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Current Evidence on Diagnosis and Management of Abdominal Compartment Syndrome

Tsalis K[1*] and Vasiliadis K[2]

[1]*Fourth Surgical Department, Aristotle University, Thessaloniki, Greece*

[2]*First Department of General Surgery, Papageorgiou Hospital, Nea Efkarpia 564 03, Thessaloniki, Greece*

[*]**Corresponding author:** Konstantinos George Tsails, Fourth Surgical Department, Aristotle University, Thessaloniki, Greece, Tel: 30-231-099-600; E-mail: ctsalis@yahoo.gr

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Abstract

The sustained intra-abdominal hypertension, leading to critical tissue hypoperfusion and permanent functional organ impairment, constitutes the life-threatening disorder of abdominal compartment syndrome (ACS). In recent years, the increased awareness of the presence, clinical importance and devastating impact of ACS has led to dramatic improvements in the diagnosis, definition and management of this lethal disorder. Because of the high incidence of ACS in high-risk patients, regular assessment of intra-abdominal pressure should be performed and timely, evidence-based therapeutic interventions should be employed not only to minimize the risk of developing intra-abdominal hypertension but also to aggressively treat the fully developed ACS. Such an approach will improve patient safety, optimize survival, and decrease morbidity. Among evidence-based measures for the management of ACS surgical decompression remains the method of choice succeeding fast and definitive treatment of the fully developed ACS.

Keywords: Abdominal compartment syndrome; Intra-abdominal pressure; Necrosectomy; Mortality

Introduction

Although intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS) are relatively young pathological entities, there has been recently an exponential growth in scientific research relating to their recognition, definition, diagnosis, prevention and treatment [1]. These developments, which are reflecting the increased awareness of the deleterious effects of IAH and ACS, led to an emphatic decrease in the mortality rate of ACS from 60% to approximately 30% during the last decade [2]. Intra-abdominal hypertension and ACS occur frequently in both medical and surgical Intensive Care Units (ICU), but they may even occur in the general ward, and the Emergency Department. Among evidence-based measures for the management of ACS surgical decompression remains the method of choice succeeding fast and definitive treatment of the fully developed ACS.

This review provides a concise approach to the diagnosis and management of IAH and ACS, with a particular emphasis on the role of surgical decompression.

Epidemiology and etiology

The incidence of IAH in critical care patients is reported to be 50%, and of these patients with IAH, 4.2% will develop ACS within their first day of hospitalization [3,4]. Several factors have the potential to increase intra-abdominal pressure (IAP) predisposing to the development of IAH and ensuing ACS. These factors can be related to the abdominal wall compliance, the total volume of intraluminal abdominal contents and the increased capillary permeability and leakage of plasma proteins into the interstitial fluid. The latter pathogenetic mechanism represents a frequent unanticipated adverse effect of aggressive fluid resuscitation therapy. In fact, the implementation of recent resuscitation protocols such as Early Goal Directed Therapy and Damage Control Resuscitation in patients with critical illnesses, can lead to the development of the undesired life-threatening consequences of IAH and ACS [5]. Other important risk factors for the development of IAH are core hypothermia, coagulopathy necessitating component therapy, severe sepsis and septic shock, liver failure associated with extant ascites, and mechanical ventilation [6,7] (Table 1).

Considering the detrimental effects of IAH and ACS, the critical care personnel need to be familiar with the current definitions related to these disorders and to be aware of their predisposing factors, signs and symptoms in order to succeed an early diagnosis that will provide the opportunity for a timely and successful management.

Aggressive crystalloid fluid resuscitation (>5,000 mL in 24 h)
Upwards of 10 units of packet red blood cells transfusion within 24 h
Core temperature <330 C
Arterial pH <7.2
Body mass index >30

Table 1: Independent contributing factors for the development ABC

Pathophysiology

Intra-abdominal hypertension and ACS can lead to multisystem dysfunction. Elevated IAP is directly transmitted to the abdominal vasculature leading on the one hand to venous occlusion and a subsequent reduction in preload and decreased cardiac output and on

the other; to arterial compression inducing a reduced arterial compliance and an increased afterload [8].

The respiratory system is also largely affected due to pressure-induced diaphragmatic splinting and elevation, producing a functional constraint of pulmonary expansion. These factors can lead to decreased pulmonary and chest wall compliance, basal collapse and atelectasis, increasing Ventilation-Perfusion (V/Q) mismatch, hypoxemia, and distorted pulmonary flow characteristics [9].

Renal dysfunction in the context of IAH and ACS is multifactorial. The IAH and the subsequent decrease in cardiac output can lead to a significant decrease in renal blood flow. Additionally, renal vein compression, combined with increased venous impedance resulting from IVC compression, can cause decreased glomerular filtration, up regulation of antidiuretic hormone, and activation of the rennin-angiotensin axis stimulating fluid retention. Acute tubular necrosis can also occur [10,11].

The gastrointestinal system is particularly vulnerable to elevated IAP in critically ill patients. The decreased gut perfusion as part of the response to critical illness compounded by the reduction in abdominal perfusion pressure can lead to the development of bowel wall edema. The latter induces a significant decrease in bowel wall oxygen delivery eventually leading to bowel ischemia and translocation of bacteria into the systemic circulation. Hepatic perfusion is also adversely affected, leading to liver dysfunction and failure [12,13].

Furthermore, the central nervous system, similarly suffer hypoperfusion. Elevated intra-abdominal and intra-thoracic pressures inhibit venous return with a consequent increase in intra-cranial pressure. Associated hypercarbia, and any consequent cerebral vasodilatation, may further raise intracranial pressure [14].

Current definitions

In 2013, the World Society of the Abdominal Compartment Syndrome (WSACS) updated the definitions and management statements relating to IAH and ACS via a new international consensus conference. The current consensus statement defines ACS as the sustained intra-abdominal pressure (IAP) >20 mmHg that is associated with new onset of organ dysfunction or failure, regardless of abdominal perfusion pressure (APP). Of note is that the WSACS suggests that, if the patient exhibits signs of new organ dysfunction or failure, this development is more clinically significant than an absolute metric value. Abdominal compartment syndrome is a separate and distinct entity from IAH, which is defined as a sustained or repeated IAP ≥ than 12 mmHg [15]. Normal or mean pressure within the non-diseased abdominal cavity ranges between 2 mmHg and 5 mmHg, while the normal IAP for critically ill adults who are usually fluid overloaded ranges between 5 mmHg and 7 mmHg [16]. Intra-abdominal pressure (IP) depends on overall body mass index and therefore, it can be elevated as high as 12 mm Hg in the obese adult. The absence of organ injury constitutes the determinant difference between obesity related chronic IAH and ACS [17].

The current WSACS consensus conference has categorized IAH into grades, based upon worsening abdominal pressures [16] (Table 2). Abdominal compartment syndrome is further classified into primary, secondary and recurrent. Primary ACS results from injury or disease of the abdomen or pelvis which frequently necessitates immediate therapeutic intervention or, from postoperative sequela requiring surgical intervention [18,19]. Primary ACS can also evolve in patients with organ damage who were initially managed non-surgically and then developed ACS [18,20]. Secondary ACS refers to disorders not originating from the abdomen or pelvis and occurs in the absence of abdominal injury [15]. Secondary ACS may develop in patients suffering from sepsis, pancreatitis or may be the result of excessive fluid resuscitation. Recurrent or "tertiary" ACS, as it was formerly known, is the reoccurrence of either primary or secondary ACS after successful medical or surgical treatment [16].

Abdominal perfusion pressure (APP), which is defined as the difference between the mean arterial pressure and the IAP, indicates the pressure available for the perfusion of abdominal organs [15,20]. Calculation of APP assesses accurately the adequacy of abdominal blood flow and therefore it has previously been considered as a reliable indirect indicator of IAH/ACS severity. In fact, APP has been evaluated as a resuscitation end point in several clinical trials which demonstrated statistically significant differences in APP between survivors and non-survivors with IAH/ACS [16,21]. Additionally, APP proved superior to global resuscitation end points such as arterial pH, base deficit, arterial lactate and urinary output in its ability to predict patient outcome. Despite these scientifically sound data, the WSACS 2013 consensus management statement could make no recommendations for the use of APP in the resuscitation or management of patients with IAH/ACS.

Grade	IAP, mm Hg	Analysis
Normal	<12	
1	12-15	Normal IAP for the obese
2	16-20	The development of organ dysfunction is unlikely
3	21-25	In this IAP range ACS is considered present only if organ(s) dysfunction coexists
4	>25	When organ(s) dysfunction coexists the terminology changes to ACS

Table 2: Current IAP grading

Diagnosis

Early diagnosis of IAH before it progresses to ACS depends on thorough knowledge of its pathophysiology in addition to a high index of clinical suspicion. Usually, the patient who is at risk for the development of IAH and ACS would be sedated, intubated on mechanical ventilation in the intensive care unit setting. The abdomen is usually very tense and distended and the extremities edematous. Furthermore, the face and neck, penis and genitalia may be swollen. Often, the ICU personnel are alerted to the possibility of IAH because of the high airway pressures in addition to signs of an impending renal failure.

In rare occasions, such as large space-occupying intra-abdominal lesions or ruptured abdominal aortic aneurysms; IAH can occur in alert-awake patients [22,23]. In these patients prominent symptoms on physical examination are painful and distended abdomen associated with orthopnea, shortness of breath and limited or even absence of diaphragmatic excursion.

Unfortunately, the clinical features of IAH are non-specific. Indeed, the sensitivity of the physical examination to detect IAH is very low and therefore, it is not considered a reliable diagnostic method. On the contrary, measuring bladder pressure is the gold standard method and

should be performed according to the current WSACS recommended protocol.

Indications for IAP Monitoring

As underlined previously, the sensitivity of physical examination in detecting IAH is significantly low, ranging between 40% and 60% [24] therefore; the diagnosis of IAH/ACS relies on accurate measurement of IAP. In fact, IAP monitoring represents a safe, inexpensive and accurate mean not only for the diagnosis of IAH but also for the proper guiding of resuscitative therapy [25]. However, there remains considerable debate over the applicability of absolute intra-abdominal pressure (IAP) ranges, in the management of patients with critical illnesses. Several studies support that an IAP>20 mmHg induces serious physiological consequences in critically ill patients [3,26,27]. Despite these data, it is well known that this absolute IAP level, does not always associated with physiological derangements [28,29]. Therefore, the current WSACS consensus conference, developed definitive evidence-based algorithms for the diagnosis and management of IAH and ACS [15].

A crucial issue in the management of IAH and ACS is early identification of patients at risk of developing this disorder [30-32]. Therefore, the risk factors of IAH and ACS should be assessed on admission and for the total duration of the critical illness according to the recognized independent risk factors for the development of these derangements (Table 1). Patients with open or blunt abdominal trauma and patients with a high body mass index, mesenteric ischemia, elevated intracranial pressure, those who sustained burns and hypotensive patients are also at risk of developing IAH [19]. It is of utmost importance that all critically ill patients should be screened for the presence of IAH or ACS upon admission or in the presence of new or progressive organ failure. Furthermore, if there are two or more risk factors present or if there is a new or progressive organ failure, then a baseline IAP measurement should be repeated and the currently recommended WSACS assessment algorithm should be implemented. In the presence of IAH, a 4 to 6 hours intervals or continuously IAP monitoring is recommended [15].

Methods of intra-abdominal pressure (IAP) measurement

A variety of methods of measuring IAP have been reported, such as gastric pressure via a nasogastric tube, inferior vena cava pressure, rectal pressure, direct IAP via direct puncture, or use of bedside ultrasound to assess the caliber and respiratory variation of the inferior vena cava [33-36]. Measurement accuracy and reproducibility, financial constraints and personnel training influence all these methods. Additionally, there is minimal standardization of the IAP assessment methods across various centers [19].

Trying to bring order to this scientific "polyphony" the WSACS has recommended the use of a standardized protocol for IAP measurement advocating the use of the modified intermittent Kron technique as the gold standard of IAP measurement [15,16]. This method assesses the IAP via bladder pressure measurement using a maximum instillation of 25 ml of sterile saline [15,16]. The measurement was performed with the transducer zeroed and positioned in line with the iliac crest and mid-axillar line, with the patient in a supine position at end-expiration and with an instillation volume of no greater than 25 ml of saline. However, the reliability of the intermittent measurement guidelines, recommended by WSACS has been recently challenged [37]. Specifically, it has been argued that the current technology of continuous IAP monitoring is superior to the intermittent technique because it provides continuous analysis of the IAP level via the bladder, which eliminates the risk of missing alterations in IAP over time, which is unavoidable when using the intermittent technique [38,39]. Despite the obvious advantages of continuous IAP monitoring, a recent study showed comparable results between the two methods [40]. Furthermore, it should be underlined that the continuous IAP measurement technique requires the use of more expensive medical equipment, which is limiting its wider use. Regardless of the current controversy regarding the most reliable method of measuring IAP, the modified intermittent Kron technique remains the gold standard.

Management

One of the most crucial issues that must be addressed when managing critically ill patients at risk for IAH and ACS is to determine on an individual basis, whether the presence of increased IAP aggravates or not their already severe clinical condition. The treating personnel should always keep in mind that it remains unsettled how much conservative treatment a critically ill patient with IAH can bear before the lethal cascade of ACS becomes irreversible and a point of no return is reached and most importantly, that the definitive management of a fully developed ACS can be accomplished only after laparotomy and temporary abdominal closure, aiming at reversing the deleterious consequences of the syndrome [41]. Apart from these strategic principles, prevention is the best treatment of ACS.

Non-operative management

The non-operative management of IAH can be divided into the following steps: sedation and paralysis to relax the abdominal wall, evacuation of intraluminal contents, drainage of large abdominal fluid collections, optimization of APP, and correct a positive fluid balance [42] (Table 3).

Sedation

Adequate sedation and sometimes paralysis should be ensured. Complete paralysis will relax the muscles of the abdominal wall allowing additional expansion of the abdominal domain and lowering of the IAP [4].

Sedation and paralysis
Evacuation of hollow viscera contents
Optimizing abdominal perfusion pressure
Drainage of large intraperitoneal fluid collections
Correction of positive fluid balance

Table 3: Parameters of non-surgical management of IAH

Evacuation of intraluminal contents

Hollow viscera distension can increase IAP substantially; therefore a simple endoluminal decompression is an effective way to decrease the IAH [43].

Drainage of large abdominal fluid collections

Patients with ascites or abdominal trauma patients may have large intraperitoneal fluid collections contributing to elevated IAP. Percutaneous drainage has shown great success in burn and oncology patients however, recurrent ACS is always possible as the fluid often re-accumulates. Therefore, a continued surveillance is mandatory. On the contrary, in the trauma patient with solid organ injury the blood will shortly after the insult loculate, making its percutaneous drainage practically impossible [44].

Optimizing abdominal perfusion pressure

Several investigators implement a more rational, well-documented method, which is based on the assessment of APP that serves as a reliable indicator of IAH/ACS severity, to clearly define the requisiteness of abdominal decompression in patients with IAH, but indefinite signs of ACS [16,23]. Despite the rationale of this approach, the 2013 WSACS consensus management statement could make no recommendations for the use of APP in the resuscitation or management of patients with IAH/ACS.

Correction a positive fluid balance

In cases of overly aggressive resuscitation in multi-trauma patients an iatrogenic ACS may develop. In such situations the pathogenic mechanism that leads to ACS might more likely be the aggressive fluid resuscitation rather than the traumatic event itself [45,46]. In such condition, the fluid excess has to be removed without however creating hypotension, hypoperfusion, or acidosis. Although gentle diuresis is an appealing perspective, excreting the third-spaced fluid excess without causing intravascular volume depletion is rather almost impossible therefore, in these cases early institution of renal replacement therapy is the most appropriate therapy [47,48].

Surgical decompression

This is unarguably the most effective and immediate way to reduce elevated IAP. Surgical decompression should be considered in the unfortunate event of failure of non-operative measures to relieve IAH. However, the indication, timing and type of decompression should be carefully balanced because of the substantial morbidity associated with all types of surgical decompression [49]. Unfortunately, there is no uniform consensus on the indications for the surgical management of ACS. As an axiom, when medical or minimally invasive measures fail to intercept the progressive decline of organ function or in the presence of a fully developed ACS, surgical management is absolutely justified. Clinical experience indicates that early compared with delayed decompression several days after the onset of the syndrome, is more effective and is associated with lower mortality [49]. Notwithstanding, there are no sound evidence existing on the proper timing of surgical management.

It should be also taken into consideration that all these therapeutic principles can be valid when ACS develops early in the course of the critical illness, as usually happens. However, if the syndrome is caused as a result of a later event such as the development of infected pancreatic necrosis following severe acute pancreatitis, then delayed surgical decompression combined with necrosectomy is a rather justifiable treatment planning. Finally, the concept of prophylactic surgical decompression in high-risk patients, namely preventing the development of ACS by leaving the abdominal cavity open, is a rather reasonable approach, which however does not supported by sound scientific evidence.

Decompressive laparotomy aims to decrease the elevated IAP and reverse organ dysfunction providing vital space for continued expansion of the abdominal viscera during ongoing resuscitation. Additionally, the technique of decompressive laparotomy should obviate excessive fascial retraction and ensure temporary abdominal coverage allowing the evacuation of fluid from the abdominal cavity. Unfortunately, there is no current surgical technique in use, which totally fulfills all these prerequisites. However, in recent years various effective surgical techniques have been developed, sharing the same basic surgical principles consisting in (a) performance of a generous fasciotomy incurring evisceration, (b) separation of the underlining hollow viscera from the abdominal wall with the use of a hermetic sealing barrier, (c) obviation of excessive fascial retraction and (d) ensuring an indiscriminate evacuation of peritoneal fluid with the use of drainage catheters or vacuum sponge materials (Table 4).

Among these principles, complete fasciotomy is of the utmost importance because the degree to which the IAP decreases is directly proportional to the degree to which the fascia is released. However, the reinstatement of organ function following surgical decompression is not immediate and can persist despite the adequacy of surgical technique, leading ultimately to death, in case of irreversibility of organ dysfunction and progression of the lethal cascade of ACS [50]. Evenly important is the principal of safe separation of the underlining hollow viscera from the abdominal wall. If this is not the case, then the small bowel will adhere to the abdominal wall within a period of approximately three days, which will predispose to inadvertent small bowel injury and possible subsequent enterocutaneous fistula development, during the future abdominal wall closure. Therefore, avoiding the adherence of bowel wall to the fascia and implementing surgical techniques to prevent retracting of the fascia far laterally, is extremely important to ensure a safe delayed abdominal wall closure. The recently employed option of suturing mesh to the edges of the divided abdominal wall, using vacuum sponges or silicone elastomer materials, provide additional effectiveness [51,52].

Finally, as last but not least step of the ideal decompressive laparotomy technique is to ensure indiscriminate evacuation of peritoneal fluid. The rationality of this measure is based on the fact that, following decompression the peritoneal fluid will continue to accumulate in the peritoneal cavity and inevitably will trap under the hermetic sealing barrier predisposing to the development of a recurrent ACS.

Targets
Decrease IAP and reverse organ dysfunction
Provide vital space for continued expansion of the abdominal viscera
Creation of an iatrogenic evisceration
Ensuring an indiscriminate evacuation of peritoneal fluid
Surgical methodology
Performance of complete fasciotomy
Separation of the underlining hollow viscera from the abdominal wall with the use of a hermetic sealing barrier
Obviation of excessive fascial retraction

Suturing a prosthetic material to the fascia to avert fascial retraction
Ensuring indiscriminate evacuation of peritoneal fluid with the use of drainage catheters or vacuum sponge materials

Table 4: Crucial therapeutic targets and methodology of decompressive laparotomy

Conclusions

Abdominal compartment syndrome is a devastating, life-threatening disorder. Although non-surgical interventions have a role in the stable and minimally symptomatic patient, however this mode of treatment should be carefully balanced. This is because the limit of how much conservative treatment a patient with IAH can bear before the lethal cascade of ACS becomes irreversible and a point of no return is reached has not yet been settled. Most importantly, it should be remembered that the definitive management of a fully developed ACS could be accomplished only after laparotomy and temporary abdominal closure, which reverses the deleterious consequences of the syndrome in a significant proportion of patients. The present review aims to serve as a modest contributor in recognizing and timely treating IAH and ACS, which will lead to a further reduction of the mortality rate associated with these disorders.

References

1. Barnes GE, Laine GA, Giam PY, Smith EE, Granger HJ (1985) Cardiovascular responses to elevation of intra-abdominal hydrostatic pressure. Am J Physiol 248: R208-213.
2. De Waele J, Desender L, De Laet I, Ceelen W, Pattyn P, et al. (2010) Abdominal decompression for abdominal compartment syndrome in critically ill patients: a retrospective study. Acta Clin Belg 65: 399-403.
3. Kim IB, Prowle J, Baldwin I, Bellomo R (2012) Incidence, risk factors and outcome associations of intra-abdominal hypertension in critically ill patients. Anaesth Intensive Care 40: 79-89.
4. Malbrain ML, Chiumello D, Pelosi P, Bihari D, Innes R, et al. (2005) Incidence and prognosis of intraabdominal hypertension in a mixed population of critically ill patients: a multiple-center epidemiological study. Crit Care Med 33: 315-322.
5. Marik PE1, Desai H (2012) Goal directed fluid therapy. Curr Pharm Des 18: 6215-6224.
6. Malbrain ML, Chiumello D, Pelosi P, Wilmer A, Brienza N, et al. (2004) Prevalence of intra-abdominal hypertension in critically ill patients: a multicentre epidemiological study. Intensive Care Med 30: 822-829.
7. Ivatury RR, Porter JM, Simon RJ, Islam S, John R, et al. (1998) Intra-abdominal hypertension after life-threatening penetrating abdominal trauma: prophylaxis, incidence, and clinical relevance to gastric mucosal pH and abdominal compartment syndrome. J Trauma 44: 1016-1021.
8. Ridings PC, Bloomfield GL, Blocher CR, Sugerman HJ (1995) Cardiopulmonary effects of raised intra-abdominal pressure before and after intravascular volume expansion. J Trauma 39: 1071-1075.
9. Runck H, Schumann S, Tacke S, Haberstroh J, Guttmann J (2012) Effects of intra-abdominal pressure on respiratory system mechanics in mechanically ventilated rats. Respir Physiol Neurobiol 180: 204-210.
10. Harman PK, Kron IL, McLachlan HD, Freedlender AE, Nolan SP (1982) Elevated intra-abdominal pressure and renal function. Ann Surg 196: 594-597.
11. Kirsch AJ, Hensle TW, Chang DT, Kayton ML, Olsson CA, et al. (1994) Renal effects of CO2 insufflation: oliguria and acute renal dysfunction in a rat pneumoperitoneum model. Urology 43: 453-459.
12. Diebel LN, Wilson RF, Dulchavsky SA, Saxe J (1992) Effect of increased intra-abdominal pressure on hepatic arterial, portal venous, and hepatic microcirculatory blood flow. J Trauma 33: 279-282.
13. Diebel LN, Dulchavsky SA, Wilson RF (1992) Effect of increased intra-abdominal pressure on mesenteric arterial and intestinal mucosal blood flow. J Trauma 33: 45-48.
14. Marinis A, Argyra E, Lykoudis P, Brestas P, Theodoraki K, et al. (2010) Ischemia as a possible effect of increased intra-abdominal pressure on central nervous system cytokines, lactate and perfusion pressures. Crit Care 14: 31.
15. Kirkpatrick A, Roberts D, De Waele J, Jaeschke R, Malbrain ML, et al. (2013) Intra-abdominal hypertension and the abdominal compartment syndrome: updated consensus definitions and clinical practice guidelines from the world society of abdominal compartment syndrome. Intensive Care Med 39: 1190-1206.
16. Malbrain ML, Cheatham ML, Kirkpatrick A, Sugrue M, Parr M, et al. (2006) Results from the International Conference of Experts on Intra-abdominal Hypertension and Abdominal Compartment Syndrome. I. Definitions. Intensive Care Med 32: 1722-1732.
17. Lambert DM, Marceau S, Forse RA (2005) Intra-abdominal pressure in the morbidly obese. Obes Surg 15: 1225-1232.
18. Cheatham ML (2009) Abdominal compartment syndrome. Curr Opin Crit Care 15: 154-162.
19. Malbrain ML, Cheatham ML, Kirkpatrick A, Sugrue M, De Waele J, et al. (2006) Abdominal compartment syndrome: it's time to pay attention! Intensive Care Med 32: 1912-1914.
20. An G1, West MA (2008) Abdominal compartment syndrome: a concise clinical review. Crit Care Med 36: 1304-1310.
21. Cheatham ML, White MW, Sagraves SG, Johnson JL, Block EF (2000) Abdominal perfusion pressure: a superior parameter in the assessment of intra-abdominal hypertension. J Trauma 49: 621-626.
22. Merlicco D, Roggia G, Lombardi M, Lattanzio L, Marzaioli R, et al. (2012) Abdominal compartment syndrome due to a giant multilobulated ovarian serous cystadenoma. Case report and review of the literature. Ann Ital Chir 83: 563-566.
23. Björck M (2012) Management of the tense abdomen or difficult abdominal closure after operation for ruptured abdominal aortic aneurysms. Semin Vasc Surg 25: 35-38.
24. Kirkpatrick AW, Brenneman FD, McLean RF, Rapanos T, Boulanger BR (2000) Is clinical examination an accurate indicator of raised intra-abdominal pressure in critically injured patients? Can J Surg 43: 207-211.
25. Malbrain MLNG, Jones F. (2006) Intra-abdominal pressure measurement techniques. In: Ivatury RR, Cheatham ML, Malbrain MLNG, Sugrue M, editors. Abdominal Compartment Syndrome. Landes Biomedical, Georgetown.
26. Murcia-Sáez IM, Sobrino-Hernandez ML, García-Lopez F, Córcoles-González V, Cortés-Monedero JL, et al. (2010) Usefulness of intra-abdominal pressure in a predominantly medical intensive care unit. J Crit Care 25: 175.
27. Cheatham ML (2008) Intraabdominal pressure monitoring during fluid resuscitation. Curr Opin Crit Care 14: 327-333.
28. Meldrum DR, Moore FA, Moore EE, Franciose RJ, Sauaia A, et al. (1997) Prospective characterization and selective management of the abdominal compartment syndrome. Am J Surg 174: 667-672.
29. Rotondo MF, Schwab CW, McGonigal MD, Phillips GR 3rd, Fruchterman TM, et al. (1993) 'Damage control': an approach for improved survival in exsanguinating penetrating abdominal injury. J Trauma 35: 375-382.
30. Lui F, Sangosanya A, Kaplan LJ (2007) Abdominal compartment syndrome: clinical aspects and monitoring. Crit Care Clin 23: 415-433.
31. Kaussen T, Otto J, Steinau G, Höer J, Srinivasan PK, et al. (2012) Recognition and management of abdominal compartment syndrome among German anesthetists and surgeons: a national survey. Ann Intensive Care 2 Suppl 1: S7.
32. Hanci V, Kiraz HA, Ã–mÃ¼r D, Ã–zkan Ã–F (2013) Intra-abdominal pressure measurement applications of the intensive care physicians: a survey. Journal of the Turkish Sociaty of Intensive Care 11: 11-7.

33. Sugrue M, Buist MD, Lee A, Sanchez DJ, Hillman KM (1994) Intra-abdominal pressure measurement using a modified nasogastric tube: description and validation of a new technique. Intensive Care Med 20: 588-590.
34. Balogh Z, De Waele JJ, Malbrain ML (2007) Continuous intra-abdominal pressure monitoring. Acta Clin Belg Suppl : 26-32.
35. De laet I, Malbrain ML, Jadoul JL, Rogiers P, Sugrue M (2007) Renal implications of increased intraabdominal pressure: Are the kidneys the canary for abdominal hypertension? Acta Clin Belg Suppl 1: 119-130.
36. Iberti TJ, Kelly KM, Gentili DR, Hirsch S, Benjamin E (1987) A simple technique to accurately determine intra-abdominal pressure. Crit Care Med 15: 1140-1142.
37. Shuster MH, Haines T, Sekula LK, Kern J, Vazquez JA (2010) Reliability of intrabladder pressure measurement in intensive care. Am J Crit Care 19: e29-39.
38. Balogh Z, Jones F, D'Amours S, Parr M, Sugrue M (2004) Continuous intra-abdominal pressure measurement technique. Am J Surg 188: 679-684.
39. De laet I, Hoste E, De Waele JJ (2008) Transvesical intra-abdominal pressure measurement using minimal instillation volumes: how low can we go? Intensive Care Med 34: 746-750.
40. Van Waes O, Jaquet J, Hop W, Morak M, Ijzermans JK (2009) Single-lumen central venous catheter for continuous and direct intra-abdominal pressure measurement. Eur J Trauma Emerg Surg 35: 1-6.
41. Carr JA (2013) Abdominal compartment syndrome: a decade of progress. J Am Coll Surg 216: 135-146.
42. De Keulenaer BL, De Waele JJ, Malbrain ML (2011) Nonoperative management of intra-abdominal hypertension and abdominal compartment syndrome: evolving concepts. Am Surg 77 Suppl 1: S34-41.
43. van der Spoel JI, Oudemans-van Straaten HM, Stoutenbeek CP, Bosman RJ, Zandstra DF (2001) Neostigmine resolves critical illness-related colonic ileus in intensive care patients with multiple organ failure--a prospective, double-blind, placebo-controlled trial. Intensive Care Med 27: 822-827.
44. Kozar RA, Moore FA, Cothren CC, Moore EE, Sena M, et al. (2006) Risk factors for hepatic morbidity following nonoperative management: multicenter study. Arch Surg 141: 451-458.
45. Madigan MC, Kemp CD, Johnson JC, Cotton BA (2008) Secondary abdominal compartment syndrome after severe extremity injury: are early, aggressive fluid resuscitation strategies to blame? J Trauma 64: 280-285.
46. Rodas EB, Malhotra AK, Chhitwal R, Aboutanos MB, Duane TM, et al. (2005) Hyperacute abdominal compartment syndrome: an unrecognized complication of massive intraoperative resuscitation for extra-abdominal injuries. Am Surg 71: 977-981.
47. Kula R, Szturz P, Sklienka P, Neiser J, Jahoda J (2004) A role for negative fluid balance in septic patients with abdominal compartment syndrome? Intensive Care Med 30: 2138-2139.
48. Oda S, Hirasawa H, Shiga H, Matsuda K, Nakamura M, et al. (2005) Management of intra-abdominal hypertension in patients with severe acute pancreatitis with continuous hemodiafiltration using a polymethyl methacrylate membrane hemofilter. Ther Apher Dial 9: 355-361.
49. De Waele JJ, Hoste EA, Malbrain ML (2006) Decompressive laparotomy for abdominal compartment syndrome--a critical analysis. Crit Care 10: R51.
50. Hershberger RC, Hunt JL, Arnoldo BD, Purdue GF (2007) Abdominal compartment syndrome in the severely burned patient. J Burn Care Res 28: 708-714.
51. Tsalis K, Vasiliadis K, Bitzani M, Blouchos K, Botsios D, et al. (2007) Management of abdominal compartment syndrome: A monoinstitutional experience. 3rd World Congress Abdominal Compartment Syndrome, Acta Clinica Belgica 62: 290.
52. Foy HM, Nathens AB, Maser B, Mathur S, Jurkovich GJ (2003) Reinforced silicone elastomer sheeting, an improved method of temporary abdominal closure in damage control laparotomy. Am J Surg 185: 498-501.