

Abdominal compartment syndrome: A concise clinical review

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Objective: There has been an increased awareness of the presence and clinical importance of abdominal compartment syndrome. It is now appreciated that elevations of abdominal pressure occur in a wide variety of critically ill patients. Full-blown abdominal compartment syndrome is a clinical syndrome characterized by progressive intra-abdominal organ dysfunction resulting from elevated intra-abdominal pressure. This review provides a current, clinically focused approach to the diagnosis and management of abdominal compartment syndrome, with a particular emphasis on intensive care.

Methods: Source data were obtained from a PubMed search of the medical literature, with an emphasis on the time period after 2000. PubMed "related articles" search strategies were likewise employed frequently. Additional information was derived from the Web site of the World Society of the Abdominal Compartment Syndrome (<http://www.wsacs.org>).

Summary and Conclusions: The detrimental impact of elevated intra-abdominal pressure, progressing to abdominal compart-

ment syndrome, is recognized in both surgical and medical intensive care units. The recent international abdominal compartment syndrome consensus conference has helped to define, characterize, and raise awareness of abdominal compartment syndrome. Because of the frequency of this condition, routine measurement of intra-abdominal pressure should be performed in high-risk patients in the intensive care unit. Evidence-based interventions can be used to minimize the risk of developing elevated intra-abdominal pressure and to aggressively treat intra-abdominal hypertension when identified. Surgical decompression remains the gold standard for rapid, definitive treatment of fully developed abdominal compartment syndrome, but nonsurgical measures can often effectively affect lesser degrees of intra-abdominal hypertension and abdominal compartment syndrome. (Crit Care Med 2008; 36:1304–1310)

KEY WORDS: intra-abdominal hypertension; intra-abdominal pressure; compartment syndrome; massive resuscitation; damage control; decompressive laparotomy; temporary abdominal closure

There has been an increased awareness of the presence and clinical importance of abdominal compartment syndrome (ACS) over the past decade. Whereas this condition was formerly recognized predominately in blunt trauma victims, it is now appreciated that elevations of abdominal pressure occur in a wide variety of critically ill patients. Critical care practitioners need to be familiar with the signs and symptoms of ACS and understand how to treat this increasingly common condition (1). This review provides a current, clinically focused approach to the diagnosis and management of ACS, with a particular emphasis on care in the intensive care unit (ICU).

Full-blown abdominal compartment syndrome is a clinical syndrome characterized by progressive intra-abdominal organ dysfunction resulting from ele-

ated intra-abdominal pressure (IAP). The term *abdominal compartment syndrome* was coined by Kron et al. (2) in 1984, when they described the pathophysiology following a ruptured abdominal aortic aneurysm. The classic description of ACS included a tense distended abdomen, increased IAP, decreased renal function, elevated peak airway pressure, hypoxia, and inadequate ventilation. Improvement of all variables was seen after decompressive laparotomy.

Definitions

Elevated IAP with progression to ACS has been described in a wide variety of clinical conditions (3–25). A number of authors have promulgated a wide variety of sometimes subtly different definitions and classification schemes (26–28). The World Society of the Abdominal Compartment Syndrome met in 2004 to try to bring order to this chaotic situation via an international consensus conference. The goal was to produce a consensus statement (accessible at <http://www.wsacs.org>) related to the definition, diagnosis, and treatment of ACS (29, 30). The consensus statement defines intra-abdominal hypertension (IAH) as an IAP ≥ 12 mm

Hg and ACS as a sustained IAP ≥ 20 mm Hg that is associated with new organ dysfunction or failure. The society also defined another variable, abdominal perfusion pressure (APP), which may be useful in discussing ACS. This value, which is equal to the mean arterial pressure (MAP) minus the IAP ($APP = MAP - IAP$) is a measure of the net pressure available for perfusion of intra-abdominal organs (31). Clinicians can think of APP in the same way that cerebral perfusion pressure is used when discussing brain perfusion in the face of intracranial hypertension. Table 1 compares the current World Society of the Abdominal Compartment Syndrome grading classification of elevated IAP to the previously widely used Burch/Meldrum classification (4, 32). The consensus conference also redefined the zero point for pressure measurements (29, 30). The updated zero point, the mid-axillary line, is the same reference point used for most other hemodynamic measurements in the critically ill. IAP should be measured in reference to this point, expressed in mm Hg, and measured at end-expiration in the complete supine position after ensuring that abdominal muscular contractions are absent.

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Table 1. Grading schemes for elevated intra-abdominal pressure

Grade	WSACS Definition (30), mm Hg ^a	Burch (4)/Meldrum (32) Classification, mm Hg ^b
I	12–15	10–15
II	16–20	16–25
III	21–25	26–35
IV	>25	>35

WSACS, World Society of the Abdominal Compartment Syndrome.

^aPressure measured with zero at midaxillary line; ^bpressure measured with zero at pubic symphysis.

Abdominal compartment syndrome can be classified as either primary ACS (33–35), due to the presence of intra-abdominal and/or retroperitoneal pathology, or secondary ACS (17, 35–37), due to generalized capillary leak with massive fluid resuscitation leading to edema of otherwise normal bowel or development of tense ascites. Examples of primary ACS include those associated with damage control laparotomies for trauma or large retroperitoneal hematomas. Secondary ACS is often seen in the postresuscitation phase for septic shock, hemorrhagic shock, or burn injury. *Recurrent ACS* refers to situations in which ACS redevelops after treatment for either primary or secondary ACS (26, 33).

It is vital to identify and recognize the underlying pathophysiologic conditions, whether primary or secondary (or even tertiary) (38), that produce elevated IAP and that may progress to ACS. In many cases the pathogenesis of ACS is multifactorial, such as primary ACS following abdominal trauma being exacerbated by secondary ACS resulting from massive fluid resuscitation. IAH and ACS share the same pathophysiology, and the clinical distinctions represent points along a continuum. Therefore, when dealing with an individual patient it is critical to identify which pathophysiologic processes are involved in the evolution of IAH/ACS.

Pathophysiology

The abdominal compartment is bounded inferiorly by the pelvic floor, circumferentially by the abdominal wall, and superiorly by the diaphragm, which separates the abdomen from the thorax. Although the diaphragm anatomically divides the chest and abdomen, the diaphragm is not a rigid barrier to transmission of increased pressures within the torso (39), although the tendency is more for intra-abdominal processes to affect intrathoracic measurements than in the other direction. The underlying pathophysiology of ACS is consistent with the patho-

genesis of compartment syndrome in other body regions (40–42). The fundamental abnormality, increased pressure within a (relatively) nonexpandable compartment, leads to aberrations in the blood flow of intracompartmental tissues, initially at the microvascular (capillary) level but eventually progressing to affect the venous return and arterial inflow.

In contrast to extremity compartment syndromes, the organ dysfunction from sustained IAH usually becomes clinically significant before actual intra-abdominal organ infarction (e.g., bowel necrosis). However, as with extremity compartment syndromes, there is a threshold of IAH at which the cycle of microvascular derangement becomes self-propagating. This point is reached when elevations in IAP increase venous outflow resistance, leading to venous congestion and further increases of intracompartmental pressures. At the point where IAP is greater than ~20 mm Hg, there is a significant reduction in effective perfusion of the capillary beds, leading to tissue ischemia and activation of inflammatory mediators. This in turn leads to increased extravascular fluid loss via capillary leak, producing more tissue fluid influx, a further net increase in intra-abdominal volume, and additional elevation of IAP, perpetuating the cycle. An additional contributing factor in the cycle of IAH/ACS is the impairment of lymphatic flow and subsequent increase in intestinal edema (43, 44).

The systemic and clinical manifestations of ACS are related to the consequences of IAH at the organ level. Expansion of the abdominal cavity from elevated IAP results in a cephalad displacement of the diaphragm with reduction in dynamic pulmonary compliance and a requirement for increasing positive airway pressure to deliver the same tidal volume (45). The cycle of impaired outflow, decreased capillary perfusion, and increasing pressure also leads to de-

creased hepatosplanchnic flow (with impaired liver function) (46–48), decreased renal blood flow (resulting in low urine output, progressing from oliguria to anuria) (49–51), compression of the inferior vena cava, and decreased venous return to the heart (with resultant decreased cardiac output, progressing to shock) (52).

Diagnosis

The diagnosis of ACS can be divided into three components: 1) identifying patients at risk; 2) recognizing the clinical signs associated with the transition of IAH to ACS; and 3) proactively carrying out diagnostic measures to confirm the suspected diagnosis. Elevations in IAP, and even the presence of ACS, may be suspected based on computed tomography radiographic findings (53–55). Risk factors for ICU patients can be seen in Table 2. A variety of means of measuring IAP have been reported, such as gastric pressure via a nasogastric tube (56), inferior vena cava pressure (57), rectal pressure, direct IAP via direct puncture, or use of bedside ultrasound to assess the caliber and respiratory variation of the inferior vena cava (58), but measurement of IAP using the bladder is the current mainstay of diagnosis (59). The currently recommended technique involves instillation of 25–50 mL of sterile saline into the bladder via a Foley catheter (29, 60). The catheter tubing is clamped, and a needle is inserted via the specimen-collection port proximal to the clamp and attached to a calibrated pressure transducer. Variation in IAP with gentle abdominal pressure confirms that there is good fidelity of pressure transduction. To ensure accuracy and reproducibility, IAP should be measured at end-expiration with the patient completely supine (30). Abdominal muscle contractions should be absent and the transducer is zeroed at the level of midaxillary line. Modifications of this system allow continuous measurement (57, 61, 62).

Patients who are at risk for possible development of ACS (35, 63–66) (Table 3) should have baseline measurement of IAP at admission to the ICU. The most common risk factor associated with ACS is a history of massive fluid resuscitation, usually with a significant crystalloid component. In this context, *massive* is defined as >5 L of intravenous fluid resuscitation within a 24-hr period. Experienced clinicians will quickly recognize that such a

Table 2. Intensive care unit conditions that may predispose to intra-abdominal hypertension (29, 35, 64–66)

Acidosis (arterial pH, ≤ 7.2)
Hypothermia (core temperature, $\leq 33^\circ\text{C}$)
Polytransfusion (transfusion, ≥ 10 units of PRBCs in 24 hrs)
Coagulopathy (platelets, $\leq 55,000/\text{mm}^3$; PTT, $>2 \times$ normal; or an INR, >1.5)
Sepsis (American-European Consensus Conference definition)
Bacteremia (positive blood cultures)
Liver dysfunction (cirrhosis with ascites, portal vein thrombosis, ischemic hepatitis)
Need for mechanical ventilation
Use of PEEP or the presence of auto-PEEP
Pneumonia

PRBCs, packed red blood cells; PTT, partial thromboplastin time; INR, international normalized ratio; PEEP, positive end-expiratory pressure.

Table 3. Common etiologic factors for intra-abdominal hypertension (29, 33, 110, 111)

Abdominal surgery
Massive fluid resuscitation (>5 L in 24 hrs)
Ileus (paralytic, mechanical, or pseudo-obstructive)
Intra-abdominal infection
Pneumoperitoneum (can include pneumoperitoneum for laparoscopy)
Hemoperitoneum

Table 4. Common clinical conditions that warrant prospective monitoring of intra-abdominal hypertension

Postoperative from abdominal surgery
Blunt or penetrating abdominal trauma
Pelvic fractures with retroperitoneal bleeding
Mechanically ventilated ICU patients with other organ dysfunction (increased SOFA or MOF score)
Abdominal packing after temporary abdominal closure for multiple trauma or liver transplantation
Open abdomen (may still develop ACS, especially if early postoperative)
Large-volume fluid resuscitation (e.g., pancreatitis, septic shock, trauma)

ICU, intensive care unit; SOFA, Sepsis-related Organ Failure Assessment; MOF, multiple organ failure; ACS, abdominal compartment syndrome.

definition of *massive resuscitation* encompasses a large number of ICU patients, underscoring the importance of always considering the possibility of ACS development and proactively assessing IAP.

Clinical Signs of ACS

The classic constellation of clinical findings associated with ACS includes in-

creased airway pressure, decreased urine output, and a tense abdomen on physical exam (2). Unfortunately, from a practical standpoint, these signs and symptoms are extremely nonspecific in critically ill patients. For example, patients undergoing large-volume fluid resuscitation frequently have impaired tissue perfusion, hypotension, and oliguria. These same patients are at risk for acute lung injury or pulmonary edema, either of which may result in increased airway pressure (67, 68). In many cases the only way to detect ACS is via active monitoring of IAP. This is especially important for the conditions delineated in Table 4. Factors involved in determining the subsequent interval and duration of IAP monitoring include the baseline pressure measurement, the ability to alter etiologic and risk factors, and the dynamic evolution of the patient's course.

Treatment

To a great extent, the best treatment for ACS is prevention. Increased recognition regarding the risk factors, setting, and pathogenesis of ACS allows earlier identification of at-risk patients. This in turn prompts earlier, more aggressive monitoring of IAP and facilitates the institution of corrective/preventive measures before full-blown ACS develops. Prevention of ACS also involves accurate determination of appropriate end points of resuscitation (69–74) in order to avoid excess fluid administration. While the specific variables used to determine end points of resuscitation may always be debated, the adage that “if some urine is good, more urine is better” is clearly obsolete. Furthermore, development of ACS is just one of the adverse clinical consequences of overly vigorous fluid administration. In the setting of IAH, traditional measures of preload may be insufficient due to a concomitant increase in intrathoracic pressure. In these cases, more precise measures of ventricular preload, such as those obtained via global end-diastolic volume (obtained via transpulmonary thermodilution) or continuous right ventricular end-diastolic volume, may be used (75, 76). Once goal-directed resuscitation end points (i.e., decrease in lactate, adequate mixed venous oxygen saturation, decrease in base deficit) are achieved, ongoing aggressive resuscitation should be stopped just as aggressively. Finally, primary ACS can be forestalled by the recognition of patients at

risk while still in the operating room. In high-risk patients it may be safest for the surgeon to use prophylactic measures, such as temporary abdominal closure, to allow the abdominal contents to expand more freely (77–80). However, when patients return to the ICU with a temporary abdominal closure, it is still imperative that the ICU team avoid overresuscitation. Monitoring the APP during this period may provide guidance in determining the trends during resuscitation, and if it is not possible to maintain an APP >60 , then additional surgical intervention should be considered.

The definitive treatment for fully manifested ACS is surgical decompression via a laparotomy (1, 81–83). When performed appropriately, surgical decompression is almost always effective, often immediately and dramatically, with rapid resolution of hypotension, oliguria, and elevated airway pressure. Surgical decompression requires the presence of a surgeon and usually requires general anesthesia. Numerous reports attest to the fact that surgical decompression can be safely performed at the bedside in the ICU (84, 85), an important factor to consider, since many patients with ACS are clinically unstable. Surgical decompression also involves a surgical stress that may serve as a second hit with respect to the immunoinflammatory status of the patient (86, 87). Additionally, the rapid resolution of the hemodynamic consequences of ACS may lead to ischemia-reperfusion injury to the visceral organs (88). Successful surgical decompression of ACS creates another problem: how to deal with the open abdominal wound. The rate of unsuccessful reclosure of the abdomen has been reported between 20% and 78% (89–91). A substantial number of these patients represent major challenges with respect to coverage of the bowel, prevention and management of enterocutaneous fistulas, and management of large ventral hernias. Because of these potential consequences to an open abdomen, attempts should be made to close the abdomen as soon as the underlying cause of the ACS has been addressed, since the incidence of failed closure and additional complications increases as time passes. This timeframe can be short but varies greatly from patient to patient, and actual timing depends greatly on the assessment of the abdomen by the surgeons involved.

While surgical decompression is unquestionably a rapid and definitive treatment, given the consequences of this pro-

Table 5. Nonsurgical treatment options for elevated intra-abdominal pressure/abdominal compartment syndrome

Gastric decompression (nasogastric suction)
Rectal decompression (enemas, rectal drainage catheter/tube)
Sedation
Neuromuscular blockade
Body positioning
Paracentesis
Prokinetic agents (stomach: cisapride, metoclopramide, domperidone, erythromycin; colon: prostigmine)
Diuretics (alone or in combination with 25% human albumin)
Venovenous hemofiltration/ultrafiltration

cedure there is increasing enthusiasm for nonsurgical treatment options of elevated IAH, IAP, and ACS (Table 5). Many of these measures are employed relatively routinely (e.g., nasogastric decompression and sedation) in critically ill patients. Prokinetic agents, enemas, or placement of a rectal decompression tube will seldom dramatically change IAP and should be used very cautiously in cases where toxic megacolon or inflammatory bowel syndrome is suspected. Likewise, the impact of body positioning should not be underestimated as a potentially confounding variable. Avoiding acute flexion at the hips (even though this is counter to the current ICU guideline to maintain a 30–45° elevation of the head of the bed) and/or employing reverse Trendelenburg can relieve pressure on the abdomen. Neuromuscular blockade may dramatically decrease IAP and can be administered quickly and safely to intubated ICU patients (28, 29, 92–96). Even small amounts of tension in the abdominal muscles can have a large impact on IAP, and this component can easily be removed with neuromuscular blockade. In many cases, decreases in IAP with neuromuscular blockade may provide sufficient time for other nonoperative measures, such as removal of excess fluid, to be effective.

Excess fluid can and should be aggressively removed (by diuretics or ultrafiltration); however, it is absolutely crucial to ensure that fluid removal does not result in impaired oxygen delivery or systemic or local perfusion. In our practice we often employ invasive hemodynamic monitoring (e.g., pulmonary artery catheter) with continuous measurements of cardiac output and mixed venous oxygen saturation to avoid hypoperfusion and exacerbation of shock. If accessible free

fluid is present in the abdomen, percutaneous catheter drainage has been reported to be a successful treatment for ACS (31, 81, 97–100). Even with primary ACS there may be moderately large pockets of accessible fluid, and removal of even small volumes can significantly lower IAP (100). Bedside ultrasound can be useful for identifying a safe window for placing the drainage catheter in the setting of abdominal distension and ACS (97). Generally, success with catheter drainage is early and dramatic. Therefore, if ACS persists after catheter drainage, definitive treatment (i.e., surgical decompression) should not be delayed in the hope that repositioning the drainage catheter will improve the situation (81).

Management of the open abdomen following surgical decompression is an area of controversy and ongoing research. There are many modifications of the classic Bogota bag abdominal closure (78, 79, 101–103); the most important consideration when performing a temporary abdominal closure is to allow for additional bowel swelling, thereby avoiding recurrent ACS. When ACS and elevated IAP have resolved, an effort to close the abdomen with autogenous tissue should be made as soon as the capillary leak and edema have resolved (34, 90, 103, 104). In this phase, aggressive, deliberate diuresis and/or ultrafiltration can be used to remove excess total body water. While the window of opportunity for primary fascial closure may vary from patient to patient, in general it is ~7 days; beyond this time the development of adhesions and early granulation tissue often leads to a “frozen abdomen” that precludes fascial closure. The chances of success can be further augmented by progressive incremental closure or increased tension of the temporary closure (80, 105–107). Additionally, the use of vacuum-assisted wound devices may aid in closing the abdomen or at least potentially reduce the size of any residual ventral hernia (81, 89). Clinicians should remain vigilant for the possibility of recurrent ACS during these efforts. Abdominoplasty, in the form of component separation and closure, has also been reported (80, 108, 109), although these sometimes extensive procedures are usually limited to more stable patients. An excellent review of issues related to management of the open abdomen has been recently published (90).

CONCLUSIONS

The detrimental impact of elevated IAP, progressing to ACS, is recognized more frequently in both surgical and medical ICUs. Because of the frequency of this condition and the subtle clinical findings, routine measurement of IAP should be performed in all high-risk ICU patients. A number of measures can be employed to minimize the risk of developing elevated IAP and to aggressively treat IAH when identified. Nonsurgical measures can effectively treat lesser degrees of IAH and ACS, but surgical decompression remains the gold standard for rapid, definitive treatment of fully developed ACS. The recent international ACS consensus conference has helped to define, characterize, and raise awareness of this condition. We hope that this review will assist in understanding and recognizing ACS and ultimately will lead to the development of even more effective clinical treatment guidelines (110, 111).

REFERENCES

1. Kimball EJ, Rollins MD, Mone MC, et al: Survey of intensive care physicians on the recognition and management of intra-abdominal hypertension and abdominal compartment syndrome. *Crit Care Med* 2006; 34:2340–2348
2. Kron IL, Harman PK, Nolan SP: The measurement of intra-abdominal pressure as a criterion for abdominal re-exploration. *Ann Surg* 1984; 199:28–30
3. Bloomfield GL, Dalton JM, Sugerman HJ, et al: Treatment of increasing intracranial pressure secondary to the acute abdominal compartment syndrome in a patient with combined abdominal and head trauma. *J Trauma* 1995; 39:1168–1170
4. Burch JM, Moore EE, Moore FA, et al: The abdominal compartment syndrome. *Surg Clin North Am* 1996; 76:833–842
5. Ciresi DL, Cali RF, Senagore AJ: Abdominal closure using nonabsorbable mesh after massive resuscitation prevents abdominal compartment syndrome and gastrointestinal fistula. *Am Surg* 1999; 65:720–724
6. Eddy VA, Key SP, Morris JA: Abdominal compartment syndrome: Etiology, detection, and management. *J Tenn Med Assoc* 1994; 87:55–57
7. Ertel W, Oberholzer A, Platz A, et al: Incidence and clinical pattern of the abdominal compartment syndrome after “damage-control” laparotomy in 311 patients with severe abdominal and/or pelvic trauma. *Crit Care Med* 2000; 28:1747–1753
8. Fietsam R, Villalba M, Glover JL, et al: Intra-abdominal compartment syndrome as a complication of ruptured abdominal aortic

- aneurysm repair. *Am Surg* 1989; 55: 396–402
9. Fraipont V, Lambermont B, Ghaye B, et al: Unusual complication after percutaneous dilatational tracheostomy: Pneumoperitoneum with abdominal compartment syndrome. *Intensive Care Med* 1999; 25: 1334–1335
 10. Gorecki PJ, Kessler E, Schein M: Abdominal compartment syndrome from intractable constipation. *J Am Coll Surg* 2000; 190:371
 11. Haan J, Johnson SB, Scalea T: Ovarian tumor causing abdominal compartment syndrome. *J Trauma* 2007; 62:768–769
 12. Hirshberg A, Mattox KL: Planned reoperation for severe trauma. *Ann Surg* 1995; 222: 3–8
 13. Hunter JG: Laparoscopic pneumoperitoneum: The abdominal compartment syndrome revisited. *J Am Coll Surg* 1995; 181: 469–470
 14. Ivy ME, Possenti PP, Kepros J, et al: Abdominal compartment syndrome in patients with burns. *J Burn Care Rehabil* 1999; 20: 351–353
 15. Lambert DM, Marceau S, Forse RA: Intra-abdominal pressure in the morbidly obese. *Obes Surg* 2005; 15:1225–1232
 16. Lohlun J, Margolis M, Gorecki P, et al: Fecal impaction causing megarectum-producing colorectal catastrophes: A report of two cases. *Dig Surg* 2000; 17:196–198
 17. Maxwell RA, Fabian TC, Croce MA, et al: Secondary abdominal compartment syndrome: An underappreciated manifestation of severe hemorrhagic shock. *J Trauma* 1999; 47:995–999
 18. Meldrum DR, Moore FA, Moore EE, et al: Jack A. Barney Resident Research Award. Cardiopulmonary hazards of perihepatic packing for major liver injuries. *Am J Surg* 1995; 170:537–540
 19. Neville HL, Lally KP, Cox CS: Emergent abdominal decompression with patch abdominoplasty in the pediatric patient. *J Pediatr Surg* 2000; 35:705–708
 20. Oelschläger BK, Boyle EM, Johansen K, et al: Delayed abdominal closure in the management of ruptured abdominal aortic aneurysms. *Am J Surg* 1997; 173:411–415
 21. Orlando R III, Eddy VA, Jacobs LM Jr, et al: The abdominal compartment syndrome. *Arch Surg* 2004; 139:415–422
 22. Pupelis G: Renal failure in acute pancreatitis: Timing of dialysis and surgery. *Przeegl Lek* 2000; 57(Suppl 5):29–31
 23. Schein M, Wittmann DH, Arahamian CC, et al: The abdominal compartment syndrome: The physiological and clinical consequences of elevated intra-abdominal pressure. *J Am Coll Surg* 1995; 180:745–753
 24. von Gruenigen VE, Coleman RL, King MR, et al: Abdominal compartment syndrome in gynecologic surgery. *Obstet Gynecol* 1999; 94:830–832
 25. Phadnis J, Pilling JE, Evans TW, et al: Abdominal compartment syndrome: A rare complication of plication of the diaphragm. *Ann Thorac Surg* 2006; 82:334–336
 26. Sugrue M: Abdominal compartment syndrome. *Curr Opin Crit Care* 2005; 11: 333–338
 27. Malbrain ML, Deeren D, De Potter TJ: Intra-abdominal hypertension in the critically ill: It is time to pay attention. *Curr Opin Crit Care* 2005; 11:156–171
 28. Morken J, West MA: Abdominal compartment syndrome in the intensive care unit. *Curr Opin Crit Care* 2001; 7:268–274
 29. Cheatham ML, Malbrain ML, Kirkpatrick A, et al: Results from the International Conference of Experts on Intra-abdominal Hypertension and Abdominal Compartment Syndrome: II. Recommendations. *Intensive Care Med* 2007; 33:951–962
 30. Malbrain ML, De laet I, Cheatham M: Consensus conference definitions and recommendations on intra-abdominal hypertension (IAH) and the abdominal compartment syndrome (ACS)—The long road to the final publications, how did we get there? *Acta Clin Belg Suppl* 2007; (1):44–59
 31. Reed SF, Britt RC, Collins J, et al: Aggressive surveillance and early catheter-directed therapy in the management of intra-abdominal hypertension. *J Trauma* 2006; 61:1359–1363
 32. Meldrum DR, Moore FA, Moore EE, et al: Prospective characterization and selective management of the abdominal compartment syndrome. *Am J Surg* 1997; 174: 667–672
 33. Malbrain ML, Cheatham ML, Kirkpatrick A, et al: Results from the International Conference of Experts on Intra-abdominal Hypertension and Abdominal Compartment Syndrome: I. Definitions. *Intensive Care Med* 2006; 32:1722–1732
 34. Miller RS, Morris JA Jr, Diaz JJ Jr, et al: Complications after 344 damage-control open celiotomies. *J Trauma* 2005; 59: 1365–1371
 35. Balogh Z, McKinley BA, Holcomb JB, 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
 36. Britt RC, Gannon T, Collins JN, et al: Secondary abdominal compartment syndrome: Risk factors and outcomes. *Am Surg* 2005; 71:982–985
 37. Kirkpatrick AW, Balogh Z, Ball CG, et al: The secondary abdominal compartment syndrome: Iatrogenic or unavoidable? *J Am Coll Surg* 2006; 202:668–679
 38. Ball CG, Kirkpatrick AW, Karmali S, et al: Tertiary abdominal compartment syndrome in the burn injured patient. *J Trauma* 2006; 61:1271–1273
 39. Wauters J, Wilmer A, Valenza F: Abdomino-thoracic transmission during ACS: Facts and figures. *Acta Clin Belg Suppl* 2007; (1):200–205
 40. Olson SA, Glasgow RR: Acute compartment syndrome in lower extremity musculoskeletal trauma. *J Am Acad Orthop Surg* 2005; 13:436–444
 41. Patel RV, Haddad FS: Compartment syndromes. *Br J Hosp Med (Lond)* 2005; 66: 583–586
 42. Hargens AR, Mubarak SJ: Current concepts in the pathophysiology, evaluation, and diagnosis of compartment syndrome. *Hand Clin* 1998; 14:371–383
 43. Lattuada M, Hedenstierna G: Abdominal lymph flow in an endotoxin sepsis model: Influence of spontaneous breathing and mechanical ventilation. *Crit Care Med* 2006; 34:2792–2798
 44. Malbrain ML, Pelosi P, De laet I, et al: Lymphatic drainage between thorax and abdomen: Please take good care of this well-performing machinery. *Acta Clin Belg Suppl* 2007; (1):152–161
 45. Pelosi P, Quintel M, Malbrain ML: Effect of intra-abdominal pressure on respiratory mechanics. *Acta Clin Belg Suppl* 2007; (1): 78–88
 46. Cresswell AB, Wendon JA: Hepatic function and non-invasive hepatosplanchnic monitoring in patients with abdominal hypertension. *Acta Clin Belg Suppl* 2007; (1): 113–118
 47. Sakka SG: Indocyanine green plasma disappearance rate as an indicator of hepatosplanchnic ischemia during abdominal compartment syndrome. *Anesth Analg* 2007; 104:1003–1004
 48. Perkins JD: Abdominal compartment syndrome: Is the liver involved? *Liver Transpl* 2006; 12:1555–1556
 49. Shear W, Rosner MH: Acute kidney dysfunction secondary to the abdominal compartment syndrome. *J Nephrol* 2006; 19: 556–565
 50. Wiebe S, Kellenberger CJ, Khoury A, et al: Early Doppler changes in a renal transplant patient secondary to abdominal compartment syndrome. *Pediatr Radiol* 2004; 34: 432–434
 51. Doty JM, Saggi BH, Sugerman HJ, et al: Effect of increased renal venous pressure on renal function. *J Trauma* 1999; 47: 1000–1003
 52. Cheatham ML, Malbrain ML: Cardiovascular implications of abdominal compartment syndrome. *Acta Clin Belg Suppl* 2007; (1): 98–112
 53. Al-Bahrani AZ, Abid GH, Sahgal E, et al: A prospective evaluation of CT features predictive of intra-abdominal hypertension and abdominal compartment syndrome in critically ill surgical patients. *Clin Radiol* 2007; 62:676–682
 54. Pickhardt PJ, Shimony JS, Heiken JP, et al: The abdominal compartment syndrome: CT findings. *AJR Am J Roentgenol* 1999; 173: 575–579
 55. Severgnini P, Inzigner G, Olvera C, et al: New and old tools for abdominal imaging in critically ill patients. *Acta Clin Belg Suppl* 2007; (1):173–82
 56. Sugrue M, Buist MD, Lee A, et al: Intra-

- abdominal pressure measurement using a modified nasogastric tube: Description and validation of a new technique. *Intensive Care Med* 1994; 20:588–590
57. Balogh Z, De Waele JJ, Malbrain ML: Continuous intra-abdominal pressure monitoring. *Acta Clin Belg Suppl* 2007; (1):26–32
 58. De laet I, Malbrain ML, Jadoul JL, et al: Renal implications of increased intra-abdominal pressure: Are the kidneys the canary for abdominal hypertension? *Acta Clin Belg Suppl* 2007; (1):119–130
 59. Iberti TJ, Kelly KM, Gentili DR, et al: A simple technique to accurately determine intra-abdominal pressure. *Crit Care Med* 1987; 15:1140–1142
 60. de Laet IE, Malbrain M: Current insights in intra-abdominal hypertension and abdominal compartment syndrome. *Med Intensiva* 2007; 31:88–99
 61. Balogh Z, Jones F, D'Amours S, et al: Continuous intra-abdominal pressure measurement technique. *Am J Surg* 2004; 188: 679–684
 62. Cheatham ML, White MW, Sargraves SG, et al: Abdominal perfusion pressure: A superior parameter in the assessment of intra-abdominal hypertension. *J Trauma* 2000; 49:621–626
 63. Malbrain ML, Chiumello D, Pelosi P, et al: Incidence and prognosis of intraabdominal hypertension in a mixed population of critically ill patients: A multiple-center epidemiological study. *Crit Care Med* 2005; 33: 315–322
 64. Malbrain ML, Chiumello D, Pelosi P, et al: Prevalence of intra-abdominal hypertension in critically ill patients: A multicentre epidemiological study. *Intensive Care Med* 2004; 30:822–829
 65. Mehta M, Darling RC III, Roddy SP, et al: Factors associated with abdominal compartment syndrome complicating endovascular repair of ruptured abdominal aortic aneurysms. *J Vasc Surg* 2005; 42: 1047–1051
 66. McNelis J, Marini CP, Jurkiewicz A, et al: Predictive factors associated with the development of abdominal compartment syndrome in the surgical intensive care unit. *Arch Surg* 2002; 137:133–136
 67. Dry SM, Bechard KM, Milford EL, et al: The pathology of transfusion-related acute lung injury. *Am J Clin Pathol* 1999; 112:216–221
 68. Crimi E, Slutsky AS: Inflammation and the acute respiratory distress syndrome. *Best Pract Res Clin Anaesthesiol* 2004; 18: 477–492
 69. McIntyre LA, Hebert PC, Fergusson D, et al: A survey of Canadian intensivists' resuscitation practices in early septic shock. *Crit Care* 2007; 11:R74
 70. Englehart MS, Schreiber MA: Measurement of acid-base resuscitation endpoints: Lactate, base deficit, bicarbonate or what? *Curr Opin Crit Care* 2006; 12:569–574
 71. Goodrich C: Endpoints of resuscitation: what should we be monitoring? *AACN Adv Crit Care* 2006; 17:306–316
 72. McKinley BA, Valdivia A, Moore FA: Goal-oriented shock resuscitation for major torso trauma: What are we learning? *Curr Opin Crit Care* 2003; 9:292–299
 73. Revell M, Greaves I, Porter K: Endpoints for fluid resuscitation in hemorrhagic shock. *J Trauma* 2003; 54(5 Suppl):S63–S67
 74. Dabrowski GP, Steinberg SM, Ferrara JJ, et al: A critical assessment of endpoints of shock resuscitation. *Surg Clin North Am* 2000; 80:825–844
 75. Schachtrupp A, Lawong G, Afify M, et al: Fluid resuscitation preserves cardiac output but cannot prevent organ damage in a porcine model during 24 h of intraabdominal hypertension. *Shock* 2005; 24:153–158
 76. Cheatham ML, Safcsak K, Block EF, et al: Preload assessment in patients with an open abdomen. *J Trauma* 1999; 46:16–22
 77. Shapiro MB, Jenkins DH, Schwab CW, et al: Damage control: Collective review. *J Trauma* 2000; 49:969–978
 78. Murdock AD: What is the standard approach to temporary abdominal closure? *J Trauma* 2007; 62(6 Suppl):S29
 79. Kirshtein B, Roy-Shapira A, Lantsberg L, et al: Use of the "Bogota bag" for temporary abdominal closure in patients with secondary peritonitis. *Am Surg* 2007; 73:249–252
 80. Howdieshell TR, Proctor CD, Sternberg E, et al: Temporary abdominal closure followed by definitive abdominal wall reconstruction of the open abdomen. *Am J Surg* 2004; 188:301–306
 81. Latenser BA, Kowal-Vern A, Kimball D, et al: A pilot study comparing percutaneous decompression with decompressive laparotomy for acute abdominal compartment syndrome in thermal injury. *J Burn Care Rehabil* 2002; 23:190–195
 82. De Waele JJ, Hoste EA, Malbrain ML: Decompressive laparotomy for abdominal compartment syndrome—A critical analysis. *Crit Care* 2006; 10:R51
 83. Hunter JD, Damani Z: Intra-abdominal hypertension and the abdominal compartment syndrome. *Anaesthesia* 2004; 59:899–907
 84. Ivatury RR, Porter JM, Simon RJ, et al: 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
 85. Diaz JJ Jr, Mejia V, Subhawong AP, et al: Protocol for bedside laparotomy in trauma and emergency general surgery: A low return to the operating room. *Am Surg* 2005; 71:986–991
 86. Rotstein OD: Modeling the two-hit hypothesis for evaluating strategies to prevent organ injury after shock/resuscitation. *J Trauma* 2003; 54(5 Suppl):S203–S206
 87. Claridge JA, Weed AC, Enelow R, et al: Laparotomy potentiates cytokine release and impairs pulmonary function after hemorrhage and resuscitation in mice. *J Trauma* 2001; 50:244–252
 88. Kacmaz A, Polat A, User Y, et al: Octreotide improves reperfusion-induced oxidative injury in acute abdominal hypertension in rats. *J Gastrointest Surg* 2004; 8:113–119
 89. Miller PR, Meredith JW, Johnson JC, et al: Prospective evaluation of vacuum-assisted fascial closure after open abdomen: Planned ventral hernia rate is substantially reduced. *Ann Surg* 2004; 239:608–614
 90. Fabian TC: Damage control in trauma: Laparotomy wound management acute to chronic. *Surg Clin North Am* 2007; 87:73–93, vi
 91. Joels CS, Vanderveer AS, Newcomb WL, et al: Abdominal wall reconstruction after temporary abdominal closure: A ten-year review. *Surg Innov* 2006; 13:223–230
 92. Macalino JU, Goldman RK, Mayberry JC: Medical management of abdominal compartment syndrome: Case report and a caution. *Asian J Surg* 2002; 25:244–246
 93. Hershberger RC, Hunt JL, Arnoldo BD, et al: Abdominal compartment syndrome in the severely burned patient. *J Burn Care Res* 2007; 28:708–714
 94. De Waele JJ, Benoit D, Hoste E, et al: A role for muscle relaxation in patients with abdominal compartment syndrome? *Intensive Care Med* 2003; 29:332
 95. De laet I, Malbrain ML: ICU management of the patient with intra-abdominal hypertension: What to do, when and to whom? *Acta Clin Belg Suppl* 2007; (1):190–199
 96. De Laet I, Hoste E, Verhoben E, et al: The effect of neuromuscular blockers in patients with intra-abdominal hypertension. *Intensive Care Med* 2007; 33:1811–1814
 97. Reckard JM, Chung MH, Varma MK, et al: Management of intraabdominal hypertension by percutaneous catheter drainage. *J Vasc Interv Radiol* 2005; 16:1019–1021
 98. Tuggle D, Skinner S, Garza J, et al: The abdominal compartment syndrome in patients with burn injury. *Acta Clin Belg Suppl* 2007; (1):136–140
 99. Corcos AC, Sherman HF: Percutaneous treatment of secondary abdominal compartment syndrome. *J Trauma* 2001; 51: 1062–1064
 100. Leppaniemi A, Johansson K, De Waele JJ: Abdominal compartment syndrome and acute pancreatitis. *Acta Clin Belg Suppl* 2007; (1):131–135
 101. Myers JA, Latenser BA: Nonoperative progressive "Bogota bag" closure after abdominal decompression. *Am Surg* 2002; 68: 1029–1030
 102. Navsaria PH, Bunting M, Omoshoro-Jones J, et al: Temporary closure of open abdominal wounds by the modified sandwich-vacuum pack technique. *Br J Surg* 2003; 90:718–722
 103. Barker DE, Green JM, Maxwell RA, et al: Experience with vacuum-pack temporary abdominal wound closure in 258 trauma and general and vascular surgical patients. *J Am Coll Surg* 2007; 204:784–792

104. Pearl ML, Rayburn WF: Choosing abdominal incision and closure techniques: A review. *J Reprod Med* 2004; 49:662–670
105. Losanoff JE, Richman BW, Jones JW: Adjustable suture-tension closure of the open abdomen. *J Am Coll Surg* 2003; 196:163–164
106. Miller PR, Thompson JT, Faler BJ, et al: Late fascial closure in lieu of ventral hernia: The next step in open abdomen management. *J Trauma* 2002; 53:843–849
107. Cothren CC, Moore EE, Johnson JL, et al: One hundred percent fascial approximation with sequential abdominal closure of the open abdomen. *Am J Surg* 2006; 192:238–242
108. Jernigan TW, Fabian TC, Croce MA, et al: Staged management of giant abdominal wall defects: Acute and long-term results. *Ann Surg* 2003; 238:349–355
109. Hultman CS, Pratt B, Cairns BA, et al: Multidisciplinary approach to abdominal wall reconstruction after decompressive laparotomy for abdominal compartment syndrome. *Ann Plast Surg* 2005; 54:269–275
110. Balogh Z, De Waele JJ, Kirkpatrick A, et al: Intra-abdominal pressure measurement and abdominal compartment syndrome: The opinion of the World Society of the Abdominal Compartment Syndrome. *Crit Care Med* 2007; 35:677–678; author reply 678–679
111. Kimball EJ, Kim W, Cheatham ML, et al: Clinical awareness of intra-abdominal hypertension and abdominal compartment syndrome in 2007. *Acta Clin Belg Suppl* 2007; (1):66–73