The secondary abdominal compartment syndrome: not just another post-traumatic complication

Chad G. Ball, MD, MSc;* Andrew W. Kirkpatrick, MD;†‡ Paul McBeth, MD‡

The secondary abdominal compartment syndrome (ACS) is defined as the presence of organ dysfunction with concurrent intra-abdominal hypertension (IAH) in a scenario lacking primary intraperitoneal injury or intervention. This state appears to be related to visceral, abdominal wall and retroperitoneal edema and ascites induced by resuscitation. Despite a diverse range of associated causes such as pancreatitis, intra-abdominal sepsis, cardiac arrest, thermal injury and extraperitoneal trauma, this class of ACS is characterized by the presence of shock requiring aggressive fluid resuscitation. Secondary ACS is an extreme condition along a continuum of raised intra-abdominal pressure (IAP) that is pathoneumonic when associated with new overt organ failure. When IAP is above normal but is not associated with organ failure, IAH is diagnosed. Because these conditions are common among critically ill patients, the measurement of IAP is crucial. It is unclear whether preventing IAH reduces progression to ACS or influences outcomes. When overt ACS is confirmed, immediate surgical decompression of the patient's abdomen via a standard laparotomy is usually required. Because many disease processes resulting in critical illness require aggressive fluid resuscitation as a primary therapy, it is likely that secondary ACS is much more common than previously believed. Further study is needed.

Le syndrome du compartiment abdominal (SCA) secondaire s'entend de la présence d'un dysfonctionnement organique conjugué à une hypertension intra-abdominale (HIA) dans un scénario où il n'y a pas d'intervention ou de traumatisme intrapéritonéal primaire. Cet état semble relié à la paroi abdominale des viscères, ainsi qu'à l'œdème rétropéritonéal et à l'ascite provoqués par la réanimation. En dépit d'un éventail diversifié de causes connexes comme la pancréatite, un sepsis intra-abdominal, l'arrêt cardiaque, le traumatisme thermique et le traumatisme extrapéritonéal, cette catégorie de SCA est caractérisée par la présence d'un choc qui nécessite une réanimation liquidienne agressive. Le SCA secondaire est un problème extrême dans un continuum d'élévation de la pression intra-abdominale (PIA) qui est pathopneumonique lorsqu'il est associé à une nouvelle défaillance organique évidente. Lorsque la PIA dépasse la normale mais n'est pas associée à une défaillance organique, on diagnostique une HIA. Comme ces problèmes sont courants chez les patients en phase critique, il est crucial de mesurer la PIA. On ne sait pas clairement si la prévention de l'hypertension intra-abdominale atténue l'évolution en SCA ou a un effet sur les résultats. Lorsque l'on confirme un SCA évident, il faut habituellement procéder sur-le-champ à la décompression chirurgicale de l'abdomen du patient par laparotomie ordinaire. Comme beaucoup de processus morbides qui aboutissent à une maladie critique nécessitent une réanimation liquidienne agressive comme thérapie de première intention, il est probable que le SCA secondaire est beaucoup plus courant qu'on le croyait auparavant. Une étude plus poussée s'impose.

A compartment syndrome is a condition of increased pressure in a confined anatomic space that adversely affects the circulation and threatens the function and viability of the tissues therein. This may arise in any closed compartment within the body. Secondary abdominal compartment syndrome (ACS) is characterized by the presence of shock requiring aggressive fluid resuscitation. Because many disease

*From the *Departments of Trauma, Surgery and Critical Care, Grady Memorial Hospital, Emory University, Atlanta, Ga., and the †Department of Critical Care Medicine and the Regional Trauma Program, and the ‡Department of Surgery, Foothills Medical Centre, University of Calgary, Calgary, Alta.*

Accepted for publication May 28, 2008

Correspondence to: *Dr. A.W. Kirkpatrick, Department of Surgery, Foothills Medical Centre, 1403-29th Street NW, Calgary AB T2N 2T9; fax 403 944-1277; andrew.kirkpatrick@calgaryhealthregion.ca*

processes resulting in critical illness require aggressive fluid resuscitation as a primary therapy, it is likely that this condition is much more common than previously believed.

Background

The World Society of the Abdominal Compartment Syndrome (WSACS) defines ACS as a sustained increase in intra-abdominal pressure (IAP) greater than 20 mm Hg concurrent with a new onset of organ dysfunction.[1] This state of organ failure frequently affects the cardiovascular, respiratory and renal systems.[2–8] The condition is uniformly fatal if left untreated.[2–8]

It is widely understood that ACS represents the end stage of a pathophysiologic spectrum that begins with normal IAP, proceeds to intra-abdominal hypertension (IAH) and ends with overt ACS. To standardize definitions and facilitate communication and research, the WSACS recently outlined working definitions of the conditions.[1,9] They also published evidence-based guidelines for the diagnosis, measurement, management and prevention of IAH and ACS. These documents will be revised regularly and are intended to provide guidance to clinicians.

Historically, ACS was often diagnosed when the effects of IAH had become overtly obvious. The signs included severe respiratory distress, elevated peak airway pressures, hypotension, diminished cardiac output and oliguria.[2,3,6] Diagnosing IAH and ACS at this point is clearly too late, as evidenced by an increasingly poor prognosis, infectious complications and death.[1–16] Because many of the effects of ACS are clinically indistinguishable from those of other common syndromes related to critical illness, it is probable that the influence of abnormal IAP is not infrequently missed in a critically ill patient with multifactorial complications. As a result, clinicians must possess a high index of suspicion and monitor IAP aggressively.

Definitions

The WSACS defines IAP as the pressure within the abdominal cavity, measured at end-expiration in a relaxed, supine patient.[1] It defines IAH as a sustained elevation of IAP greater than 12 mm Hg.[1] It further classifies IAH into grades I–IV (Box 1). The WSACS defines ACS as a sustained increase of IAP greater than 20 mm Hg that is associated with the onset of organ dysfunction.[1] Because abdominal perfusion pressure (APP) is potentially a more accurate predictor of visceral perfusion, and a theoretically superior outcome of resuscitation, it is often included in the definition of ACS (APP < 60 mm Hg).[1,17] This index is derived by subtracting the IAP from the mean arterial pressure, and it should be considered in relation to the overall physiologic status of the patient.

The ACS can also be subcategorized based on its causes. Primary or "surgical" ACS is associated with an injury or disease in the abdominopelvic region that requires surgical or angiographic intervention.[1] This is also considered to be "classic" ACS. Patients with primary ACS typically have intraperitoneal or retroperitoneal bleeding, solid organ injury, damage control surgery (e.g., packing of liver hemorrhage) or transplantation. Primary ACS also includes bleeding pelvic fractures.[1]

Secondary or "medical" ACS, the focus of our review, is a fundamentally unique entity because it occurs in patients without a primary intraperitoneal injury or intervention.[1] This terminology represents a significant departure from the same descriptors employed in the lexicon of peritonitis.[10]

Finally, tertiary ACS happens when ACS recurs despite attempts at prophylactic or therapeutic treatment of either primary or secondary IAH or ACS.[1,11] Examples may include persistent ACS despite surgical decompression or an entirely new episode of ACS after the fascia has been reapproximated following temporary abdominal closure.

Incidence and cause

IAH and ACS are diseases related to critical illness. Their incidence reflects both the acuity of the population and a clinician's diligence in measuring IAP. The reported incidence of IAH and ACS is about 32.1% and 4.2%, respectively, in the mixed intensive care unit (ICU) population.[12] Rates of IAH have also been reported for patients with severe burns (36.7%–70%)[4,7,13] and traumatic injuries (2%–50%)[6,14–16] and for patients who had major abdominal procedures (31.5%–40.7%).[14–16,18–21] Overt ACS occurs in 1%–31% of those with burns[4,7,13,22] and in 0.5%–36% of those with injuries.[15,16,23–29] An institution with both an aggressive fluid resuscitation protocol and diligent IAP monitoring reported an incidence of 8% for secondary ACS and an incidence of 6% for primary ACS in severely injured trauma patients who presented with shock.[18] When compared with patients with primary ACS, secondary IAH has also been shown to occur later in a patient's hospital admission and to be associated with more prolonged and severe elevations of IAP and with higher mortality.[30]

Box 1. World Society of the Abdominal Compartment Syndrome definitions of intra-abdominal hypertension and abdominal compartment syndrome

Condition	Measured intra-abdominal pressure
Normal	< 12 mm Hg
IAH	
Grade I	12–15 mm Hg
Grade II	16–20 mm Hg
Grade III	21–25 mm Hg
Grade IV	> 25 mm Hg
ACS	> 20 mm Hg + new organ failure **or** APP* < 60 mm Hg + new organ failure

ACS = abdominal compartment syndrome; APP = abdominal perfusion pressure; IAH = intra-abdominal hypertension.
*Mean arterial pressure minus intra-abdominal pressure.

The occurrence of secondary ACS appears to be directly related to visceral, abdominal wall and retroperitoneal edema and ascites induced by resuscitation.[1,2,4,7–9,12,13,16,17,28,31–43] Despite a diverse range of associated conditions, secondary ACS is characterized by the presence of shock requiring aggressive fluid resuscitation (Box 2). This situation is compounded by the swelling of the intestinal wall and luminal distension that accompanies shock/resuscitation. The recognition that aggressive crystalloid fluid resuscitation in shock necessitates an obligatory loss of fluid to the intracellular or "third" space has nearly eliminated renal failure as a complication of burns.[44,45] Unfortunately this benefit must be balanced against the possibility that excess crystalloid resuscitation may contribute to an increased occurrence of IAH and, therefore, secondary ACS. The phenomenon of "fluid creep" is an important problem in modern burn care, and it likely applies to other areas of critical care.[46,47] This concept was most recently supported in a large pan-European ICU study of sepsis that showed a positive fluid balance as the strongest prognostic factor for death.[48]

Box 2. Risk factors for IAH/ACS

- Acidosis (pH < 7.2)
- Hypothermia (core temperature < 33°C)
- Polytransfusion (> 10 U packed red blood/24 h)
- Coagulopathy (platelets < 55 000/mm^3, or activated partial thromboplastin time twice the normal level or higher, or prothrombin time > 15 s, or international standardized ratio > 1.5)
- Sepsis (American-European Consensus Conference definitions)
- Bacteremia
- Intra-abdominal infection/abscess
- Peritonitis
- Liver dysfunction/cirrhosis with ascites
- Mechanical ventilation
- Use of positive end expiratory pressure (PEEP) or the presence of auto-PEEP
- Pneumonia
- Abdominal surgery, especially with tight fascial closures
- Massive fluid resuscitation (> 5l colloid or crystalloid/24 h)
- Gastroparesis/gastric distention/ileus
- Volvulus
- Hemoperitoneum/pneumoperitoneum
- Major burns
- Major trauma
- High body mass index (> 30 kg/m^2)
- Intra-abdominal or retroperitoneal tumours
- Prone positioning
- Massive incisional hernia repair
- Acute pancreatitis
- Distended abdomen
- Damage control laparotomy
- Laparoscopy with excessive inflation pressures
- Peritoneal dialysis

IAH = intra-abdominal hypertension; ACS = abdominal compartment syndrome. Adapted from Malbrain et al.[1]

Pathophysiology

The pathophysiology of secondary ACS affects the entire body and is identical to primary ACS. Cardiac output is reduced owing to decreased preload and right heart volumes. Although increased systemic vascular resistance initially maintains apparent blood pressure, decreases in preload from the pooling of blood in splanchnic and lower extremity vascular beds eventually lead to reduced central venous return.[3,35,49–53] Cardiac underfilling also occurs despite apparently increased central hemodynamic measurements (central venous pressure [CVP] and pulmonary artery occlusion pressure). As respiratory compliance decreases, mechanical ventilation with increased ventilatory pressures and decreased volumes becomes difficult.[54,55] The partial pressures of oxygen will decrease, and carbon dioxide will increase.[56,57] Even modest IAH appears to exacerbate acute lung injury and the acute respiratory distress syndrome (ARDS). When IAP levels greater than 20 mm Hg are applied to critically ill animals, a dramatic exacerbation of ARDS-associated pulmonary edema is evident.[58] Furthermore, elevated IAP results in a stiffer chest wall with much lower transpulmonary pressures, and therefore less susceptibility to ventilator-induced lung injury.[59,60] Oliguria is a common manifestation of the ACS because renal failure and IAH are dose-dependant.[3,19,20] These effects are exaggerated by hypovolemia and positive end-expiratory pressure.[56,61]

Beyond the heart, lungs and kidneys, almost every other organ system is altered by IAH, even if the effects are not clinically overt. Also, IAH appears to contribute to increased intracerebral pressure (ICP) via transmitted intrathoracic pressure[62–65] to the extent that laparotomies have been reported to reduce ICP in patients with secondary ACS.[66] Patients in shock are at a particularly high risk for splanchnic malperfusion because even modest elevations in IAP greatly reduce hepatic and splanchnic perfusion.[67–70] This effect is exacerbated by prior hemorrhage[71] and is observed at much lower IAPs than required to induce other clinical features of ACS. As a result, subtle organ failure, concurrent to multisystem disease compatible with other causes, may be difficult to ascertain.

Clinical settings

Secondary ACS or IAH are typically described in patients with either physical or thermal injuries. Patients with burns are at a particularly high risk for secondary ACS when the burns cover 70% or more of the body surface area (BSA). Patients with smaller burns and concomitant inhalation injuries are also at risk for secondary ACS.[7,13,22,33] It should be noted that circumferential abdominal eschar is not a prerequisite for the condition.[7,22,33] As stated previously, patients with an intraperitoneal injury who develop IAH or ACS are classified as having primary IAH or ACS. However, if no intraperitoneal injury is present, IAH or ACS may still develop if massive fluid resuscitation is required to treat hemodynamic instability resulting from injuries that are anatomically distant from the peritoneal cavity.[49] Secondary visceral swelling from massive resuscitation is

especially common in patients with displaced pelvic fractures. This scenario is classified as secondary ACS if the pelvic hematoma is not the primary cause of IAH or ACS. These patients typically have hypothermia, coagulopathy and acidosis, and they require ongoing resuscitation even after admission to the ICU.[32] Those with secondary ACS were reported to have longer stays in the emergency department, receive substantially more crystalloid fluid and experience prolonged delays before undergoing therapeutic angiography.[28] These findings reinforce the need for early hemorrhage control rather than ongoing fluid resuscitation.

In addition, IAH and ACS likely influence the clinical course of many critically ill patients with sepsis. This is a result of both primary intraperitoneal disease and the massive fluid resuscitation that is often required to stabilize hemodynamics in patients with severe sepsis or septic shock. Severe sepsis is a leading cause of death in ICUs throughout the world, with mortality approaching 30%[72,73] and an incidence that has nearly doubled over the last 11 years.[74] Recently updated international guidelines continue to prioritize fluid resuscitation to obtain a CVP between 8 and 12 mm Hg.[75] This typically requires aggressive fluid administration during the first 24 hours of management.[76] If IAH is recognized, even higher targets are required. Whether this increased emphasis on early (the period of maximal risk for secondary ACS) aggressive fluid resuscitation in the case of sepsis will lead to an increased incidence of IAH, and hence secondary ACS, remains unknown. In our own ICU, we detected IAH (IAP > 12 mm Hg) in 87% of study participants with sepsis.[77] Furthermore, elevated IAP correlated with organ dysfunction, the need for continuous renal replacement therapy and mortality.[77] This is supported by similar observations,[12,78] including a recent report that noted sepsis to be the leading cause (39%) of secondary IAH.[30]

Current indications and techniques to measure intra-abdominal pressure

Clinical examination has repeatedly been shown to be inaccurate in detecting elevated IAP.[6,79] Although numerous modalities are available, the current gold standard technique for noninvasive measurements uses the patient's urinary bladder for pressure transduction.[19,80–83] It is recommended that the patient be supine and that the transducer be zeroed at the iliac crest in the midaxillary line.[1] We believe a given clinician can select any technique, as long as it is familiar to ICU nursing staff and easily repeated in the daily care of a patient.

Indications for measuring IAP should include all risk factors for the development of IAH or ACS. These indications are numerous and have recently been summarized by the WSACS.[1] We feel it is prudent to screen all critically ill patients upon admission to an ICU, or as a minimum, all those at risk for IAH and ACS. In the case of secondary ACS, patients at high risk include but are not limited to those with abdominal infection (e.g., pancreatitis, peritonitis), thermal injury and intraperitoneal or retroperitoneal hemorrhage, as well as any patient who has received large volumes of fluid resuscitation.[1] Although authors have become increasingly conservative with the threshold volumes of crystalloid considered to be significant,[7,12,22,29,34,35] there does not appear to be a single predictive factor that is reliable and accurate in predicting secondary IAH or ACS other than critical illness. Studies performed at the University of Colorado have noted that the time required to diagnose secondary ACS in nontrauma patients was twice that required in patients with injuries, likely owing to a decreased awareness of this phenomenon outside of trauma.[35,49] Given the evidence that IAH is an independent predictor of mortality, these concepts can no longer be ignored in critically ill patients.[4,7,12,22,25,29,32,33,50] Infrequent monitoring of IAP among patients in the ICU is in many ways synonymous to interpreting hemodynamic indices without considering thoracic pressures (i.e., peak and plateau airway and positive end expiratory pressures) and must be avoided.[84,85]

Management

The WSACS has outlined a suggested management strategy for IAH/ACS that is largely derived from consensus and retrospective data.[9] The association currently recommends surgical decompression for secondary ACS when it is refractory to medical treatment options. It remains highly controversial among clinicians as to whether a particular level of IAH should automatically necessitate surgical decompression in the absence of associated organ failure. Most surgeons are reluctant to perform laparotomies in patients who do not meet the complete definition of secondary ACS.[1,9,86,87] Although the current gold standard for overt ACS is surgical decompression of the abdomen via a laparotomy, alternative medical strategies have been described in the treatment of IAH. Although there are no randomized prospective data, authors have suggested using hypertonic crystalloid and colloid-based resuscitation,[1,88,89] neuromuscular blockade, body positioning, percutaneous catheter decompression, sedation, analgesia, nasogastric and colonic decompression, escharotomy or tangential burn wound excision, externally applied continuous negative abdominal pressure devices, and the careful use of diuretics and continuous renal replacement therapies once the initial resuscitation is complete.[1,4,7,22,32,38,65,90] We recommend routine bowel decompression via nasogastric tubes, selective rectal decompression when colonic distension is documented and percutaneous removal of intra-abdominal fluid when demonstrated in patients with secondary ACS. When this fails and/or

ACS develops, we proceed with an emergent decompressive laparotomy.

Although laparotomy often resolves the ACS, a minority of patients (8%–16%) will not obtain a subsequent primary fascial closure.[91–94] In those who survive, the open abdomen is fraught with inherent complications that include fistulae, surgical site infections, sepsis, fluid and electrolyte imbalances from exposed bowel, prolonged ventilatory requirements and massive ventral hernia.[2,39,95] Because the management options are numerous, the appropriate therapy of this condition is becoming a distinct emerging discipline. It is therefore an oversimplification to state that these patients may require absorbable mesh interposition, skin grafting, nonabsorbable mesh and/or component separation repairs.

Summary

Secondary ACS is defined as the onset of organ failure with concurrent IAH in a patient who has not experienced an injury or intervention. This condition is directly related to visceral, abdominal wall and retroperitoneal edema and ascites induced by resuscitation. As a result, secondary ACS is typically characterized by the presence of shock requiring aggressive fluid resuscitation, and therefore includes patients with almost any form of critical illness. With an incidence approaching 30%, ACS can no longer be ignored. The measurement of IAP must occur more often than it is currently among critically ill patients. Furthermore, when overt ACS is confirmed, patients typically require immediate surgical decompression via a standard laparotomy. In conclusion,

- IAH and ACS are common;
- secondary ACS is defined as the onset of organ failure in a patient with IAH > 20 mm Hg;
- secondary ACS occurs in patients without abdominal trauma or intraperitoneal surgery;
- shock and aggressive fluid resuscitation are common among patients with secondary ACS;
- the most common treatment for secondary ACS is an emergent decompressive laparotomy; and
- monitoring IAP to identify ACS is crucial in all critically ill patients.

Competing interests: None declared.

Contributors: Drs. Ball and Kirkpatrick conceived and designed the study. All authors acquired, analyzed and interpreted the data; wrote and reviewed the manuscript critically; and approved the final version for publication.

References

1. Malbrain ML, Cheatham ML, Kirkpatrick AW, et al. Results from the International Conference of Experts on Intra-abdominal Hypertension and Abdominal Compartment Syndrome. I. Definitions. *Intensive Care Med* 2006;32:1722-32.
2. Burch JM, Moore EE, Moore FA, et al. The abdominal compartment syndrome. *Surg Clin North Am* 1996;76:833-42.
3. Nathens AB, Brenneman FD, Boulanger BR. The abdominal compartment syndrome. *Can J Surg* 1997;40:254-8.
4. 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;23:190-5.
5. Schein M, Wittman DH, Aprahamian CC, et al. The abdominal compartment syndrome: the physiological and clinical consequences of elevated intra-abdominal pressure. *J Am Coll Surg* 1995;180:745-52.
6. Kirkpatrick AW, Brenneman FD, McLean RF, et al. Is clinical examination an accurate indicator of raised intra-abdominal pressure in critically injured patients? *Can J Surg* 2000;43:207-11.
7. Ivy ME, Atweh NA, Palmer J, et al. Intra-abdominal hypertension and abdominal compartment syndrome in burn patients. *J Trauma* 2000;49:387-91.
8. Morken J, West MA. Abdominal compartment syndrome in the intensive care unit. *Curr Opin Crit Care* 2001;7:268-74.
9. Cheatham ML, Malbrain ML, Kirkpatrick AW, et al. Results from the international conference of experts on intra-abdominal hypertension and abdominal compartment syndrome. II. Recommendations. *Intensive Care Med* 2007;33:951-62.
10. Nathens AB, Rotstein OD, Marshall JC. Tertiary peritonitis: clinical features of a complex nosocomial infection. *World J Surg* 1998;22:158-63.
11. Ball CG, Kirkpatrick AW, Karmali S, et al. Tertiary abdominal compartment syndrome in the burn patient. *J Trauma* 2006;61:1271-3.
12. Malbrain ML, Chiumello D, Pelosi P, et al. Incidence and prognosis of intra-abdominal hypertension in a mixed population of critically ill patients: a multiple-center epidemiological study. *Crit Care Med* 2005;33:315-22.
13. Greenhalgh DG, Warden GD. The importance of intra-abdominal pressure measurements in burned children. *J Trauma* 1994;36:685-90.
14. 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;44:1016-23.
15. Hong JJ, Cohn SM, Perez JM, et al. Prospective study of the incidence and outcome of intra-abdominal hypertension and the abdominal compartment syndrome. *Br J Surg* 2002;89:591-6.
16. Balogh Z, McKinley BA, Cocanour CS, et al. Supranormal trauma resuscitation causes more cases of abdominal compartment syndrome. *Arch Surg* 2003;138:637-43.
17. Cheatham ML, White MW, Sagraves SG, et al. Abdominal perfusion pressure: a superior parameter in the assessment of intra-abdominal hypertension. *J Trauma* 2000;49:621-6.
18. Biancofiore G, Bindi ML, Romanelli AM. Bet al. Intra-abdominal pressure monitoring in liver transplant recipients: a prospective study. *Intensive Care Med* 2003;29:30-6.
19. Sugrue M, Buist MD, Hourihan F, et al. Prospective study of intra-abdominal hypertension and renal function after laparotomy. *Br J Surg* 1995;82:235-8.
20. Sugrue M, Jones F, Deane SA, et al. Intra-abdominal hypertension is an independant cause of postoperative renal impairment. *Arch Surg* 1999;134:1082-5.

21. Sugrue M, Jones F, Lee A, et al. Intra-abdominal pressure and gastric intramucosal pH: Is there an association? *World J Surg* 1996;20:988-91.

22. Hobson KG, Young KM, Ciraulo A, et al. Release of abdominal compartment syndrome improves survival in patients with burn injury. *J Trauma* 2002;53:1129-34.

23. Meldrum DR, Moore FA, Moore EE, et al. Prospective characterization and selective management of the abdominal compartment syndrome. *Am J Surg* 1997;174:667-72.

24. Morris JA Jr, Eddy VA, Blinman TA, et al. The staged celiotomy for trauma. Issues in unpacking and reconstruction. *Ann Surg* 1993;217:576-84.

25. Raeburn CD, Moore EE, Biffl WL, et al. The abdominal compartment syndrome is a morbid complication of postinjury damage control surgery. *Am J Surg* 2001;182: 542-6.

26. 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;28:1747-53.

27. Offner PJ, de Souza AL, Moore EE, et al. Avoidance of abdominal compartment syndrome in damage-control laparotomy after trauma. *Arch Surg* 2001;136:676-81.

28. 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;54:848-61.

29. Maxwell RA, Fabian T, Croce M, et al. Secondary abdominal compartment syndrome: an underappreciated manifestation of severe hemorrhagic shock. *J Trauma* 1999;47:995-9.

30. Reintam A, Parm P, Kitus R, et al. Primary and secondary intra-abdominal hypertension-different impact on ICU outcome. *Intensive Care Med* 2008 May 1. [Epub ahead of print]

31. 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.

32. Balogh Z, McKinley BA, Cocanour CS, et al. Secondary abdominal compartment syndrome is an elusive early complication of traumatic shock resuscitation. *Am J Surg* 2002;184:538-44.

33. Ivy ME, Possenti PP, Kepros J, et al. Abdominal compartment syndrome in patients with burns. *J Burn Care Rehabil* 1999;20:351-3.

34. Malbrain MLNG, Chiumello D, Pelosi P, et al. Prevalence of intra-abdominal hypertension in critically ill patients: a multicentre epidemiological study. *Intensive Care Med* 2004;30:822-9.

35. Biffl WL, Moore EE, Burch JM, et al. Secondary abdominal compartment syndrome is a highly lethal event. *Am J Surg* 2001;182:645-8.

36. Leppäniemi A, Johansson K, De Waele JJ. Abdominal compartment syndrome and acute pancreatitis. *Acta Clin Belg Suppl* 2007;(1):S131-5.

37. Keskinen P, Leppäniemi A, Pettila V, et al. Intra-abdominal pressure in severe acute pancreatitis. *World J Surg* 2007;2:2.

38. Corcos AC, Sherman HF. Percutaneous treatment of secondary abdominal compartment syndrome. *J Trauma* 2001;51: 1062-4.

39. Reckard JM, Chung MH, Varma MK, et al. Management of intraabdominal hypertension by percutaneous catheter drainage. *J Vasc Interv Radiol* 2005;16: 1019-21.

40. Brown TLH, Muller MJ. Damage limitation in burn surgery. *Injury* 2004;35: 697-707.

41. Leppaniemi A, Kemppainen E. Recent advances in the surgical management of necrotizing pancreatitis. *Curr Opin Crit Care* 2005;11:349-52.

42. Watson RA, Howdieshell TR. Abdominal compartment syndrome. *South Med J* 1998;91:326-32.

43. Kirkpatrick AW, Balogh Z, Ball CG, et al. The secondary abdominal compartment syndrome: Iatrogenic or unavoidable? *J Am Coll Surg* 2006;202:668-79.

44. Pruitt BA. Protection from excessive resuscitation: "Pushing the pendulum back." *J Trauma* 2000;49:567-8.

45. Pruitt BA. The development of the International Society for Burn Injuries and progress in burn care: the whole is greater than the sum of its parts. *Burns* 1999;25: 683-96.

46. Pruitt BA Jr. Does hypertonic burn resuscitation make a difference? *Crit Care Med* 2000;28:277-8.

47. Saffle JI. The phenomenon of "fluid creep" in acute burn resuscitation. *J Burn Care Res* 2007;28:382-95.

48. Vincent JL, Sakr Y, Sprung CL, et al. Sepsis in European intensive care units: results of the SOAP study. *Crit Care Med* 2006;34: 344-53.

49. Cothren CC, Moore EE, Johnson JL, et al. Outcomes in surgical versus medical patients with the secondary abdominal compartment syndrome. *Am J Surg* 2007; 194:804-7.

50. Kopelman T, Harris C, Miller R, et al. Abdominal compartment syndrome in patients with isolated extraperitoneal injuries. *J Trauma* 2000;49:744-9.

51. Sanchez NC, Tenofsky PL, Dort JM, et al. What is normal intra-abdominal pressure? *Am Surg* 2001;67:243-8.

52. Sugerman HJ. Effects of intra-abdominal pressure in severe obesity. *Surg Clin North Am* 2001;81:1063-75.

53. *Illustrated Stedman's medical dictionary*. Baltimore: Williams & Wilkins, 1982.

54. Cullen DJ, Coyle JP, Teplick R, et al. Cardiovascular, pulmonary, and renal effects of massively increased intra-abdominal pressure in critically ill patients. *Crit Care Med* 1989;17:118-21.

55. Meldrum DR, Moore FA, Moore EE, et al. Cardiopulmonary hazards of perihepatic packing for major liver injuries. *Am J Surg* 1995;170:537-42.

56. Richardson JD, Trinkle JK. Hemodynamic and respiratory alterations with increased intra-abdominal pressure. *J Surg Res* 1976;20:401-4.

57. Malbrain MLNG. Is it wise not to think about intraabdominal hypertension in the ICU? *Curr Opin Crit Care* 2004;10:132-45.

58. Quintel M, Pelosi P, Caironi P, et al. An increase of abdominal pressure increases pulmonary edema in oleic acid-induced lung injury. *Am J Respir Crit Care Med* 2004;169:534-41.

59. Gattinoni L, Chiumello D, Carlesso E, et al. Bench-to-bedside review: chest wall elastance in acute lung injury/acute respiratory distress syndrome patients. *Crit Care* 2004;8:350-5.

60. Kirkpatrick AW, Meade MO, Mustard RA, et al. Strategies of invasive ventilatory support in ARDS. *Shock* 1996;6(Suppl 1): S17-22.

61. Kashtan J, Green JF, Parsons EQ, et al. Hemodynamic effects of increased abdominal pressure. *J Surg Res* 1981;30: 249-55.

62. Malbrain MLNG, Deeren D, De Potter TJR. Intra-abdominal hypertension in the critically ill: It is time to pay attention. *Curr Opin Crit Care* 2005;11:156-71.

63. Citerio G, Vascotto E, Villa F, et al. Induced abdominal compartment syndrome increases intracranial pressure in neurotrauma patients: a prospective study. *Crit Care Med* 2001;29:1466-71.

64. Bloomfield G, Saggi B, Blocher C, et al. Physiologic effects of externally applied continuous negative abdominal pressure for intra-abdominal hypertension. *J Trauma* 1999;46:1009-16.

65. Bloomfield GL, Dalton JM, Sugerman HJ, et al. Treatment of increasing intracranial pressure secondry to the acute abdominal compartment syndrome in a patient with combined abdominal and head trauma. *J Trauma* 1995;39:1168-70.

66. Miglietta MA, Salzano LJ, Chiu WC, et al. Decompressive laparotomy: a novel approach in the management of severe intracranial hypertension. *J Trauma* 2003; 55:551-5.

67. Caldwell CB, Ricotta JJ. Changes in visceral blood flow with elevated intraabdominal pressure. *J Surg Res* 1987;43: 14-20.

68. Diebel LN, Dulchavsky SA, Brown WJ. Splanchnic ischemia and bacterial translocation in the abdominal compartment syndrome. *J Trauma* 1997;43:852-5.

69. Diebel LN, Wilson RF, Dulchavsky SA, et al. Effect of increased intra-abdominal pressure on hepatic arterial, portal venous, and hepatic microcirculatory blood flow. *J Trauma* 1992;33:279-83.

70. Diebel LN, Dulchavsky SA, Wilson RF. Effect of increased intra-abdominal pressure on mesenteric arterial and intestinal mucosal blood flow. *J Trauma* 1992;33:45-9.

71. Friedlander MH, Simon RJ, Ivatury R, et al. Effect of hemorrhage on superior mesenteric artery flow during increased intraabdominal pressures. *J Trauma* 1998;45: 433-9.

72. Angus DC, Linde-Zwirble WT, Lidicker J, et al. Epidemiology of severe sepsis in the United States: analysis of incidence, outcomes, and associated costs of care. *Crit Care Med* 2001;29:1303-10.

73. Slade E, Tamber PS, Vincent JL. The surviving sepsis campaign: raising awareness to reduce mortality. *Crit Care* 2003;7:1-2.

74. Dombrovskiy VY, Martin AA, Sunderram J, et al. Rapid increase in hospitalization and mortality rates for severe sepsis in the United States: a trend analysis from 1993 to 2003. *Crit Care Med* 2007;35: 1244-50.

75. Dellinger RP, Levy MM, Carlet JM. Surviving sepsis campaign: international guidelines for management of severe sepsis and septic shock: 2008. *Intens Care Med* 2007, epub.

76. Dellinger RP, Carlet JM, Masur H, et al. Surviving sepsis campaign guidelines for management of severe sepsis and septic shock. *Crit Care Med* 2004;32:858-73.

77. McBeth PB, Zygun DA, Widder S, et al. Effect of patient positioning on intraabdominal pressure monitoring. *Am J Surg* 2007;193:644-7.

78. Reintam A, Parm P, Kitus R, et al. Intraabdominal hypertension as a risk factor of death in patients with severe sepsis or septic shock. *Crit Care Med* 2007;11 (Suppl 2):P319.

79. Sugrue M, Bauman A, Jones F, et al. Clinical examination is an inaccurate predictor of intraabdominal pressure. *World J Surg* 2002;26:1428-31.

80. Noblett KL, Jensen JK, Ostergard DR. The relationship of body mass index to intra-abdominal pressure as measured by multichannel cystometry. *Int Urogynecol J Pelvic Floor Dysfunct* 1997;8:323-6.

81. Kron IL. A simple technique to accurately determine intra-abdominal pressure. *Crit Care Med* 1989;17:714-5.

82. Iberti TJ, Lieber CE, Benjamin E. Determination of intra-abdominal pressure using a transuretheral bladder catheter: clinical validation of the technique. *Anesthesiology* 1989;70:47-50.

83. Cheatham ML, Safcsak K. Intra-abdominal pressure: A revised method for measurement. *J Am Coll Surg* 1998;186:594-5.

84. Pelosi P, Calzia E, Asfar P. It's time to measure intra-abdominal pressur eto optimize hemodynamics! *Intensive Care Med* 2007;33:6-8.

85. Duperret s, Lhuillier f, Piriou V, et al. Increased intra-abdominal pressure affects respiratory variations in arterial pressure in normovolemic and hypovolemic mechanically ventilated healthy pigs. *Intensive Care Med* 2007;33:163-71.

86. Mayberry JC, Goldman RK, Mullins RJ, et al. Surveyed opinion of American trauma surgeons on the prevention of the abdominal compartment syndrome. *J Trauma* 1999;47:509-14.

87. Kirkpatrick AW, Laupland KB, Karmali S, et al. Spill your guts! Perceptions of Trauma Association of Canada member surgeons regarding the open abdomen and the abdominal compartment syndrome. *J Trauma* 2006;60:279-86.

88. Oda J, Ueyama M, Yamashita K, et al. Hypertonic lactated saline resuscitation reduces the risk of abdominal compartment syndrome. *J Trauma* 2006;60:64-71.

89. O'Mara MS, Slater H, Goldfarb IW, et al. A prospective, randomized evaluation of intra-abdominal pressures with crystalloid and colloid resuscitation in burn patients. *J Trauma* 2005;58:1011-8.

90. Valenza F, Bottino N, Canavasi K, et al. Intra-abdominal pressure may be decreased non-invasively by continuous negative extra-abdominal pressure (NEXAP). *Intensive Care Med* 2003;29:2063-7.

91. 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;53:843-9.

92. 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;239:608-14.

93. Garner GB, Ware DN, Cocanour CS, et al. Vacuum-assisted wound closure provides early fascial reapproximation in trauma patients with open abdomens. *Am J Surg* 2001;182:630-8.

94. Suliburk JW, Ware DN, Balogh Z, et al. Vacuum-assisted wound closure achieves early fascial closure of open abdomens after severe trauma. *J Trauma* 2003;55: 1155-60.

95. Walsh GL, Chiasson P, Hedderich G, et al. The open abdomen: the marlex mesh and zipper technique: a method of managing intraperitoneal infection. *Surg Clin North Am* 1988;68:25-40.