Langenbeck's Arch Surg (2001) 386:53–61
DOI 10.1007/s004230000185

ORIGINAL ARTICLE

K.-M. Sieh
Kent-Man Chu
John Wong

Intra-abdominal hypertension and abdominal compartment syndrome

Received: 26 May 2000
Received in revised form: 6 September 2000
Accepted: 4 October 2000
Published online: 12 January 2001

K.-M. Sieh · K.-M. Chu (✉) · J. Wong
Department of Surgery,
University of Hong Kong Medical Centre,
Queen Mary Hospital, Hong Kong, China
e-mail: chukm@hkucc.hku.hk
Tel.: +852-2855-4637
Fax: +852-2819-4221

Abstract *Background:* The effects of increased intra-abdominal pressure in various organ systems have been noted over the past century. The concept of abdominal compartment syndrome has gained more attention in both trauma and general surgery in the last decade. This article reviews the current understanding and management of intra-abdominal hypertension and abdominal compartment syndrome. *Methods:* Relevant information was gathered from a Medline search of the English literature, previous review and original articles, references cited in papers, and by checking the latest issues of appropriate journals. *Results and conclusion:* Akin to compartment syndrome in extremities, the pathophysiological effects of increased intra-abdominal pressure developed well before any clinical evidence of compartment syndrome. These effects include cardiovascular, pulmonary, renal and intracranial derangement, reduction of intestinal and hepatic blood flow, and reduction of abdominal wall compliance. Although abdominal compartment syndrome is more commonly noted in patients with abdominal trauma, it is now evident that non-trauma surgical patients could also develop the condition. Early initiation of treatment for intra-abdominal hypertension is currently advocated in view of the possibility of subclinical progress to the full-blown abdominal compartment syndrome.

Keywords Intra-abdominal hypertension · Abdominal compartment syndrome

Introduction

Consequences and physiological effects of increased intra-abdominal pressure (IAP), or intra-abdominal hypertension (IAH), have been described since the late 19th century in both animal and human models [1, 2]. In 1890, Heinricius showed that IAH (27–46 cm H_2O) was fatal in feline and porcine models [1]. Such deaths were initially attributed to respiratory dysfunction. Subsequently, cardiovascular and renal dysfunction were found to be associated with IAH in the 1910s [1, 3, 4]. In 1951, Baggot [5] reported the high mortality associated with forced closure of the abdomen with distended bowel, addressing the fatal effect of severe IAH. However, the recognition of the abdomen as a compartment and the concept of IAH resulting in abdominal compartment syndrome (ACS) has only recently received attention. Kron and associates [6] first used the term ACS in the 1980s, and it is now broadly defined as organ dysfunction attributable to increased IAP [2].

Patients with ACS typically present with a tense abdomen with distension, increased peak respiratory pressure, intractable hypercapnia, and oliguria [7, 8]. It has also been found recently that ACS increased the risk of disruption of small-bowel anastomoses [9]. IAH and ACS are, therefore, taking on more importance not just to trauma surgeons but to all practicing surgeons.

Table 1 Factors predisposing to increased intra-abdominal pressure [8, 12, 13,14,15, 16]

Acute	Spontaneous abdominal events	Acute pancreatitis, hemoperitoneum, ileus, intestinal obstruction, intra-abdominal sepsis, peritonitis, mesenteric venous thrombosis, bowel ischemia, pneumoperitoneum, retroperitoneal hematoma, ruptured abdominal aortic aneurysm
	Abdominal trauma	Hemoperitoneum, post-resuscitation visceral edema, retroperitoneal hematoma
	Abdominal operation	Aortic surgery, laparoscopy under pneumoperitoneum, liver transplantation, packing, reduction of a massive hernia or gastroschisis
	Post-operative	Acute gastric dilatation, hemoperitoneum, ileus, intra-abdominal collection, peritonitis
	Others	Burn, massive fluid resuscitation, pneumatic antishock garment
Chronic		Ascites, central obesity, chronic ambulatory peritoneal dialysis, massive abdominal tumor, pregnancy

Etiology

The normal IAP is zero (that is, atmospheric) or less [4, 10, 11]. There are many situations associated with clinically significant elevation of IAP (Table 1) [8, 12, 13, 14, 15, 16]. A chronic increase in IAP, as in pregnant ladies, results in gradual stretching of the abdominal wall, which becomes more compliant with time. The acute deterioration associated with ACS is, therefore, rarely seen in chronic elevation of IAP. Any insult that results in an acute rise in the volume of the abdominal contents may lead to IAH and ACS. Such insult may originate spontaneously from intra-abdominal events, such as ruptured abdominal aortic aneurysm.

A typical example occurs in a victim with abdominal trauma requiring laparotomy for hemostasis. A large volume of fluid is usually given for resuscitation, and this may result in visceral, retroperitoneal and abdominal wall edema. During closure, the laparotomy wound edges are brought together under tension over the bulging abdominal contents. Post-operatively, mechanical ventilation is often necessary, which may further aggravate IAH [17, 18]. The pathophysiological effects of IAH will be discussed later.

Abdominal insult, however, is not an absolute association of the ACS, which has been described in patients sustaining trauma remote from the abdomen [19]. In the literature, ACS has often been described after abdominal trauma; but one should keep in mind that ACS can occur in a variety of surgical settings, including major, life-threatening hemorrhage and shock, massive volume resuscitation, prolonged operation with coagulopathy, and hypothermia [7, 12]. All these factors are often additive, resulting in or aggravating multiple system organ failure [12].

Table 2 Pathophysiological effects of intra-abdominal hypertension

System	Parameter	Effect
Cardiovascular	Cardiac output	↓
	Venous return	↓
	Peripheral vascular resistance	↑
	Intrathoracic pressure	↑
	Heart rate	↑
	Mean blood pressure	–
Pulmonary	Pulmonary compliance	↓
	Peak inspiratory pressure	↑
	Pulmonary vascular resistance	↑
	Total lung capacity	↓
	Functional residual capacity	↓
	Residual volume	↓
Renal	Renal vascular resistance	↑
	Renal arterial flow	↓
	Glomerular filtration rate	↓
Abdominal viscera	Splanchnic blood flow	↓
Abdominal wall	Abdominal wall blood flow	↓
	Abdominal wall compliance	↓
Intracranial	Intracranial pressure	↑
	Cerebral perfusion pressure	↓

Pathophysiology

The fundamental problem in ACS is the acute expansion of the intra-abdominal volume in excess of the capacity of the abdominal cavity [2]. IAH causes changes in almost all organ systems (Table 2), and these changes occur in a graded fashion according to the level of IAP [12, 20]. For the sake of clarity, these changes are categorized under different organ systems. In an individual patient, such changes occur simultaneously and may affect each other.

Cardiovascular derangement

Increased IAP leads to a reduction in cardiac output (CO) [21, 22, 23, 24, 25]. Although this effect may be seen with IAP as low as 10–15 mmHg, it is most consistently seen at an IAP greater than 20 mmHg [8, 12, 25]. The decrease in CO is related to diminished venous return, increased peripheral resistance, or increased intrathoracic pressure [7, 17, 25]. Venous return is reduced by a number of mechanisms [8, 12, 25, 26]. Increased IAP leads to reduction in caval and retroperitoneal venous flow [27]. Venous flow is also reduced by functional narrowing of the inferior vena cava at the suprahepatic, subdiaphragmatic level, where the high pressure zone of the abdomen meets the lower pressure zone of the thorax [28, 29]. Elevation of peripheral vascular resistance is likely to be related to mechanical compression of capillary beds [26]. IAH increases intrathoracic pressure by elevating the diaphragm. As a result, ventricular filling pressure increases while ventricular compliance decreases. All these factors (diminished venous return, increased peripheral resistance, and increased intrathoracic pressure) lead to a reduced stroke volume with compensatory increase in heart rate. The blood pressure usually remains unchanged [25].

Other compounding factors may influence the hemodynamic effects of IAH. Such factors include, for example, volume status of the patient, the use of inhalational anesthetics, and application of mechanical ventilation [18, 27, 30].

Pulmonary derangement

A common presentation of ACS is respiratory failure, which is characterized by high ventilatory pressure, hypoxia, and hypercapnia [2, 12, 24, 31]. Elevation of the diaphragm causes reduction in static and dynamic pulmonary compliance and increase in peak inspiratory pressure as well as pulmonary vascular resistance [25, 32, 33, 34]. Increase in IAP also reduces total lung capacity, functional residual capacity, and residual volume [35]. All these factors lead to ventilation–perfusion abnormalities with resulting hypoxia and hypercapnia [24, 33]. Mechanical ventilation with high positive end-expiratory pressure (PEEP) is required to maintain adequate oxygenation, but this may lead to further physiological abnormalities. Abdominal decompression improves the respiratory abnormalities [8, 24, 36].

Renal derangement

An IAP of 15–20 mmHg may produce oliguria, while IAP of 30 mmHg or higher may lead to anuria [24, 37, 38, 39]. The renal effects of IAH are related to increased renal vascular resistance and decreased renal arterial flow as well as glomerular filtration rate (GFR) [3]. These, in turn, are due to reduced cardiac output, direct compression of the kidney, and obstruction of renal venous outflow. Compression of abdominal aorta and renal arteries contribute to the increase in renal vascular resistance [37]. The increased release of antidiuretic hormone, renin, and aldosterone with IAH also contributes to the renal effects [40]. Ureteric compression is not a contributory factor for renal impairment since ureteric stenting did not prevent oliguria [37]. Renal failure could be reversed by timely abdominal decompression [38]. The importance of IAH in critically ill patients as an independent cause of postoperative renal impairment has been highlighted recently [41].

Abdominal visceral effect

Caldwell and Ricotta demonstrated a reduction in blood flow to all abdominal viscera except adrenal glands in response to IAH in an animal model [42]. Increased IAP reduces splanchnic blood flow [23, 43, 44]. Mesenteric arterial, intestinal mucosal, hepatic arterial, hepatic microcirculatory, and portal venous blood flow have all been shown to be reduced by IAH [43, 44]. Such reductions have a number of potential causes. Reduction of CO as a result of IAH would lead to a decrease in perfusion. It has, however, been shown that reduction in splanchnic blood flow could still occur if the CO and systemic blood pressure were maintained at normal levels [43]. Increased IAP also leads to venous outflow obstruction and hence increases splanchnic vascular resistance. It was also postulated that mesenteric vasoconstriction could be related to the release of vasoactive agents such as catecholamines and angiotensin in reaction to IAH [44]. Small-bowel ischemia and elevated portal venous pressure cause visceral edema, an event which may further aggravate IAH [44].

It has also been noted that while a decrease in mesenteric and intestinal mucosal blood flow first occurred at an IAP of 20 mmHg, hepatic and portal blood flow dropped at merely 10 mmHg [44]. Significant IAH may lead to gut ischemia and infarction. In fact, intestinal ischemia and infarction have been described during prolonged laparoscopy, despite apparently normal hemodynamics and renal function [45].

As a result of hypoperfusion, intestinal mucosal pH is significantly reduced [20, 46]. Several investigators have demonstrated that measurement of intramucosal pH using a gastric tonometer may help in the early detection of splanchnic hypoperfusion in patients with IAH [46]. IAH and intestinal ischemia may also lead to bacterial translocation and free oxygen radical production [47]. Nevertheless, whether bacterial translocation with IAH contributes to the development of septic com-

plications and multiple organ failure in ACS remains controversial [12, 47].

Abdominal wall problem

The direct compressive effect of IAH causes reduction in abdominal wall blood flow causing ischemia and edema [48]. This may then contribute to abdominal wound complications such as dehiscence or necrotizing fasciitis. IAH also reduces abdominal wall compliance. The abdominal wall pressure-volume (i.e., compliance) curve is not linear. As IAP increases, the stiffness of the abdominal wall increases. Consequently, progressively smaller increases in volume are required to effect a given increase in IAP [21]. Abdominal wall compliance may be further affected by tissue edema as a result of fluid resuscitation.

Intracranial derangement

Increased intracranial pressure (ICP) and diminished cerebral perfusion pressure (CPP) have been described in both animal and human studies [49, 50, 51, 52, 53]. Such changes appear to be the result of increased intrathoracic and central venous pressures, which then impede the cerebral venous outflow. The development of pseudotumour cerebri, or benign intracranial hypertension, in morbidly obese individuals has been attributed to the chronic elevation of IAP [54, 55].

Special situations with laparoscopy

The advent of laparoscopic surgery has revolutionized the management of a number of surgical conditions in the last decade. At the same time, however, the pathophysiological effects of pneumoperitoneum have become more evident. In general, the effects are similar to those of IAH. A rise in systemic vascular resistance and fall in CO have been found during laparoscopic cholecystectomy [56, 57]. In fact, the hemodynamic effects of pneumoperitoneum have been likened to that of heart failure [58]. Besides IAH, other major influences on cardiocirculatory status during laparoscopic surgery include posture and carbon dioxide insufflation [56, 58, 59, 60]. Head-up position is associated with a decrease in preload and an increase in afterload while the opposite effects are seen with head-down position [56]. Systemic absorption of carbon dioxide after insufflation results in acidemia and release of vasopressin, which contribute partly to the cardiocirculatory effects of carbon dioxide pneumoperitoneum [59, 60]. However, pneumoperitoneum and IAH also increase the release of catecholamine, but this increase is independent of the gas used for pneumoperitoneum [61].

The pulmonary effects of pneumoperitoneum include high ventilatory pressure and hypercapnia [62]. The use of carbon dioxide also results in its higher arterial and end-tidal levels [62]. The renal effects of pneumoperitoneum are similar to those reported in IAH [63, 64, 65, 66, 67, 68, 69]. The most frequently reported effect is oliguria [70]. Plasma renin and aldosterone levels are increased by pneumoperitoneum [71, 72]. Acute renal failure as a consequence of pneumoperitoneum has not been reported, but inadvertent fluid loading in an attempt to correct oliguria may result in pulmonary edema [65]. Reduction in splanchnic blood flow with resultant mesenteric ischemia and derangement of liver function were also noted with pneumoperitoneum [73, 74, 75]. Fatal cases of intestinal ischemia following laparoscopic cholecystectomy have been reported, although these were protracted operations in high-risk patients [45, 76]. In an elective setting for an otherwise healthy individual, laparoscopic surgery should not produce any clinically significant ill effects. However, its use in patients who are critically ill, having existing multiple organ failure or cardiopulmonary dysfunction, should be cautioned. While gasless laparoscopy may avoid some of the pathophysiological effects of pneumoperitoneum, it provides less satisfactory exposure [65]. In order to lessen the unwanted effects of pneumoperitoneum, the lowest possible pressure for an adequate exposure should be used.

Diagnostic laparoscopy has been advocated for patients with blunt or penetrating abdominal trauma in order to avoid unnecessary laparotomy [77, 78, 79, 80]. The use of laparoscopy in such patients, however, has been controversial owing to the known pathophysiological effects of pneumoperitoneum. Many victims of abdominal trauma have limited cardiopulmonary reserve; increase in IAP in such patients may further jeopardize their hemodynamic status. Most published studies have, therefore, included only patients with stable hemodynamics. However, major blunt abdominal trauma is frequently associated with intracranial injury. In view of the possibility of elevating ICP and decreasing CPP with pneumoperitoneum [81], laparoscopy in patients with suspected head injury should only be performed after careful consideration.

Diagnosis

ACS is a clinical syndrome characterized by increased IAP with evidence of organ dysfunction. A diagnosis of ACS requires (1) recognition of patients at risk; (2) detection of the clinical manifestations; and (3) measurement of IAP. The patients at risk include those who have factors predisposing to increased IAP (Table 1). The typical clinical manifestations include tensely distended abdomen with rising peak ventilatory pressure, elevated central venous pressure, decreased CO, hypoxia, hyper-

capnia, and oliguria The recognition of ACS is not difficult provided the diagnosis is kept in mind.

IAP could be measured using either direct or indirect methods. IAP was first measured directly using an intraperitoneal cannula or needle connected to a saline manometer [4, 10, 11]. Direct measurement using an intraperitoneal catheter connected to a pressure transducer was subsequently used [25, 29, 33, 43]. Owing to the invasiveness of direct methods, indirect methods by measuring either the intragastric or intravesical pressure have been introduced more recently. Intragastric pressure could be obtained by connecting a nasogastric tube or gastrostomy tube to saline or water manometer [82]. Nowadays, measurement of intravesical or urinary bladder pressure (UBP) is the most common indirect method of determining IAP [33, 83, 84]. The bladder wall behaves like a passive diaphragm when the bladder volume is between 50 ml and 100 ml [6]. During the measurement, 50 ml saline is instilled into the bladder through a transurethral catheter which is connected to a manometer. With the patient in supine position, UBP is measured with the zero reference point at the symphysis pubis. Measurement of UBP to determine IAP may be invalid in patients with neurogenic or small contracted bladder.

Computed tomography (CT) findings in patients with ACS were reported recently [85]. These included tense infiltration of the retroperitoneum out of proportion to peritoneal disease, extrinsic compression of the inferior vena cava by retroperitoneal hemorrhage or exudate, and massive abdominal distention with an increased ratio of anteroposterior-to-transverse abdominal diameter (positive round belly sign; ratio >0.80) [85]. Other less frequent CT findings included direct renal compression or displacement, bowel-wall thickening with enhancement, and bilateral inguinal herniation [85].

There is no consensus on criteria for the diagnosis of ACS and the level of IAP at which ACS will occur. Moreover, patients respond differently to the same level of IAP [7]. Consequently, the diagnosis and the need for treatment depend on the clinical assessment of individual patients.

Management

Prevention is better than cure. Every clinician should remember the risk factors for IAH and be vigilant to avoid the progression to ACS. Moreover, forceful closure of an abdominal wound should be avoided in patients with risk factors for IAH.

Indications for abdominal decompression

Abdominal decompression (decompressive laparotomy) is the only treatment for ACS. The main controversy is when to intervene. Most centers will perform abdominal decompression in the presence of definite organ failure. Kron suggested using a UBP above 25 mmHg (1 mmHg equals 1.36 cm H_2O approximately) as a criterion for surgical decompression when it is associated with oliguria [6]. Others considered clinical manifestation a more important parameter for decompression with UBP as a supportive guide only [2, 86].

Table 3 Grading system for abdominal compartment syndrome [7]

Grade	Bladder pressure (cm H_2O)	Recommendation
1	10–15	Maintain normovolemia
2	16–25	Clinical correlation
3	26–35	Decompression
4	>35	Decompression

Burch proposed a grading system upon which treatment can be based (Table 3) [7]. He recommended clinical correlation at moderately elevated IAP (grade 2) and surgical decompression in more severe IAH (grades 3, 4) irrespective of clinical manifestation of ACS.

Similarly, Meldrum recommended a liberal policy using physiological criteria and UBP as the guide for surgical decompression [31]. Prompt decompression was recommended in the presence of an IAP of greater than 20 mmHg and any significant physiological abnormalities such as elevated peak airway pressure or oliguria. A higher survival rate was achieved than with other studies [2, 31, 86]. Recent studies also suggested that intestinal ischemia and mucosal acidosis began at much lower IAP, long before ACS became clinically evident [20]. Early and aggressive management of IAH was therefore advocated [20]. Measurement of gut mucosal pH using a gastric tonometer may help in the early detection of splanchnic hypoperfusion [46]. Early decompression in patients with mucosal acidosis may avoid the occurrence of full-blown ACS.

Peri-operative management

Hypotension may occur after decompression because of the abrupt drop in central filling pressure and systemic vascular resistance [12, 87, 88]. Moreover, bleeding accompanying coagulopathy may further aggravate the hypotensive episode. Supraventricular arrhythmia and episodes of asystole have also been reported [2, 6, 86]. One proposed mechanism for these events is the reperfusion syndrome [2]. There is rapid delivery to the systemic circulation of by-products of anaerobic metabolism from the reperfused visceral and lower extremities after decompression [2].

Consequently, prior to any decompressive laparotomy, the patient should be well prepared with adequate in-

travascular volume. Coagulopathy should be adequately corrected and hypothermia should be avoided. The infusion of normal saline with mannitol and sodium bicarbonate before decompression was advocated to minimize the effects of reperfusion [2, 86]. The anesthetist plays an important role in the perioperative management of the patient [89]. Monitoring and maintenance of intravascular volume must be undertaken at the time of release of the IAP [8, 90]. The PEEP can usually be reduced following decompression.

Decompressive laparotomy

The conduct of decompressive laparotomy depends on the clinical situation. It is affected by the laparotomy findings, previous operation, etiology of IAH, previous damage control procedure, and the means of closure. In general, the abdomen should be thoroughly explored, definite bleeding sources should be controlled, and fascial closure of the abdominal wall could be attempted if the volume of abdominal content was sufficiently reduced [2, 87]. In most instances, however, the marked edema of bowel may render a formal fascial closure impossible. Some forms of temporary abdominal closure are then required. Many centers advocate the use of UBP to guide the closure of abdomen [91]. The choice between primary fascial closure and temporary abdominal closure can be made according to the assessment of IAP and the likelihood of development of ACS [2, 87].

Damage control

Another important concept, especially in the context of abdominal trauma, is the performance of planned reoperation (damage control or staged repair) [86, 92, 93, 94]. It is required in approximately 10% of all trauma victims who require laparotomy [86, 95]. There are three main indications for damage control [93, 96, 97]. First, the presence of hypothermia, acidosis, and coagulopathy may lead to death on the operating table if the operation is not terminated quickly. Second, uncontrollable bleeding may require maneuvers like temporary packing or balloon catheter tamponade. Lastly, in the presence of massive visceral edema, temporary closure of the abdomen helps avoid the development of IAH and ACS. In order to expedite the operation, any devitalized intestines could be resected with staplers and intestinal continuity is not restored. Similarly, vascular injuries may be treated with ligation, vascular shunts, or extra-anatomic bypass rather than with definitive repair [98, 99].

Temporary abdominal closure

There are numerous methods for temporary abdominal closure [100, 101]. The principle is to maintain tension-free, secure, and watertight coverage of the viscera. This helps to minimize fluid loss and protects the viscera from damage. Leaving the abdomen open allows fluid loss as well as bowels to eviscerate and is therefore not an appropriate option. Temporary closure can be achieved by closing only the skin with towel clips or sutures [7, 8] if the IAP is not high. Towel clip closure is done with 25 to 40 towel clips, depending on the length of incision [96]. This method is quick and easy but it also carries two main disadvantages [97]: (1) there is a possibility of closure with tension and thus the development of ACS and (2) the presence of towel clips may obscure any subsequent radiological imaging.

In the presence of marked tension, bridging the wound gap by some form of coverage or prosthesis is necessary. Such coverage could be effected using a variety of methods including, for example, combination of surgical towels and adhesive drapes, non-adhesive drapes stapled to skin, or sterile rayon cloth under retention sutures [12, 102, 103]. Another inexpensive option is to suture in place a sterilized opened 3-l genitourinary irrigation bag, also known as the “Bogota bag” after its first description by Londoni in Bogota, Columbia [7]. The advantages of such plastic coverage are cheapness, softness, and transparency, which permit inspection of the intra-abdominal contents.

Alternatively, absorbable or non-absorbable and porous or nonporous prostheses have also been recommended [7, 8, 87, 104, 105]. Non-absorbable materials include polypropylene and polytetrafluoroethylene, while absorbable materials include polyglactin and polyglycolic acid [103]. The choice of prosthesis depends on the circumstances as well as individual preferences. The use of Velcro or zipper allows easy re-exploration of the peritoneal cavity [106, 107].

Definitive abdominal closure

When further laparotomy is not required, definitive closure of the abdominal wall could either be achieved during the same hospitalization or be delayed for several months. Early definitive closure during the same hospitalization is preferred [7]. In general, definitive closure is recommended when the condition of patient is stabilized with restoration of tissue oxygenation and vascular volume, as well as reversal of coagulopathy and hypothermia [86, 93]. After a prolonged period of temporary closure, primary fascial closure may not be possible. In such patients, various methods of closure are available. Skin grafting of a bridging mesh is one possible option [7, 87]. Alternatively, bilateral medial myocutaneous ad-

vancement of the rectus muscle may avoid the use of prosthesis in selected patients [108, 109].

Conclusion

IAH exerts physiological derangement in a graded fashion. IAH and ACS can be considered as different stages of the pathophysiology of increased IAP. Rapid progression of IAH will lead to ACS, which is characterized by multiple system organ dysfunction, commonly cardiopulmonary and renal impairment. Recent studies showed that ischemic insult of the gut as a result of splanchnic hypoperfusion appeared well before clinical manifestation of ACS. In view of the association between bowel ischemia and higher incidence of septic complication and multiple organ failure, early decompression had been recently advocated.

The mortality of ACS is high. Recognizing patients at risk, monitoring for signs of ACS, and early initiation of treatment could help to reduce the morbidity and mortality of the syndrome. ACS is a clinical entity of increasing importance not just to trauma surgeons but to all surgeons alike.

References

1. Coombs HC (1920) The mechanism of the regulation of intra-abdominal pressure. Am J Physiol 61:159–163
2. Eddy V, Nunn C, Morris JA Jr (1997) Abdominal compartment syndrome. The Nashville experience. Surg Clin North Am 77:801–812
3. Bradley SE, Bradley GP (1947) The effect of increased intra-abdominal pressure on renal function. J Clin Invest 26:1010–1015
4. Emerson H (1911) Intra-abdominal pressures. Arch Intern Med 7:754–784
5. Baggot MG (1951) Abdominal blowout: a concept. Curr Res Anesth Analg 30:295–298
6. Kron IL, Harman PK, Nolan SP (1984) The measurement of intra-abdominal pressure as a criterion for abdominal re-exploration. Ann Surg 199:28–30
7. Burch JM, Moore EE, Moore FA, Franciose R (1996) The abdominal compartment syndrome. Surg Clin North Am 76:833–842
8. Schein M, Wittmann DH, Aprahamian CC, Condon RE (1995) The abdominal compartment syndrome: the physiological and clinical consequences of elevated intra-abdominal pressure. J Am Coll Surg 180:745–753
9. Behrman SW, Bertken KA, Stefanacci HA, Parks SN (1998) Breakdown of intestinal repair after laparotomy for trauma: incidence, risk factors, and strategies for prevention. J Trauma 45:227–231
10. Overholt RH (1931) Intraperitoneal pressure. Arch Surg 22:691–703
11. Wagner GW (1926) Studies on intra-abdominal pressure. Am J Med 171:697–707
12. Saggi BH, Sugerman HJ, Ivatury RR, Bloomfield GL (1998) Abdominal compartment syndrome. J Trauma 45:597–609
13. Ivy ME, Possenti PP, Kepros J, Atweh NA, M DA, Palmer J, Pineau M, Burns GA, Caushaj PF (1999) Abdominal compartment syndrome in patients with burns. J Burn Care Rehabil 20:351–353
14. Fietsam R Jr, Villalba M, Glover JL, Clark K (1989) Intra-abdominal compartment syndrome as a complication of ruptured abdominal aortic aneurysm repair. Am Surg 55:396–402
15. Meldrum DR, Moore FA, Moore EE, Haenel JB, Cosgriff N, Burch JM (1995) Jack A. Barney Resident Research Award. Cardiopulmonary hazards of perihepatic packing for major liver injuries. Am J Surg 170:537–540
16. Sugerman H, Windsor A, Bessos M, Wolfe L (1997) Intra-abdominal pressure, sagittal abdominal diameter and obesity comorbidity. J Intern Med 241:71–79
17. Burchard KW, Ciombor DM, McLeod MK, Slothman GJ, Gann DS (1985) Positive end expiratory pressure with increased intra-abdominal pressure. Surg Gynecol Obstet 161:313–318
18. Moffa SM, Quinn JV, Slotman GJ (1993) Hemodynamic effects of carbon dioxide pneumoperitoneum during mechanical ventilation and positive end-expiratory pressure. J Trauma 35:613–617
19. Burrows R, Edington J, Robbs JV (1995) A wolf in wolf's clothing–the abdominal compartment syndrome. S Afr Med J 85:46–48
20. Ivatury RR, Diebel L, Porter JM, Simon RJ (1997) Intra-abdominal hypertension and the abdominal compartment syndrome. Surg Clin North Am 77:783–800
21. 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–R213
22. Burchard KW (1990) Cardiovascular, pulmonary, and renal effects of massively increased intra-abdominal pressure in critically ill patients. Crit Care Med 18:120
23. Chang MC, Miller PR, Agostino RJ, Meredith JW (1998) Effects of abdominal decompression on cardiopulmonary function and visceral perfusion in patients with intra-abdominal hypertension. J Trauma Inj Infect Crit Care 44:440–445
24. Cullen DJ, Coyle JP, Teplick R, Long MC (1989) Cardiovascular, pulmonary, and renal effects of massively increased intra-abdominal pressure in critically ill patients. Crit Care Med 17:118–121
25. Richardson JD, Trinkle JK (1976) Hemodynamic and respiratory alterations with increased intra-abdominal pressure. J Surg Res 20:401–404
26. Caldwell CB, Ricotta JJ (1987) Changes in visceral blood flow with elevated intraabdominal pressure. J Surg Res 43:14–20
27. Kashtan J, Green JF, Parsons EQ, Holcroft JW (1981) Hemodynamic effects of increased abdominal pressure. J Surg Res 30:249–255
28. Takata M, Wise RA, Rotham JL (1990) Effects of abdominal pressure on venous return: abdominal vascular zone conditions. J Appl Physiol 69:1961–1972
29. Rubinson RM, Vasko JS, Doppman JL, Morrow AG (1967) Inferior caval obstruction from increased intra-abdominal pressure. Arch Surg 94:766–770
30. Diamant M, Benumof JL, Saidman LJ (1978) Hemodynamics of intra-abdominal pressure. Anesthesiology 48:23–27
31. Meldrum DR, Moore FA, Moore EE, Franciose RJ, Sauaia A, Burch JM (1997) Prospective characterization and selective management of the abdominal compartment syndrome. Am J Surg 174:667–672

32. Safran DS, Orlando R (1994) Physiologic effects of pneumoperitoneum. Am J Surg 167:281–286
33. 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
34. Obeid F, Saba A, Fath J, Guslits B, Chung R, Sorensen V, Buck J, Horst M (1995) Increases in intra-abdominal pressure affect pulmonary compliance. Arch Surg 130:544–547
35. Mutoh T, Lamm WJ, Embree LJ (1991) Abdominal distension alters regional pleural pressures and chest wall mechanics in pigs in vivo. J Appl Physiol 70:2611–2618
36. 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
37. Harman PK, Kron IL, McLachlan HD, Freedlender AE, Nolan SP (1982) Elevated intra-abdominal pressure and renal function. Ann Surg 196:594–597
38. Richards WO, Scovill W, Shin B, Reed W (1983) Acute renal failure associated with increased intra-abdominal pressure. Ann Surg 197:183–187
39. Tan IK, Kua JS (1998) Abdominal compartment syndrome and acute anuria. Nephrol Dial Transplant 13:2651–2653
40. Bloomfield GL, Blocher CR, Fakhry IF, Sica DA, Sugerman HJ (1997) Elevated intra-abdominal pressure increases plasma renin activity and aldosterone levels. J Trauma 42:997–1004
41. Sugrue M, Jones F, Deane SA, Bishop G, Bauman A, Hillman K (1999) Intra-abdominal hypertension is an independent cause of postoperative renal impairment. Arch Surg 134:1082–1085
42. Caldwell CB, Ricotta JJ (1986) Evaluation of intra-abdominal pressure and renal hemodynamics. Curr Surg 43:495–498
43. 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
44. 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
45. Paul A, Troidl H, Peters S, Stuttmann R (1994) Fatal intestinal ischaemia following laparoscopic cholecystectomy. Br J Surg 81:1207
46. Ivatury RR, Porter JM, Simon RJ, Islam S, John R, Stahl WM (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
47. Diebel LN, Dulchavsky SA, Brown WJ (1997) Splanchnic ischemia and bacterial translocation in the abdominal compartment syndrome. J Trauma 43:852–855
48. Diebel L, Saxe J, Dulchavsky S (1992) Effect of intra-abdominal pressure on abdominal wall blood flow. Am Surg 58:573–575
49. Bloomfield GL, Dalton JM, Sugerman HJ, Ridings PC, DeMaria EJ, Bullock R (1995) Treatment of increasing intracranial pressure secondary to the acute abdominal compartment syndrome in a patient with combined abdominal and head trauma. J Trauma 39:1168–1170
50. Bloomfield GL, Ridings PC, Blocher CR, Marmarou A, Sugerman HJ (1996) Effects of increased intra-abdominal pressure upon intracranial and cerebral perfusion pressure before and after volume expansion. J Trauma 40:936–941
51. Bloomfield GL, Ridings PC, Blocher CR, Marmarou A, Sugerman HJ (1997) A proposed relationship between increased intra-abdominal, intrathoracic, and intracranial pressure. Crit Care Med 25:496–503
52. Rosenthal RJ, Friedman RL, Kahn AM, Martz J, Thiagarajah S, Cohen D, Shi Q, Nussbaum M (1998) Reasons for intracranial hypertension and hemodynamic instability during acute elevations of intra-abdominal pressure: observations in a large animal model. J Gastrointest Surg 2:415–425
53. Irgau I, Koyfman Y, Tikellis JI (1995) Elective intraoperative intracranial pressure monitoring during laparoscopic cholecystectomy. Arch Surg 130:1011–1013
54. Sugerman HJ, Felton WL III, Salvant JB Jr, Sismanis A, Kellum JM (1995) Effects of surgically induced weight loss on idiopathic intracranial hypertension in morbid obesity. Neurology 45:1655–1659
55. Sugerman HJ, DeMaria EJ, Felton WL III, Nakatsuka M, Sismanis A (1997) Increased intra-abdominal pressure and cardiac filling pressures in obesity-associated pseudotumor cerebri. Neurology 49:507–511
56. Hirvonen EA, Poikolainen EO, Paakkonen ME, Nuutinen LS (2000) The adverse hemodynamic effects of anesthesia, head-up tilt, and carbon dioxide pneumoperitoneum during laparoscopic cholecystectomy. Surg Endosc 14:272–277
57. Joris JL, Noirot DP, Legrand MJ, Jacquet NJ, Lamy ML (1996) Hemodynamic changes during laparoscopic cholecystectomy. Anesth Analg 76:1067–1071
58. Struthers AD, Cuschieri A (1998) Cardiovascular consequences of laparoscopic surgery. Lancet 352:568–570
59. Ho HS, Saunders CJ, Gunther RA, Wolfe BM (1995) Effector of hemodynamics during laparoscopy: CO2 absorption or intra-abdominal pressure? J Surg Res 59:497–503
60. Mann C, Boccara G, Pouzeratte Y, Eliet J, Serradeil Le Gal C, Vergnes C, Bichet DG, Guillon G, Fabre JM, Colson P (1999) The relationship among carbon dioxide pneumoperitoneum, vasopressin release, and hemodynamic changes. Anesth Analg 89:278–283
61. Mikami O, Fujise K, Matsumoto S, Shingu K, Ashida M, Matsuda T, Hirvela ER (1998) High intra-abdominal pressure increases plasma catecholamine concentrations during pneumoperitoneum for laparoscopic procedures. Arch Surg 133:39–43
62. Schulze S, Lyng KM, Bugge K, Perner A, Bendtsen A, Thorup J, Nielsen HJ, Rasmussen V, Rosenberg J (1999) Cardiovascular and respiratory changes and convalescence in laparoscopic colonic surgery: comparison between carbon dioxide pneumoperitoneum and gasless laparoscopy. Arch Surg 134:1112–1118
63. Chiu AW, Azadzoi KM, Hatzichristou DG, Siroky MB, Krane RJ, Babayan RK (1994) Effects of intra-abdominal pressure on renal tissue perfusion during laparoscopy. J Endourol 8:99–103
64. Chiu AW, Chang LS, Birkett DH, Babayan RK (1995) The impact of pneumoperitoneum, pneumoretroperitoneum, and gasless laparoscopy on the systemic and renal hemodynamics. J Am Coll Surg 181:397–406
65. Hunter JG (1995) Laparoscopic pneumoperitoneum: the abdominal compartment syndrome revisited. J Am Coll Surg 181:469–470
66. Iwase K, Takenaka H, Ishizaka T, Ohata T, Oshima S, Sakaguchi K (1993) Serial changes in renal function during laparoscopic cholecystectomy. Eur Surg Res 25:203–212
67. Kirsch AJ, Hensle TW, Chang DT, Kayton ML, Olsson CA, Sawczuk IS (1994) Renal effects of CO2 insufflation: oliguria and acute renal dysfunction in a rat pneumoperitoneum model. Urology 43:453–459
68. Lee BR, Cadeddu JA, Molnar Nadasdy G, Enriquez D, Nadasdy T, Kavoussi LR, Ratner LE (1999) Chronic effect of pneumoperitoneum on renal histology. J Endourol 13:279–282

69. McDougall EM, Monk TG, Wolf JS Jr, Hicks M, Clayman RV, Gardner S, Humphrey PA, Sharp T, Martin K (1996) The effect of prolonged pneumoperitoneum on renal function in an animal model. J Am Coll Surg 182:317–328
70. Hamilton BD, Chow GK, Inman SR, Stowe NT, Winfield HN (1998) Increased intra-abdominal pressure during pneumoperitoneum stimulates endothelin release in a canine model. J Endourol 12:193–197
71. O'Leary E, Hubbard K, Tormey W, Cunningham AJ (1996) Laparoscopic cholecystectomy: haemodynamic and neuroendocrine responses after pneumoperitoneum and changes in position. Br J Anaesth 76:640–644
72. Koivusalo AM, Kellokumpu I, Scheinin M, Tikkanen I, Halme L, Lindgren L (1996) Randomized comparison of the neuroendocrine response to laparoscopic cholecystectomy using either conventional or abdominal wall lift techniques. Br J Surg 83:1532–1536
73. Andrei VE, Schein M, Margolis M, Rucinski JC, Wise L (1998) Liver enzymes are commonly elevated following laparoscopic cholecystectomy: is elevated intra-abdominal pressure the cause? Dig Surg 15:256–259
74. Richmond BK, Lucente FC, Boland JP (1997) Laparoscopy-associated mesenteric vascular events: description of an evolving clinical syndrome. J Laparoendosc Adv Surg Tech A 7:363–367
75. Windberger UB, Auer R, Keplinger F, Langle F, Heinze G, Schindl M, Losert UM (1999) The role of intra-abdominal pressure on splanchnic and pulmonary hemodynamic and metabolic changes during carbon dioxide pneumoperitoneum. Gastrointest Endosc 49:84–91
76. Jaffe V, Russell RC (1994) Fatal intestinal ischaemia following laparoscopic cholecystectomy. Br J Surg 81:1827–1828
77. Brandt CP, Priebe PP, Jacobs DG (1994) Potential of laparoscopy to reduce non-therapeutic trauma laparotomies. Am Surg 60:416–420
78. Ditmars ML, Bongard F (1996) Laparoscopy for triage of penetrating trauma: the decision to explore. J Laparoendosc Surg 6:285–291
79. Fabian TC, Croce MA, Stewart RM, Pritchard FE, Minard G, Kudsk KA (1993) A prospective analysis of diagnostic laparoscopy in trauma. Ann Surg 217:557–564
80. Mutter D, Nord M, Vix M, Evrard S, Marescaux J (1997) Laparoscopy in the evaluation of abdominal stab wounds. Dig Surg 14:39–42
81. Josephs LG, Este McDonald JR, Birkett DH, Hirsch EF (1994) Diagnostic laparoscopy increases intracranial pressure. J Trauma 36:815–818
82. Collee GG, Lomax DM, Ferguson C, Hanson GC (1993) Bedside measurement of intra-abdominal pressure (IAP) via an indwelling naso-gastric tube: clinical validation of the technique. Intensive Care Med 19:478–480
83. Iberti TJ, Lieber CE, Benjamin E (1989) Determination of intra-abdominal pressure using a transurethral bladder catheter: clinical validation of the technique. Anesthesiology 70:47–50
84. Cheatham ML, Safcsak K (1998) Intra-abdominal pressure: a revised method for measurement. J Am Coll Surg 186:594–595
85. Pickhardt PJ, Shimony JS, Heiken JP, Buchman TG, Fisher AJ (1999) The abdominal compartment syndrome: CT findings. Am J Roentgenol 173:575–579
86. Morris JA Jr, Eddy VA, Blinman TA, Rutherford EJ, Sharp KW (1993) The staged celiotomy for trauma. Issues in unpacking and reconstruction. Ann Surg 217:576–584
87. Reeves ST, Pinosky ML, Byrne TK, Norcross ED (1997) Abdominal compartment syndrome. Can J Anaesth 44:308–312
88. Shelly MP, Robinson AA, Hesford JW, Park GR (1987) Haemodynamic effects following surgical release of increased intra-abdominal pressure. Br J Anaesth 59:800–805
89. Ham AA, Coveler LA (1997) Anesthetic considerations in damage control surgery. Surg Clin North Am 77:909–920
90. Nathens AB, Brenneman FD, Boulanger BR (1997) The abdominal compartment syndrome. Can J Surg 40:254–258
91. Rizzo A, Davis PC, Hamm CR, Powell RW (1996) Intraoperative vesical pressure measurements as a guide in the closure of abdominal wall defects. Am Surg 62:192–196
92. Hirshberg A, Wall MJ, Jr., Mattox KL (1994) Planned reoperation for trauma: a two year experience with 124 consecutive patients. J Trauma 37:365–369
93. Hirshberg A, Mattox KL (1995) Planned reoperation for severe trauma. Ann Surg 222:3–8
94. Moore EE, Burch JM, Franciose RJ, Offner PJ, Biffl WL (1998) Staged physiologic restoration and damage control surgery. World J Surg 22:1184–1190
95. Rotondo MF, Schwab CW, McGonigal MD, Phillips GR, Fruchterman TM, Kauder DR, Latenser BA, Angood PA (1993) 'Damage control': an approach for improved survival in exsanguinating penetrating abdominal injury. J Trauma 35:375–382
96. Burch JM, Ortiz VB, Richardson RJ, Martin RR, Mattox KL, Jordan GL Jr (1992) Abbreviated laparotomy and planned reoperation for critically injured patients. Ann Surg 215:476–483
97. Hirshberg A, Walden R (1997) Damage control for abdominal trauma. Surg Clin North Am 77:813–820
98. Pourmoghadam KK, Fogler RJ, Shaftan GW (1997) Ligation: an alternative for control of exsanguination in major vascular injuries. J Trauma 43:126–130
99. Porter JM, Ivatury RR, Nassoura ZE (1997) Extending the horizons of "damage control" in unstable trauma patients beyond the abdomen and gastrointestinal tract. J Trauma 42:559–561
100. Brock WB, Barker DE, Burns RP (1995) Temporary closure of open abdominal wounds: the vacuum pack. Am Surg 61:30–35
101. Ciresi DL, Cali RF, Senagore AJ (1999) Abdominal closure using nonabsorbable mesh after massive resuscitation prevents abdominal compartment syndrome and gastrointestinal fistula. Am Surg 65:720–724
102. Bender JS, Bailey CE, Saxe JM, Ledgerwood AM, Lucas CE (1994) The technique of visceral packing: recommended management of difficult fascial closure in trauma patients. J Trauma 36:182–185
103. Brasel KJ, Weigelt JA (2000) Damage control in trauma surgery. Curr Opin Crit Care 6:276–280
104. Albrecht R, Diebel LN, Moorman D, Ciresi DL (1999) Abdominal closure using nonabsorbable mesh after massive resuscitation prevents abdominal compartment syndrome and gastrointestinal fistula. Am Surgeon 65:724–725
105. Mayberry JC, Mullins RJ, Crass RA, Trunkey DD (1997) Prevention of abdominal compartment syndrome by absorbable mesh prosthesis closure. Arch Surg 132:957–961
106. Cuesta MA, Doblas M, Castaneda L, Bengoechea E (1991) Sequential abdominal reexploration with the zipper technique. World J Surg 15:74–80
107. Tsiotos GG, Luque de Leon E, Soreide JA, Bannon MP, Zietlow SP, Baerga Varela Y, Sarr MG (1998) Management of necrotizing pancreatitis by repeated operative necrosectomy using a zipper technique. Am J Surg 175:91–98
108. Fayman MS, Schein M, Saadia R (1990) Abdominal wall reconstruction after open management of the septic abdomen. S Afr J Surg 28:62–65
109. Fabian TC, Croce MA, Pritchard FE, Minard G, Hickerson WL, Howell RL, Schurr MJ, Kudsk KA (1994) Planned ventral hernia. Staged management for acute abdominal wall defects. Ann Surg 219:643–650