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**
  - None

- **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 no more than 25 mL of priming fluid (to allow bladder detrusor muscle relaxation), and in the absence of abdominal muscle contractions.

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

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.

Approved 5/18/04
Revised 2/21/08

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 (Figure 1). 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.

It should be noted that the IAP ranges associated with these grades have been revised downward in recent years as the detrimental impact of elevated IAP on end-organ function has been recognized.

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%.

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%

Incidence of Intra-abdominal Hypertension (IAH) and Abdominal
Compartment Syndrome (ACS) Among ICU Patients (2)
*** - no data available

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.

- 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

Independently Associated Risk Factors for IAH and/or ACS

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).

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. As 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 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). 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).

Angiocatheter technique

Bard E-Z Lok technique
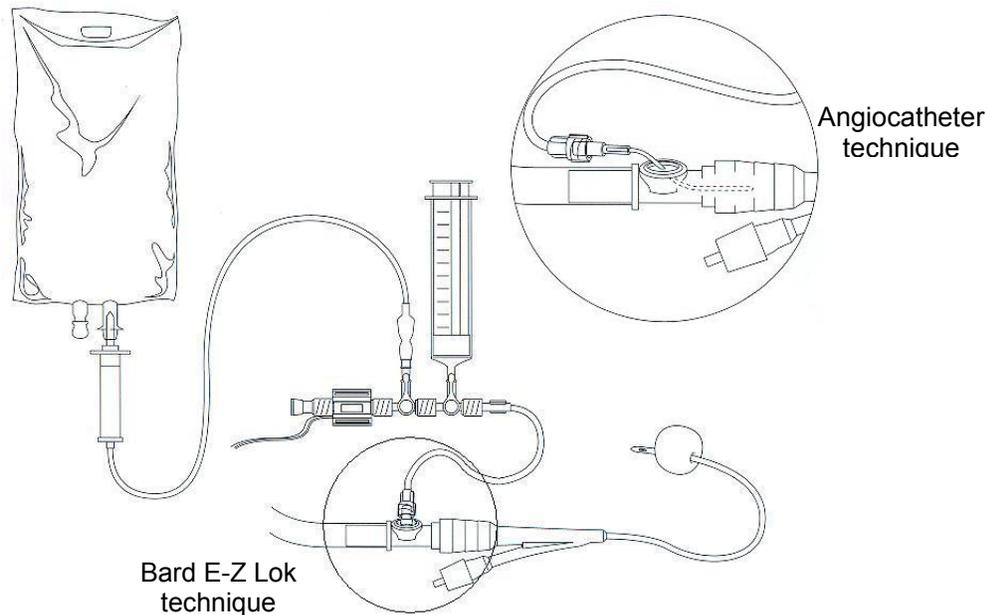
Technique: A standard intravenous (IV) infusion set is connected to 500 mL of normal saline, two three-way stopcocks, a 20 mL Luer lock syringe, and a disposable pressure transducer. A short segment of arterial pressure tubing is used to connect the stopcocks to the Bard EZ-Lok™ Sampling Port urinary 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). 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.

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).

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.

REFERENCES

1. Malbrain MLNG, Cheatham ML, Kirkpatrick A, Sugrue M, Parr M, De Waele J, Balogh Z, Leppäniemi A, Olvera C, Ivatury R, D'Amours S, Wilmer A, Wendon J, Hillman K. Results from the conference of experts on intra-abdominal hypertension and abdominal compartment syndrome. Part I: Definitions.. *Intensive Care Med* 2006; 32:1722-1732.

2. Cheatham ML, Malbrain MLNG, Kirkpatrick A, Sugrue M, Parr M, De Waele J, Balogh Z, Leppäniemi A, Olvera C, Ivatury R, D'Amours S, Wendon J, Hillman K, Wilmer A. Results from the conference of experts on intra-abdominal hypertension and abdominal compartment syndrome. Part II: Recommendations.. *Intensive Care Med* 2007; 33:951-962.
   consensus recommendations for all aspects of IAH/ACS care

3. Malbrain MLNG, Chiumello D, Pelosi P, Wilmer A, Brienza N, Malcagni V, et al. Prevalence of intra-abdominal hypertension in critically ill patients: a multicentre epidemiological study. *Intensive Care Med* 2004; 30:822-829.

4. Cheatham ML, Safcsak K. Is the evolving management of IAH/ACS improving survival?. *Acta Clinica Belgica* 2007; 62(Supplement 1); 268.

5. Kimball EJ, Kim W, Cheatham ML, Malbrain MLNG. Clinical awareness of intra-abdominal hypertension and abdominal compartment syndrome in 2007. *Acta Clinica Belgica* 2007; 62(Supplement 1); 66-73.

6. Daugherty EL, Hongyan Liang , Taichman D, Hansen-Flaschen J, Fuchs BD. Abdominal compartment syndrome is common in medical intensive care unit patients receiving large-volume resuscitation. *J Intensive Care Med* 2007; 22:294-299.

7. Ivatury RR, Porter JM, Simon RJ, Islam S, John R, Stahl WM. Intra-abdominal hypertension after life-threatening penetrating abdominal trauma: Prophylaxis, incidence, and clinical relevance to gastric mucosal pH and abdominal compartment syndrome. *J Trauma* 1998; 44:1016-1021.

8. Kirkpatrick AW, De Waele JJ, Ball CG, Ranson K, Widder S, Laupland KB. The secondary and recurrent abdominal compartment syndrome. *Acta Clinica Belgica* 2007; 62(Supplement 1); 60-65.

9. Balogh Z, McKinley BA, Holcomb JB, Miller CC, Cocanour CS, et al. Both primary and secondary abdominal compartment syndrome can be predicted early and are harbingers of multiple organ failure. *J Trauma* 2003; 54:848-859.

10. Balogh Z, McKinley BA, Cocanour CS, Kozar RA, Holcomb JB, et al. Secondary abdominal compartment syndrome is an elusive early complication of traumatic shock resuscitation. *Am J Surg* 2002; 184:538-543.

11. Kirkpatrick AW, Balogh Z, Ball CG, Ahmed N, Chun R, McBeth P, Kirby A, Zygun DA. The secondary abdominal compartment syndrome: iatrogenic or unavoidable? *J Am Coll Surg* 2006; 202:668-679.

12. Balogh Z, Moore FA. Postinjury secondary abdominal compartment syndrome. In: Ivatury RR, Cheatham ML, Malbrain MLNG, Sugrue M, editors. Abdominal Compartment Syndrome. Landes Biomedical, Georgetown, 2006.

13. Balogh Z, McKinley BA, Cocanour CS, Kozar RA, Valdivia A, Sailors RM, et al. Supranormal trauma resuscitation causes more cases of abdominal compartment syndrome. *Arch Surg* 2003; 138:637-642.

14. Cheatham ML, Safcsak K, Llerena LE, Morrow CE, Block EFJ. Long-term physical, mental, and functional consequences of abdominal decompression. *J Trauma* 2004; 56:237-242.

15. Malbrain MLNG, Cheatham ML. Results of the international survey on clinical awareness of intra-abdominal hypertension and abdominal compartment syndrome in critically ill patients. *Acta Clinica Belgica* 2007; 62(Supplement 1); 247.

16. Pelosi P, Quintel M, Malbrain MLNG. Effect of intra-abdominal pressure on respiratory mechanics. *Acta Clinica Belgica* 2007; 62(Supplement 1); 78-88.

17. Cheatham ML, Malbrain MLNG. Cardiovascular implications of abdominal compartment syndrome. *Acta Clinica Belgica* 2007; 62(Supplement 1); 98-112.

18. Cheatham ML, Malbrain MLNG. Cardiovascular implications of elevated intra-abdominal pressure. In: Ivatury RR, Cheatham ML, Malbrain MLNG, Sugrue M, editors. Abdominal Compartment Syndrome. Landes Biomedical, 2006.

19. Cheatham ML, Malbrain MLNG. Abdominal perfusion pressure. In: Ivatury RR, Cheatham ML, Malbrain MLNG, Sugrue M, editors. Abdominal Compartment Syndrome. Landes Biomedical, Georgetown, 2006.
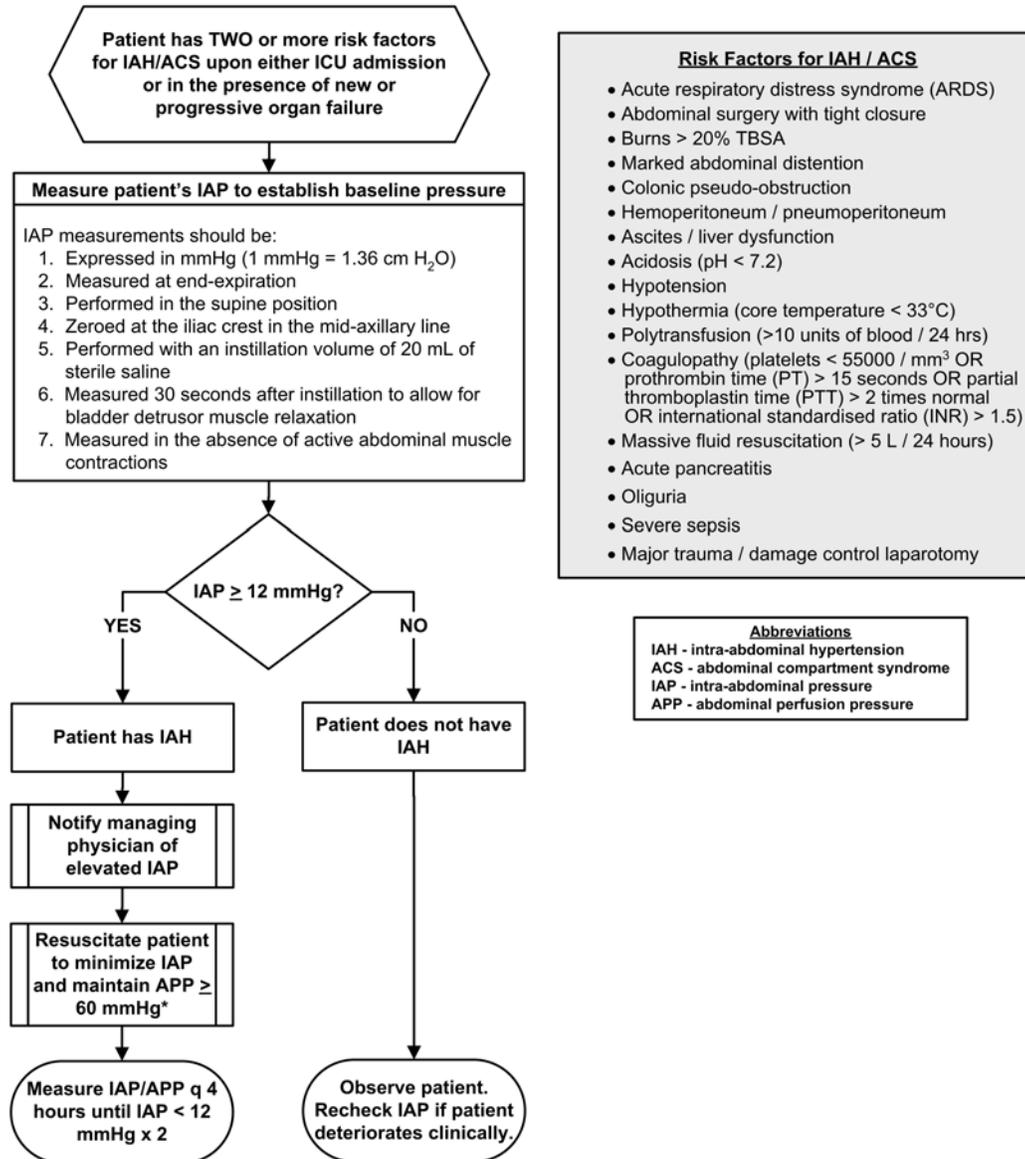
20. Sugrue M, Jones F, Deane SA, Bishop G, Bauman A, Hillman K. Intra-abdominal hypertension is an independent cause of postoperative renal impairment. *Arch Surg* 1999; 134:1082-1085.
21. Sugrue M, Buist MD, Hourihan F, Deane S, Bauman A, Hillman K. Prospective study of intra-abdominal hypertension and renal function after laparotomy. *Br J Surg* 1995; 82:235-238.
22. Serpytis M, Ivaskevicius J. Relationship between intrabdominal pressure (IAP), fluid balance (FB), and systemic inflammatory response syndrome (SIRS) after major abdominal surgery. *Acta Clinica Belgica* 2007; 62(Supplement 1); 249.
23. McNelis J, Marini CP. Predictive factors associated with the development of abdominal compartment syndrome in the SICU. *Acta Clinica Belgica* 2007; 62(Supplement 1); 255.
24. Sugrue M, Bauman A, Jones F et al. Clinical examination is an inaccurate predictor of intraabdominal pressure. *World J Surg* 2002; 26:1428-1431.
25. Kirkpatrick AW, Brenneman FD, McLean RF, Rapanos T, Boulanger BR. Is clinical examination an accurate indicator of raised intra-abdominal pressure in critically injured patients? *Can J Surg* 2000; 43:207-211.
26. Malbrain MLNG, Jones F. Intra-abdominal pressure measurement techniques. In: Ivatury RR, Cheatham ML, Malbrain MLNG, Sugrue M, editors. Abdominal Compartment Syndrome. Landes Biomedical, Georgetown, 2006.
27. De Waele JJ, De laet I, Malbrain MLNG. Rational intraabdominal pressure monitoring: How I do it? *Acta Clinica Belgica* 2007; 62(Supplement 1); 16-25.
28. Balogh Z, De Waele JJ, Malbrain MLNG. Continuous intra-abdominal pressure monitoring *Acta Clinica Belgica* 2007; 62(Supplement 1); 26-32.
29. Arsenkov L, Karagozov S, Antonik S, Nikolovski A. Intraoperative measurement of IAP: How, when, why. *Acta Clinica Belgica* 2007; 62(Supplement 1); 264.
30. Kimball EJ, Mone MC, Wolfe TR, Baraghoshi GK, Alder SC. Reproducibility of bladder pressure measurements in critically ill patients. *Intensive Care Med* 2007; 33:1195–1198.
31. Malbrain MLNG, De laet I, Viane D, Schoonheydt K, Dits H. In vitro validation of a novel method for continuous intra-abdominal pressure monitoring. *Intensive Care Med* 2008 (In press)
32. De Waele J, Cheatham ML, De Keulenaer B, Widder S, Kirkpatrick A, Cresswell B, Malbrain M, Bodnar Z, Meija J, Reis R, Parr M, Schulze R, Compano S. The optimal zero reference transducer position for intra-abdominal pressure measurement: A multicenter analysis. *Acta Clinica Belgica* 2007; 62(Supplement 1); 247.
33. McBeth PB, Zygun DA, Widder S, Cheatham M, Zengerink I, Glowa J, Kirkpatrick AW. Effect of patient positioning on intra-abdominal pressure monitoring*. Am J Surgery* 2007; 193:644–647.
34. De Waele J, Pletinckx P, Blot S, Hoste E. Saline volume in transvesical intra-abdominal pressure measurement: enough is enough. *Intensive Care Med* 2006; 32:455-459.
35. Malbrain ML, Deeren DH. Effect of bladder volume on measured intravesical pressure: a prospective cohort study. *Crit Care* 2006; 10:R98.
36. Ejike JC, Mathur M. Bladder volumes for accurate intra-abdominal pressure measurements in children. *Acta Clinica Belgica* 2007; 62(Supplement 1); 269.
37. Chiumello D, Tallarini F, Chierichetti M, Polli F, Li Bassi G, Motta G, Azzari S, Carsenzola C, Gattinoni L. The effect of different volumes and temperatures of saline on the bladder pressure measurement in critically ill patients. *Critical Care* 2007, 11:R82.
38. De laet I, Hoste E, DeWaele JJ. Transvesical intra-abdominal pressure measurement using minimal instillation volumes: how low can we go? *Intensive Care Med* (In press)
39. Vasquez DG, Berg-Copas GM, Wetta-Hall R. Influence of semi-recumbent position on intra-abdominal pressure as measured by bladder pressure. *Journal of Surgical Research* 2007; 139: 280–285.
40. Cheatham ML, De Waele J, De Keulenaer B, Widder S, Kirkpatrick A, Cresswell B, Malbrain M, Bodnar Z, Meija J, Reis R, Parr M, Schulze R, Compano S. The effect of body position on intra-abdominal pressure measurement: A multicenter analysis. *Acta Clinica Belgica* 2007; 62(Supplement 1); 246.
41. Vianne D, De laet I, Vermeiren G, Schoonheydt K, Dits D, Malbrain MLNG. Effect of different body positions on intra-abdominal pressure estimated with 3 different methods via the bladder and stomach. *Acta Clinica Belgica* 2007; 62(Supplement 1); 257.

42. De laet I, Vianne D, Vermeiren G, Schoonheydt K, Dits H, Malbrain MLNG. Effect of head of bed elevation on intra-abdominal pressure estimated via bladder and stomach: Preliminary results on the validation of the gastromanometer. *Acta Clinica Belgica* 2007; 62(Supplement 1); 259.

43. Pracca F, Gorrasi J, Moraes L, Iturralde A, Puppo C, Biestro A, Cancela M. Effects of PEEP and bed recumbent angle on intraabdominal pressure measurement. *Acta Clinica Belgica* 2007; 62(Supplement 1); 264.

44. De Keulenauer BL, Chana G, Maddox I, Powell B, Jenkins I. Intraabdominal pressure measurements in lateral decubitus and supine position. *Acta Clinica Belgica* 2007; 62(Supplement 1); 269.

45. Pelosi P, Tubiolo D, Mascheroni D, Vicardi P, Crotti S, et al. Effects of the prone position on respiratory mechanics and gas exchange during acute lung injury. *Am J Respir Crit Care Med* 1998; 157:387-93.

46. Hering R, Wrigge H, Vorwerk R, Brensing KA, Schroder S, Zinserling J, et al. The effects of prone positioning on intraabdominal pressure and cardiovascular and renal function in patients with acute lung injury. *Anesth Analg* 2001; 92:1226-1231.

Approved 5/18/04
Revised 2/21/08

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

Patient has TWO or more risk factors for IAH/ACS upon either ICU admission or in the presence of new or progressive organ failure

Measure patient's IAP to establish baseline pressure

IAP measurements should be:
1. Expressed in mmHg (1 mmHg = 1.36 cm H_2O)
2. Measured at end-expiration
3. Performed in the supine position
4. Zeroed at the iliac crest in the mid-axillary line
5. Performed with an instillation volume of 20 mL of sterile saline
6. Measured 30 seconds after instillation to allow for bladder detrusor muscle relaxation
7. Measured in the absence of active abdominal muscle contractions

IAP ≥ 12 mmHg?

YES → **Patient has IAH**

NO → **Patient does not have IAH**

Notify managing physician of elevated IAP

Resuscitate patient to minimize IAP and maintain APP ≥ 60 mmHg*

Measure IAP/APP q 4 hours until IAP < 12 mmHg x 2

Observe patient. Recheck IAP if patient deteriorates clinically.

Risk Factors for IAH / ACS

- Acute respiratory distress syndrome (ARDS)
- Abdominal surgery with tight closure
- Burns > 20% TBSA
- Marked abdominal distention
- Colonic pseudo-obstruction
- Hemoperitoneum / pneumoperitoneum
- Ascites / liver dysfunction
- Acidosis (pH < 7.2)
- Hypotension
- Hypothermia (core temperature < 33°C)
- Polytransfusion (>10 units of blood / 24 hrs)
- Coagulopathy (platelets < 55000 / mm^3 OR prothrombin time (PT) > 15 seconds OR partial thromboplastin time (PTT) > 2 times normal OR international standardised ratio (INR) > 1.5)
- Massive fluid resuscitation (> 5 L / 24 hours)
- Acute pancreatitis
- Oliguria
- Severe sepsis
- Major trauma / damage control laparotomy

Abbreviations
IAH - intra-abdominal hypertension
ACS - abdominal compartment syndrome
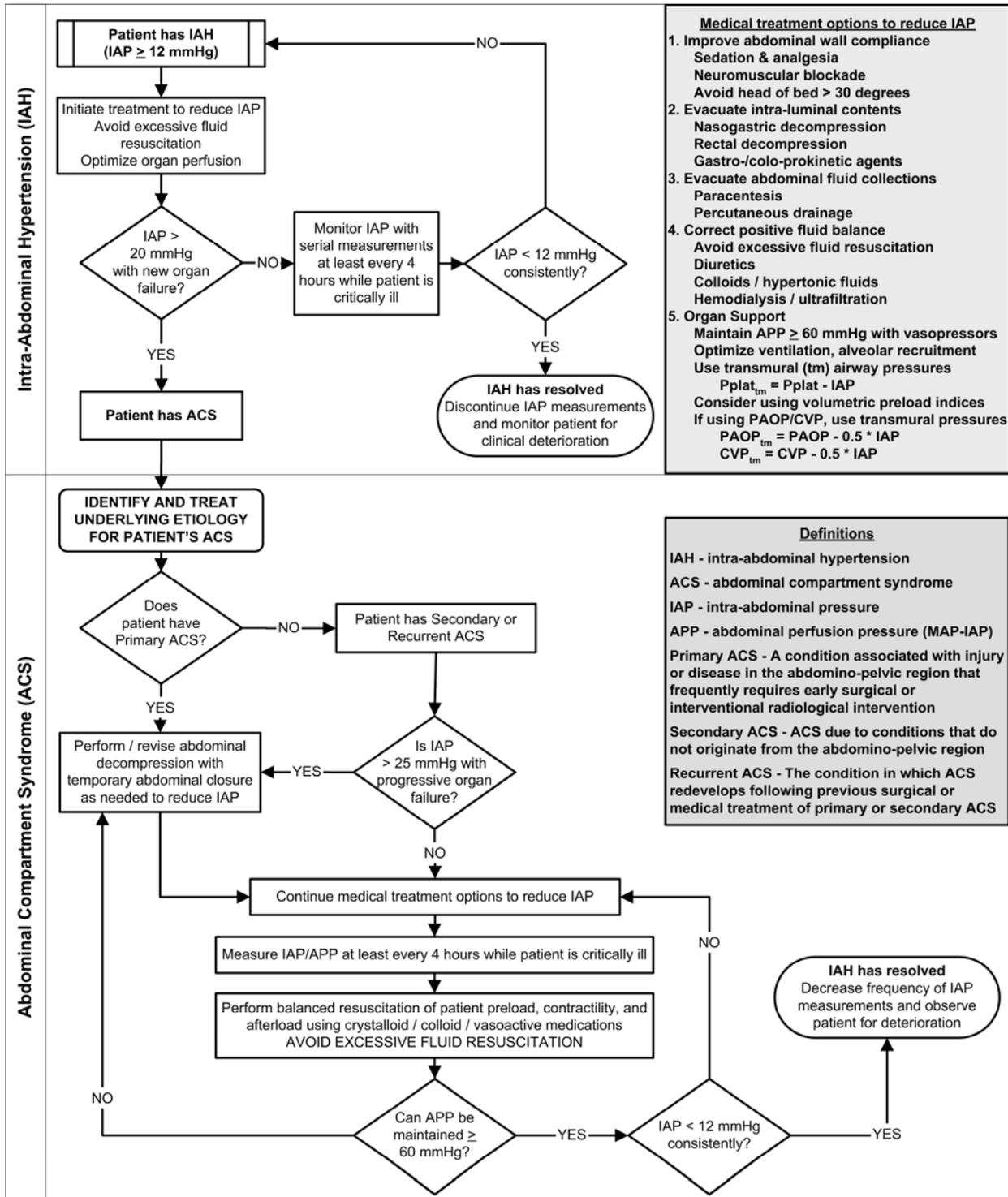IAP - intra-abdominal pressure
APP - abdominal perfusion pressure

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

IAH Assessment algorithm

Adapted from *Intensive Care Medicine* 2007;33(6):951-962 and used with the permission of the World Society of the Abdominal Compartment Syndrome (WSACS)

Figure 2

Intra-Abdominal Hypertension (IAH)

Patient has IAH
(IAP ≥ 12 mmHg)

— NO →

Initiate treatment to reduce IAP
Avoid excessive fluid resuscitation
Optimize organ perfusion

IAP > 20 mmHg with new organ failure?

— NO →

Monitor IAP with serial measurements at least every 4 hours while patient is critically ill

IAP < 12 mmHg consistently?

YES

IAH has resolved
Discontinue IAP measurements and monitor patient for clinical deterioration

YES

Patient has ACS

Medical treatment options to reduce IAP
1. Improve abdominal wall compliance
   Sedation & analgesia
   Neuromuscular blockade
   Avoid head of bed > 30 degrees
2. Evacuate intra-luminal contents
   Nasogastric decompression
   Rectal decompression
   Gastro-/colo-prokinetic agents
3. Evacuate abdominal fluid collections
   Paracentesis
   Percutaneous drainage
4. Correct positive fluid balance
   Avoid excessive fluid resuscitation
   Diuretics
   Colloids / hypertonic fluids
   Hemodialysis / ultrafiltration
5. Organ Support
   Maintain APP ≥ 60 mmHg with vasopressors
   Optimize ventilation, alveolar recruitment
   Use transmural (tm) airway pressures
      $Pplat_{tm}$ = Pplat - IAP
   Consider using volumetric preload indices
   If using PAOP/CVP, use transmural pressures
      $PAOP_{tm}$ = PAOP - 0.5 * IAP
      CVP_{tm} = CVP - 0.5 * IAP

Abdominal Compartment Syndrome (ACS)

IDENTIFY AND TREAT UNDERLYING ETIOLOGY FOR PATIENT'S ACS

Does patient have Primary ACS?

— NO →

Patient has Secondary or Recurrent ACS

YES

Perform / revise abdominal decompression with temporary abdominal closure as needed to reduce IAP

— YES —

Is IAP > 25 mmHg with progressive organ failure?

NO

Definitions
IAH - intra-abdominal hypertension
ACS - abdominal compartment syndrome
IAP - intra-abdominal pressure
APP - abdominal perfusion pressure (MAP-IAP)

Primary ACS - A condition associated with injury or disease in the abdomino-pelvic region that frequently requires early surgical or interventional radiological intervention

Secondary ACS - ACS due to conditions that do not originate from the abdomino-pelvic region

Recurrent ACS - The condition in which ACS redevelops following previous surgical or medical treatment of primary or secondary ACS

Continue medical treatment options to reduce IAP

Measure IAP/APP at least every 4 hours while patient is critically ill

Perform balanced resuscitation of patient preload, contractility, and afterload using crystalloid / colloid / vasoactive medications
AVOID EXCESSIVE FLUID RESUSCITATION

Can APP be maintained ≥ 60 mmHg?

NO

— YES —

IAP < 12 mmHg consistently?

NO

— YES —

IAH has resolved
Decrease frequency of IAP measurements and observe patient for deterioration

IAH/ACS Management algorithm

Adapted from *Intensive Care Medicine* 2007;33(6):951-962 and used with the permission of the World Society of the Abdominal Compartment Syndrome (WSACS)