

Overview of the recent definitions and terminology for acute gastrointestinal injury, intra-abdominal hypertension and the abdominal compartment syndrome*

Aperçu des définitions et de la terminologie récentes concernant les lésions gastro-intestinales aiguës, l'hypertension intra-abdominale et le syndrome du compartiment abdominal

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Abstract This paper summarizes the latest definitions and terminology on acute gastrointestinal injury, intra-abdominal hypertension and the abdominal compartment syndrome in the intensive care unit patients as published recently by the Working Group on Abdominal Problems (WGAP) of the European Society of Intensive Care Medicine (ESICM) and the World Society on Abdominal Compartment Syndrome (WSACS). The uniformity created is a great step forward for the evaluation of gastrointestinal function and abdominal hypertension in the critically ill and should assist in further research.

Keywords Gastrointestinal function · Abdominal pressure · Gastrointestinal symptoms · Intra-abdominal hypertension · Abdominal compartment syndrome

Résumé Ce document résume les dernières définitions et la terminologie sur la défaillance aiguë gastro-intestinale, l'hypertension intra-abdominale et le syndrome du compartiment abdominal chez les patients de réanimation, tel que publiées récemment par le groupe de travail sur les problèmes abdominaux (WGAP) de l'European Society of intensive Care Medicine (ESICM) et la Société mondiale du syndrome du compartiment abdominal (WSACS). Ces définitions consensuelles sont une avancée majeure en matière de normalisation et constituent une bonne base pour la recherche future. L'uniformité créée est un grand pas en avant pour l'évaluation de la fonction gastro-intestinale et l'hypertension abdominale chez les personnes gravement malades.

Mots clés Fonction gastro-intestinale · Pression abdominale · Symptômes gastro-intestinaux · Hyperpression intra-abdominale · Syndrome du compartiment abdominal

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Introduction

Terminology and definitions form the basis of research and are of extreme importance for the comparison of published studies. It is the first step for exploring a topic and allows better clinical communication. Until recently there were no objective and clinically relevant definitions for gastrointestinal (GI) dysfunction in critical illness. Several definitions were used and this led to great confusion [1,2]. The main cause for the lack of a good definition of acute gastrointestinal injury (AGI) is the lack of (bio)markers evaluating or quantifying GI function. A good measurable parameter is needed for the assessment of GI dysfunction as any other organ failure that is evaluated with the sequential organ failure assessment (SOFA) score. For this purpose, the Working Group on Abdominal Problems (WGAP) of the European Society of

Intensive Care Medicine (ESICM) developed recently the descriptive definitions for GI dysfunction in intensive care patients on the basis of the available evidence and current understanding of the pathophysiology of AGI [3].

Because a good measurable parameter is still lacking for the assessment of gastrointestinal dysfunction in contrast to other organ failure that is evaluated with the sequential organ failure assessment (SOFA) score, the definitions on AGI are only descriptive.

As GI symptoms are subjective and dependent on feeding practices, IAP is the only numerical bedside variable reflecting pathological changes in abdominal compartment. Even though the relations between GI dysfunction and intra-abdominal hypertension (IAH) are still unclear, IAP must be considered as a valuable additional tool for monitoring the dynamics of GI dysfunction.

In critical ill patients, an increase in intra-abdominal pressure (IAP) is often present (in up to 25% of patients on admission). Increased IAP may then lead to IAH and abdominal compartment syndrome (ACS). Also recently, the World Society on Abdominal Compartment Syndrome (WSACS, www.wsacs.org) has revised the original 2006 consensus definitions and the 2007 management recommendations [4–6]. In this paper, an overview is given with a summary of the new definitions for AGI, IAH and ACS together with some management recommendations.

Methods

The WGAP and WSACS used the Grading of Recommendations, Assessment, Development, and Evaluation (GRADE) system to provide consistency in identifying and rating the quality of available evidence and the strength of management suggestions and recommendations (Table 1). The parts of this article on AGI were based on the open access 2012 consensus definitions paper on AGI published under CC BY Licence, doi: 10.1007/s00134-011-2459-y [3]. For further reading we refer to the full consensus definitions paper (<http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3286505/>) and the online electronic supplemental material (<http://link.springer.com/article/10.1007/s00134-011-2459-y>). The parts of this article on IAH and ACS were based on the 2013 revised consensus definitions paper, published under CC BY Licence, doi: 10.1007/s00134-013-2906-z [7]. For further reading we refer to the full consensus definitions paper (<http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3680657/>) and the online electronic supplemental material (<http://link.springer.com/article/10.1007/s00134-013-2906-z>). Other parts were adapted from the recent book: Malbrain MLNG, De Waele J: Core Critical Care Series: Intra-abdominal hypertension. Edited by Vuylsteke A. Cam-

Table 1 Grading of the quality of evidence and strength of recommendations

Quality of evidence	Rationale
A (High)	Randomized controlled trials or meta-analyses
B (Moderate)	Downgraded RCTs: related to publication bias, inconsistency, imprecision, indirectness or other limitations
C (Low)	Well-done observational studies
D (Very low)	Expert opinion or case reports
Strength of recommendation	
Grade 1 (Strong)	We recommend
Grade 2 (Weak)	We suggest

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Results

Definitions on Acute Gastrointestinal Injury

The consensus definitions and terminology of the WGAP about AGI are listed here below. The WGAP suggests using the following terminology and definitions [3]:

DEFINITION 1 - Gastrointestinal function

The human GI tract has many functions including facilitating digestion to absorb nutrients and water, barrier control to modulate absorption of intraluminal microbes (and their products), endocrine and immune functions. Perfusion, secretion, motility and a coordinated gut–microbiome interaction are prerequisites for an adequate function. It needs to be underlined that because we currently lack the tool or marker to measure GI function we cannot reliably decide about normal GI function in the acute setting.

DEFINITION 2 – AGI

AGI is malfunctioning of the GI tract in critically ill patients due to their acute illness. According to severity different grades of AGI can be distinguished.

• DEFINITION 2.1 – AGI grade I

AGI grade I (risk of developing GI dysfunction or failure) - the function of the GI tract is partially impaired, expressed as

GI symptoms related to a known cause and perceived as transient. *Rationale* The condition is clinically seen as occurrence of GI symptoms after an insult, which expectedly has temporary and self-limiting nature. *Examples* Postoperative nausea and/or vomiting during the first days after abdominal surgery, postoperative absence of bowel sounds, diminished bowel motility in the early phase of shock. *Management* The general condition is usually improving and specific interventions for GI symptoms are not needed, except the replacement of fluid requirements by intravenous infusions. Early enteral feeding, started within 24–48 h after the injury, is recommended (grade 1B). The use of drugs impairing GI motility (e.g. catecholamines, opioids) has to be limited whenever possible (grade 1C).

• DEFINITION 2.2 – AGI grade II

AGI grade II (GI dysfunction) - the GI tract is not able to perform digestion and absorption adequately to satisfy the nutrient and fluid requirements of the body. There are no changes in the general condition of the patient related to GI problems. *Rationale* The condition is characterized by acute occurrence of GI symptoms requiring therapeutic interventions for achievement of nutrient and fluid requirements. This condition occurs without previous GI interventions or is more severe than might be expected in relation to the course of preceding abdominal procedures. *Examples* Gastroparesis with high gastric residuals or reflux, paralysis of the lower GI tract, diarrhoea, IAH grade I (IAP 12–15 mmHg), visible blood in gastric content or stool. Feeding intolerance is present if at least 20 kcal/kg BW/day via enteral route cannot be reached within 72 h of feeding attempt. *Management* Measures to treat the condition and to prevent the progression to gastrointestinal failure (GIF) need to be undertaken (e.g. treatment of IAH (grade 1D)); or measures to restore the motility function of GI tract, such as prokinetic therapy (grade 1C). Enteral feeding should be started or continued; in cases of high gastric residuals/reflux or feeding intolerance, regular challenges with small amounts of enteral nutrition (EN) should be regularly considered (grade 2D). In patients with gastroparesis, initiation of postpyloric feeding should be considered in this state, when prokinetic therapy is not effective (grade 2D).

• DEFINITION 2.3 – AGI grade III

AGI grade III (GIF) - there is a loss of GI function, where restoration of GI function is not achieved despite interventions and the general condition is not improving. *Rationale* Clinically seen as sustained intolerance to enteral feeding without improvement after treatment (e.g. erythromycin, postpyloric tube placement), leading to persistence or worsening of MODS. *Examples* Despite treatment, feeding intolerance

is persisting – high gastric residuals, persisting GI paralysis, occurrence or worsening of bowel dilatation, progression of IAH to grade II (IAP 15–20 mmHg), low abdominal perfusion pressure (APP) (below 60 mmHg). Feeding intolerance is present and possibly associated with persistence or worsening of MODS. *Management* Measures to prevent worsening of GIF are warranted (e.g. monitoring and targeted treatment of IAH (grade 1D)). Presence of undiagnosed abdominal problem (cholecystitis, peritonitis, bowel ischemia) should be excluded. The medications promoting GI paralysis have to be discontinued as far as possible (grade 1C). Early parenteral feeding (within the first 7 days of intensive care unit (ICU) stay) supplementary to insufficient enteral nutrition is associated with higher incidence of hospital infections and should be avoided (grade 2B). Challenges with small amounts of EN should be regularly considered (grade 2D).

• DEFINITION 2.4 – AGI grade IV

AGI grade IV (GIF with severe impact on distant organ function)—AGI has progressed to become directly and immediately life-threatening, with worsening of MODS and shock. *Rationale* The situation when AGI has led to an acute critical deterioration of the general condition of the patient with distant organ dysfunction(s). *Examples* Bowel ischaemia with necrosis, GI bleeding leading to haemorrhagic shock, Ogilvie's syndrome, ACS requiring decompression. *Management* The condition requires laparotomy or other emergency interventions (e.g. colonoscopy for colonic decompression) for life-saving indications (grade 1D). There is no proven conservative approach to resolve this situation. As differentiation of the acute GI problem from previously existing chronic condition might be very difficult, we suggest using the same definitions also in cases where the condition (e.g. GI bleeding, diarrhoea) might be due to a chronic GI disease (e.g. Crohn's disease). In patients on chronic parenteral feeding, GIF (equal to AGI III) should be considered chronically present, and no new acute interventions to restore function are indicated. However, monitoring of IAH and exclusion of the new acute abdominal problems should be performed similarly as in AGI grade III management.

• DEFINITION 2.5 - Primary and secondary AGI

DEFINITION 2.5.1 – Primary AGI

Primary AGI is associated with primary disease or direct injury to organs of the GI system (first hit). *Rationale* The condition may usually be observed early (during the first day) after the insult to the GI system. *Examples* Peritonitis, pancreatic or hepatic pathology, abdominal surgery, abdominal trauma.

DEFINITION 2.5.2 – Secondary AGI

Secondary AGI develops as the consequence of a host response in critical illness without primary pathology in the GI system (second hit). *Rationale* The condition develops without direct insult to the GI tract. *Examples* GI malfunction in a patient with pneumonia, cardiac pathology, non-abdominal surgery or trauma, postresuscitation state.

DEFINITION 3 - Feeding intolerance syndrome (FI)

FI is a general term indicating intolerance of enteral feeding for whatever clinical reason (vomiting, high gastric residuals, diarrhoea, GI bleeding, presence of entero-cutaneous fistulas, etc.). *Rationale* Diagnosis is based on complex clinical evaluation. There is no single clear-cut symptom or value that defines FI. Several symptoms are commonly present. FI should be considered present if at least 20 kcal/kg BW/day via enteral route cannot be reached within 72 h of feeding attempt or if enteral feeding has to be stopped for whatever clinical reason. FI should not be considered as present if enteral feeding is electively not prescribed or is withheld/interrupted due to procedures. FI in special conditions: in a patient with postpyloric feeding, FI is defined similarly to gastric feeding. If a patient is not fed enterally due to the presence of enteroatmospheric fistulas, FI should be considered present. If the patient undergoes a surgical intervention for ACS or for changing of surgical dressings of an open abdomen, FI should be considered present immediately after surgery unless enteral feeding can be administered. *Management* FI requires efforts to maintain/restore GI function: limiting the use of drugs impairing motility, applying prokinetics and/or laxatives (grade 1C) and controlling IAP. Challenges with small amounts of EN should be regularly considered. In patients not tolerating enteral feeding, supplemental parenteral nutrition should be considered (grade 2D). Recent data suggest that delay for 1 week with parenteral nutrition enhances recovery when compared to early intravenous feeding (grade 2B).

DEFINITION 4 - Gastrointestinal symptoms

• DEFINITION 4.1 – Vomiting

Vomiting (emesis) is the occurrence of any visible regurgitation of gastric content irrespective of the amount. *Rationale* Vomiting is commonly defined as the oral expulsion of GI contents resulting from contractions of gut and thoraco-abdominal wall musculature. Vomiting is contrasted with regurgitation, which is the effortless passage of gastric contents into the mouth. In ICU patients the forcefulness of the act is often not detectable; therefore, regurgitation and vomiting should be assessed together. *Management* Several guidelines for prevention and management of postoperative

nausea and vomiting are available. However, no studies have addressed management of vomiting in mechanically ventilated ICU patients; therefore, no specific recommendation can be given.

• DEFINITION 4.2 – Gastric residual volume

Gastric residual volume could be considered high if a single volume exceeds 200 ml. *Rationale* There is no sufficient scientific evidence or physiological ground to define precise values for high gastric residuals. Measurement of gastric residuals is neither standardized nor validated. It has been suggested that gastric residual volume greater than 200 ml should prompt careful bedside evaluation, but automatic cessation of enteral nutrition solely on the basis on residual volumes of 200–500 ml should be avoided. Despite the lack of scientific evidence, the members of the WGAP arbitrarily use total volumes of gastric residuals above 1,000 ml/24 h as a sign of abnormal gastric emptying, which requires specific attention. *Management* Intravenous administration of metoclopramide and/or erythromycin is recommended for management of high gastric residuals, whereas cisapride is no longer approved (grade 1B). Routine use of motility agents is not recommended (grade 1A). Acupuncture stimulation may facilitate gastric emptying in neurosurgical ICU patients (grade 2B). Use of opioids and deep sedation should be avoided/reduced if possible. Cessation of gastric feeding is suggested if residual volumes exceed 500 ml per single measurement. Here, postpyloric feeding should be considered (grade 2D). Routine application of postpyloric feeding is not advocated (grade 2D). Postpyloric feeding may cause severe small bowel dilatation and perforation in rare cases.

• DEFINITION 4.3 – Diarrhoea

Diarrhoea is defined as having three or more loose or liquid stools per day with a stool weight greater than 200–250 g/day (or greater than 250 ml/day). *Rationale* Normal bowel frequency ranges from three times a week to three times a day. Secretory, osmotic, motor and exudative diarrhoea may be distinguished, but in the ICU it is often better to distinguish between disease-, food/feeding- and drug-related diarrhoea. *Management* Symptomatic therapy—replacement of fluids and electrolytes, haemodynamic stabilization and organ protection (e.g. correction of hypovolemia to prevent impairment of renal function) forms the basic management (grade 1D). At the same time, trigger mechanisms need to be discovered and when possible stopped (e.g. laxatives, sorbitol, lactulose, antibiotics) or treated (e.g. malabsorption, inflammatory bowel disease). Feeding-related diarrhoea in critically ill patients may require reduction of infusion rate, repositioning of feeding tube or dilution of nutrition formula. Changing formula by

adding soluble fibre prolongs transit time (grade 1C). Only in cases of severe or recurrent *Clostridium difficile*-associated diarrhoea is oral vancomycin superior to metronidazole (grade 2C).

• DEFINITION 4.4 – GI bleeding

GI bleeding is any bleeding into the GI tract lumen, confirmed by macroscopic presence of blood in vomited fluids, gastric aspirate or stool. *Rationale* Asymptomatic, endoscopically evident mucosal damage occurs in the majority of ICU patients. Clinically evident GI bleeding reflecting considerable damage to GI mucosa may be seen in 5–25% of ICU patients. Clinically important bleeding, defined as overt bleeding in association with haemodynamic compromise or the need for blood transfusions, occurs in 1.5–4% of mechanically ventilated patients. *Management* In cases of clinically evident GI bleeding, the haemodynamic status dictates the approach. In cases of bleeding with haemodynamic instability, endoscopy is the diagnostic tool of choice, but when bleeding is ongoing and massive, precluding adequate endoscopic assessment, angiography is appropriate (grade 2C). Early upper GI endoscopy (less than 24 h) is recommended (grade 1A), except for patients with acute variceal bleeding in whom a more expedite procedure (less than 12 h) should be considered (grade 2C). Epinephrine injection can be used in combination with another method, such as clips, thermocoagulation or sclerosant injection (grade 1A). Routine second endoscopy is not recommended, but in cases of rebleeding, a second attempt for endoscopic therapy is recommended (grade 1A). In cases of a negative upper endoscopy with evidence of GI bleeding, colonoscopy should be performed, followed by small bowel exploration using push enteroscopy if colonoscopy is negative (grade 2C). In cases of ongoing bleeding with negative endoscopies, abdominal surgery with intraoperative endoscopy or interventional radiology should be considered (grade 2C).

• DEFINITION 4.5 – Paralysis of lower GI tract

Paralysis of lower GI tract (paralytic ileus) is the inability of the bowel to pass stool due to impaired peristalsis. Clinical signs include absence of stool for three or more consecutive days without mechanical obstruction. Bowel sounds may or may not be present. *Rationale* Outside of the ICU, the terms constipation and obstipation include uncomfortable or infrequent bowel movements, hard stool and painful defaecation. Because these symptoms may not be expressed in ICU patients, it is suggested to use the term paralysis of lower GI tract. A cut-off level of 3 days has been used in most of the epidemiological ICU studies. *Management* Inhibitory drugs for GI motility (e.g. catecholamines, sedatives, opioids) must be withdrawn if possible and conditions

impairing motility (e.g. hyperglycaemia, hypokalaemia) corrected (grade 1C). Because of their delayed onset of action, laxative drugs must be started early or given prophylactically (grade 1D). Because of unknown long-term efficacy and safety, the routine use of opioid antagonists cannot be recommended (grade 2B). Prokinetics like domperidone, metoclopramide and erythromycin are used to stimulate the upper GI tract (stomach and small bowel), whereas neostigmine stimulates small bowel and colonic motility. Despite the lack of well-controlled studies and sufficient evidence, we recommend a standardised approach in using prokinetics for management of motility disorders (grade 1D).

• DEFINITION 4.6 – Abnormal bowel sounds

Abnormal bowel sounds *Rationale* Normal frequency of bowel sounds may range between 5 and 35 sounds/min; the clinical significance of abnormal bowel sounds is not clear. No technique of auscultation has been proven to be superior. The authors suggest auscultation for at least 1 min in two quadrants, repeated at least once within a tight time frame. Palpation of the abdomen before the auscultation may stimulate peristalsis causing subsequent bowel sounds that may not have been there otherwise. *Management* There are no special management suggestions for absent/abnormal bowel sounds.

DEFINITION 4.6.1 – Absent peristalsis

Absent peristalsis - No bowel sounds are heard at cautious auscultation. *Rationale* Complete lack of bowel sounds is abnormal. However, it should be recognized that presence of bowel sounds does not confirm normal motility, and that reoccurrence of bowel sounds does not correlate with improvement of paralysis.

DEFINITION 4.6.2 – Hyperperistalsis

Hyperperistalsis is present if excessive bowel sounds are heard on auscultation. *Rationale* Hyperperistalsis is a state of excessive motility of the digestive tract. It can be present during bowel obstruction occurring in parts of the bowel as attempts to overcome obstruction.

• DEFINITION 4.7 – Bowel dilatation

Bowel dilatation is present if colonic diameter exceeds 6 cm (greater than 9 cm for caecum) or small bowel diameter exceeds 3 cm, diagnosed either on plain abdominal X-ray or CT scan. *Rationale* Bowel dilatation is a common sign of obstruction at any level of the GI tract. Bowel dilatation may also appear without an obstruction; the terms toxic megacolon following colitis and acute colonic pseudo-obstruction or Ogilvie's syndrome are used to describe acute severe colonic dilatation. *Management* Next to the correction of fluid and electrolyte imbalance, nasogastric decompression may be helpful (grade 1D), although routine usage of

nasogastric tubes after elective laparotomy is not recommended (grade 1A). After exclusion of mechanical obstruction, intravenous neostigmine could be considered in patients with a caecal diameter > 10 cm and without improvement within 24 h (grade 2B). Colonoscopy is recommended for non-surgical decompression in patients with a caecal diameter > 10 cm and no improvement after 24–48 h of conservative treatment (grade 1C). Colonoscopic decompression is effective in up to 80% of cases, but carries a certain morbidity/mortality risk. Conservative treatment together with colonoscopy may be continued for 48–72 h unless the 389 caecum is >12 cm wide (grade 2C). In cases of unresponsiveness to conservative treatment, surgery is indicated due to the threatening risk of perforation (grade 1D). Usage of a laparoscopic technique with thoracic epidural anaesthesia where appropriate enhances bowel function after abdominal surgery (grade 1B), and may therefore prevent bowel dilatation.

Definitions on Intra-abdominal Hypertension

The new and updated WSACS definitions regarding IAH and ACS are summarized in Table 2 and will be discussed in detail hereafter [8,9]. Table 3 contains the list of risk factors for increased IAP, IAH and ACS [8]. When the abdomen is open, one has to consider the critical complications like fixation of the abdomen and development of enteroatmospheric fistulae (EAF). Since the standard treatment for ACS is still decompressive laparotomy (DL), which leaves the patient with an open abdomen (OA), a new classification for OA was also proposed by the WSACS (Table 4), in order to facilitate comparison of patient groups with similar determinants of outcomes and complications. This classification is an update of the original Björck et al. classification [10]. Although the pathophysiology of raised IAP in children is quite similar to adults, cut-off values for definitions and IAP measurement technique are somewhat different and those definitions that were amended for paediatric use are listed in Table 5 but will not be discussed herein [11]. The WSACS consensus management recommendations are listed in Table 6.

DEFINITION 1 - What is IAP? IAP is the steady-state pressure concealed within the abdominal cavity

Since the abdominal compartment is defined by a combination of rigid (the spine, rib cage and bony pelvis) and semi-rigid borders (the muscular abdominal wall, the diaphragm and the pelvic muscles), it is vulnerable to the development of raised compartmental pressure (CP), just like any other identifiable compartment in the human body [12]. The IAP is directly affected by the volume of the solid organs or hollow viscera, the presence of ascites, blood or other space-occupying lesions (such as tumours or a gravid uterus) and

the presence of conditions that limit expansion of the abdominal wall (such as circular burn eschars, obesity, tight closure, velcro belt, prone positioning or ascites) [4].

DEFINITION 2 - Abdominal perfusion pressure, $APP = MAP - IAP$

Analogous to cerebral perfusion pressure (CPP), APP has been proposed as a more accurate predictor of visceral perfusion and as an endpoint for resuscitation. APP, by considering both arterial inflow (MAP) and restrictions to venous outflow (IAP), is statistically superior to either parameter alone in predicting patient survival from IAH/ACS [13,14]. Retrospective analysis showed that APP was superior to other common resuscitation endpoints in patients with elevated IAP including arterial pH, base deficit, arterial lactate and hourly urinary output [15]. It seems prudent to maintain APP above 60 mmHg, although hard prospective evidence for this statement is still lacking.

DEFINITION 3 - Measurement; IAP should be expressed in mmHg and measured at end-expiration in the complete supine position after ensuring that abdominal muscle contractions are absent and with the transducer zeroed at the level of the mid-axillary line

IAP measurements are essential to the diagnosis of IAH/ACS as physical examination is notoriously poor in identifying elevated IAP [16,17]. Studies also showed that the abdominal perimeter does not correlate well with true IAP [18]. Several key principles must be followed to ensure accurate and reproducible measurements [19]. Early studies using water manometers reported their results in cm H₂O while subsequent studies using electronic pressure transducers reported IAP in mmHg (1 mmHg = 1.36 cm H₂O). This led to confusion and difficulty in comparing studies [20]. IAP varies with respiration and is most consistently measured at end-expiration.

DEFINITION 4 - The reference standard for intermittent IAP measurement is via the bladder with a maximal instillation volume of 25 mL of sterile saline

For an IAP measurement, the residual urine from the bladder is completely drained and the bladder catheter is blocked, followed by instillation of 25 mL sterile saline after which the IAP can be measured. Multiple studies have demonstrated that, for the bladder technique, instillation of volumes in excess of 25 mL may artificially increase IAP, leading to potentially erroneous measurements and inappropriate therapy [21–23] and recent studies have suggested that volumes as low as 2–5 mL are sufficient [24].

Table 2 Definitions regarding intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS) according to the 2006 and 2013 WSACS guidelines update (adapted from Malbrain et al. [4] and Kirkpatrick et al. [8])

Def	2006 definitions	Def	2013 definitions
1	IAP is the steady-state pressure concealed within the abdominal cavity.	1	IAP is the steady-state pressure concealed within the abdominal cavity.
2	APP = MAP – IAP	2	APP = MAP – IAP
3	FG = GFP – PTP = MAP – 2 * IAP		REJECTED
4	IAP should be expressed in mmHg and measured at end-expiration in the complete supine position after ensuring that abdominal muscle contractions are absent and with the transducer zeroed at the level of the mid-axillary line.	3	IAP should be expressed in mmHg and measured at end-expiration in the complete supine position after ensuring that abdominal muscle contractions are absent and with the transducer zeroed at the level of the mid-axillary line.
5	The reference standard for intermittent IAP measurement is via the bladder with a maximal instillation volume of 25 mL of sterile saline.	4	The reference standard for intermittent IAP measurements is via the bladder with a maximal instillation volume of 25 mL of sterile saline.
6	Normal IAP is approximately 5–7 mmHg in critically ill adults.	5	IAP is approximately 5–7 mmHg and around 10 mmHg in critically ill adults.
7	IAH is defined by a sustained or repeated pathologic elevation of IAP \geq 12 mmHg.	6	IAH is defined by a sustained or repeated pathologic elevation of IAP \geq 12 mmHg.
8	IAH is graded as follows: • Grade I: IAP 12–15 mmHg • Grade II: IAP 16–20 mmHg • Grade III: IAP 21–25 mmHg • Grade IV: IAP > 25 mmHg	7	IAH is graded as follows: • Grade I: IAP 12–15 mmHg • Grade II: IAP 16–20 mmHg • Grade III: IAP 21–25 mmHg • Grade IV: IAP > 25 mmHg
9	ACS is defined as a sustained IAP \geq 20 mmHg (with or without an APP < 60 mmHg) that is associated with new organ dysfunction/failure.	8	ACS is defined as a sustained IAP \geq 20 mmHg (with or without an APP < 60 mmHg) that is associated with new organ dysfunction/failure.
10	Primary ACS is a condition associated with injury or disease in the abdomino-pelvic region that frequently requires early surgical or interventional radiological intervention.	9	Primary ACS is a condition associated with injury or disease in the abdomino-pelvic region that frequently requires early surgical or interventional radiological intervention.
11	Secondary ACS refers to conditions that do not originate from the abdomino-pelvic region.	10	Secondary ACS refers to conditions that do not originate from the abdomino-pelvic region.
12	Recurrent ACS refers to the condition in which ACS redevelops following previous surgical or medical treatment of primary or secondary ACS.	11	Recurrent ACS refers to the condition in which ACS redevelops following previous surgical or medical treatment of primary or secondary ACS.
		12	NEW: A poly-compartment syndrome is a condition where two or more anatomical compartments have elevated compartmental pressures.

(Suite page suivante)

Table 2 (suite)

Def	2006 definitions	Def	2013 definitions
13			NEW: Abdominal compliance quantifies the ease of abdominal expansion, is determined by the elasticity of the abdominal wall and diaphragm and is expressed as the change in intra-abdominal volume per change in intra-abdominal pressure in L/mmHg.
14			NEW: An open abdomen (OA) is any abdomen requiring a temporary abdominal closure due to the skin and fascia not being closed after laparotomy. The technique of temporary abdominal closure should be explicitly described.
15			NEW: The open abdomen is classified with the following grading system: 1 – No Fixation 1A: clean, no fixation 1B: contaminated, no fixation 1C: enteric leak, no fixation 2 – Developing Fixation 2A: clean, developing fixation 2B: contaminated, developing fixation 2C: enteroatmospheric/cutaneous fistula, developing fixation 3 and 4 – Frozen Abdomen 3: frozen abdomen, no fistula 4: frozen abdomen with enteroatmospheric/cutaneous fistula
16			NEW: Lateralization of the abdominal wall refers to the phenomenon whereby the musculature and fascia of the abdominal wall, most well seen by the rectus abdominis muscles and their enveloping fascia, move laterally away from the midline with time.

ACS: abdominal compartment syndrome; FG: filtration gradient; GFP: glomerular filtration pressure; IAH: intra-abdominal hypertension; IAP: intra-abdominal pressure; MAP: mean arterial pressure; OA: open abdomen; PTP: proximal tubular pressure.

Table 3 Risk factors for intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS) [7]
Risk factor
<i>Diminished abdominal wall compliance</i>
Abdominal surgery
Major trauma
Major burns
Prone positioning
<i>Increased intra-luminal contents</i>
Gastroparesis/gastric distension/ileus
Ileus
Colonic pseudo-obstruction
Volvulus
<i>Increased intra-abdominal contents</i>
Acute pancreatitis
Distended abdomen
Haemoperitoneum/pneumoperitoneum or intra-peritoneal fluid collections
Intra-abdominal infection/abscess
Intra-abdominal or retroperitoneal tumours
Laparoscopy with excessive insufflation pressures
Liver dysfunction/cirrhosis with ascites
Peritoneal dialysis
<i>Capillary leak/fluid resuscitation</i>
Acidosis/Damage control laparotomy
Hypothermia
Increased APACHE-II or SOFA score
Massive fluid resuscitation or positive fluid balance
Polytransfusion
<i>Others/miscellaneous</i>
Age
Bacteremia
Coagulopathy
Increased head of bed angle
Massive incisional hernia repair
Mechanical ventilation
Obesity or increased body mass index
PEEP > 10
Peritonitis
Pneumonia
Sepsis
Shock or hypotension
APACHE-II: acute physiology and chronic health evaluation-II; PEEP: positive end expiratory pressure; SOFA: sequential organ failure assessment.

DEFINITION 5 - Normal IAP is approximately 5–7 mmHg and around 10 mmHg in critically ill adults

Normal IAP ranges from sub-atmospheric to zero mmHg but values from 5 to 7 mmHg have also been reported [25].

Table 4 Classification scheme for the complexity of the open abdomen, adapted from Björck et al. [10]	
1 No fixation	
1A	Clean, no fixation
1B	Contaminated, no fixation
1C	Enteric leak, no fixation
2 Developing fixation	
2A	Clean, developing fixation
2B	Contaminated, developing fixation
2C	Enteric leak, developing fixation
3 Frozen abdomen	
3A	Clean, frozen abdomen
3B	Contaminated, frozen abdomen
4 Established enteroatmospheric fistula, frozen abdomen	

Table 5 Definitions regarding intra-abdominal pressure (IAP) for paediatric use according to the 2013 WSACS guidelines update (adapted from Kirkpatrick et al. [7])	
IAP measurement	The reference standard for intermittent IAP measurement in children is via the bladder using 1 mL/kg as an instillation volume and a minimal instillation volume of 3 mL and a maximum installation volume of 25 mL of sterile saline.
‘Normal’ IAP	IAP in critically ill children is approximately 4–10 mmHg.
IAH	IAH in children is defined by a sustained or repeated pathological elevation in IAP > 10 mmHg.
ACS	ACS in children is defined as a sustained elevation in IAP of greater than 10 mmHg associated with new or worsening organ dysfunction that can be attributed to elevated IAP.
For those items not mentioned in Table 2, the definitions of Table 1 apply also to children.	

From a physiological point of view, sub-atmospheric values do not make sense in supine critically ill mechanically ventilated patients using the mid-axillary line as a zero reference point and may rather reflect a measurement error [26]. Morbid obesity or pregnancy may be associated with asymptomatic IAP elevations of up to 15 mmHg, which are seemingly well tolerated due to their chronic nature and generally occult effects [26,27]. Such levels, occurring acutely, however, can have significant pathophysiologic impact upon organ perfusion and result in organ dysfunction and/or overt ACS. Recent abdominal surgery, sepsis, organ failure and need for mechanical ventilation are all associated with elevated IAP [28–32]. The clinical importance of any IAP measurement must always be assessed in view of the

Table 6 Final 2013 WSACS consensus management statements and recommendations [7]**Recommendations**

1. We recommend measuring IAP when any known risk factor for IAH/ACS is present in a critically ill or injured patient [GRADE 1C]
2. Studies should adopt the trans-bladder technique as the standard IAP measurement technique [not GRADED]
3. We recommend use of protocolized monitoring and management of IAP versus not [GRADE 1C]
4. We recommend efforts and/or protocols to avoid sustained IAH as compared to inattention to IAP among critically ill or injured patients [GRADE 1C]
5. We recommend decompressive laparotomy in cases of overt ACS compared to strategies that do not use decompressive laparotomy in critically ill adults with ACS [GRADE 1D]
6. We recommend that among ICU patients with open abdominal wounds, conscious and/or protocolized efforts be made to obtain an early or at least same-hospital-stay abdominal fascial closure [GRADE 1D]
7. We recommend that among critically ill/injured patients with open abdominal wounds, strategies utilizing negative pressure wound therapy should be used versus not [GRADE 1C]

Suggestions

1. We suggest that clinicians ensure that critically ill or injured patients receive optimal pain and anxiety relief [GRADE 2D]
2. We suggest brief trials of neuromuscular blockade as a temporizing measure in the treatment of IAH/ACS [GRADE 2D]
3. We suggest that the potential contribution of body position to elevated IAP be considered among patients with, or at risk of, IAH or ACS [GRADE 2D]
4. We suggest liberal use of enteral decompression with nasogastric or rectal tubes when the stomach or colon are dilated in the presence of IAH/ACS [GRADE 1D]
5. We suggest that neostigmine be used for the treatment of established colonic ileus not responding to other simple measures and associated with IAH [GRADE 2D]
6. We suggest using a protocol to try and avoid a positive cumulative fluid balance in the critically ill or injured patient with, or at risk of, IAH/ACS after the acute resuscitation has been completed and the inciting issues have been addressed [GRADE 2C]
7. We suggest use of an enhanced ratio of plasma/packed red blood cells for resuscitation of massive haemorrhage versus low or no attention to plasma/packed red blood cell ratios [GRADE 2D]
8. We suggest use of percutaneous catheter drainage (PCD) to remove fluid (in the setting of obvious intraperitoneal fluid) in those with IAH/ACS when this is technically possible compared to doing nothing [GRADE 2C]. We also suggest using PCD to remove fluid (in the setting of obvious intraperitoneal fluid) in those with IAH/ACS when this is technically possible compared to immediate decompressive laparotomy as this may alleviate the need for decompressive laparotomy [GRADE 2D]
9. We suggest that patients undergoing laparotomy for trauma suffering from physiologic exhaustion be treated with the prophylactic use of the open abdomen versus intraoperative abdominal fascial closure and expectant IAP management [GRADE 2D]
10. We suggest not to routinely utilize the open abdomen for patients with severe intraperitoneal contamination undergoing emergency laparotomy for intra-abdominal sepsis unless IAH is a specific concern [GRADE 2B]
11. We suggest that bioprosthetic meshes should not be routinely used in the early closure of the open abdomen compared to alternative strategies [GRADE 2D]

No recommendations

1. We could make no recommendation regarding use of abdominal perfusion pressure in the resuscitation or management of the critically ill or injured
2. We could make no recommendation regarding use of diuretics to mobilize fluids in haemodynamically stable patients with IAH after the acute resuscitation has been completed and the inciting issues have been addressed
3. We could make no recommendation regarding use of renal replacement therapies to mobilize fluid in haemodynamically stable patients with IAH after the acute resuscitation has been completed and the inciting issues have been addressed
4. We could make no recommendation regarding the administration of albumin versus not, to mobilize fluid in haemodynamically stable patients with IAH after acute resuscitation has been completed and the inciting issues have been addressed
5. We could make no recommendation regarding the prophylactic use of the open abdomen in non-trauma acute care surgery patients with physiologic exhaustion versus intraoperative abdominal fascial closure and expectant IAP management
6. We could make no recommendation regarding use of an acute component separation technique versus not, to facilitate earlier abdominal fascial closure

ACS: abdominal compartment syndrome; IAP: intra-abdominal pressure; IAH: intra-abdominal hypertension; PCD: percutaneous catheter drainage.

baseline IAP for the individual patient. Table 3 lists some well-recognised risk factors for IAH/ACS, and this is an area of active research by the WSACS.

DEFINITION 6 - Intra-abdominal hypertension; IAH is defined by a sustained or repeated pathologic elevation of IAP ≥ 12 mmHg

Pathological IAP is a continuum ranging from mild, asymptomatic elevations in IAP to marked elevations in IAP with severe consequences on virtually all organ systems in the body. The exact IAP that defines IAH has long been a subject of debate; however, the majority of animal studies show that visceral organ perfusion begins to decrease at an IAP of 10–15 mmHg [12]. It is at this level that cardiac, renal, hepatic and GI perfusion becomes compromised to the point that anaerobic metabolism may ensue followed by organ dysfunction and failure.

DEFINITION 7 - Grading; IAH is graded as follows:

- Grade I: IAP 12–15 mmHg;
- grade II: IAP 16–20 mmHg;
- grade III: IAP 21–25 mmHg;
- grade IV: IAP > 25 mmHg.

Patients with prolonged untreated elevations in IAP commonly manifest inadequate perfusion and subsequent organ failure. The more severe the degree of IAH, the more urgent is the need to reduce the pressure (either medically or surgically). Grading systems help to improve communication and improve clinical research.

DEFINITION 8 - Abdominal compartment syndrome; ACS is defined as a sustained IAP > 20 mmHg (with or without an APP < 60 mmHg) that is associated with new organ dysfunction/failure

ACS is best remembered as the presence of significant IAH with new-onset organ failure. Failure to recognize and appropriately treat ACS is often fatal whereas prevention and timely intervention are associated with marked improvements in organ function and patient survival. In contrast to IAH, ACS is not graded, but rather is considered an ‘all or nothing’ phenomenon. There are three different types of ACS, which are listed below under definitions 9–11.

DEFINITION 9 - Primary ACS is a condition associated with injury or disease in the abdomino-pelvic region that frequently requires early surgical or radiological intervention

Primary ACS is characterized by acute or subacute IAH of relatively brief duration occurring as a result of intra-

abdominal pathology, such as abdominal trauma (and lesions to spleen or liver), ruptured abdominal aortic aneurysm, haemoperitoneum, acute pancreatitis, secondary peritonitis, retroperitoneal haemorrhage or liver transplantation. It is most commonly encountered in the traumatically injured or postoperative surgical patient.

DEFINITION 10 - Secondary ACS refers to conditions that do not originate from the abdomino-pelvic region

Secondary ACS is characterized by subacute or chronic IAH that develops due to extra-abdominal pathology, such as sepsis, capillary leak and major burns, or other conditions requiring massive fluid resuscitation [33–36]. It is most commonly encountered in the medical or burn patient and can be considered iatrogenic related to excessive crystalloid resuscitation [37]. Secondary IAH can be considered as a frequent iatrogenic and thus avoidable problem. Recent studies show a relative decrease in secondary ACS compared to primary ACS due to increased awareness [38].

DEFINITION 11 - Recurrent ACS refers to the condition in which ACS redevelops following previous surgical or medical treatment of primary or secondary ACS

Recurrent ACS is a redevelopment of ACS symptoms following resolution of an earlier episode of either primary or secondary ACS. It may occur despite the presence of an open abdomen or as a new ACS episode following definitive closure of the abdominal cavity. Recurrent ACS (formerly termed tertiary IAH/ACS) is associated with high morbidity and mortality [39–41].

DEFINITION 12 - Poly-compartment syndrome; PCS is a condition where two or more anatomical compartments have elevated compartmental pressures

A compartment syndrome (CS) is defined as an increased pressure in a closed anatomic space which threatens the viability of enclosed and surrounding tissue [4]. Within the body there are four major compartments among many: the head, the chest, the abdomen and the extremities. Within each compartment, a CS can affect individual organs and the CS can be associated with different causal disease states. The abdominal compartment has unique topographic properties because it is ‘up-stream’ from the lower extremities and ‘down-stream’ from the chest. Therefore, it may influence the pathophysiology of these compartments. Scalea et al. were the first to introduce the term multiple CS (MCS) in a study of 102 patients with increased IAP, intrathoracic (ITP) and intracranial pressure (ICP) after severe brain injury [42]. He suggested that different compartments within the body are not isolated and

independent entities but instead are closely connected. Because the term multi or multiple CS is nowadays mostly used in relation to multiple limb trauma with CS needing fasciotomy, the term poly-compartment syndrome (PCS) was finally coined in 2007 in order to avoid further confusion [43,44]. Because of the clinical importance of diverse aspects of PCS, further classification in the future seems warranted. First, PCS can either be primary or secondary or a combination of both, in view of the potential effect on organ function [35]. A primary CS is defined as a pathological rise of CP in a compartment due to physical tissue or organ injury within the compartment (i.e. intracranial haematoma or limb fracture). In secondary CS, there is no primary injury in the affected compartment and symptoms are solely based on pressure transmission from one compartment to another (i.e. ACS that develops following a tension pneumothorax) [45]. Different conditions precipitate the occurrence of PCS: severe burns, massive fluid resuscitation, severe sepsis or prolonged hypotension.

DEFINITION 13 - Abdominal compliance; It is a measure of the ease of abdominal expansion, and is determined by the elasticity of the abdominal wall and diaphragm and is expressed as a change in intra-abdominal volume per change in intra-abdominal pressure

The abdominal compliance quantifies the ease of abdominal expansion and is determined by the elasticity of the anterior and lateral abdominal wall and to a smaller degree the diaphragm, whereas the more rigid spine and pelvis only minimally if at all affect abdominal elasticity. The abdominal compliance changes with changes in abdominal volume. It can be expressed as the slope on a volume–pressure curve and the slope will depend on its position on the abdominal volume–pressure curve as explained further.

DEFINITION 14 - Open abdomen; It is any abdomen requiring a temporary abdominal closure due to the skin and fascia not being closed after laparotomy. In order to facilitate research in this controversial field, the technical details regarding the type of temporary closure should be explicitly stated

The open abdomen continues to be variably defined, even in contemporary reviews, and surveys even among trauma surgeons reveal confusion as to exactly what anatomy constitutes an ‘open abdomen’ [46]. Surveys asking this simple question have noted surprising confusion and disagreement in regards to skin closure without fascial closure, or visceral containment with mesh interposition between fasciae with or without skin or soft-tissue closure.

DEFINITION 15 - Grading; the open abdomen is classified with a grading system

Planning to successfully and safely close any open abdomen must begin immediately after the abdomen is first left open. Clinical studies performed in this field should address abdominal closure rates considering the indications for open abdominal management, and compare abdominal closure problems of similar difficulty. This requires an open abdomen classification systems, two of which have been previously proposed, by Swan et al. [47] and Björck et al. [10]. The WSACS recognizes two critical complications which should be considered in managing an open abdomen; namely fixation of the abdominal contents to the abdominal wall, and the development of enteroatmospheric fistulae (EAF). The classification of Björck was therefore amended to reflect this hierarchy of challenges to the patient and this is listed in Table 4.

DEFINITION 16 - Lateralization of the abdominal wall refers to the phenomenon whereby the musculature and fascia of the abdominal wall, most well seen by the rectus abdominis muscles and their enveloping fascia, move laterally away from the midline with time

There are many recognized complications of the open abdomen, such as EAF; heat, fluid and protein losses; catabolism; and increased nursing resources, among many others. However, loss of domain, wherein the peritoneal contents no longer reside naturally within the confines of the abdominal wall, may be an overlooked concern. Although not well studied or reported, this phenomenon is increasingly being understood both as influencing the degree of complexity involved in abdominal wall reconstruction, and as an undesirable outcome that temporary abdominal closures aim to avoid [48].

Discussion

The initial definitions on IAH and ACS from 2006 and the therapeutic recommendations from 2007 formed the basis for research in this field. Since their initial publication, these consensus papers have been cited over 500 times confirming the need for standardisation. However, 7 years after the initial publications it became clear we are still lacking good level evidence for good recommendations with regard to the patient groups in which IAP measurements are mandatory, or when they become obsolete, or with regard to support interventions to lower IAP.

The terminology and definitions on AGI, IAH and ACS that are summarized herein have the purpose of providing a basis for further research on gastro-intestinal dysfunction and intra-abdominal hypertension and are useful for the daily

practice in the ICU. Until recently several definitions/symptoms have been used for AGI and there was no golden standard for evaluating the GI function/dysfunction [1,2,49]. The main problem remains, namely that there is still no objective measurement to evaluate the GI function. Again in these new definitions there are no useful objective measurable parameters that we can use at the bedside. The lack of good serum (bio)markers is probably the main cause for the lack of studies on the assessment of the GI dysfunction in the ICU and explains why the GI tract has not been included in organ failure scores like MODS, LODS or SOFA. Plasma citrulline and intestinal fatty acid binding protein (i-FABP) have been proposed as possible markers for small bowel function (and total enterocyte mass), but their clinical use in diagnosis and management of GI dysfunction is still unclear [50]. However, the present definitions and terminology for the GI function, IAP, IAH and ACS are definitely a great step forward and they form a good basis for future research.

A revival for studies on the GI tract may come from the recent big fluid trials that support the use of crystalloids over colloids like CHRYSTMAS [51], FEAST [52], 6S [53], FINNAKI [54], CHEST [55] and CRISTAL [56], with the ALBIOS study probably being published in the very near future. The European Medicines Agency (EMA)'s Pharmacovigilance Risk Assessment Committee (PRAC) has also just completed its review of HES solutions following an assessment of new information and commitments from companies for additional studies and risk minimization activities. The committee confirmed that HES solutions must no longer be used to treat patients with sepsis (bacterial infection in the blood) or burn injuries or critically ill patients, because of an increased risk of kidney injury and mortality. HES solutions may, however, continue to be used in patients to treat hypovolemia (low blood volume) caused by acute blood loss (especially in the perioperative setting of early goal-directed treatment in the operating room), provided that appropriate measures are taken to reduce potential risks and that additional studies are carried out. It may happen that after a couple of months in view of the CRISTAL data, a revision or fine-tuning of the PRAC statement may occur but the chances are very small. Meanwhile we'll have to adapt our resuscitation protocols avoiding starches and above all while we will use more crystalloids we must avoid fluid overload [57]. This is going to be the biggest challenge and it implicates that we must use crystalloids in a different way. While we have been using them in the past mainly for maintenance at a max rate of 84–200 ml/hour (or a bit more in burns), we should now treat them as a 'colloid' for resuscitation and give them in boluses of 250–500 ml over 15–30 min and then stop and re-assess macrohaemodynamics.

Fluid resuscitation indeed remains a two-edged sword as stated and for those clinicians who see a lot of trauma, col-

loids can be very useful for resuscitation especially if they are unlikely to need transfusion. Resuscitation to similar endpoints with colloids versus crystalloids gives virtually zero oedema when using the former. The use of (balanced) crystalloids is always associated with some conjunctival and bowel oedema, the latter leading to abdominal hypertension and ACS [7]. The mainly young trauma patients are very resilient and deal with oedema fairly well but there is slower feeding and more open abdomens due to bowel oedema. With the use of colloids, open abdomens became rare, whereas Bogota bags were almost inevitable when only crystalloids were used (personal communication Dr Eric Hodgson, Durban, South Africa). The use of colloids results in similar survival and shorter length of ICU and hospital stay when compared to crystalloids (in the setting of perioperative goal-directed therapy) [58–62]. The debate continues...

The mechanism of injury by capillary leak is widely recognized and accepted in the lung, where it is classified as acute lung injury (ALI) and acute respiratory distress syndrome (ARDS) [63]. The same pathological process occurs in the gut, but this concept is much slower to seep through into general ICU practice [64]. However, the role of the gut as the motor of organ dysfunction syndrome cannot be denied and difficulties in assessing gut function should not deter us from recognizing that concept. Even more than other organ dysfunction syndromes, some consider the gut as being the motor of the multiple organ dysfunction syndrome so AGI could have a negative impact on distant organ systems through the development of IAH, and could contribute to the development of acute kidney injury and ALI.

Recently the multicentre gastrointestinal failure (GIF) trial tried to develop a GI dysfunction score predicting 28-day mortality for adult patients needing mechanical ventilation; however, the pilot study did not allow to develop a valid score that was able to improve the accuracy of the SOFA score [65]. Such attempts must be continued, perhaps combining clinical evaluation with new biomarkers like i-FABP or citrulline.

Conclusion

The current definitions on AGI, IAH and ACS are not static, but dynamic, as they may evolve over time in case new measurements, parameters or biomarkers for GI function/dysfunction are developed or discovered. They are a first step in standardization of future studies on these topics.

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