GM, Mutlu EA, Factor P:* GI complications in patients receiving mechanical ventilation. Chest 2001; 119: 1222–1241.
12. *Sheth SG, LaMont JT:* Toxic megacolon. Lancet 1998; 351: 509–513.
13. *Fruhwald S, Holzer P, Metzler H:* Gastrointestinal motility in acute illness. Wien Klin Wochenschr 2008; 120: 6–17. doi: 10.1007/s00508-007-0920-2.
14. *Fauci AS, Braunwald E, Kasper DL et al.:* Harrison's principles of internal medicine, 17th ed. McGraw-Hill Medical, New York 2007.
15. *Garrett K, Tsuruta K, Walker S, Jackson S, Sweat M:* Managing nausea and vomiting. Current strategies. Crit Care Nurs 2003; 23: 31–50; quiz 51–32.
16. *Doig GS, Heighes PT, Simpson F, Sweetman EA:* Early enteral nutrition reduces mortality in trauma patients requiring intensive care: a meta-analysis of randomised controlled trials. Injury 2011; 42: 50–56. doi: 10.1016/j.injury.2010.06.008.
17. *Elpern EH, Stutz L, Peterson S, Gurka DP, Skipper A:* Outcomes associated with enteral tube feedings in a medical intensive care unit. Am J Crit Care 2004; 13: 221–227.
18. *Montejo JC:* Enteral nutrition-related gastrointestinal complications in critically ill patients: a multicenter study. The Nutritional and Metabolic Working Group of the Spanish Society of Intensive Care Medicine and Coronary Units. Crit Care Med 1999; 27: 1447–1453.
19. *Montejo JC, Grau T, Acosta J et al.:* Multicenter, prospective, randomized, single-blind study comparing the efficacy and gastrointestinal complications of early jejunal feeding with early gastric feeding in critically ill patients. Crit Care Med 2002; 30: 796–800.
20. *O'Leary-Kelley CM, Puntillo KA, Barr J, Stotts N, Douglas MK:* Nutritional adequacy in patients receiving mechanical ventilation who are fed enterally. Am J Crit Care 2005; 14: 222–231.
21. *Reintam Blaser A, Parm P, Kitus R, Starkopf J:* Intra-abdominal hypertension and gastrointestinal symptoms in mechanically ventilated patients. Crit Care Res Pract 2011; 2011: 982507. doi: 10.1155/2011/982507.
22. *Metheny NA, Clouse RE, Chang YH, Stewart BJ, Oliver DA, Kollef MH:* Tracheobronchial aspiration of gastric contents in critically ill tube-

-fed patients: frequency, outcomes, and risk factors. Crit Care Med 2006; 34: 1007–1015.

23. *Reignier J, Mercier E, Le Gouge A et al.:* Effect of not monitoring residual gastric volume on risk of ventilator-associated pneumonia in adults receiving mechanical ventilation and early enteral feeding: a randomized controlled trial. JAMA 2013; 309: 249–256. doi: 10.1001/jama.2012.196377.

24. *McClave SA, Snider HL:* Clinical use of gastric residual volumes as a monitor for patients on enteral tube feeding. JPEN J Parenter Enteral Nutr 2002; 26 (6 Suppl): S43–48.

25. *Hurt RT, McClave SA:* Gastric residual volumes in critical illness: what do they really mean? Crit Care Clin 2010; 26: 481–490, viii-ix. doi: 10.1016/j.ccc.2010.04.010.

26. *Van Stappen J, Pigozzi C, Tepaske R et al.:* Validation of a novel method for measuring intra-abdominal pressure and gastric residual volume in critically ill patients. Anaesthesiol Intensive Ther 2014; 46: 245–254. doi: 10.5603/AIT.2014.0042.

27. *Neumann DA, DeLegge MH:* Gastric versus small-bowel tube feeding in the intensive care unit: a prospective comparison of efficacy. Crit Care Med 2002; 30: 1436–1438.

28. *McClave SA, Martindale RG, Vanek VW et al.:* Guidelines for the provision and assessment of nutrition support therapy in the adult critically ill patient: Society of Critical Care Medicine (SCCM) and American Society for Parenteral and Enteral Nutrition (A.S.P.E.N.). J Parenter Enteral Nutr 2009; 33: 277–316. doi: 10.1177/0148607109335234.

29. *Vincent JL, Preiser JC:* When should we add parenteral to enteral nutrition? Lancet 2013; 381: 354–355. doi: 10.1016/S0140-6736(12)61893-5.

30. *Montejo JC, Minambres E, Bordeje L et al.:* Gastric residual volume during enteral nutrition in ICU patients: the REGANE study. Intensive Care Med 2010; 36: 1386–1393. doi: 10.1007/s00134-010-1856-y.

31. *Steinberg KP:* Stress-related mucosal disease in the critically ill patient: risk factors and strategies to prevent stress-related bleeding in the intensive care unit. Crit Care Med 2002; 30 (6 Suppl): S362–364.

32. *Cook DJ, Fuller HD, Guyatt GH et al.:* Risk factors for gastrointestinal bleeding in critically ill patients. Canadian Critical Care Trials Group. NEJM 1994; 330: 377–381.

33. *Basso N, Bagarani M, Materia A, Fiorani S, Lunardi P, Speranza V:* Cimetidine and antacid prophylaxis of acute upper gastrointestinal bleeding in high risk patients. Controlled, randomized trial. Am J Surg 1981; 141: 339–341.

34. *Bresalier RS, Grendell JH, Cello JP, Meyer AA:* Sucralfate suspension versus titrated antacid for the prevention of acute stress-related gastrointestinal hemorrhage in critically ill patients. AJM 1987; 83: 110–116.

35. *Poleski MH, Spanier AH:* Cimetidine versus antacids in the prevention of stress erosions in critically ill patients. Am J Gastroenterol 1986; 81: 107–111.

36. *Stothert JC, Jr., Simonowitz DA, Dellinger EP et al.:* Randomized prospective evaluation of cimetidine and antacid control of gastric pH in the critically ill. Ann Surg 1980; 192: 169–174.

37. *Dellinger RP, Levy MM, Rhodes A et al:* Surviving sepsis campaign: international guidelines for management of severe sepsis and septic shock, 2012. Intensive Care Med 2013; 39: 165–228. doi: 10.1007/s00134-012-2769-8.

38. *Whelan K, Judd PA, Preedy VR, Taylor MA:* Enteral feeding: the effect on faecal output, the faecal microflora and SCFA concentrations. Proc Nutr Soc 2004; 63: 105–113.

39. *Wiesen P, Van Gossum A, Preiser JC:* Diarrhoea in the critically ill. Curr Opin Crit Care 2006; 12: 149–154.

40. *Bleichner G, Blehaut H, Mentec H, Moyse D:* Saccharomyces boulardii prevents diarrhea in critically ill tube-fed patients. A multicenter, randomized, double-blind placebo-controlled trial. Intensive Care Med 1997; 23: 517–523.

41. *Riddle DJ, Dubberke ER:* Clostridium difficile infection in the intensive care unit. Infect Dis Clin North Am 2009; 23: 727–743. doi: 10.1016/j.idc.2009.04.011.

42. *Grundfest-Broniatowski S, Quader M, Alexander F, Walsh RM, Lavery I, Milsom J:* Clostridium difficile colitis in the critically ill. Dis Colon Rectum 1996; 39: 619–623.

43. *Kyne L, Merry C, O'Connell B, Kelly A, Keane C, O'Neill D:* Factors associated with prolonged symptoms and severe disease due to Clostridium difficile. Age Ageing 1999; 28: 107–113.

44. *Lobo DN:* Fluid, electrolytes and nutrition: physiological and clinical aspects. Proc Nutr Soc 2004; 63: 453–466.

45. *Lobo DN, Bostock KA, Neal KR, Perkins AC, Rowlands BJ, Allison SP:* Effect of salt and water balance on recovery of gastrointestinal function after elective colonic resection: a randomised controlled trial. Lancet 2002; 359: 1812–1818.

46. *Malbrain ML, Chiumello D, Pelosi P et al.:* Incidence and prognosis of intraabdominal hypertension in a mixed population of critically ill patients: a multiple-center epidemiological study. Crit Care Med 2005; 33: 315–322.

47. *Saunders MD:* Acute colonic pseudo-obstruction. Gastrointest Endosc Clin N Am 2007; 17: 341–360, vi-vii.

48. *Vanek VW, Al-Salti M:* Acute pseudo-obstruction of the colon (Ogilvie's syndrome). An analysis of 400 cases. Dis Colon Rectum 1986; 29: 203–210.

49. *Gustafsson UO, Scott MJ, Schwenk W et al.:* Guidelines for perioperative care in elective colonic surgery: Enhanced Recovery After Surgery (ERAS(R)) Society recommendations. Clin Nutr 2012; 31: 783–800. doi: 10.1016/j.clnu.2012.08.013.

50. *Staniland JR, Ditchburn J, De Dombal FT:* Clinical presentation of acute abdomen: study of 600 patients. Br Med J 1972; 3(5823): 393–398.

51. *Martin RF, Rossi RL:* The acute abdomen. An overview and algorithms. Surg Clin North Am 1997; 77: 1227–1243.

52. *Reintam A, Kern H, Starkopf J:* Defining gastrointestinal failure. Acta Clin Belg Suppl 2007; 62: 168–172. doi: 10.1179/acb.2007.62.s1.022.

53. *Reintam A, Parm P, Kitus R, Starkopf J, Kern H:* Gastrointestinal Failure score in critically ill patients: a prospective observational study. Crit Care 2008; 12: R90. doi: 10.1186/cc6958.

54. *Reintam A, Parm P, Redlich U et al.:* Gastrointestinal failure in intensive care: a retrospective clinical study in three different intensive care units in Germany and Estonia. BMC Gastroenterol 2006; 6: 19.

55. *Blot S, De Waele JJ:* Critical issues in the clinical management of complicated intra-abdominal infections. Drugs 2005; 65: 1611–1620.

56. *Malbrain ML, Cheatham ML, Kirkpatrick A et al.:* Results from the International Conference of Experts on Intra-abdominal Hypertension and Abdominal Compartment Syndrome. I. Definitions. Intensive Care Med 2006; 32: 1722–1732.

57. *Kirkpatrick AW, Roberts DJ, De Waele J et al.:* Intra-abdominal hypertension and the abdominal compartment syndrome: updated consensus definitions and clinical practice guidelines from the World Society of the Abdominal Compartment Syndrome. Intensive Care Med 2013; 39: 1190–1206. doi: 10.1007/s00134-013-2906-z.

58. *Malbrain ML, De laet I, Cheatham M:* Consensus conference definitions and recommendations on intra-abdominal hypertension (IAH) and the abdominal compartment syndrome (ACS) — the long road to the final publications, how did we get there? Acta Clin Belg Suppl 2007; 62: 44–59. doi: 10.1179/acb.2007.62.s1.007.

59. *Malbrain ML, De Laet I:* AIDS is coming to your ICU: be prepared for acute bowel injury and acute intestinal distress syndrome. Intensive Care Med 2008; 34: 1565–1569. doi: 10.1007/s00134-008-1135-3.

60. *Malbrain ML, De laet I:* It's all in the gut: introducing the concept of acute bowel injury and acute intestinal distress syndrome. Crit Care Med 2009; 37: 365–366. doi: 10.1097/CCM.0b013e3181935001.

61. *Malbrain ML, Vidts W, Ravyts M, De Laet I, De Waele J:* Acute intestinal distress syndrome: the importance of intra-abdominal pressure. Minerva Anestesiol 2008; 74: 657–673.

62. *Hedenstierna G, Larsson A:* Influence of abdominal pressure on respiratory and abdominal organ function. Curr Opin Crit Care 2012; 18: 80–85. doi: 10.1097/MCC.0b013e32834e7c3a.

63. *Cheatham ML, Malbrain ML:* Cardiovascular implications of abdominal compartment syndrome. Acta Clin Belg Suppl 2007; 62: 98–112. doi: 10.1179/acb.2007.62.s1.013.

64. *De laet I, Citerio G, Malbrain ML:* The influence of intraabdominal hypertension on the central nervous system: current insights and clinical recommendations, is it all in the head? Acta Clin Belg Suppl 2007; 62: 89–97. doi: 10.1179/acb.2007.62.s1.012.

65. *De laet I, Malbrain ML, Jadoul JL, Rogiers P, Sugrue M:* Renal implications of increased intra-abdominal pressure: are the kidneys the canary for abdominal hypertension? Acta Clin Belg Suppl 2007; 62: 119–130. doi: 10.1179/acb.2007.62.s1.015.

66. *Pelosi P, Quintel M, Malbrain ML:* Effect of intra-abdominal pressure on respiratory mechanics. Acta Clin Belg Suppl 2007; 62: 78–88. doi: 10.1179/acb.2007.62.s1.011.

67. *Starkopf J, Tamme K, Blaser AR:* Should we measure intra-abdominal pressures in every intensive care patient? Ann Intensive Care 2012, 2 (Suppl 1): S9. doi: 10.1186/2110-5820-2-S1-S9.

68. *Dalfino L, Tullo L, Donadio I, Malcangi V, Brienza N:* Intra-abdominal hypertension and acute renal failure in critically ill patients. Intensive Care Med 2008; 34: 707–713.

69. *Kim IB, Prowle J, Baldwin I, Bellomo R:* Incidence, risk factors and outcome associations of intra-abdominal hypertension in critically ill patients. Anaesth Intensive Care 2012; 40: 79–89.

70. *Malbrain ML, Chiumello D, Pelosi P et al.:* Prevalence of intra-abdominal hypertension in critically ill patients: a multicentre epidemiological study. Intensive Care Med 2004; 30: 822–829.

71. *Reintam Blaser A, Parm P, Kitus R, Starkopf J:* Risk factors for intra-abdominal hypertension in mechanically ventilated patients. Acta Anaesthesiol Scand 2011; 55: 607–614. doi: 10.1111/j.1399-6576.2011.02415.x.

72. *Vidal M, Ruiz Weisser J, Gonzalez F et al.:* Incidence and clinical effects of intraabdominal hypertension in critically ill patients. Crit Care Med 2008; 36: 1823–1831. doi: 10.1097/CCM.0b013e31817c7a4d.

73. *Malbrain ML, Chiumello D, Cesana BM et al.:* A systematic review and individual patient data meta-analysis on intra-abdominal hypertension in critically ill patients: the wake-up project. World initiative on Abdominal Hypertension Epidemiology, a Unifying Project (WAKE-Up!). Minerva Anestesiol 2014; 80: 293–306.

74. *Oda J, Yamashita K, Inoue T et al.:* Acute lung injury and multiple organ dysfunction syndrome secondary to intra-abdominal hypertension and abdominal decompression in extensively burned patients. J Trauma 2007; 62: 1365–1369.

75. *Djavani Gidlund K, Wanhainen A, Bjorck M:* Intra-abdominal hypertension and abdominal compartment syndrome after endovascular repair of ruptured abdominal aortic aneurysm. Eur J Vasc Endovasc Surg 2011; 41: 742–747. doi: 10.1016/j.ejvs.2011.02.021.

76. *De Waele JJ, Hoste E, Blot SI, Decruyenaere J, Colardyn F:* Intra-abdominal hypertension in patients with severe acute pancreatitis. Crit Care 2005; 9: R452–457.

77. *Regueira T, Bruhn A, Hasbun P et al.:* Intra-abdominal hypertension: incidence and association with organ dysfunction during early septic shock. J Crit Care 2008; 23: 461–467.

78. *Scollay JM, de Beaux I, Parks RW:* Prospective study of intra-abdominal pressure following major elective abdominal surgery. World J Surg 2009; 33: 2372–2377. doi: 10.1007/s00268-009-0191-3.

79. *Sugrue M, Jones F, Deane SA, Bishop G, Bauman A, Hillman K:* Intra-abdominal hypertension is an independent cause of postoperative renal impairment. Arch Surg 1999; 134: 1082–1085.

80. *Reintam A, Parm P, Kitus R, Kern H, Starkopf J:* Primary and secondary intra-abdominal hypertension-different impact on ICU outcome. Intensive Care Med 2008; 34: 1624–1631. doi: 10.1007/s00134-008-1134-4.

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