World J Surg (2009) 33:1123–1127
DOI 10.1007/s00268-009-0040-4

World Journal of Surgery

Intra-Abdominal Pressure and Abdominal Compartment Syndrome in Acute General Surgery

Michael Sugrue · Yasir Buhkari

Published online: 29 April 2009

Abstract

Background Intra-abdominal pressure (IAP) is a harbinger of intra-abdominal mischief, and its measurement is cheap, simple to perform, and reproducible. Intra-abdominal hypertension (IAH), especially grades 3 and 4 (IAP > 18 mmHg), occurs in over a third of patients and is associated with an increase in intra-abdominal sepsis, bleeding, renal failure, and death.

Patients and methods Increased IAP reading may provide an objective bedside stimulus for surgeons to expedite diagnostic and therapeutic work-up of critically ill patients. One of the greatest challenges surgeons and intensivists face worldwide is lack of recognition of the known association between IAH, ACS, and intra-abdominal sepsis. This lack of awareness of IAH and its progression to ACS may delay timely intervention and contribute to excessive patient resuscitation.

Conclusions All patients entering the intensive care unit (ICU) after emergency general surgery or massive fluid resuscitation should have an IAP measurement performed every 6 h. Each ICU should have guidelines relating to techniques of IAP measurement and an algorithm for management of IAH.

M. Sugrue (✉) · Y. Buhkari
Department of Surgery, Letterkenny General Hospital and Galway University Hospitals, Letterkenny, Donegal, Ireland
e-mail: acstrauma@hotmail.com

Y. Buhkari
e-mail: dryasirimran@hotmail.com

Introduction

While the term "abdominal compartment syndrome" (ACS) was coined by Fietsam in 1989, intra-abdominal hypertension (IAH) has been recognized as a clinical entity for nearly 150 years [1, 2]. Intra-abdominal hypertension is defined as a "sustained or repeated pathological elevation in intra-abdominal pressure (IAP) $\geq$ 12 mmHg," whereas ACS is defined as "sustained IAP > 20 mmHg (with or without an [abdominal perfusion pressure] APP < 60 mmHg) that is associated with new organ dysfunction/ failure" [3]. Abdominal compartment syndrome can be classified as primary, secondary, or recurrent.

Intra-abdominal hypertension is measured as an IAP of 12 mmHg or greater. There are four classifications: grade one, 12–15 mmHg; grade two, 16–20 mmHg; grade three, 21–25 mmHg; and grade four, > 25 mmHg.

The true prevalence of ACS has been difficult to ascertain from past studies because of the varied definitions of IAH and ACS applied. A multicenter point prevalence prospective study of IAH (defined as IAP $\geq$ 12 mmHg) conducted over a one-day period in 13 Intensive Care Units (ICU) from Belgium, Italy, Austria, Israel, Brazil, and Australia, found the prevalence of IAH to be 50.5% and the prevalence of ACS to be 8.2% [4].

General surgery has evolved over the last two decades with changes in subspecialization resulting in the loss of many patients to individual surgical specialties including vascular, trauma, breast, endocrine, colorectal, hepatobiliary, and transplant. What is clear is that acute general surgery incorporating emergency surgery will remain in the realm of the general surgeon. It is in this subgroup of general surgery patients that IAH is most common.

Intra-abdominal hypertension has adverse effects on cardiovascular, respiratory, gastrointestinal, renal, and neurological function [5–12]. General surgeons should therefore have a clear understanding of IAH and its potential progression to ACS. Despite a plea for guidelines regarding monitoring of IAH [13, 14], there is a general reluctance to adopt the concept of monitoring and understanding the consequences of IAH [15–18].

History and background

Intra-abdominal pressure has become an important physiological concept in medicine. It was probably Wendt, as early as 1867, who first described the association between raised abdominal pressure and renal impairment [2]. Although Weber in 1851 and Donders in 1854 identified the adverse effects of increased respiratory pressures on venous return in both humans and animals, they did not discover the effect of IAP on venous circulation and stasis [19]. Until the early part of this century, there was a poor understanding of the concept of IAP. Most investigators felt that the IAP was negative or subatmospheric. In 1911, Emerson first recognized, during studies on dogs, cats, and rabbits that IAP was usually positive and found those significant rises in IAP causing cardiac failure [20]. Almost 50 years later, in the early 1980s Kron et al., from the University of Virginia, introduced the concept of abdominal exploration and decompression to improve outcome in patients with tense abdomens after surgery [21]. They standardized the measurement of IAP using a urinary catheter.

In 1982 Harmon suggested that renal vascular resistance was probably the single most important contributor to renal dysfunction in patients with increased IAP [22]. Within 2 years, Kron and colleagues suggested that IAP could be used as a criterion for abdominal decompression [21]. Fietsam et al., from Michigan, described ACS in four patients post-aortic surgery [1].

It was not until the 1990s that our understanding of IAH in general surgery gained prominence, as more studies reported evaluation of the effect of IAP during laparoscopy and concepts related to damage control in both trauma and abdominal surgery. For example, it was recognized that during laparoscopic cholecystectomy a significant reduction in hepatic blood flow was related to an increase in IAP [23].

It was Sugrue and colleagues, in the 1990s, who reported the first two large prospective studies of IAH in general surgery patients [10, 24]. These studies identified not only the relative frequency of raised IAP and IAH in a general surgery cohort but also the importance of IAH in those patients undergoing emergency general surgery.

Pathophysiology

Intra-abdominal hypertension results in a variable series of pathophysiologic sequalae depending on the underlying diagnosis, the presence or absence of sepsis, persistence of bleeding, state of abdominal compliance, and volume status. Because the impact of IAH will vary from patient to patient, IAP readings cannot be taken in isolation. This relates to where the patient lies on the abdominal compliance curve: IAH commences by exerting negative effects at the cellular level, with alterations in mRNA, as well as intracellular enzyme and nitrous oxidation [25]. The effect then becomes regional, with reduction in visceral blood flow, increase in organ vascular resistance, and reduced local perfusion. Uncorrected IAH will result in significantly reduced abdominal perfusion pressure (APP). The APP has been shown to be an even more sensitive indicator of negative outcome than IAP [26]. What started at the cellular level moves from regional to global with increased afterload and reduced pre-load. Abdominal compartment syndrome becomes apparent with the progression of IAH, especially in patients undergoing laparotomy where intra-abdominal sepsis is confounded by postoperative pain and reduced ventilation. Mechanical problems that are commonly seen as ACS progresses include decreased functional residual capacity, ventilation perfusion mismatch, and increased intrathoracic pressures with resultant decreased oxygen delivery.

How and when in whom should pressure be measured?

Intra-abdominal pressure should be measured, ideally continuously, to provide real-time readings in the serious ill patient [27]. Historically the benchmark standard has been the Kron technique or minor modifications of it. Intra-abdominal pressure is measured in line with the WCACS recommendations; that is, at end-expiration with the patient lying supine and the transducer zeroed at the level of the mid-axillary line with an instillation volume of 25 ml saline. Continuous readings are popular among nurses, but unfortunately the technique still requires a three-way catheter, which is not routinely used in general surgery. In the ICU, IAP is a fairly steady state (exceptions occur during laparoscopy and endoscopy), and two daily readings are generally adequate where an intermittent technique is used. When there is concern of bleeding, the frequency should be increased to 4 hourly. Different commercial devices make the procedure easy; for the ICU the AbViser (Wolfe Tory Medical, Salt Lake City, UT, USA) is ideal and for the ward patient the U manometer is useful.

All patients admitted to ICU after emergency general surgery should have IAP measurement performed because

Table 1 Risk factors for abdominal compartment syndrome (ACS)

Acute respiratory failure
Abdominal surgery
Major trauma
Major burns
Prone positioning
High body mass index
Gastroparesis
Ileus
Colonic pseudo-obstruction
Hemoperitoneum
Pneumoperitoneum
Liver dysfunction/ascites
Acidosis
Hypotension
Hypothermia
Polytransfusion
Coagulopathy
Massive fluid resuscitation
Pancreatitis
Oliguria
Sepsis

we know that 40% will have grade two or greater IAH. Risk factors for the development of ACS are shown in Table 1.

Which general surgery patients are at risk of IAH?

Patients undergoing emergency surgery are at much greater risk than patients undergoing elective procedures. Those who are likely to develop secondary and tertiary peritonitis are highest risk, and those with severe pancreatitis, severe burns, or post-aortic surgery are the group on the "fringe" of general surgery most likely to be affected. A recent study of 102 consecutive patients undergoing laparotomy for secondary peritonitis found that the postoperative detection of IAH preceded the diagnosis of postoperative peritonitis by 4 ± 2.4 days [28].

Prevention

The key to prevention is to understand the causes of IAH that lead to ACS (Tables 2 and 3). While 45% are multifactorial, fluid overload coupled with intra-abdominal sepsis, bowel obstruction, and hemorrhage are the main individual causes. Earlier diagnosis and treatment of intra-abdominal sepsis will result in less fluid administration and prevention of tissue edema [29, 30]. Excellence in technical surgery coupled with appropriate correction of coagulopathy will decrease postoperative hematoma and bleeding. While trauma surgeons have embraced prophylactic abdominal decompression, this procedure has not been popular among general surgeons [31], and it is probably underutilized. The use of AbdoVAC (KCI Medical, San Antonio, TX, USA) and other proprietary devices facilitates re-exploration in patients who need decompression for IAH and ACS.

Optimizing clinical care will reduce adverse outcomes, which has been demonstrated in areas at the periphery of acute general surgery, such as pelvic fracture, where early hemorrhage control will reduce mortality significantly and in the process reduce the prevalence of ACS [32].

Treatment

An increase in IAP resulting in grade three and four IAH indicates a need for more urgent and precise treatment. The World Society on the Abdominal Compartment Syndrome has published Web based guidelines (www.wsacs.org). The key is detection and treatment of sepsis, postoperative hemorrhage, and efforts to avoid aggressive fluid resuscitation.

Diagnosis of tertiary peritonitis requires an aggressive diagnostic approach, with abdominal CT scanning and prompt drainage of pus. Detection of postoperative hemorrhage requires good nursing care and clear pathways for communication between junior staff and consultants. Ideally, frontline ward and ICU staff should have regular performance evaluations to ensure a reduction in errors, which are fairly common. Apart from the direct association between sepsis and bleeding, IAH exerts negative effects on colon healing and visceral blood flow [33, 34]

Challenges

Surgeons have an innate ability to deny the obvious. This may reflect a protective mechanism in dealing with the pain, both physical and psychological, of their patients and the patients' relatives. Surgeons are often reluctant to consider a second look (either directed by CT or otherwise) because they may feel that recurrent problems are unlikely after they have performed an operation. Similarly, although it has been shown that the use of regular IAP measurements and early detection of IAH can alert the attending surgical team of an impending abdominal catastrophe, few ICUs around the world regularly measure IAP. Also, we know that girth measurements and clinical estimates of IAP are often inaccurate.

In patients with grades three to four IAH, the treating surgical and ICU team should make it a priority to locate the underlying problem, and provide interim hemodynamic

Table 2 Nature of surgery and intra-abdominal pressure (IAP) level (mmHg)

Type of surgery	Overall		Emergency		Elective		Elective versus emergency
	n	IAP ≥ 18 (%)	*n*	IAP ≥ 18 (%)	*n*	IAP ≥ 18 (%)	
Upper gastrointestinal	131	54 (41)	96	44 (46)	35	10 (29)	$\chi^2 = 2.48$ $p = 0.12$
Lower gastrointestinal	70	27 (39)	54	25 (46)	16	2 (13)	$\chi^2 = 4.61$ $p < 0.05$
Vascular	57	24 (42)	20	12 (12)	37	12 (32)	$\chi^2 = 3.00$ $p = 0.08$
Other	5	2 (20)	4	2 (40)	1	0 (0)	$\chi^2 = 0.70$ $p = 0.40$
Total	263	104 (40)	174	83 (48)	89	24 (25)	

Table 3 Predominant cause of increased IAP (%)

Combination of factors	45
Tissue edema	24
Intra-abdominal sepsis	15
Ileus/bowel obstruction	9
Hemorrhage/hematoma	6
Fluid collection/ascites	1

Table 4 The odds ratios for the occurrence of increased IAP and complications in patients with a pHi < 7.32

Condition	Odds ratio	95%CI	Π^2 df = 1
IAP ≥ 20 mmHg	11.3	3.2–43.5	17.5 $p < 0.01$
MAP < 80 mmHg	6.4	1.5–32.2	6.8 $p < 0.01$
Sepsis	8.7	0.97–198.9	3.7 $p = 0.06$
Renal impairment	63.7	7.6–1396.9	28.3 $p < 0.01$
Re-laparotomy	5.8	1.0–42.9	4.1 $p = 0.04$
Death	18.0	2.2–394.9	9.7 $p < 0.01$

pHi; *MAP* mean arterial pressure

support until the focus of bleeding, sepsis, or obstruction has been dealt with. Not all patients require surgical exploration, and often percutaneous drainage of an abscess or ascites will be all that is required.

Future

Awareness of the likelihood that approximately 5% of acute general surgical patients will develop IAH leading to ACS will allow surgeons and intensivists to make informed decisions about patient care. Pressure readings, although important, cannot be considered in isolation but should involve the concept of perfusion. Little work has been done to date on APP, but it appears to be better predictor than IAP of death and sepsis. Measurement of visceral and regional abdominal blood flow have shown some promise, but techniques such as gastric tonometry, while predictive of sepsis in general surgery patients, were cumbersome to perform and fell out of favor. A strong correlation between pHi as measured with a gastric tonometry and IAP, is shown in Table 4 [35].

In conclusion more than one third of patients undergoing acute abdominal general surgery will develop IAH, and one third of those will develop ACS. At present, only a third of surgeons routinely measure IAP. We can surely improve on this.

References

1. Fietsam R Jr, Villalba M, Glover JL et al (1989) Intra-abdominal compartment syndrome as a complication of ruptured abdominal aortic aneurysm repair. Am Surg 55:396–402
2. Wendt E (1867) Ueber den einfluss des intraabdominalen Druckes auf die absonderungsgechwindigkeit des harnes. Arch Physiol Heilk 52:7–575
3. Malbrain ML, Cheatham ML, Kirkpatrick A 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
4. Malbrain ML, Chiumello D, Pelosi P et al (2004) Prevalence of intra-abdominal hypertension in critically ill patients: a multicentre epidemiological study. Intensive Care Med 30:822–829
5. Cheatham ML, Malbrain ML (2007) Cardiovascular implications of abdominal compartment syndrome. Acta Clin Belg Suppl 1:98–112
6. Oda J, Yamashita K, Inoue T et al (2007) Acute lung injury and multiple organ dysfunction syndrome secondary to intra-abdominal hypertension and abdominal decompression in extensively burned patients. J Trauma 62:1365–1369
7. Quintel M, Pelosi P, Caironi P et al (2004) An increase of abdominal pressure increases pulmonary edema in oleic acid-induced lung injury. Am J Respir Crit Care Med 169:534–541
8. Sukhotnik I, Bejar J, Srugo I et al (2006) Adverse effects of increased intra-abdominal pressure on small bowel structure and bacterial translocation in the rat. J Laparoendosc Adv Surg Tech A 16:404–410
9. Moore-Olufemi SD, Xue H, Allen SJ et al (2005) Effects of primary and secondary intra-abdominal hypertension on mesenteric lymph

flow: implications for the abdominal compartment syndrome. Shock 23:571–575
10. Sugrue M, Jones F, Deane SA et al (1999) Intra-abdominal hypertension is an independent cause of postoperative renal impairment. Arch Surg 134:1082–1085
11. Kirkpatrick AW, Colistro R, Laupland KB et al (2007) Renal arterial resistive index response to intraabdominal hypertension in a porcine model. Crit Care Med 35:207–213
12. Scalea TM, Bochicchio GV, Habashi N et al (2007) Increased intra-abdominal, intrathoracic, and intracranial pressure after severe brain injury: multiple compartment syndrome. J Trauma 62:647–656
13. Sugrue M (2002) Intra-abdominal pressure: time for clinical practice guidelines? Intensive Care Med 28:389–391
14. Cheatham ML, Malbrain ML, Kirkpatrick A et al (2007) Results from the International Conference of Experts on Intra-abdominal Hypertension and Abdominal Compartment Syndrome. II. Recommendations. Intensive Care Med 33:951–962
15. Kimball EJ, Rollins MD, Mone MC et al (2006) Survey of intensive care physicians on the recognition and management of intra-abdominal hypertension and abdominal compartment syndrome. Crit Care Med 34:2340–2348
16. Kirkpatrick AW, Laupland KB, Karmali S et al (2006) Spill your guts! Perceptions of Trauma Association of Canada member surgeons regarding the open abdomen and the abdominal compartment syndrome. J Trauma 60:279–286
17. Ravishankar N, Hunter J (2005) Measurement of intra-abdominal pressure in intensive care units in the United Kingdom: a national postal questionnaire study. Br J Anaesth 94:763–766
18. De Laet IE, Hoste EA, De Waele JJ (2007) Survey on the perception and the management of the abdominal compartment syndrome among Belgian surgeons. ACAT Chir Belg 107:648–652
19. Donders F (1854) Deutsch Arch Klin Med 4:230–239
20. Emerson H (1911) Intra-abdominal pressures. Arch Intern Med 7:754–784
21. Kron IL, Harmon PK, Nolan SP (1984) The measurement of intra-abdominal pressure as a criterion for abdominal re-exploration. Ann Surg 196:594–597
22. Harmon KP, Kron IL, McLachlan DH et al (1982) Elevated intra-abdominal pressure and renal function. Ann Surg 196:594–597
23. Eleftheriadis E, Kotzampassi K, Botsios D et al (1996) Splanchnic ischaemia during laparoscopic cholecystectomy. Surg Endosc 10:324–326
24. Sugrue M, Buist MD, Hourihan F et al (1995) Prospective study of intra-abdominal hypertension and renal function after laparotomy. Br J Surg 82:235–238
25. Bishara B, Karram T, Khatib S et al (2007) Impact of pneumoperitoneum on renal perfusion and excretory function: beneficial effect of nitroglycerine. Surg Endosc 23:568–576
26. Cheatham ML, White MW, Sagraves SG et al (2000) Abdominal perfusion pressure; a superior parameter in assessment of intra-abdominal hypertension. J Trauma 49:621–627
27. Balogh Z, Jones F, D'Amours S et al (2004) Continuous intra-abdominal pressure measurement technique. Am J Surg 188:679–684
28. Basu A, Pai DR (2008) Early elevation of intra-abdominal pressure after laparotomy for secondary peritonitis: a predictor of relaparotomy? World J Surg 32:1851–1856
29. Daugherty EL, Liang H, Taichman D et al (2007) Abdominal compartment syndrome is common in medical intensive care unit patients receiving large-volume resuscitation. J Intensive Care Med 2007 22:294–299
30. Balogh Z, McKinley BA, Holcomb JB et al (2003) Both primary and secondary abdominal compartment syndrome can be predicted early and are harbingers of multiple organ failure. J Trauma 54: 848–859
31. Ivatury RR, Porter JM, Simon RJ et al (1998) Intra-abdominal hypertension after life-threatening penetrating abdominal trauma: prophylaxis, incidence, and clinical relevance to gastric mucosal pH. J Trauma 44:1016–1021
32. Balogh Z, Caldwell E, Heetveld M et al (2005) Institutional practice guidelines on management of pelvic fracture-related hemodynamic instability: do they make a difference? J Trauma 58:778–782
33. Diebel LN, Dulchavsky SA, Wilson RF (1992) Effect of increased intra-abdominal pressure on mesenteric arterial and intestinal mucosal blood flow. J Trauma 33:45–49
34. Ploat C, Arikan Y, Vatansev C et al (2002) The effect of increased intraabdominal pressure on colonic anastomosis. Surg Endosc 16:1314–1319
35. Sugrue M, Jones F, Lee A et al (1996) Intra-abdominal pressure and gastric intramucosal pH: is there an association? World J Surg 20:988–991