Current Approach to the Evaluation and Management of Abdominal Compartment Syndrome in Pediatric Patients

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Abstract: Abdominal compartment syndrome is an emergent condition caused by increased pressure within the abdominal compartment. It can be caused by a number of etiologies, which are associated with decreased abdominal wall compliance, increased intraluminal or intraperitoneal contents, or edema from capillary leak or fluid resuscitation. The history and physical examination are of limited utility, and the criterion standard for diagnosis is intra-abdominal pressure measurement, which is typically performed via an intravesical catheter. Management includes increasing abdominal wall compliance, evacuating gastrointestinal or intraperitoneal contents, avoiding excessive fluid resuscitation, and decompressive laparotomy in select cases.

Key Words: abdominal compartment syndrome, critical care, increased abdominal pressure, intra-abdominal hypertension

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TARGET AUDIENCE

This CME activity is intended for all practitioners who care for pediatric patients presenting with possible abdominal compartment syndrome, which may include general pediatricians, pediatric emergency physicians, general emergency physicians, pediatric intensive care physicians, pediatric surgeons, and general surgeons.

LEARNING OBJECTIVES

After completion of this article, the reader should be better able to:

1. Discuss the risk factors for pediatric abdominal compartment syndrome.
2. Describe diagnostic testing that can be utilized to identify this syndrome.
3. Explain the management strategies for pediatric abdominal compartment syndrome.

Abdominal compartment syndrome (ACS) is a surgical emergency that requires prompt recognition and early intervention to avoid serious complications.[1] The effects of increased intra-abdominal pressure (IAP) were first described in 1863, but it received relatively little attention until the 1980s.[2–5] It was at this time that researchers and clinicians began to learn how increased IAP could lead to multisystem organ dysfunction including respiratory failure and renal impairment.[6,7] This discovery fueled research to better understand the pathophysiology behind ACS and its precursor, intra-abdominal hypertension (IAH), so that superior therapeutic interventions could be developed. The term ACS was officially coined by Fietsam et al[8] in 1989.

Abdominal compartment syndrome can be challenging to diagnose. However, it is critical to recognize the symptoms early because delays in diagnosis are associated with increased morbidity and mortality.[9,10] Unfortunately, it may be particularly difficult to diagnose ACS in the pediatric population owing to lack of provider familiarity with the diagnosis as well as the inherent challenges of eliciting a reliable history and physical examination in critically ill pediatric patients. A recent survey suggested that pediatricians are not as familiar with ACS compared with providers who care for adults.[11] In addition, IAPs are not routinely measured in the pediatric population.[12–14]

A recent multicenter study demonstrated that 44% of newly admitted, critically ill pediatric patients had IAH.[15] Other studies have found that the incidence of ACS specifically in pediatric intensive care units was 1%,[16,17] but that it increased to 18.7% among ventilated children.[18] Overall mortality rates owing to ACS range from 32% to 68%,[5,19,20] and mortality rates specifically in pediatric patients range from 40% to 64%.[16,18,21,22] Although mortality due to ACS in adults has decreased significantly, mortality due to ACS in children has remained relatively stable.[23–25] Part of this may be attributed to poor recognition of ACS in the pediatric population resulting in delays in diagnosis and management. Therefore, it is essential that providers are well versed in the evaluation and management of ACS.

ANATOMY AND PATHOPHYSIOLOGY

The World Society of the Abdominal Compartment Syndrome defines IAH as an IAP greater or equal to 12 mm Hg, whereas ACS is defined as an IAP greater than 20 mm Hg leading to new organ dysfunction or failure.[26] However, children have a lower baseline IAP than adults, so ACS may occur when IAP reaches 10 mm Hg.[27,28]

Although traditionally thought of as a postsurgical complication, ACS occurs in both medical and surgical patients, including those with nonabdominal surgeries.[29] Etiologies can be subdivided into 4 major categories: diminished abdominal wall compliance, increased intraluminal contents, increased intraperitoneal contents, and capillary leak leading to extravascular fluid accumulation.[27] Examples of diminished abdominal wall compliance includes gastroschisis, omphalocele, constricting dressings, burn eschars, obesity, and tight closure after abdominal surgery.[30] Increased intraluminal contents may result from constipation, ileus, and Hirschsprung disease.[5] Increased intraperitoneal contents may be caused by hepatomegaly, splenomegaly, intra-abdominal tumors, ascites, and massive hemorrhage.[5] Capillary leak and fluid replacement may occur in the setting of sepsis, severe pancreatitis, and extensive burns.[31,32]

Abdominal compartment syndrome is often classified into primary, secondary, or recurrent ACS based upon the underlying cause and duration. Primary ACS occurs in the presence of abdominal pathology and can include causes such as infectious enterocolitis, abdominal surgery, bowel perforation, and bowel

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obstruction.[3,4,18] Secondary ACS occurs when the elevated IAP results in end organ dysfunction in the absence of abdominal injury and can occur in the setting of trauma, sepsis, shock states, and aggressive fluid hydration.[3,4,18] Finally, recurrent ACS occurs when a patient with a prior episode of ACS redevelops ACS due to either a primary or secondary etiology.[3,4]

HISTORY AND PHYSICAL EXAMINATION

Children presenting with ACS are usually critically ill and may be limited in their ability to communicate and provide a history.[22] Early symptoms of ACS may include abdominal pain, abdominal distension, or increased work of breathing.[33] Oliguria is one of the first findings in ACS and is caused by renal venous congestion as a result of renal vein compression.[34] However, symptoms are often generalized and nonspecific.[35]

The physical examination has also been shown to be unreliable and a poor predictor of increased IAP.[36,37] Patients with ACS may present with tachypnea, hypoventilation, abdominal distension, abdominal tenderness, tachycardia, hypotension, sepsis, or signs of peritonitis suggesting intestinal perforation or ischemia.[9,25] Abdominal assessment of girth has been shown to be an unreliable index of IAP.[36] One study found that attending intensivists using clinical examination to diagnose elevated IAP in postoperative patients had a sensitivity of 60.9%, specificity of 80.5%, and positive predictive value of only 45.2%.[37] In another study, clinical estimation of increased IAP had a sensitivity of 56%, a specificity of 87%, and a positive predictive value of 35%.[38] Because of the aforementioned limitations, more invasive testing is generally required to confirm the diagnosis in these patients.

DIAGNOSTIC TESTING

Laboratory testing may demonstrate evidence of severe organ dysfunction (eg, elevated lactate, creatinine, or liver function testing).[3,4] However, there are currently no laboratory studies that are sufficiently sensitive or specific to facilitate the diagnosis.

Imaging studies may be helpful in suggesting the diagnosis or elucidating underlying causes but cannot effectively rule out the diagnosis. On chest radiograph, the presence of unilateral diaphragmatic elevation, pleural effusions, or lobar collapse may suggest the diagnosis of ACS.[3,4,39] Computed tomography findings suggestive of ACS include massive abdominal distension, bowel wall thickening, compression of the inferior vena cava, bilateral inguinal herniation, direct renal compression, or tense infiltration of the retroperitoneal space.[39–43] In addition, computed tomography may identify potential etiologies of elevated IAP such as ascites, hematomas, or large intra-abdominal masses that can suggest the diagnosis, as well as guide interventions.[44]

Intra-abdominal pressure can be measured in several ways. Measuring it directly using intraperitoneal catheters or pressure transducer needles placed directly into the peritoneal space is highly accurate; however, the roles for this are limited owing to its invasiveness and the potential risk of traumatic bowel injury.[45] Consequently, IAP is usually measured indirectly using pressure transducers that can be passed into the stomach (nasogastric), bladder (intravesical), or colon (intracolonic). Intravesical pressure monitoring via Foley catheter is by far the most commonly used method of measuring IAP in adults and children.[27,46]

To measure intravesical pressure, patients are first placed in the supine position. A Foley catheter is inserted, and the bladder is drained. A pressure device is then attached to the Foley catheter, and a pressure transducer is connected to this system. While the pressure transducer is placed at the patient's mix-axillary line, isotonic saline is instilled into the bladder through a 3-way stopcock and the catheter is then clamped. For children weighing between 2.7 and 25 kg, 1 mL/kg of isotonic saline (up to a maximum of 25 mL) is recommended for instillation volume.[47] In children more than 25 kg, 25 mL of isotonic saline is the recommended instillation volume.[20] No appropriate volume has been determined for infants weighing under 2.7 kg.[32] The transducer is zeroed, and the pressure readings on the monitor are recorded.[48] When assessing IAP, it is recommended to wait 30 seconds after instilling saline to allow the detrusor muscles to relax.[27,49] In addition, the measurement should be obtained at end exhalation to reduce false positives.[27,49] Once the measurement has been obtained, the provider should unclamp the Foley to fully decompress the bladder.

Intra-abdominal hypertension is defined as a prolonged IAP greater than or equal to 10 mm Hg, whereas ACS is defined as an IAP greater than 20 mm Hg with associated end-organ dysfunction.[26,35] Although these definitions were initially designated for adult patients, newer literature suggests that the IAP threshold may be lower in pediatric patients.[27] Intra-abdominal hypertension is graded from I to IV, with increased morbidity and mortality associated with higher grades (Table 1).[35]

Abdominal perfusion pressure is defined as mean arterial pressure minus the IAP. Abdominal perfusion pressure has been studied as a resuscitative end point in ACS and was found to be superior to mean arterial pressure and intravesicular pressure in predicting patient survival from IAH and ACS.[50] Abdominal perfusion pressure has shown clinical superiority when compared with other resuscitative end points such as base deficit, arterial pH, hourly urinary output, and arterial lactate.[50]

MANAGEMENT

Once ACS is identified, the key is to reduce the IAP and improve organ function. Providers should aim to lower the IAP to below 10 mm Hg and keep the abdominal perfusion pressure to 35 mm Hg or greater in infants or greater than 50 mm Hg in children.[29] The management of ACS has 5 components: evacuate intraluminal contents, remove intra-abdominal space-occupying fluid or masses, improve abdominal wall compliance, optimize fluid administration and perfusion, and consider decompressive laparotomy.[9,20,27,29,51,52]

Evacuate Intraluminal Contents

Ileus is a common finding in critically ill patients. When present, nasogastric drainage can be a simple first step to decrease IAP in these patients with consideration of a rectal cannula or enema for distal decompression.[27] Prokinetic agents like metoclopramide, erythromycin, or neostigmine should also be considered.[27] For opioid-induced constipation, methylnaltrexone can exert prokinetic effects.[27] If the patient does not respond to pharmacologic approaches, endoscopy can be performed to facilitate decompression of the colon in select cases.[4,9,53]

Remove Intra-abdominal Space-Occupying Fluid or Masses

Paracentesis should be considered in patients with ACS who have significant ascites.[4,5,27,53] However, placement of a percutaneous drainage catheter may be preferable because it allows

TABLE 1. Grading of IAH in Children (Ejike 2011)[35]

IAH Grade	IAP
Grade I	10–12 mm Hg
Grade II	13–15 mm Hg
Grade III	16–19 mm Hg
Grade IV	≥20 mm Hg

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ongoing drainage of the intraperitoneal fluid and may help avoid the need for decompressive laparotomy in selected patients with secondary ACS.[3,54–56] One study of pediatric patients with ACS who had massive ascites found that percutaneous drainage of the abdominal fluid significantly lowered the IAP and improved survival.[57]

Improve Abdominal Wall Compliance

Providers should first remove restrictive abdominal bandages or binders.[27] Next, it is important to ensure adequate pain control and sedation.[3,27,58,59] Among adult patients, administration of analgesics through an epidural catheter was found to reduce the IAP when compared with intravenous agents.[60,61] However, further studies are needed to determine the benefit of this in pediatric patients.

Neuromuscular blockade has also been demonstrated to decrease the IAP in patients with IAH.[62–64] Therefore, a trial of neuromuscular blocking agents should be considered when other measures are ineffective.[4] However, routine or prolonged use of neuromuscular blockers should be avoided owing to their potential adverse effects (eg, atelectasis, pneumonia, prolonged weaning).[27,62–64] In general, these agents should primarily be used as a bridging technique while awaiting decompressive laparotomy.[27,60]

Optimize Fluid Administration and Perfusion

Excessive fluid resuscitation is an independent predictor of IAH and should be avoided.[3,25,65–67] In patients who are hypotensive or septic with ACS, early vasopressor use should be considered.[27,66,68,69] Diuretics are unlikely to be effective in these patients owing to third spacing of the fluid combined with renal dysfunction from the ACS.[70] Renal replacement therapy using continuous hemofiltration or ultrafiltration may be considered in select cases with significant fluid overload.[3,66,71]

Decompressive Laparotomy

Decompressive laparotomy is recommended if the above measures are unsuccessful.[5,18,20,26,27,53,72–74] The timing of the decompression is important, as one study of patients with pancreatitis-induced ACS showed significantly worse outcomes when decompression was performed more than 4 days after admission.[10] Importantly, when ACS is relieved with laparotomy, there is often a sudden improvement in both the patient's respiratory and cardiovascular function.[75] However, a reperfusion effect similar to that seen with limb compartment syndrome can occur.[75] This causes a large release of intracellular contents into the circulation that has the potential to cause cardiac arrhythmias and metabolic acidosis.[75,76] It has been reported that the risk of asystolic cardiac arrest can be up to 25% at the time of decompression.[77]

COMPLICATIONS

Abdominal compartment syndrome can result in damage to a number of different organ systems. Intestinal ischemia and necrosis due to decreased perfusion can lead to bacterial translocation across the gut mucosa and into the systemic circulation, as well as the release of inflammatory mediators that can worsen a patient's hemodynamic status.[3,5,28,78–80] In addition, abdominal wall muscle and fascial ischemia may contribute to infectious and noninfectious wound complications (eg, dehiscence, herniation, necrotizing fasciitis) often seen in this patient population.[81]

The upward displacement of the diaphragm with IAH can result in compressive pulmonary atelectasis and ventilation-perfusion mismatch.[5] Transmission of this elevated pressure from the abdomen to the thoracic cavity through the diaphragm can lead to increased intrathoracic pressure, reduced functional residual capacity, smaller tidal volumes, and increased work of breathing.[5] These can subsequently lead to hypoxia, hypercapnia, and eventually respiratory failure.[49] The increased IAP may also worsen preload by compressing the inferior vena cava, as well as afterload by compressing the aorta.[81] The inferior vena cava compression can lead to venous stasis in the lower extremities, increasing the risk of deep venous thrombosis or even limb compartment syndrome.[82] Moreover, the decrease in cardiac output can lead to splanchnic and renal hypoperfusion, further worsening the aforementioned conditions.[5,83,84]

Increased IAP also reduces venous return from the kidneys by direct compression, leading to an increase in renal vascular resistance, reduced renal blood flow, and decreased glomerular filtration rate.[70] This can lead to reduced ability to clear toxins, as well as fluid retention which can further increase the IAP.[85] Finally, IAH causes an increase in intracranial pressure owing to reduced central venous return caused by the increased intrathoracic pressure.[81,86–88]

Patients surviving ACS may also suffer long-term sequelae including depression, chronic organ failure, hernias, and prolonged need for rehabilitation.[49,89]

DISPOSITION

Patients with ACS are generally critically ill and require admission to the intensive care unit. However, it can be easy to overlook this disorder in these patients. Therefore, providers should assess for the risk of ACS in critically ill patients, particularly if patients will be in the Emergency Department for an extended period of time.

CONCLUSIONS

Abdominal compartment syndrome is an emergent condition caused by increased pressure within the abdominal compartment. This article reviews the pathophysiology, historical and physical examination findings, diagnostic strategies, and treatment for this dangerous condition. Knowledge of this can assist providers in effectively identifying and managing this condition.

REFERENCES

1. Saggi BH, Sugerman HJ, Ivatury RR, et al. Abdominal compartment syndrome. *J Trauma*. 1998;45:597–609.
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. Papavramidis TS, Marinis AD, Pliakos I, et al. Abdominal compartment syndrome — intra-abdominal hypertension: defining, diagnosing, and managing. *J Emerg Trauma Shock*. 2011;4:279–291.
4. Sosa G, Gandham N, Landeras V, et al. Abdominal compartment syndrome. *Dis Mon*. 2019;65:5–19.
5. Cheatham ML. Abdominal compartment syndrome: pathophysiology and definitions. *Scand J Trauma Resusc Emerg Med*. 2009;17:10.
6. Wesley JR, Drongowski R, Coran AG. Intragastric pressure measurement: a guide for reduction and closure of the silastic chimney in omphalocele and gastroschisis. *J Pediatr Surg*. 1981;16:264–270.
7. Hershenson MB, Brouillette RT, Klemka L, et al. Respiratory insufficiency in newborns with abdominal wall defects. *J Pediatr Surg*. 1985;20: 348–353.
8. Fietsam R Jr., 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. Hecker A, Hecker B, Hecker M, et al. Acute abdominal compartment syndrome: current diagnostic and therapeutic options. *Langenbecks Arch Surg*. 2016;401:15–24.

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10. Mentula P, Hienonen P, Kemppainen E, et al. Surgical decompression for abdominal compartment syndrome in severe acute pancreatitis. *Arch Surg*. 2010;145:764–769.
11. Ejike JC, Newcombe J, Baerg J, et al. Understanding of abdominal compartment syndrome among pediatric healthcare providers. *Crit Care Res Pract*. 2010;2010:876013.
12. Schierz IA, Giuffrè M, Piro E, et al. Predictive factors of abdominal compartment syndrome in neonatal age. *Am J Perinatol*. 2014;31:49–54.
13. Kaussen T, Steinau G, Srinivasan PK, et al. Recognition and management of abdominal compartment syndrome among German pediatric intensivists: results of a national survey. *Ann Intensive Care*. 2012;2(suppl 1):S8.
14. Newcombe J, Mathur M, Bahjri K, et al. Pediatric critical care nurses' experience with abdominal compartment syndrome. *Ann Intensive Care*. 2012;2(suppl 1):S6.
15. Horoz OO, Yildizdas D, Asilioglu N, et al. The prevalance of and factors associated with intra-abdominal hypertension on admission day in critically ill pediatric patients: A multicenter study. *J Crit Care*. 2015;30:584–588.
16. Beck R, Halberthal M, Zonis Z, et al. Abdominal compartment syndrome in children. *Pediatr Crit Care Med*. 2001;2:51–56.
17. Diaz FJ, Fernandez Sein A, Gotay F. Identification and management of abdominal compartment syndrome in the pediatric intensive care unit. *P R Health Sci J*. 2006;25:17–22.
18. Ejike JC, Humbert S, Bahjri K, et al. Outcomes of children with abdominal compartment syndrome. *Acta Clin Belg*. 2007;62(suppl 1):141–148.
19. Shaheen AW, Crandall ML, Nicolson NG, et al. Abdominal compartment syndrome in trauma patients: new insights for predicting outcomes. *J Emerg Trauma Shock*. 2016;9:53–57.
20. 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.
21. Pearson EG, Rollins MD, Vogler SA, et al. Decompressive laparotomy for abdominal compartment syndrome in children: before it is too late. *J Pediatr Surg*. 2010;45:1324–1329.
22. Thomas S, Kriplani D, Crane C, et al. Outcomes in pediatric patients with abdominal compartment syndrome. *J Pediatr Surg*. 2017;52: 1144–1147.
23. Vidal MG, Ruiz Weisser J, Gonzalez F, et al. Incidence and clinical effects of intra-abdominal hypertension in critically ill patients. *Crit Care Med*. 2008;36:1823–1831.
24. De Waele J, Desender L, De Laet I, et al. Abdominal decompression for abdominal compartment syndrome in critically ill patients: a retrospective study. *Acta Clin Belg*. 2010;65:399–403.
25. Carr JA. Abdominal compartment syndrome: a decade of progress. *J Am Coll Surg*. 2013;216:135–146.
26. 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.
27. Kirkpatrick AW, Roberts DJ, De Waele J, et al. Intra-abdominal hypertension and the abdominal compartment syndrome: updated consensus definitions and clinical practice guidelines from the World Society of the Abdominal Compartment Syndrome. *Intensive Care Med*. 2013;39:1190–1206.
28. Sukhotnik I, Riskin A, Bader D, et al. Possible importance of increased intra-abdominal pressure for the development of necrotizing enterocolitis. *Eur J Pediatr Surg*. 2009;19:307–310.
29. De Waele J, Ejike JC, Leppaniemi A, et al. Intra-abdominal hypertension and abdominal compartment syndrome in pancreatitis, paediatrics, and trauma. *Anaesthesiol Intensive Ther*. 2015;47:219–227.
30. DeCou JM, Abrams RS, Miller RS, et al. Abdominal compartment syndrome in children: experience with three cases. *J Pediatr Surg*. 2000;35:840–842.
31. Newcombe J, Mathur M, Ejike JC. Abdominal compartment syndrome in children. *Crit Care Nurse*. 2012;32:51–61.
32. Ejike JC, Mathur M. Abdominal decompression in children. *Crit Care Res Pract*. 2012;2012:180797.
33. Sugrue M, Jones F, Deane SA, et al. Intraabdominal hypertension is an independent cause of postoperative renal impairment. *Arch Surg*. 1999; 134:1082–1085.
34. Doty JM, Saggi BH, Blocher CR, et al. Effects of increased renal parenchymal pressure on renal function. *J Trauma*. 2000;48:874–877.
35. Ejike JC, Mathur M, Moores DC. Abdominal compartment syndrome: focus on the children. *Am Surg*. 2011;77(suppl 1):S72–S77.
36. Fairclough JA, Mintowt-Czyz J, Mackie I, et al. Abdominal girth; an unreliable measure of intra-abdominal bleeding. *Injury*. 1984;16:85–87.
37. Sugrue M, Bauman A, Jones F, et al. Clinical examination is an inaccurate predictor or intraabdominal pressure. *World J Surg*. 2002;26:1428–1431.
38. 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;43:207–211.
39. Je BK, Kim HK, Horn PS. Abdominal compartment syndrome in children: clinical and imaging features. *AJR Am J Roentgenol*. 2019;212:655–664.
40. Patel A, Lall CG, Jennings SG, et al. Abdominal compartment syndrome. *AJR Am J Roentgenol*. 2007;189:1037–1043.
41. Pickhardt PJ, Shimony JS, Heiken JP, et al. The abdominal compartment syndrome: CT findings. *AJR Am J Roentgenol*. 1999;173:575.
42. Sugrue G, Malbrain MLNG, Pereira B, et al. Modern imaging techniques in intra-abdominal hypertension and abdominal compartment syndrome: a bench to bedside overview. *Anaesthesiol Intensive Ther*. 2018;50:234–242.
43. Bouveresse S, Piton G, Badet N, et al. Abdominal compartment syndrome and intra-abdominal hypertension in critically ill patients: diagnostic value of computed tomography. *Eur Radiol*. 2019;29:3839–3846.
44. De Laet IE, Malbrain M. Current insights in intra-abdominal hypertension and abdominal compartment syndrome. *Med Intensiva*. 2007;31:88–99.
45. Suominen PK, Pakarinen MP, Rautiainen P, et al. Comparison of direct and intravesical measurement of intraabdominal pressure in children. *J Pediatr Surg*. 2006;41:1381–1385.
46. Zhou JC, Zhao HC, Pan KH, et al. Current recognition and management of intra-abdominal hypertension and abdominal compartment syndrome among tertiary Chinese intensive care physicians. *J Zhejiang Univ Sci B*. 2011;12:156–162.
47. Ejike JC, Bahjri K, Mathur M. What is the normal intra-abdominal pressure in critically ill children and how should we measure it? *Crit Care Med*. 2008;36:2157–2162.
48. Fusco MA, Martin SR, Chang MC. Estimation of intra-abdominal pressure by bladder pressure measurement: validity and methodology. *J Trauma*. 2001;50:297–302.
49. Cheatham ML, Safcsak K, Sugrue M. Long-term implications of intra-abdominal hypertension and abdominal compartment syndrome: physical, mental, and financial. *Am Surg*. 2011;77(suppl 1):S78–S82.
50. Cheatham ML, White MW, Sagraves SG, et al. Abdominal perfusion pressure: a superior parameter in the assessment of intra-abdominal hypertension. *J Trauma*. 2000;49:621–626.
51. De Waele J, De LI, Kirkpatrick A, et al. Intra-abdominal hypertension and abdominal compartment syndrome. *Am J Kidney Dis*. 2011;57:159–169.
52. Cheatham ML, Safcsak K. Is the evolving management of intra-abdominal hypertension and abdominal compartment syndrome improving survival? *Crit Care Med*. 2010;38:402–407.
53. Ivatury RR. Abdominal compartment syndrome: a century later, isn't it time to accept and promulgate? *Crit Care Med*. 2006;34:2494–2495.

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54. Corcos AC, Sherman HF. Percutaneous treatment of secondary abdominal compartment syndrome. *J Trauma*. 2001;51:1062–1064.
55. Savino JA, Cerabona T, Agarwal N, et al. Manipulation of ascitic fluid pressure in cirrhotics to optimize hemodynamic and renal function. *Ann Surg*. 1988;208:504–511.
56. Sugrue M, Buhkari Y. Intra-abdominal pressure and abdominal compartment syndrome in acute general surgery. *World J Surg*. 2009;33: 1123–1127.
57. Liang YJ, Huang HM, Yang HL, et al. Controlled peritoneal drainage improves survival in children with abdominal compartment syndrome. *Ital J Pediatr*. 2015;41:29.
58. Malbrain ML, Chiumello D, Pelosi P, et al. Prevalence of intra-abdominal hypertension in critically ill patients: a multi- center epidemiological study. *Intensive Care Med*. 2004;30:822–829.
59. 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;33:315–322.
60. De Keulenaer B, Regli A, De Laet IE, et al. What's new in medical management strategies for raised intra-abdominal pressure: evacuating intra-abdominal contents, improving abdominal wall compliance, pharmacotherapy, and continuous negative extra-abdominal pressure. *Anaesthesiol Intensive Ther*. 2015;47:54–62.
61. Hakobyan RV, Mkhoyan GG. Epidural analgesia decreases intraabdominal pressure in postoperative patients with primary intra-abdominal hypertension. *Acta Clin Belg*. 2008;63:86–92.
62. Deeren D, Dits H, Malbrain M. Correlation between intra-abdominal and intracranial pressure in nontraumatic brain injury. *Intensive Care Med*. 2005;31:1577–1581.
63. 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.
64. De Laet I, Hoste E, Verholen E, et al. The effect of neuromuscular blockers in patients with intra-abdominal hypertension. *Intensive Care Med*. 2007;33:1811–1814.
65. Daugherty EL, Liang H, Taichman D, et al. Abdominal compartment syndrome is common in medical intensive care unit patients receiving large-volume resuscitation. *J Intensive Care Med*. 2007;22:294–299.
66. Kula R, Szturz P, Sklienka P, et al. A role for negative fluid balance in septic patients with abdominal compartment syndrome. *Intensive Care Med*. 2004;30:2138–2139.
67. Kula R, Szturz P, Sklienka P, et al. Negative fluid balance in patients with abdominal compartment syndrome—case reports. *Acta Chir Belg*. 2008;108:346–349.
68. Hunt L, Frost SA, Hillman K, et al. Management of intra-abdominal hypertension and abdominal compartment syndrome: a review. *J Trauma Manag Outcomes*. 2014;8:2.
69. Regli A, De Keulenaer B, De Laet I, et al. Fluid therapy and perfusional considerations during resuscitation in critically ill patients with intra-abdominal hypertension. *Anaesthesiol Intensive Ther*. 2015;47: 45–53.
70. Harman PK, Kron IL, Mclachlan HD, et al. Elevated intra-abdominal pressure and renal function. *Ann Surg*. 1982;196:594–597.
71. Oda S, Hirasawa H, Shiga H, et al. Management of intraabdominal hypertension in patients with severe acute pancreatitis with continuous hemodiafiltration using a polymethyl methacrylatemembrane hemofilter. *Ther Apher Dial*. 2005;9:355–961.
72. De Waele J, Hoste EA, Malbrain ML. Decompressive laparotomy for abdominal compartment syndrome–a critical analysis. *Crit Care*. 2006; 10:R51.
73. De Laet I, Ravyts M, Vidts W, et al. Current insights in intra-abdominal hypertension and abdominal compartment syndrome: open the abdomen and keep it open! *Langenbecks Arch Surg*. 2008;393:833–847.
74. Van Damme L, De Waele JJ. Effect of decompressive laparotomy on organ function in patients with abdominal compartment syndrome: a systematic review and meta-analysis. *Crit Care*. 2018;22:179.
75. Moore AF, Hargest R, Martin M, et al. Intra-abdominal hypertension and the abdominal compartment syndrome. *Br J Surg*. 2004;91:1102–1110.
76. Gottlieb M, Adams S, Landas T. Current approach to the evaluation and management of acute compartment syndrome in pediatric patients. *Pediatr Emerg Care*. 2019;35:432–437.
77. Basu NN, Cottam S. Abdominal compartment syndrome. *Surgery (Oxford)*. 2006;24:260–262.
78. Rezende-Neto JB, Moore EE, Melo de Andrade MV, et al. Systemic inflammatory response secondary to abdominal compartment syndrome: stage for multiple organ failure. *J Trauma*. 2002;53:1121–1128.
79. Sun Z, Wang X, Lasson A, et al. Roles of platelet-activating factor, interleukin-1beta and interleukin-6 in intestinal barrier dysfunction induced by mesenteric arterial ischemia and reperfusion. *J Surg Res*. 1999;87:90–100.
80. Sun Z, Lasson A, Olanders K, et al. Gut barrier permeability, reticuloendothelial system function and protease inhibitor levels following intestinal ischaemia and reperfusion-effects of pretreatment with N-acetyl-L-cysteine and indomethacin. *Dig Liver Dis*. 2002;34:560–569.
81. Malbrain ML, De Laet IE, De Waele JJ, et al. Intra-abdominal hypertension: definitions, monitoring, interpretation and management. *Best Pract Res Clin Anaesthesiol*. 2013;27:249–270.
82. Thabet FC, Ejike JC. Intra-abdominal hypertension and abdominal compartment syndrome in pediatrics. A review. *J Crit Care*. 2017;41: 275–282.
83. Maluso P, Olson J, Sarani B. Abdominal compartment hypertension and abdominal compartment syndrome. *Crit Care Clin*. 2016;32:213–222.
84. Malbrain ML, De Waele JJ, De Keulenaer BL. What every ICU clinician needs to know about the cardiovascular effects caused by abdominal hypertension. *Anaesthesiol Intensive Ther*. 2015;47:388–399.
85. De Waele JJ, De Laet I. Intra-abdominal hypertension and the effect on renal function. *Acta Clin Belg*. 2007;62(suppl 2):371–374.
86. De Laet I, Citerio G, Malbrain ML. The influence of intraabdominal hypertension on the central nervous system: current insights and clinical recommendations, is it all in the head? *Acta Clin Belg*. 2007;62(suppl 1): 89–97.
87. Youssef AM, Hamidian Jahromi A, Vijay CG, et al. Intra-abdominal hypertension causes reversible blood-brain barrier disruption. *J Trauma Acute Care Surg*. 2012;72:183–188.
88. Bloomfield GL, Ridings PC, Blocher CR, et al. Effects of increased intra-abdominal pressure upon intracranial and cerebral perfusion pressure before and after volume expansion. *J Trauma*. 1996;40:936–941.
89. Sugrue M. Abdominal compartment syndrome. *Curr Opin Crit Care*. 2005;11:333–338.

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