INTRA-ABDOMINAL PRESSURE MONITORING

SUMMARY

Elevated intra-abdominal pressure (IAP) is commonly encountered in the critically ill, has detrimental effects on all organ systems, and is associated with significant morbidity and mortality. Serial IAP measurements are essential to the diagnosis, management, and fluid resuscitation of patients who develop intra-abdominal hypertension (IAH) and/or abdominal compartment syndrome (ACS). Intravesicular pressure (IVP) is easily measured and should be monitored in all patients believed to be at risk for significant elevations in IAP.

RECOMMENDATIONS

- **Level 1**
  - IAP should be measured with consistent body position to allow consistent trending of IAP. The transducer should be set at a consistent reference point.
- **Level 2**
  - Patients should be screened for IAH/ACS risk factors upon ICU admission and in the presence of new or progressive organ failure.
  - If two or more risk factors for IAH/ACS are present, a baseline IAP measurement should be obtained.
  - If IAH is present on baseline assessment, serial IAP measurements should be performed throughout the patient's critical illness.
- **Level 3**
  - IVP should be monitored using a closed technique.
  - IAP should be in mmHg (1 mmHg = 1.36 cm H_2O).
  - IAP should be measured in the supine position, at end-expiration, with the transducer zeroed at the mid-axillary line, 30-60 seconds after instillation of 10-25 mL of priming fluid (to allow bladder detrusor muscle relaxation), and in the absence of abdominal muscle contractions.
  - Femoral venous pressure can be used for continuous IAP monitoring to facilitate early detection of ACS if IAP is above 20 mmHg.

INTRODUCTION

Elevated intra-abdominal pressure (IAP) is frequently encountered among a variety of patient populations and causes significant morbidity and mortality (1-15). Increased recognition of its prevalence among the critically ill, combined with advances in both the diagnosis and management of intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS), have resulted in significant improvements in patient survival (4,5). IAP measurements are essential to the diagnosis and management of IAH/ACS. The World Society of the Abdominal Compartment Syndrome (WSACS) has

EVIDENCE DEFINITIONS

- **Class I:** Prospective randomized controlled trial.
- **Class II:** Prospective clinical study or retrospective analysis of reliable data. Includes observational, cohort, prevalence, or case control studies.
- **Class III:** Retrospective study. Includes database or registry reviews, large series of case reports, expert opinion.
- **Technology assessment:** A technology study which does not lend itself to classification in the above-mentioned format. Devices are evaluated in terms of their accuracy, reliability, therapeutic potential, or cost effectiveness.

LEVEL OF RECOMMENDATION DEFINITIONS

- **Level 1:** Convincingly justifiable based on available scientific information alone. Usually based on Class I data or strong Class II evidence if randomized testing is inappropriate. Conversely, low quality or contradictory Class I data may be insufficient to support a Level I recommendation.
- **Level 2:** Reasonably justifiable based on available scientific evidence and strongly supported by expert opinion. Usually supported by Class II data or a preponderance of Class III evidence.
- **Level 3:** Supported by available data, but scientific evidence is lacking. Generally supported by Class III data. Useful for educational purposes and in guiding future clinical research.

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previously published evidence-based medicine consensus guidelines for the measurement of IAP and treatment of IAH/ACS (1,2).

DEFINITIONS

Intra-abdominal pressure (IAP) is the pressure concealed within the abdominal cavity (1). IAP increases with inspiration and decreases with expiration (16). It is 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). Normal IAP is approximately 5-7 mmHg in the critically ill, but varies by disease severity with an IAP of 20-30 mmHg being common in patients with severe sepsis or an acute abdomen (1). An IAP in excess of 15 mmHg is associated with significant end-organ dysfunction and failure.

Analogous to the widely accepted concept of cerebral perfusion pressure, abdominal perfusion pressure (APP), calculated as mean arterial pressure (MAP) minus IAP, has been proposed as a more accurate predictor of visceral perfusion and an endpoint for resuscitation (1,2,17-19). APP, by considering both arterial inflow (MAP) and restrictions to venous outflow (IAP), has been demonstrated to be statistically superior to MAP or IAP alone as well as to other common resuscitation endpoints such as arterial pH, base deficit, arterial lactate, and hourly urinary output in predicting survival from IAH/ACS. A target APP of 60 mmHg has been demonstrated to correlate with improved survival from IAH/ACS (2,19).

Intra-abdominal hypertension (IAH) is defined as a sustained or repeated pathologic elevation of IAP $\geq$ 12 mmHg (1,2). 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.

Abdominal compartment syndrome (ACS) is defined as a sustained increase in IAP > 20 mmHg (with or without an APP < 60 mmHg) that is associated with new organ dysfunction / failure (1,2). The most common clinical findings are hypotension, refractory metabolic acidosis, persistent oliguria, elevated peak airway pressures, refractory hypercarbia, hypoxemia, and intracranial hypertension. ACS may be classified as primary (a condition associated with injury or disease in the abdomino-pelvic region that frequently requires early surgical or interventional radiological intervention), secondary (a condition that does not originate from the abdomino-pelvic region), or recurrent (a condition in which ACS redevelops following previous surgical or medical treatment of primary or secondary ACS) (1,2,8-12).

INCIDENCE

Originally thought to be a disease solely of the traumatically injured, IAH and ACS have now been recognized to occur in a wide variety of patient populations (1-3,5,6,15). The reported incidences of IAH and ACS have varied significantly, however, due to the historical lack of a common nomenclature. Unrecognized, the mortality of IAH and ACS has been reported to be as high as 100%.

Incidence of Intra-abdominal Hypertension (IAH) and Abdominal Compartment Syndrome (ACS) Among ICU Patients (2)

Population	IAH	ACS
Medical	18-78%	4-36%
Surgical	32-43%	4-8%
Trauma	2-50%	0.5-36%
Burn	37-70%	1-20%
Pediatric	***	0.6-19%

*** - no data available

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Numerous risk factors for the development of IAH/ACS have been suggested (2,3,7,9,20-23). Three large-scale prospective trials have identified the following independent risk factors for the development of IAH/ACS (3,7,9). A number of other non-independent risk factors for IAH/ACS have also been reported.

Independent Risk Factors for IAH and/or ACS

- Abdominal surgery or trauma
- High volume fluid resuscitation (> 3500 ml/24 hours)
- Ileus
- Pulmonary, renal, or liver dysfunction
- Damage control laparotomy
- Hypothermia; acidosis
- Anemia
- Oliguria
- Hyperlactatemia
- High gastric regional minus end-tidal carbon dioxide tension

Given the broad range of potential etiologic factors and the significant associated morbidity and mortality of IAH/ACS, a high index of suspicion and low threshold for IAP measurement appears appropriate in the patient possessing any of these risk factors. Figure 1 depicts an algorithm for the initial evaluation of patients at risk for IAH (2). The WSACS strongly recommends that patients should be screened for IAH/ACS risk factors upon ICU admission and in the presence of new or progressive organ failure.

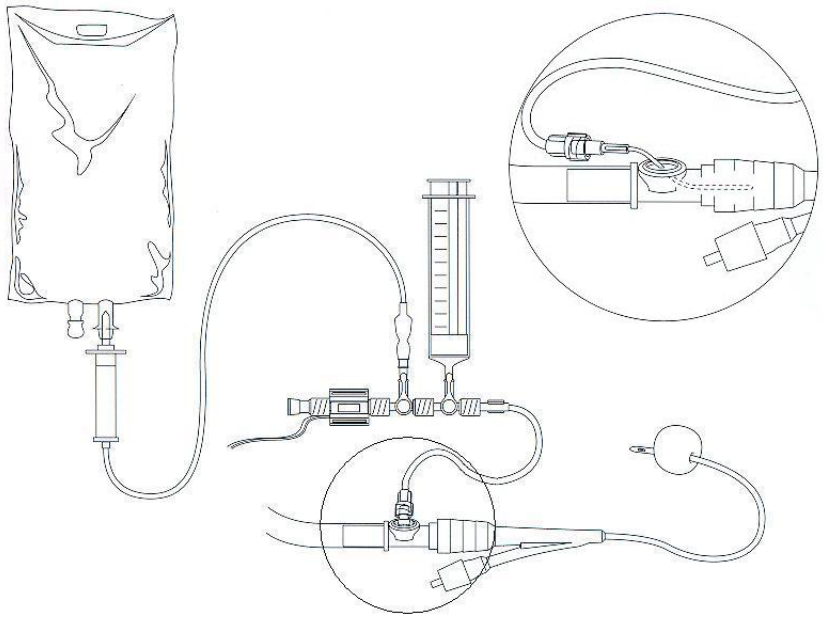
IAP MEASUREMENT

Physical examination is inaccurate in detecting elevated IAP with reported sensitivities of 40-60% (24,25). The diagnosis of IAH/ACS is therefore dependent upon the accurate and frequent measurement of IAP. IAP monitoring is a cost-effective, safe, and accurate tool for identifying the presence of IAH and guiding resuscitative therapy for ACS (2,26-29). Given the favorable risk-benefit profile of IAP monitoring and the significant associated morbidity and mortality of IAH/ACS, the WSACS recommends that if two or more risk factors for IAH/ACS are present, a baseline IAP measurement should be obtained (2). Further, if IAH is detected, serial IAP measurements should be performed throughout the patient's critical illness (Figure 1).

The accuracy and reproducibility of IAP measurements are of paramount importance in the management of IAH/ACS (26,27,30). While direct intraperitoneal catheter determinations are ideal, a variety of less-invasive techniques for determining IAP have been devised including measurement of intravesicular (bladder), intragastric, intracolonic, and intrauterine pressure (26,27). Currently, over 90% of IAP measurements worldwide are performed using the intravesicular method (15). Continuous methods for monitoring IAP have been reported and are rapidly gaining favor (26-28,31). Femoral venous pressure measurement correlates with intravesicular pressure measurement and should be considered for early detection of ACS if IAP is >20mmhg.

Regardless of the technique utilized, several key principles must be followed to ensure accurate and reproducible measurements from patient to patient (2,27). IAP should be expressed in mmHg (1 mmHg = 1.36 cm H_2O) and measured at end-expiration after ensuring that abdominal muscle contractions are absent. Measurement of IAP should be accomplished using a consistent external reference point to zero the catheter (47,48). Head of bed elevation appears to significantly increase IAP measurements, the patient should be in the complete supine position with the transducer zeroed in the mid-axillary line at the level of the iliac crest (32,33). A maximal instillation volume of 10-25 mL of sterile saline (3 mL/kg for children) should be used for the intravesicular technique as recent studies have demonstrated that larger volumes of fluid can lead to falsely elevated IAP measurements (32-40, 49). Room temperature saline significantly increases IAP, presumably due to bladder detrusor contraction (37). As a result IAP

determination should be performed 30-60 seconds after instillation of the priming fluid to allow bladder detrusor muscle relaxation (2,37).



Technique: A standard intravenous (IV) infusion set is connected to 500 mL of normal saline, two three-way stopcocks, a 20 mL Luer lock syringe, and a disposable pressure transducer. A short segment of arterial pressure tubing is used to connect the stopcocks to the Bard EZ-Lok™ Sampling Port urinary drainage tubing (C.R. Bard, Inc., Covington, GA). Alternatively, an 18-gauge plastic intravenous infusion catheter or needleless cannula is inserted into the culture aspiration port of the urinary drainage tubing and the needle removed. The infusion catheter, cannula, or sampling port is attached to the first stopcock via pressure tubing. After being flushed with saline and "zeroed" at the level of the mid-axillary line (with the patient in the supine position), the urinary drainage tubing is clamped immediately distal to the catheter. The stopcocks are turned "off" to the patient and pressure transducer and 20 mL of saline is aspirated from the IV bag and instilled into the bladder. The stopcocks are turned "off" to the syringe and IV tubing. The clamp on the urinary drainage tubing is momentarily released to ensure that all air is flushed from the urinary catheter. After a stabilization period of 30-60 seconds to allow for bladder detrusor muscle relaxation, with the patient in the complete supine position and after ensuring that abdominal muscle contractions are absent, IAP is measured at end-expiration on the bedside monitor. The patient's IAP should be expressed in mmHg (1 mmHg = 1.36 cm H_2O). After IAP determination, the clamp is removed, the bladder allowed to drain, and the volume of saline utilized subtracted from the patient's urinary output for that hour.

Head of bed elevation is widely recommended to reduce the incidence of ventilator associated pneumonia. A number of recent studies have assessed the potential impact of such changes in body position on IAP measurements (33,39-44,48). These studies have routinely found that head of bed elevation significantly increases IAP compared to supine measurements. Such increases in IAP become clinically significant (increase > 2 mmHg) when the patient's head of bed exceeds 20 degrees elevation, well below that currently practiced in many intensive care units. As a result, supine IAP measurements may underestimate the patient's true IAP if the head of bed is being elevated between measurements. Prone positioning for acute lung injury has also been demonstrated to significantly increase IAP (45,46). Until further research is available to fully clarify this issue, the WSACS recommends that all IAP measurements be performed in the supine position and that the potential contribution of body position in elevating IAP should be considered in patients with moderate to severe IAH or ACS (2). Alternatively, the

patient may be maintained in the reverse Trendelenberg position to maintain head of bed elevation while avoiding compression of the abdomen by the chest. This technique has the added benefit of utilizing gravity to decrease cephalad compression of the abdominal viscera upon the thoracic cavity, thereby reducing IAP. For serial measurements of IAP a consistent body position should be utilized to prevent variation in readings (48,49).

In addition to serial measurements of IAP, current evidence suggests that maintenance of an APP ≥ 60 mmHg also represents an important and valuable resuscitation endpoint in patients with elevated IAP (17-19). Failure to maintain an APP ≥ 60 mmHg by day three of IAH resuscitation has been demonstrated to be predictive of survival (19). If APP remains inadequate despite restoration of intravascular preload, vasoactive medications such as norepinephrine should be utilized to raise APP above 60 mmHg, especially if the patient's afterload is abnormally low. Restoration of adequate intravascular volume, guided by accurate estimates of intravascular preload, must precede institution of vasoactive medications in order to avoid visceral malperfusion and acidosis. The use of such medications may facilitate restoration of both abdominal and systemic perfusion with lower resuscitation fluid volumes than have been traditionally required, thus reducing the risk of over-resuscitation and secondary ACS (13,23). Figure 2 illustrates the WSACS algorithm for resuscitation and management of the patient with IAH/ACS (2).

TREATMENT

The WSACS recommends measuring IAP when two or more risk factors for IAH/ACS is present in a critically ill patient. A protocolized manner of measurement should be utilized (Figure 3). When IAH/ACS is detected, treatment should be directed to evacuate intraluminal content, evacuate intra-abdominal space occupying lesions, improve abdominal wall compliance, optimize fluid administration, and optimize systemic/regional perfusion. If IAH/ACS is refractory to medical management, laparotomy should be performed for abdominal decompression (51).

CONCLUSIONS

Serial IAP measurements represent an important physiologic parameter that should be monitored in any patient who demonstrates risk factors for IAH/ACS. IAP is both a diagnostic measurement, given the inaccuracy of clinical examination in detecting the presence of IAH, and a therapeutic measurement, as IAP guided resuscitation correlates with improved survival. Goal-directed resuscitation using IAP and APP to determine fluid requirements and response to therapy should be considered the standard of care for any patient with IAH/ACS.

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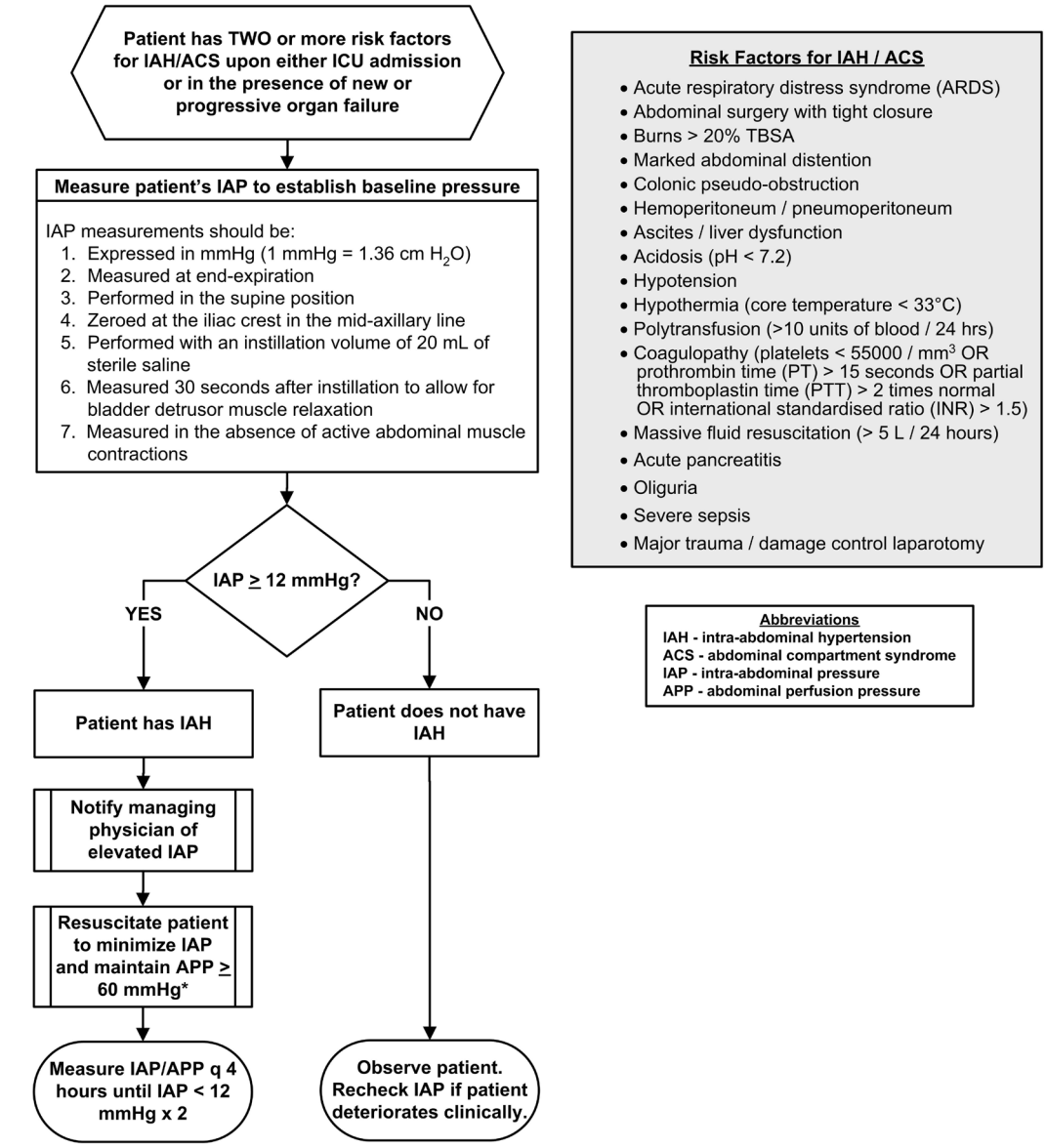
Figure 1

INTRA-ABDOMINAL HYPERTENSION (IAH) ASSESSMENT ALGORITHM

- Patients should be screened for IAH/ACS risk factors upon ICU admission and with new or progressive organ failure.
- If two or more risk factors are present, a baseline IAP measurement should be obtained.
- If IAH is present (IAP ≥ 12 mmHg) , serial IAP measurements should be performed every 4 hours throughout the patient's critical illness OR until two consecutive measurements demonstrate an IAP < 10 mmHg.
- Abdominal perfusion pressure (APP = MAP - IAP) should be documented with each IAP on the patient's flowsheet



* ***Intensive Care Medicine*** **2006;32(11):1722-1732 & 2007;33(6):951-962**

IAH Assessment algorithm

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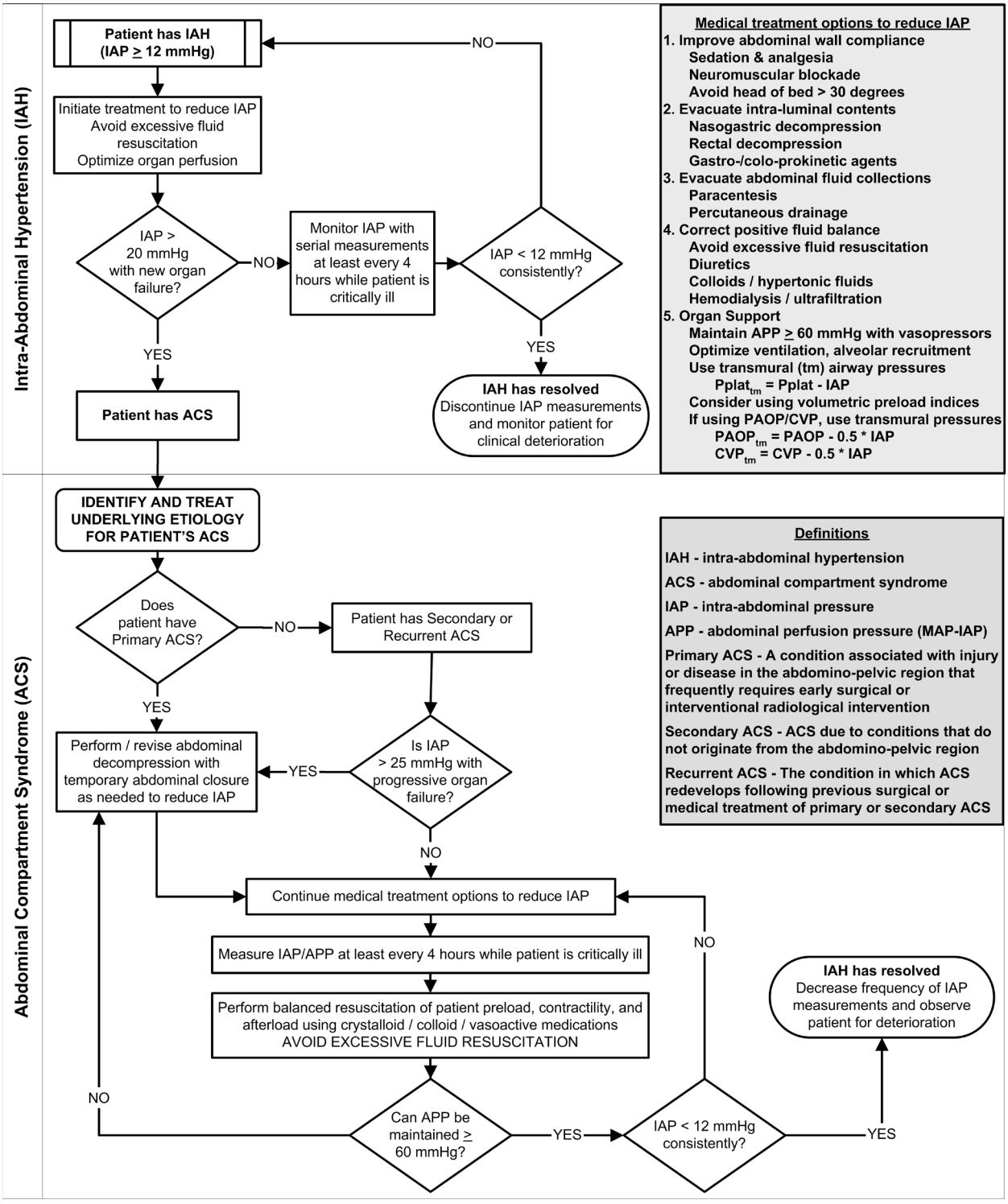
Figure 2



IAH/ACS Management algorithm

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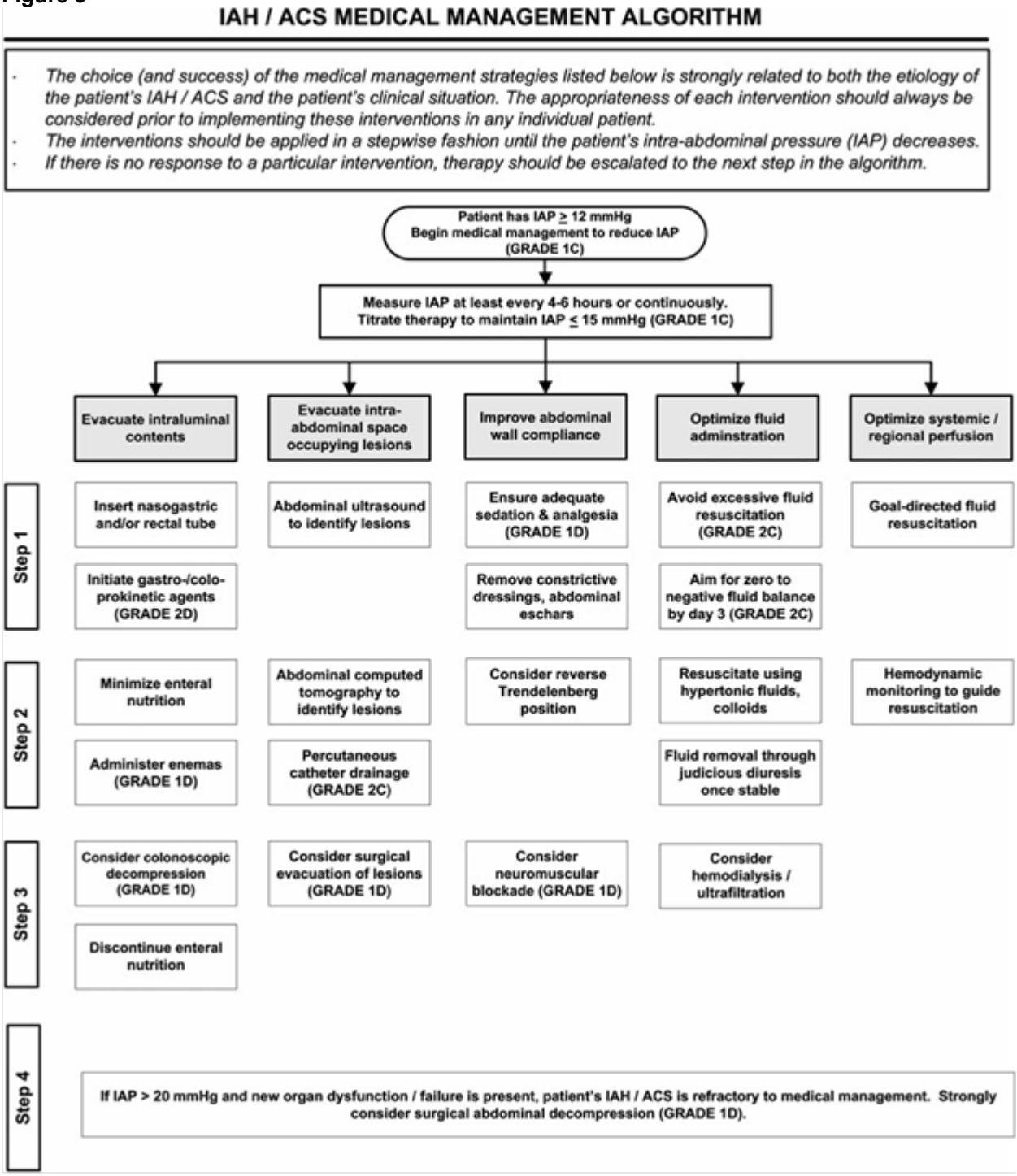
Figure 3



IAH/ACS Management algorithm

Updated intra-abdominal hypertension (IAH)/abdominal compartment syndrome (ACS) management algorithm adapted from Intensive Care Medicine (2013) 39:1190-1206.

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