Recognition and management of intra-abdominal hypertension and the abdominal compartment syndrome

A Ben Cresswell

Abstract

Intra-abdominal pressure (IAP) can be elevated by a variety of surgical and medical causes. Abnormally high IAPs can lead to multi-system organ dysfunction due to a combination of direct pressure effects and the release of endotoxins which can be life threatening.

The key to the successful management of the condition relies on a low threshold for making the diagnosis, an ability to accurately measure and interpret the IAP and the prompt management by appropriate techniques that may be surgical, non-surgical or both.

Despite an increased interest and recognition of the importance of IAP measurement amongst modern clinicians, correct knowledge of the consensus definitions and recommendations for management remains poor and this article will describe a practical approach to assessing and managing affected patients with a final description of techniques for reconstructing complex abdominal wall defects.

Keywords Abdominal compartment syndrome; critical care; intra-abdominal hypertension; intra-abdominal pressure; laparostomy

What is intra-abdominal pressure?

The term intra-abdominal pressure (IAP) refers to the dynamic pressure generated within the closed compartment of the abdomen and pelvis, and is a function of the interaction between the pressure exerted by the abdominal contents and the mechanical properties of the abdominal wall itself. The contents of the abdomen can be divided into those which are absolute, that is, the minimum volume occupied by the abdominal viscera and associated tissues and those which are variable – by either gaseous distension or the accumulation of fluid in the form of visceral oedema, ileus or ascites. It is alterations in these variable components, acting against the semi-rigid constraints of the abdominal wall (the compliance of which may vary with pathology), which leads to the problem of an acute rise in IAP during a critical illness.

The concept of the anatomical compartment is well-established in terms of intracranial[1] and muscle compartment[2] monitoring. Here, an increase in the volume of the compartment contents results in a measurable increase in intra-compartmental pressure, which can lead to organ damage. The situation is relatively straightforward in these cases because the compartment walls, bone in the case of the skull and rigid fascia in the case of muscle compartments, represent a fixed and non-distensible vessel. An expansion within the contents can lead to a reduction in blood flow and a direct pressure effect within the compartment which is both clinically evident, in terms of reducing conscious level or limb pain and measurable using intracranial or intra-fascial pressure transducers. The situation within the abdomen is similar in some respects but rather more complex.

The abdominal compartment is enclosed by both non-distensible (the bony pelvis, the posterior wall and the intra-thoracic portion) and distensible (the lateral and anterior abdominal wall) components, the contents of which are homogenous and contain fluid, gas and the abdominal viscera (including the retroperitoneal structures). End-organ function in this context is much more difficult to define and elevated pressures can have variable effects on the cardiovascular, respiratory, renal, gastrointestinal (GI) and endocrine systems – which may be related to the direct transmission of pressure, more indirectly by the release of endotoxins or by a combination of both. In this context the various organ systems, although all demonstrably affected by raised IAP, vary in their actual susceptibility and defining critical levels for each system is difficult. Early signs of abdominal compartment syndrome (ACS) include oliguria, acidosis, hypotension, rising ventilatory pressures and hypoxia. If left untreated, acute kidney and lung injuries may develop and visceral ischaemia can result in bowel infarction and ischaemic hepatitis/acute liver failure. Pressure may also be transmitted via the chest to the central nervous system and rising intracranial pressure secondary to ACS is an ominous sign.

Causes of raised IAP are numerous, and can be categorized as either primary (associated with injury or disease in the abdominopelvic region), secondary (associated with conditions that do not originate from the abdomen such as sepsis, burns and massive fluid resuscitation) and tertiary, or recurrent ACS developing after treatment for primary or secondary ACS (Table 1). As such, individual causes are in no way confined to surgical or traumatic injuries and diseases, so practically all critically ill patients remain at risk of developing this condition and a large multi-centre epidemiological study has shown a prevalence of intra-abdominal hypertension (IAH) within the general ITU population of around 50%, with 8% of patients fulfilling pressure criteria for ACS.[3]

ACS as a surgical entity, was first noted by military surgeons, and Gross in 1940,[4] was reported as describing a '… continual battle, often somewhat brutal, while try to pack intestinal loops into a cavity which was too small to receive them'. Consensus definitions for the condition have only been agreed as recently as 2006, however, following the inaugural meeting of the World Society of the Abdominal Compartment Syndrome (WSACS)[5] (Table 2). Normal IAP is considered to be less than 12 mmHg, a pressure of 12–20 mmHg corresponds to IAH and greater than 20 mmHg in the context of new organ dysfunction is known as ACS.

The key to the early diagnosis of raised IAP is clearly a high level of clinical suspicion coupled with the prompt and accurate pressure measurement and such a proactive approach has been shown to significantly improve outcome.[6]

A Ben Cresswell *MD(Res) FRCS (Gen Surg) is a Consultant Hepatopancreatobiliary Surgeon at Hampshire Hospitals NHS Foundation Trust and the Basingstoke Hepatobiliary Unit, UK.*
Conflict of interest: none declared.

Please cite this article in press as: Ben Cresswell A, Recognition and management of intra-abdominal hypertension and the abdominal compartment syndrome, Surgery (2016), http://dx.doi.org/10.1016/j.mpsur.2016.08.007

Causes of raised intra-abdominal pressure

Type of ACS	Underlying causes
Primary	• Pancreatitis • Intra-abdominal sepsis/abscess/peritonitis • Abdominal trauma • Abdominal aortic aneurysm • Peritoneal/retro-peritoneal haematoma • GI obstruction/pseudo-obstruction • Gastroparesis • Volvulus • Pneumoperitoneum • Major abdominal surgery (acute or elective) • Liver dysfunction/ascites
Secondary	• Acidosis • Hypothermia • Massive transfusion • Coagulopathy • Sepsis • Mechanical ventilation • Use of PEEP • Pneumonia • Major burns • Major trauma • High BMI • Prone positioning
Tertiary	• Recurrent ACS despite previous decompression

ACS, abdominal compartment syndrome; BMI, body mass index; PEEP, positive end-expiratory pressure.

Table 1

WSACS consensus definitions on IAP[5]

Term	Definition
IAP	The steady-state pressure concealed within the abdominal cavity. IAP should be expressed in mmHg and measured at end expiration in the completely supine position
Indirect IAP measurement	The reference standard for intermittent IAP measurement is via the bladder
IAH	IAH is defined by a sustained or repeated pathologic elevation of IAP greater than 12 mmHg
ACS	ACS is defined as a sustained IAP greater than 20 mmHg that is associated with new organ failure or dysfunction
Primary ACS	A condition associated with injury or disease in the abdomino-pelvic region
Secondary ACS	ACS secondary to conditions that do not originate from the abdomino-pelvic region
Indication for IAP monitoring	• if two or more causes for IAH/ACS present, baseline IAP measurement should be obtained and • if IAH is present, serial IAP measurements should be performed throughout the patient's critical illness

ACS, abdominal compartment syndrome; IAH, intra-abdominal hypertension; IAP, intra-abdominal pressure; WSACS, World Society of the Abdominal Compartment Syndrome.

Table 2

Measuring IAP

Numerous techniques exist for the measurement of IAP, which can be achieved either by directly inserting a catheter to measure intra-peritoneal pressure, or by indirectly measuring the pressure within the stomach or urinary bladder. For most clinical settings, the measurement of intra-vesical (bladder) pressure is considered the gold standard. Measurements can be performed cheaply using some standard ITU consumables, which should be widely available on all units, or by the use of one of the specialized devices that have been developed for the purpose — both approaches are described below. Irrespective of the technique chosen — the key is attention to detail for the accurate measurement of pressure and then serial recordings to guide management.

Measurement of IAP using ITU consumables

This technique was first described by Kron in 1984[7] and subsequently modified to involve the use of an electronic pressure transducer — such as that used to measure central venous pressure or arterial pressure — to measure the pressure within the urinary bladder, via a continuous column of fluid via the urinary catheter (Figure 1).

Step 1. Prepare the equipment you will need and ensure that you have assistance.

- A sterile dressing pack — containing a towel and some sterile gloves and an appropriate trolley.
- An alcohol wipe.
- An electronic pressure transducer with a length of plastic tubing connected to a green (21G) hypodermic needle.
- A pressurized bag of saline, to 'run-through' the transducer and tubing.
- A clamp to occlude the urinary catheter.
- A 'bladder-tip' 50-ml syringe containing sterile saline.

Step 2. Prepare the patient and the equipment.

- Patients who are awake, should have the procedure explained to them and be reassured that they should experience no discomfort or distress during the measurement process. They must be positioned supine whilst the measurement is taken and the process should take no longer than 5 minutes to perform.
- The pressure transducer should be connected to the standard ITU monitor and be 'run-through' with sterile saline (the ITU staff will be able to help you with this if you are unfamiliar with the procedure).
- The transducer should be mounted at the height of the patient's anterior—superior iliac spine in the mid-axillary line and zeroed to air at this level.

Please cite this article in press as: Ben Cresswell A, Recognition and management of intra-abdominal hypertension and the abdominal compartment syndrome, Surgery (2016), http://dx.doi.org/10.1016/j.mpsur.2016.08.007