Abdominal Compartment Syndrome

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Rao R. Ivatury

9.1 Introduction

Abdominal compartment syndrome (ACS) has tremendous relevance in the care of critically ill or injured patients, because of the effects of elevated pressure within the confined space of the abdomen on multiple organ systems. Our knowledge of this lethal syndrome has evolved over the past 15 years. Of note, we now recognize that ACS should be viewed as the end result of a progressive, unchecked rise in IAP, called intra-abdominal hypertension (IAH), as illustrated in Fig. 9.1. We also have learnt that the adverse effects of elevated IAP occur at lower levels than previously thought [1].

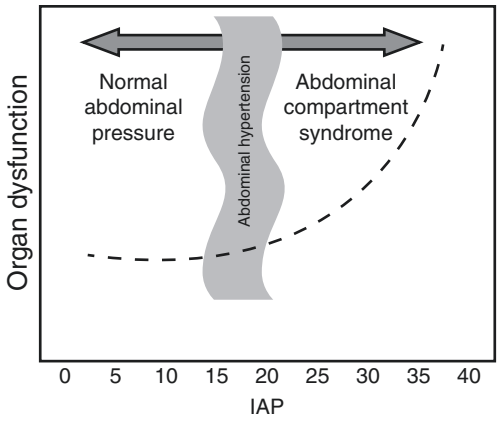

Fig. 9.1 Progression of IAP (intra-abdominal pressure) from normal to IAH (intra-abdominal hypertension) to abdominal compartment syndrome with organ failures

9.2 Historical Background

The pathophysiology of IAH has been known since late 1800s [1]. Marey (1863), Braune (1865), Schatz (1872), Wendt of Germany (1873), Oderbrecht of Germany (1875), Quinke of Germany (1878), Mosso and Pellacani of Italy (1882), and Heinricius of Germany (1890) all described the ill effects of IAH. In the next century, Emerson (1911), Bradley (1947), Gross (1948), Olerud (1953), Kashtan (1981), Harman (1982), Richards (1983), and Kron, Harman, and Nolan (1984) were the greatest contributors in the field. Fietsam et al. from William Beaumont Hospital, Royal Oak, Michigan, were the architects of the term intra-abdominal compartment syndrome [2]. They described it: "In four patients with ruptured abdominal aortic aneurysms….manifested by increased ventilatory pressure, increased central venous pressure, and decreased urinary output associated with massive abdominal distension not due to bleeding. This set of findings constitutes an intra-abdominal compartment syndrome … Opening the abdominal incision was associated with dramatic improvements." As with many advances in

R.R. Ivatury, MD, FACS, FCCM
Virginia Commonwealth University,
Richmond, VA, USA
e-mail: raoivatury@gmail.com

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medicine, this precious knowledge was forgotten, rediscovered, and forgotten again. Many proponents of the syndrome faced skepticism and ridicule till the clinical syndrome was rediscovered in patients with life-threatening abdominal injuries undergoing "damage-control" surgery. In this group of patients, IAH became a prime cause of avoidable morbidity and mortality [3–11].

Our knowledge of IAH and ACS was spurred by the shared experiences of trauma centers dealing with the nightly horrors of "America's uncivil war" (CW Schwab). The phenomena were codified by trauma surgeons who popularized the clinical practice of such advances as IAP monitoring by bladder pressure and non-closure of fascia after laparotomy ("open abdomen"). The consequent results were nothing short of dramatic [3, 4, 8]. Further advances were also realized through the efforts of a remarkable group of clinical researchers interested in the subject. After a preliminary meeting in 2001 in Sydney, Sugrue and associates formally established the World Society of Abdominal Compartment Syndrome (WSACS) in 2004 in Noosa in Australia. This society, though a small group of motivated intensivists, redefined the current concepts of IAH and ACS through multinational clinical trials, literature review and analysis, multiple publications including a monograph on the subject [1] and guideline and consensus development [12–15].

With this historic background, this chapter will summarize the WSAC consensus definitions, a brief review of pathophysiology, and WSACS recommendations and algorithms. It will then evaluate their impact on the current status of IAH in critically injured or ill patients and offer some projections for the future. The issues of "open abdomen" approach for prevention of IAH and ACS, while highly relevant, are beyond the scope of this chapter, however.

9.3 Current Definitions of IAH and ACS [12, 13]

IAP Intra-abdominal pressure (IAP) is the pressure concealed within the abdominal cavity. 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. The reference standard for intermittent IAP measurement is via the bladder with a maximal instillation volume of 25 mL of sterile saline. Normal IAP is approximately 5–7 mmHg in critically ill adults.

IAH Intra-abdominal hypertension is defined by a sustained or repeated pathologic elevation of IAP ≥12 mmHg. It is graded as follows: grade I, IAP 12–15 mmHg; grade II, IAP 16–20 mmHg; grade III, IAP 21–25 mmHg; and grade IV, IAP >25 mmHg.

ACS Abdominal compartment syndrome (ACS) is defined as a sustained IAP >20 mmHg (with or without an APP [MAP-IAP] <60 mmHg) that is associated with new organ dysfunction/failure. It should be noted that this definition moves ACS to a much earlier point in the trajectory of clinical course than the traditional fully manifested syndrome with multiorgan failure.

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 refers to conditions that do not originate from the abdominopelvic region. Recurrent ACS refers to the condition in which ACS redevelops following previous surgical or medical treatment of primary or secondary ACS.

Risk Factors for IAH/ACS [12–15] ACS can develop in both nonsurgical and surgical patients. These factors include diminished abdominal wall compliance (abdominal surgery, major trauma, major burns, prone positioning), increased intraluminal contents (e.g., acute pancreatitis, hemoperitoneum/pneumoperitoneum or intraperitoneal fluid collections, intra-abdominal infection/abscess, intra-abdominal or retroperitoneal tumors, liver dysfunction/cirrhosis with ascites, capillary leak/fluid resuscitation from massive fluid resuscitation or positive fluid balance), and damage-control laparotomy. Other miscellaneous causes include bacteremia, coagulopathy, massive incisional hernia repair, obesity or increased body mass index, PEEP, peritonitis, and sepsis.

9.4 Pathophysiology of ACS

IAH affects multiple organ systems in a graded fashion.

9.4.1 Cardiovascular Effects

Elevation in IAP leads to a reduction in cardiac output (CO) [9–11], most consistently seen at an IAP >20 mmHg. This is due to a combination of decreased inferior vena caval flow and an increased thoracic pressure (which decreases both inferior and superior vena caval flow). Other contributory factors include cardiac compression, decreased ventricular end-diastolic volumes, and marked increase in systemic afterload. This may lead to spuriously elevated central venous pressure, pulmonary artery pressure, and pulmonary artery occlusion ("wedge") pressure. Combined with a reduced CO, this may erroneously suggest a state of hypervolemia or biventricular failure [1, 9–11]. Improvement in CO after a saline fluid bolus may be therapeutic and clarify the diagnostic conundrum.

9.4.2 Pulmonary Dysfunction

With an acute elevation in IAP, respiratory failure characterized by high ventilatory pressures, hypoxia, and hypercarbia eventually develops [9–11]. Diaphragmatic elevation leads to a reduction in static and dynamic pulmonary compliance. The increase in IAP also reduces total lung capacity, functional residual capacity, and residual volume [9–11]. These lead to ventilation-perfusion abnormalities and hypoventilation producing hypoxia and hypercarbia, respectively. A porcine model by Simon et al. demonstrated that prior hemorrhage and resuscitation (ischemia-reperfusion injury) exacerbate the cardiopulmonary sequelae of IAH [16]. Chronic elevation of IAP, as in central obesity, also produces these derangements in the form of obesity hypoventilation syndrome (OHS) [11]. Abdominal decompression improves the acute respiratory failure almost immediately [9–11].

9.4.3 Renal Sequelae

Oliguria progressing to anuria and prerenal azotemia unresponsive to volume expansion characterize the renal dysfunction of ACS [1, 9–18]. Oliguria can be seen at IAP of 15–20 mmHg, while increases to 30 mmHg or above lead to anuria. Volume expansion to a normal CO and the use of dopaminergic agonists or loop diuretics may be ineffective in these patients. However, decompression and reduction in IAP promptly reverses oliguria, usually inducing a vigorous diuresis [17, 18]. The mechanisms of renal derangements with IAH involve reduced absolute and proportional renal arterial flow, increased renal vascular resistance with changes in intrarenal regional blood flow, reduced glomerular filtration, and increased tubular sodium and water retention [1, 17, 18].

9.4.4 Abdominal Visceral Abnormalities

Mesenteric arterial, hepatic arterial, intestinal mucosal, hepatic microcirculatory, and portal venous blood flow all have been shown to be reduced with IAH in animal models [19, 20]. Clinically, many investigators demonstrated that gut mucosal acidosis, demonstrable by intramucosal pH (pHi) measured with gastric tonometry, is a sensitive change after ACS [1, 4, 21]. Further increases in IAP may lead to intestinal infarction, often present in the ileum and right colon without arterial thrombosis. Prolonged low-grade elevation of IAP may be associated with bacterial translocation in rat and murine models [22]. Thus, despite normal systemic hemodynamics, profound splanchnic ischemia can be ongoing with IAH. It has been suggested that such ischemia is associated with an increased incidence of multisystem organ failure, sepsis, and increased mortality [1, 4, 9–11]. Furthermore, laboratory evidence suggests that prior hemorrhage and resuscitation actually lowers the critical levels of IAP at which mesenteric ischemia begins [23]. Many investigators note a relationship between IAH, sepsis, multisystem organ failure, and the need for reoperation and mortality. These are

some of the strongest arguments for the routine measurement of *IAP in critically ill patients.*

9.4.5 Abdominal Wall Abnormalities

Increased IAP has been shown to reduce abdominal wall blood flow by the direct, compressive effects of IAH under conditions of stable systemic perfusion, leading to local ischemia and edema [24]. This can decrease abdominal compliance (defined as a measure of the ease of abdominal expansion, which is determined by the elasticity of the abdominal wall and diaphragm and expressed as the change in intra-abdominal volume per change in IAP) and exacerbate IAH. Abdominal wall muscle and fascial ischemia may contribute to such wound complications as dehiscence, herniation, and necrotizing fasciitis.

9.4.6 Intracranial Derangements

Elevated intracranial pressure (ICP) and reduced cerebral perfusion pressure (CPP) have been described with acute changes in IAP in animal models and in human studies [25]. In animal models, the changes in ICP and CPP are independent of changes in pulmonary or cardiovascular function and appear to be the direct result of elevated intrathoracic and central venous pressures with impairment of cerebral venous outflow. Reduction in IAP by surgical decompression reverses this derangement. Furthermore, chronic elevation in IAP has been implicated as an important etiologic factor in the development of benign intracranial hypertension, or pseudotumor cerebri, in the morbidly obese [11].

9.4.7 Polycompartment Syndrome [25]

A polycompartment syndrome, where two or more anatomical compartments have elevated compartmental pressures, is a potential companion of IAH, e.g., intra-abdominal leading to intrathoracic and consequent intracranial hypertension. IAH helps to explain the severe pathophysiological condition occurring in patients with cardiorenal, hepatopulmonary, and hepatorenal syndromes. When more than one compartment is affected, an exponential detrimental effect on end-organ function in both immediate and distant organs may occur. The compliance of each compartment is the key to determining the transmission of a given compartmental pressure from one compartment to another. In high-risk patients, these interactions must be considered for optimal management [25].

9.5 Recommendations in Management

The following are the recommendations from WSACS [13] in the clinical pursuit of IAH and ACS based on the GRADE methodology (grading, assessment, development, and evaluation). Quality of evidence is graded from high (A) to very low (D). Recommendations range from strong recommendations to weaker suggestions.

The *recommendations* include use of protocolized monitoring and management of IAP [GRADE 1C], efforts and/or protocols to avoid sustained IAH among critically ill or injured patients [GRADE 1C], decompressive laparotomy [27] in cases of overt ACS [GRADE 1D], conscious and/or protocolized efforts be made among ICU patients with open abdominal wounds to obtain an early or at least same-hospital-stay abdominal fascial closure [GRADE 1D], and among critically ill/injured patients with open abdominal wounds, use of strategies utilizing negative pressure wound therapy [GRADE 1C].

The *suggestions* include the following: clinicians ensuring that critically ill or injured patients receive optimal pain and anxiety relief [GRADE 2D]; brief trials of neuromuscular blockade as a temporizing measure in the treatment of IAH/ACS [GRADE 2D]; considering the potential contribution of body position to elevated IAP among patients with, or at risk of, IAH or ACS

[GRADE 2D]; liberal use of enteral decompression with nasogastric or rectal tubes when the stomach or colon is dilated in the presence of IAH/ACS [GRADE 1D]; neostigmine be used for the treatment of established colonic ileus not responding to other simple measures and associated with IAH [GRADE 2D]; 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 [GRADE 2C]; use of an enhanced ratio of plasma/packed red blood cells for resuscitation of massive hemorrhage versus low or no attention to plasma/packed red blood cell ratios [GRADE 2D]; use of percutaneous decompression to remove fluid (in the setting of obvious intraperitoneal fluid) in those with IAH/ACS as, when this is technically possible [GRADE 2C], this may also alleviate the need for decompressive laparotomy [GRADE 2D]; trauma patients with physiologic exhaustion undergoing abbreviated laparotomy be treated with the prophylactic use of the open abdomen and expectant IAP management [GRADE 2D]; not routinely utilizing the open abdomen for patients with severe intraperitoneal contamination undergoing emergency laparotomy for intra-abdominal sepsis unless IAH is a specific concern [GRADE 2B]; and avoiding the routine use of bioprosthetic meshes in the early closure of the open abdomen compared to alternative strategies [GRADE 2D].

The WSACS noted that the evidence did not support any recommendations about the use of abdominal perfusion pressure (MAP-IAP) in the resuscitation or management of the critically ill or injured and the use of diuretics, albumin, or renal replacement therapy to mobilize fluids in hemodynamically stable patients with IAH after the acute resuscitation has been completed.

9.6 Management of IAH and ACS [13]

The most effective approach in the management of IAH and ACS is best summarized by the algorithms recommended by WSACS (Figs. 9.2 and 9.3).

9.7 Current Status of IAH and ACS

As noted earlier, the efforts of WSACS made a profound impact on our understanding of the disease and our clinical approach. Anticipation of the complication, measures of prophylaxis, earlier recognition, and intervention: all of these translated into fewer organ failures and better survival. The complications of the open abdomen may be offsetting some of these benefits. More advanced care of "laparotomy," however, is minimizing this weakness.

In a prospective, observational study, Cheatham and Safcsak [28] studied 478 consecutive patients who were treated with open abdomen for IAH and ACS according to "a continually revised management algorithm" and noted a significantly increased patient survival to hospital discharge from 50% to 72% ($p = 0.015$) and an increase in same-admission primary fascial closure from 59% to 81% over the period of the study, one of the first clinical series showing better outcomes with a management focus on IAP that did not increase resource utilization. Balogh and associates [29] prospectively analyzed 81 consecutive severely injured shock/trauma patients (mean ISS 29) admitted to the intensive care unit. They had a protocol of two-hourly intra-abdominal pressure (IAP) monitoring. No patient developed ACS, even though 61 (75%) had IAH. One patient with IAH and one without died. Multiorgan failure occurred in one patient without IAH (5%) vs. 4 with IAH (7%). The authors commented that monitoring and intervening for a less serious IAH and the avoidance of the deadly ACS are remarkable successes of critical care in the last decade. Recent evidence concluded that the current practice of restricted fluid resuscitation and liberal use of damage-control strategies among trauma patients along with monitoring for IAH has lowered the prevalence of ACS [30]. While established trauma centers and academic institutions were eliminating ACS by aggressive application of the concepts narrated in this chapter, these paradigms apparently have yet to be promulgated widely.

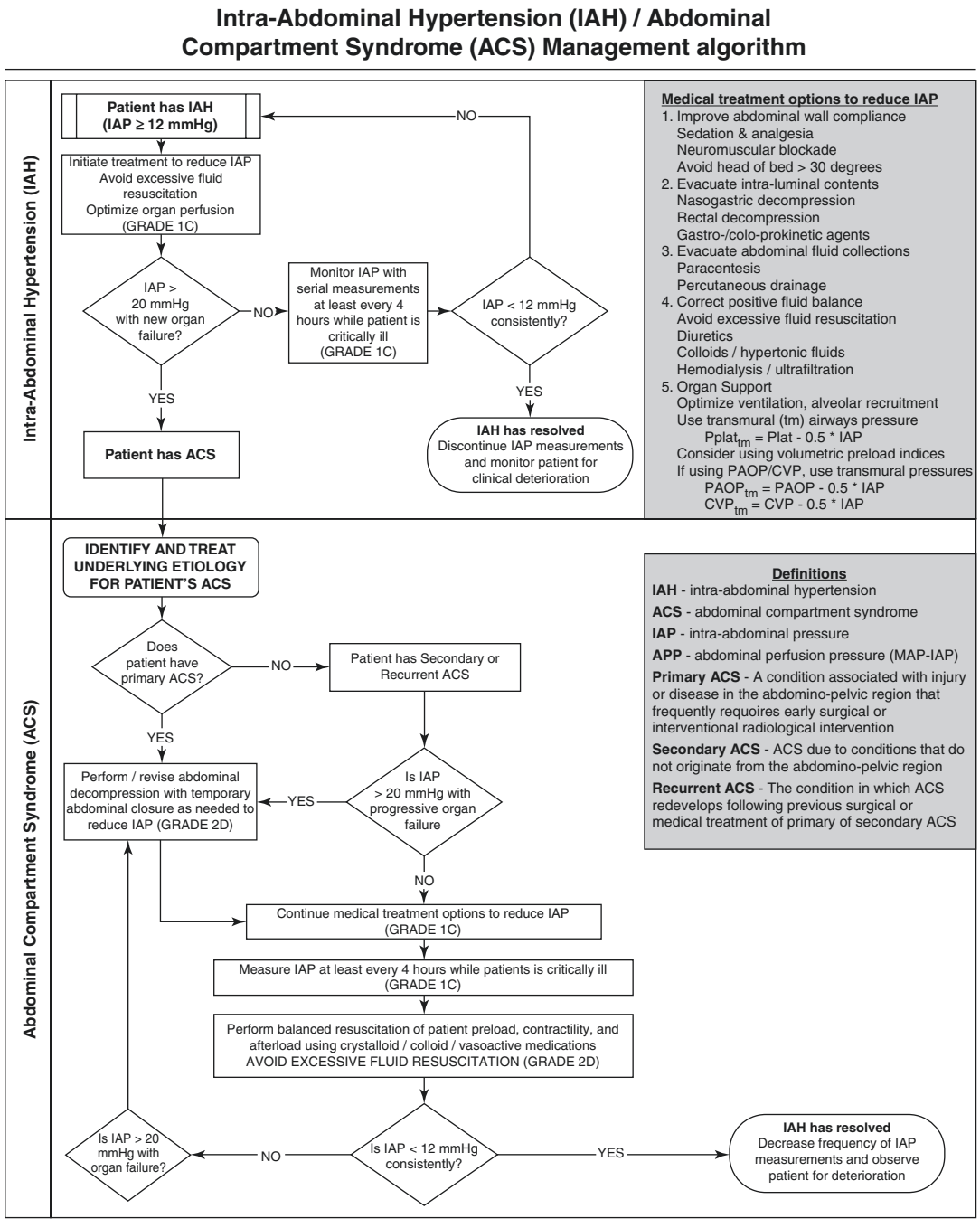

Fig. 9.2 Management algorithm for IAH and ACS (Reproduced from Kirkpatrick et al. [13])

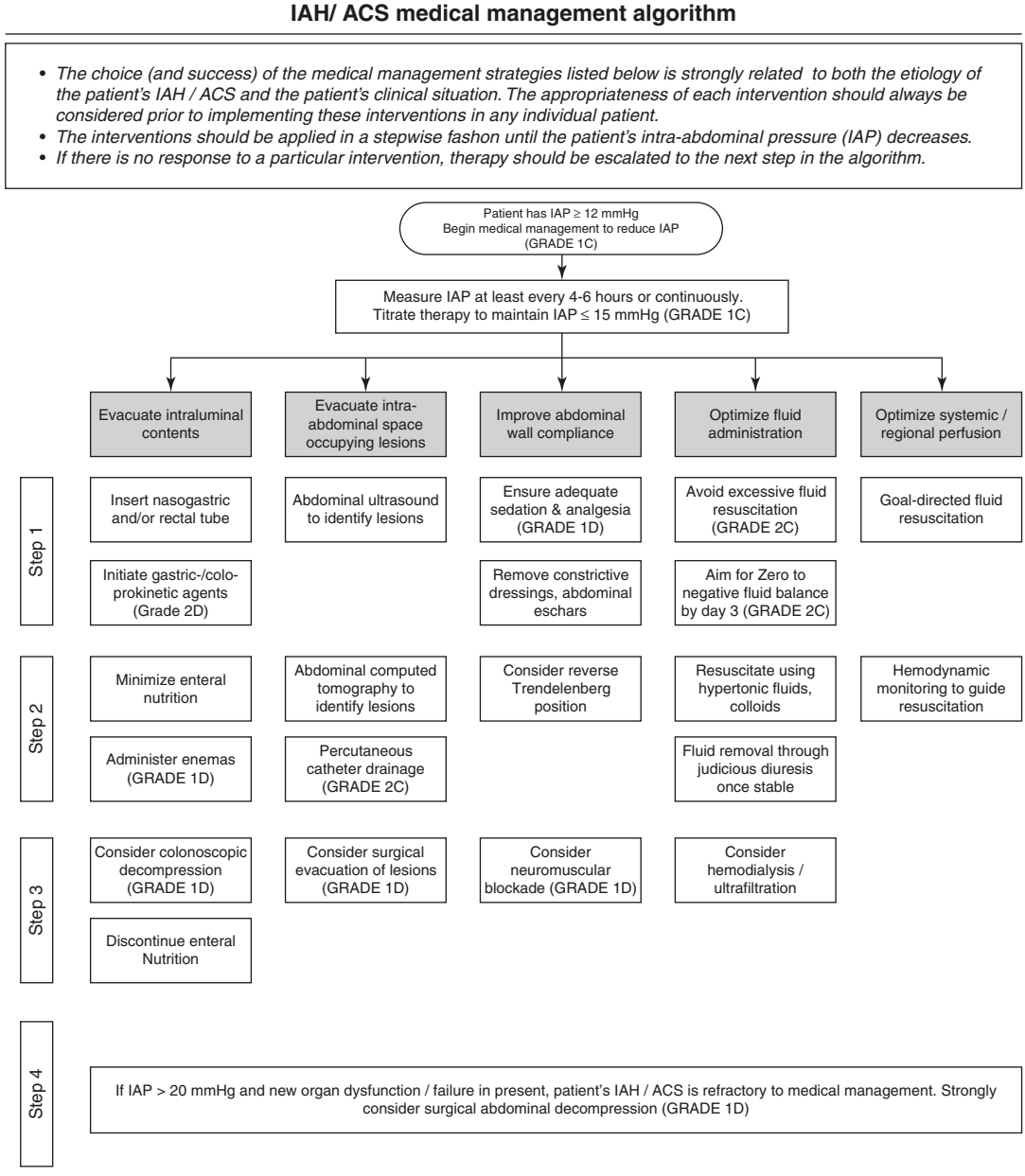

Fig. 9.3 Medical management of IAH and ACS (Reproduced from Kirkpatrick et al. [13])

9.8 Awareness and Appreciation of IAH and ACS

In 2013 the WSACS distributed a survey of 13 questions to 10,000 members of the WSACS, the European Society of Intensive Care Medicine (ESICM), and the Society of Critical Care Medicine (SCCM). A total of 2244 clinicians responded (response rate, 22.4%), a majority from North America. The majority of responders (85%) were familiar with IAP/IAH/ACS, but only 28% were aware of the WSACS consensus definitions. Overall knowledge scores were low (43 ± 15%). Respondents that were aware of the WSACS had a better score compared to those who were not (49.6% vs. 38.6%, $P < 0.001$), suggesting ignorance of established consensus definitions and guidelines [31]. Another study [32] surveyed Dutch surgeons with a literature-based and expert consensus survey. Sixty of 87 (69%) invited surgeons completed the questionnaire. Many of these surgeons exhibited a good knowledge of IAH and ACS, but only 27% used this in their daily practice. Another survey tried to clarify the current understanding and clinical management of intra-abdominal hypertension (IAH)/abdominal compartment syndrome (ACS) among Chinese intensive care physicians in tertiary hospitals [33]. The study concluded that urgent systematic education is absolutely necessary for most intensive care physicians in China to help to establish clear diagnostic criteria and appropriate management. A similar lack of application of definitions and guidelines was reported among German pediatric intensivists [34] and Australian critical care nurses [35].

In summary, IAH and ACS are common complications in the care of the critically ill or injured patients, medical or surgical, young or old. They can cause profound morbidity and mortality, if unanticipated, unrecognized, and uncontrolled. Appropriate monitoring and early intervention, based on the precepts of WSACS, can minimize organ failures, morbidity, and mortality. It appears, however, that the dissemination of the current knowledge of IAH and ACS is yet incomplete. It is definitely time to promulgate the pathophysiology of increased pressure in rigid compartments [36].

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