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Results from the International Conference of Experts on Intra-abdominal Hypertension and Abdominal Compartment Syndrome. I. Definitions

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Abstract *Objective:* Intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS) have been increasingly recognized in the critically ill over the past decade. The variety of definitions proposed has led to confusion and difficulty in comparing one study to another. *Design:* An international consensus group of critical care specialists convened at the second World Congress on Abdominal Compartment Syndrome to standardize definitions for IAH and ACS based upon the current understanding of the pathophysiology surrounding these two syndromes. *Methods:* Prior to the conference the authors developed a blueprint for the various definitions, which was further refined both during and after the conference. The present article serves as the final report of the 2004 International ACS Consensus Defi-

nitions Conference and is endorsed by the World Society of Abdominal Compartment Syndrome (WSACS). *Results:* IAH is redefined as an intra-abdominal pressure (IAP) at or above 12 mmHg. ACS is redefined as an IAP above 20 mmHg with evidence of organ dysfunction/failure. ACS is further classified as either primary, secondary, or recurrent based upon the duration and cause of the IAH-induced organ failure. Standards for IAP monitoring are set forth to facilitate accuracy of IAP measurements from patient to patient. *Conclusions:* State-of-the-art definitions for IAH and ACS are proposed based upon current medical evidence as well as expert opinion. The WSACS recommends that these definitions be used for future clinical and basic science research. Specific guidelines and recommendations for clinical management of patients with IAH/ACS are published in a separate review.

Introduction

Interest in and clinical investigation into intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS) as causes of significant morbidity and mortality among the critically ill have increased exponentially over the past decade [1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 21, 22, 23, 24, 25, 26, 27, 28, 29, 30, 31, 32, 33, 34, 35, 36, 37]. Given the prevalence of elevated intra-abdominal pressure (IAP) as well as earlier detection and appropriate therapeutic management of IAH and ACS, significant decreases in patient morbidity and mortality have been achieved [2, 10, 11, 15, 17, 19, 23, 31]. As our understanding of the pathophysiology surrounding these two syndromes has evolved, IAP measurements have been identified as essential to the diagnosis and management of both IAH and ACS and have gained increasing prominence in intensive care units worldwide [38, 39, 40, 41, 42, 43, 44, 45, 46, 47, 48, 49, 50]. The accuracy and reproducibility of the methods promoted for measuring IAP, however, have been variable [1, 2, 38, 51, 52]. Similarly, the threshold values used to define the presence of IAH and ACS have lacked consensus. Some use the terms IAH and ACS interchangeably, resulting in conflicting definitions, confusion, and the inability to compare the results of published clinical trials [3, 53, 54, 55].

Given the growing awareness of IAH and ACS, and in response to an outcry for consensus from clinicians worldwide, this article proposes state-of-the-art definitions for IAH and ACS as well as standardized techniques for IAP monitoring to facilitate future research and improve patient care [4, 6, 36, 55].

Methods

While preparing for the second World Congress on Abdominal Compartment Syndrome (WCACS), several European, Australasian, and North American surgical, trauma, and medical critical care specialists recognized the lack of uniformity among current definitions for IAH and ACS. Confusion surrounding IAP monitoring and threshold IAP values inherent in the above definitions

Table 1 Consensus definitions list (*ACS* abdominal compartment syndrome, *APP* abdominal perfusion pressure, *FG* filtration gradient, *GFP* glomerular filtration pressure, *IAH* intra-abdominal hypertension, *IAP* intra-abdominal pressure, *MAP* mean arterial pressure, *PTP* proximal tubular pressure)

Definition 1	IAP is the steady-state pressure concealed within the abdominal cavity.
Definition 2	APP = MAP − IAP.
Definition 3	FG = GFP − PTP = MAP − 2 × IAP.
Definition 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 midaxillary line.
Definition 5	The reference standard for intermittent IAP measurement is via the bladder with a maximal instillation volume of 25 ml sterile saline.
Definition 6	Normal IAP is approx. 5–7 mmHg in critically ill adults.
Definition 7	IAH is defined by a sustained or repeated pathological elevation in IAP $\geq$ 12 mmHg.
Definition 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.
Definition 9	ACS is defined as a sustained IAP > 20 mmHg (with or without an APP < 60 mmHg) that is associated with new organ dysfunction/failure.
Definition 10	Primary ACS is a condition associated with injury or disease in the abdominopelvic region that frequently requires early surgical or interventional radiological intervention.
Definition 11	Secondary ACS refers to conditions that do not originate from the abdominopelvic region.
Definition 12	Recurrent ACS refers to the condition in which ACS redevelops following previous surgical or medical treatment of primary or secondary ACS.

was also noted. In early 2004, after extensively reviewing the existing literature, the authors suggested a conceptual framework for standardizing the definitions of IAH and ACS. They also suggested a general technique for IAP monitoring based upon current understanding of the pathophysiology of these two syndromes. This proposal was reviewed and further refined in anticipation of the WCACS meeting, which was endorsed by the European Society of Intensive Care Medicine.

The WCACS meeting was held 6–8 December 2004, in Noosa, Queensland, Australia, and was attended by 160 multidisciplinary critical care physicians and nurses from around the world. Consensus definitions were extensively discussed during the conference and a writing committee was formed to develop this article. After the conference participants corresponded electronically, providing feedback to questions and issues raised during the conference. This article serves as the final report of the 2004 International ACS Consensus Definitions Conference and is endorsed by the World Society of Abdominal Compartment Syndrome (WSACS).

During the whole writing process the authors kept up to date with the recent published literature on abdominal hypertension and the abdominal compartment syndrome. However, in order to be concise some recent references were not included in the list. The reader must take into account that as pointed out in the title this manuscript is the reflection of a consensus meeting of experts in the field, therefore some of the statements are based on expertise and clinical judgement and cannot be justified by a reference. A summary of the proposed consensus definitions is listed in Table 1.

Definitions

Intra-abdominal pressure

The abdomen can be considered a closed box with walls either rigid (costal arch, spine, and pelvis) or flexible (abdominal wall and diaphragm). The elasticity of the walls and the character of its contents determine the pressure within the abdomen at any given time [40, 51]. Since the abdomen and its contents can be considered as relatively noncompressive and primarily fluid in character, behaving in accordance to Pascal's law, the IAP measured at one point may be assumed to represent the IAP throughout the abdomen [38, 51]. IAP is therefore defined as the steady-state pressure concealed within the abdominal cavity. IAP increases with inspiration (diaphragmatic contraction) and decreases with expiration (diaphragmatic relaxation) [40]. It is also directly affected by the volume of the solid organs or hollow viscera (which may be either empty or filled with air, liquid or fecal matter), the presence of ascites, blood or other space-occupying lesions (such as tumors or a gravid uterus), and the presence of conditions that limit expansion of the abdominal wall (such as burn eschars or third-space edema).

- Definition 1: The intra-abdominal pressure (IAP) is the steady-state pressure concealed within the abdominal cavity.

Abdominal perfusion pressure

Analogous to the widely accepted and clinically utilized concept of cerebral perfusion pressure, calculated as mean arterial pressure (MAP) minus intracranial pressure (ICP), abdominal perfusion pressure (APP), calculated as MAP minus IAP, has been proposed as a more accurate predictor of visceral perfusion and a potential endpoint for resuscitation [11, 12, 56, 57]. APP, by considering both arterial inflow (MAP) and restrictions to venous outflow (IAP), has been demonstrated to be statistically superior to either parameter alone in predicting patient survival from IAH and ACS [57]. Further, multiple regression analysis has identified that APP is also superior to other common resuscitation endpoints including arterial pH, base deficit, arterial lactate, and hourly urinary output [11]. A target APP of at least 60 mmHg has been demonstrated to correlate with improved survival from IAH and ACS [11, 12, 57].

- Definition 2: APP = MAP – IAP.

Filtration gradient

Inadequate renal perfusion pressure (RPP) and renal filtration gradient (FG) have been proposed as key factors in the development of IAP-induced renal failure [57, 58, 59]. The FG is the mechanical force across the glomerulus and equals the difference between the glomerular filtration pressure (GFP) and the proximal tubular pressure (PTP). In the presence of IAH, PTP may be assumed to equal IAP and thus GFP can be estimated as MAP minus IAP. Thus changes in IAP will have a greater impact upon renal function and urine production than will changes in MAP. As a result, oliguria is one of the first visible signs of IAH [60, 61, 62].

- Definition 3: FG = GFP – PTP = MAP – 2 × IAP.

IAP measurement

Recent studies have shown that clinical judgement or physical examination is far from accurate in predicting a pa-

tient's IAP [41, 42]. With recognition of the importance of IAP monitoring in the diagnosis and management of IAH/ACS, a variety of methods for intermittent IAP measurement via either direct (i.e., needle puncture of the abdomen during peritoneal dialysis or laparoscopy) and indirect (i.e., transduction of intravesicular or "bladder," gastric, colonic or uterine pressure via balloon catheter) techniques have been suggested [38, 39, 40, 41, 42, 43, 44, 45, 46, 47, 48, 49, 50, 63, 64, 65]. Of these methods, the bladder technique has achieved the most widespread adoption worldwide due to its simplicity and minimal cost [38, 39, 46, 51]. Recently, several methods for continuous IAP measurement via the stomach, peritoneal cavity and bladder have been validated [43, 44, 45, 50]. Although these techniques seem promising, further clinical validation is necessary before their general use can be recommended.

Regardless of the technique utilized several key principles must be followed to ensure accurate and reproducible measurements from patient to patient. Early IAH studies utilized water manometers to determine IAP with results reported in cmH_2O [51, 66, 67]. Subsequent studies using electronic pressure transducers reported IAP in mmHg (1 mmHg = 1.36 cmH_2O). This has led to confusion and difficulty in comparing studies. Of further confusion has been the question of the zero reference point for the abdomen. Various authors have suggested using the symphysis pubis, the phlebostatic axis and the midaxillary line, each of which may result in different IAP measurements within the same patient [51]. Changes in body position (i.e., supine, prone, head of bed elevated) and the presence of both abdominal and bladder detrusor muscle contractions have also been demonstrated to impact upon the accuracy of IAP measurements [38]. Perhaps the greatest disparity among IAP measurement techniques has been the debate as to the proper priming-volume to be instilled into the bladder to ensure a conductive fluid column between bladder wall and transducer [68, 69]. Several studies have shown that high volumes may increase bladder pressure, especially at higher IAPs, such that measurements no longer reflect true abdominal pressure [46]. In an attempt to standardize and improve the accuracy and reproducibility of IAP measurements, the following definitions are proposed:

- Definition 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 midaxillary line.
- Definition 5: The reference standard for intermittent IAP measurement is via the bladder with a maximal instillation volume of 25 ml sterile saline.

Normal and pathological IAP values

In the strictest sense normal IAP ranges from subatmospheric to 0 mmHg [13]. Certain physiological conditions, however, such as morbid obesity or pregnancy may be associated with chronic IAP elevations of 10–15 mmHg to which the patient has adapted with an absence of significant pathophysiology [70, 71, 72, 73, 74, 75, 76, 77]. In contrast, children commonly demonstrate low IAP values [47]. The clinical importance of any IAP must be assessed in view of the baseline steady-state IAP for the individual patient.

In the critically ill, IAP is frequently elevated above the patient's normal baseline. Recent abdominal surgery, sepsis, organ failure, need for mechanical ventilation, and changes in body position are all associated with elevations in IAP (Table 2) [5, 13, 4, 15, 16, 17, 18, 19, 10, 21, 22, 67]. While some elevations are transient (lasting seconds to minutes), most are prolonged (lasting hours to days), potentially resulting in organ dysfunction and failure [78]. Before a diagnosis of IAH can be made, a sustained increase in IAP reflecting a new pathological

Table 2 Risk factors for IAH/ACS

Acidosis (pH < 7.2)
Hypothermia (core temperature < 33°C)
Polytransfusion (> 10 U packed red blood/24 h)
Coagulopathy (platelets < 55,000/mm^3 *or* activated partial thromboplastin time two times normal or higher *or* prothrombin time < 50% *or* international standardized ratio > 1.5)
Sepsis (American-European Consensus Conference definitions)
Bacteremia
Intra-abdominal infection/abscess
Peritonitis
Liver dysfunction/cirrhosis with ascites
Mechanical ventilation
Use of positive end expiratory pressure (PEEP) or the presence of auto-PEEP
Pneumonia
Abdominal surgery, especially with tight fascial closures
Massive fluid resuscitation (> 5 l colloid or crystalloid/24 h)
Gastroparesis/gastric distention/ileus
Volvulus
Hemoperitoneum/pneumoperitoneum
Major burns
Major trauma
High body mass index (> 30)
Intra-abdominal or retroperitoneal tumors
Prone positioning
Massive incisional hernia repair
Acute pancreatitis
Distended abdomen
Damage control laparotomy
Laparoscopy with excessive inflation pressures
Peritoneal dialysis

phenomenon or entity within the abdominal cavity must be demonstrated [23, 24, 25, 79].

- Definition 6: Normal IAP is approx. 5–7 mmHg in critically ill adults.

Intra-abdominal hypertension

Pathological IAP is a continuum ranging from mild IAP elevations without clinically significant adverse effects to substantial increases in IAP with grave consequences to virtually all organ systems in the body [53, 54, 59, 78, 80, 81, 82, 83, 84]. Although the use of a single IAP value to define IAH could be questioned, it is important that consensus on this point be reached in order to facilitate performing and interpreting future studies.

The exact IAP that defines IAH has long been a subject of debate. Early descriptions in the surgical literature favored an IAP of 15–18 mmHg (20–25 cmH_2O). Burch and coauthors [26] defined an early grading system for IAH/ACS (in cmH_2O) by which to guide therapy: grade I, 7.5–11 mmHg (10–15 cmH_2O); grade II, 11–18 mmHg (15–25 cmH_2O); grade III, 18–25 mmHg (25–35 cmH_2O); and grade IV, higher than 25 mmHg (> 35 cmH_2O). Burch et al. suggested that most patients with grade III and all patients with grade IV should undergo abdominal decompression.

The literature currently defines IAH variously between 12 and 25 mmHg, frequently based upon the deleterious effects on renal, cardiac, and gastrointestinal function witnessed at IAP levels as low as 10–15 mmHg [1, 2, 6, 15, 16, 19, 24, 25, 26, 27, 28, 29, 30, 31, 40, 42, 54, 52, 85, 86, 87, 88, 89, 90, 91, 92, 93, 94, 95, 96, 97, 98]. A recent multicenter study aimed at establishing the prevalence, cause, and predisposing factors associated with IAH in a mixed population of intensive care patients defined IAH as a maximal IAP value of 12 mmHg or higher [2]. The incorporation of pressures at which organ dysfunction becomes apparent in the majority of patients is appropriate in defining IAH [36, 54, 99]. While IAP clearly fluctuates in response to a patient's constantly changing physiology, the majority of studies to date have utilized maximal IAP values to define IAH rather than the potentially more relevant mean or median [52]. Given the familiarity of this methodology in institutions worldwide, all pressure values subsequently referred to herein correspond to the maximal IAP values from standardized intermittent bladder pressure measurements unless stated otherwise.

- Definition 7: IAH is defined by a sustained or repeated pathological elevation in IAP $\geq$ 12 mmHg.

The more severe the degree of IAH, the more urgent is the need for decompression of the abdomen (either medically or surgically) with resolution of the damaging pressure [100, 101, 102]. Based upon our current understanding of IAH/ACS, a modification of the original Burch et al. grading system is appropriate to stratify patients with elevated IAP and guide clinical treatment.

- Definition 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

IAH may also be subclassified according to the duration of symptoms into one of four groups [36]. *Hyperacute* IAH represents elevations in IAP that last but a few seconds or minutes as a result of laughing, straining, coughing, sneezing, defecation or physical activity. *Acute* IAH develops over a period of hours and is seen primarily in surgical patients as a result of trauma or intra-abdominal hemorrhage. This fulminant example of IAH commonly leads to rapid development of ACS. *Subacute* IAH occurs over a period days and is the form most commonly encountered in medical patients [103, 104]. It results from a combination of causal factors and predisposing conditions (Table 2). *Chronic* IAH develops over a period of months (i.e., pregnancy) or years (i.e., morbid obesity, intra-abdominal tumor, peritoneal dialysis, chronic ascites or cirrhosis) and may place patients at risk for developing either acute or subacute IAH when critically ill [74, 83, 105, 106, 107, 108, 109, 110, 111]. Developing over a protracted time course, the abdominal wall adapts and progressively distends in response to increasing IAP allowing time for the body to adapt physiologically. While only the latter three are of major importance in the critically ill, clinical consideration of these IAH subtypes is useful in anticipating patients at risk for ACS.

Abdominal compartment syndrome

IAH clearly represents a continuum with IAP varying from patient to patient and from moment to moment according to underlying causal factors, cardiac filling status, presence of organ failure and preexisting comorbidities (Fig. 1) [53, 54, 78, 99, 112, 113]. Critical IAP in the majority of patients, as outlined above, appears to reside somewhere between 10 and 15 mmHg [1, 48]. It is at this pressure that reductions in microcirculatory blood flow occur, and the initial development of organ dysfunction and failure is first witnessed [82, 90, 92, 93, 94, 95, 114, 115]. ACS is the natural progression of these pressure-induced end-organ

changes and develops if IAH is not recognized and treated in a timely manner. Although the critical IAP that defines ACS is subject to debate, of greater importance than any one absolute IAP value is the development of organ dysfunction and failure [9].

ACS has been variably defined over the years based upon the existing understanding of its pathophysiology. Fietsam et al. [96] first described a syndrome in four surgical patients who developed oliguria, hypoxia, hypercarbia, high peak inspiratory pressures, and a tense abdomen. To separate IAH from ACS, Ivatury et al. [22] characterized ACS by the presence of a tensely distended abdomen, elevated intra-abdominal and peak airway pressures, inadequate ventilation with hypoxia and hypercarbia, impaired renal function, and a documented improvement of these features after abdominal decompression. ACS was thereby seen as a late manifestation of uncontrolled IAH. Meldrum et al. [30] defined ACS as an IAP higher than 20 mmHg complicated by one of the following: peak airway pressure above 40 cmH_2O, oxygen delivery index less than 600 ml O_2 min^{-1} m^{-2} or urine output under 0.5 ml $kg^{-1}h^{-1}$. Similar characteristics in different combinations and with additions of persistently low pHi, labile blood pressure, diminished cardiac output, tachycardia with or without hypotension, or oliguria have subsequently been used by other authors [32, 33, 34].

These definitions were later adapted and used to form the generally accepted definition called the "triad" of ACS: (a) a pathological state caused by an acute increase in IAP above 20 to 25 mmHg, which (b) adversely affects end-organ function or can cause serious wound complications, and in which (c) abdominal decompression has beneficial effects [32, 35]. Failure to recognize and appropriately treat ACS is uniformly fatal whereas prevention and/or timely intervention is associated with marked improvements in organ function and overall patient survival [88].

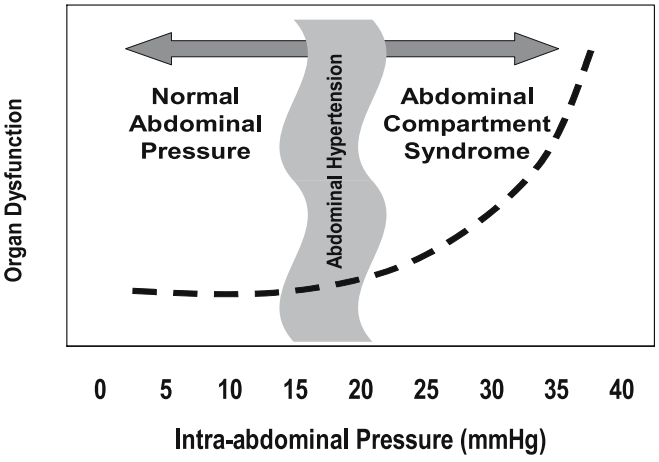

Fig. 1 Distinctions between normal intra-abdominal pressure, IAH, and ACS. *Shaded area* illustrating IAH may undergo shifts to the right or left depending on the clinical scenario (adapted from [36])

A more accurate definition of ACS will enhance the comparison of studies from different centers and will be helpful in designing future clinical trials. Such a definition must incorporate a numerical IAP value with the significant clinical consequences of prolonged IAH, such as the development of organ failure. In two recent studies, Malbrain et al. [1, 2] defined ACS as an IAP of 20 mmHg or higher with failure of one or more organ systems as depicted by a Sequential Organ Failure Assessment organ score or 3 or more [116]. In contrast to IAH, ACS should not be graded, but rather considered as an "all or nothing" phenomenon [54].

- Definition 9: ACS is defined as a sustained IAP > 20 mmHg (with or without an APP < 60 mmHg) that is associated with new organ dysfunction/failure.

Classification of IAH/ACS

Although initially considered a disease of the traumatically injured, IAH/ACS is now recognized as a cause of significant organ failure, morbidity and mortality in all critically ill patient populations [1, 36, 40]. Given the broad multitude of predisposing conditions that may lead to the development of IAH/ACS, we believe it is useful to classify ACS as either primary, secondary, or recurrent according to the duration and cause of the patient's IAH [9].

The duration of IAH, in conjunction with the acuity of onset as described above, is commonly of greater prognostic value than the absolute increase in IAP. Patients with prolonged untreated elevations in IAP commonly manifest inadequate perfusion and subsequent organ failure [9]. Preexisting comorbidities, such as chronic renal failure, pulmonary disease, or cardiomyopathy, play an important role in aggravating the effects of elevated IAP and may reduce the threshold of IAH that causes clinical manifestations of ACS [9, 51, 54]. The cause of the patient's IAH is similarly of vital importance and may be determined as being either intra-abdominal, as occurs in surgical or trauma patients following damage control laparotomy, or extra-abdominal, as occurs in medical patients with sepsis or burn patients who require aggressive fluid resuscitation [3, 15, 117, 118].

Primary ACS (formerly termed surgical, postoperative, or abdominal ACS) is characterized by the presence of acute or subacute IAH of relatively brief duration occurring as a result of an intra-abdominal cause such as abdominal trauma, ruptured abdominal aortic aneurysm, hemoperitoneum, acute pancreatitis, secondary peritonitis, retroperitoneal hemorrhage, or liver transplantation [54, 99]. It is most commonly encountered

Table 3 Clinical application of IAH/ACS classification (*ACS* abdominal compartment syndrome, *APP* abdominal perfusion pressure, *IAH* intra-abdominal hypertension, *IAP* intra-abdominal pressure)

Patient scenario	Duration	Class	Cause	IAH grade
Chronic liver failure complicated with a pneumonia and an IAP of 18 mmHg	Chronic	Primary	Medical	II
Blunt thoracoabdominal trauma with severe liver injury, hypotension, high airway pressures; initial IAP 40 mmHg	Acute	Primary	Trauma	IV
Chronic liver failure complicated with variceal bleeding and cardiorespiratory collapse and an IAP of 22 mmHg	Acute	Primary	Medical	III
Blunt abdominal trauma with severe liver injury; damage control laparotomy performed with successful resolution of primary ACS; abdominal closure is performed 2 weeks later; oliguria develops on postoperative day 3 with IAP 28 mmHg and APP < 50 mmHg	Subacute	Recurrent	Trauma	IV
Penetrating cardiac injury, with cardiorespiratory collapse requiring massive resuscitation; cardiac injury repaired but IAP increases above 21 mmHg on the third day of hospitalization	Subacute	Secondary	Trauma	III
Septic shock related to a pneumonia with an IAP of 13 mmHg on admission	Acute	Secondary	Medical	I
Septic shock due to intestinal perforation and an IAP of 25 mmHg before going to the operating theater	Acute	Primary	Surgical	IV
Severe burns to abdomen and chest develops an IAP > 20 mmHg on day 7	Subacute	Secondary	Burn	III
Chronic renal failure on low molecular weight heparins develops a rectus sheath, psoas, and retroperitoneal hematoma with an IAP of 25 mmHg	Acute	Primary	Medical	IV

in the traumatically injured or postoperative surgical patient.

- Definition 10: Primary ACS is a condition associated with injury or disease in the abdominopelvic region that frequently requires early surgical or interventional radiological intervention.

Secondary ACS (formerly termed medical or extra-abdominal ACS) is characterized by the presence of subacute or chronic IAH that develops as a result of an extra-abdominal cause such as sepsis, capillary leak, major burns, or other conditions requiring massive fluid resuscitation [3, 8, 15, 54, 103, 104, 118, 119]. It is most commonly encountered in the medical or burn patient [4, 6, 36, 104].

- Definition 11: Secondary ACS refers to conditions that do not originate from the abdominopelvic region.

Recurrent ACS (formerly termed tertiary ACS) represents a redevelopment of ACS symptoms following resolution of an earlier episode of either primary or secondary ACS [54]. It is most commonly associated with the development of acute IAH in a patient who is recovering from IAH/ACS and therefore represents a "second-hit" phenomenon. It may occur despite the presence of an open abdomen (known as the "open abdomen compartment syndrome") or as a new ACS episode following definitive closure of the abdominal wall [120]. Recurrent ACS, due to the patient's current or recent critical illness, is associated with significant morbidity and mortality [10].

- Definition 12: Recurrent ACS refers to the condition in which ACS redevelops following previous surgical or medical treatment of primary or secondary ACS.

Occasionally patients demonstrate signs and symptoms consistent with both primary and secondary ACS. An example is a patient who develops sepsis with fluid overload after initial surgical stabilization for trauma [4, 118]. This overlap of clinical conditions and potential causes has added to the confusion regarding the definition of ACS. Nevertheless, the majority of IAH/ACS patients may be assigned to one of these three classes. The clinical application of such a classification system is depicted in Table 3.

Summary

Significant progress has been made over the past decade towards understanding the cause and pathophysiology surrounding IAH and ACS. This review proposes state-of-the-art definitions for IAH and ACS that are based upon current medical evidence as well as expert opinion. No clinical definition can include all possible conditions and variations of an inherently complex phenomenon. Nevertheless, the WSACS hopes that this consensus document

will serve as a practical yet comprehensive framework for both interpreting past research and planning future clinical trials, perhaps allowing the development of more accurate and appropriate definitions as our understanding of IAH and ACS is further enhanced. Specific guidelines and recommendations for the clinical management of patients with IAH/ACS are published in a separate review.

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