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Review

The cardio-respiratory effects of intra-abdominal hypertension: Considerations for critical care nursing practice

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ABSTRACT

Intra-abdominal hypertension can be classified as either primary or secondary. Primary intra-abdominal hypertension is often associated through trauma or diseases of the abdominopelvic region such as pancreatitis or abdominal surgery, while secondary intra-abdominal hypertension is the result of extra-abdominal causes such as sepsis or burns. The critically ill patient offers some challenges in monitoring in particular secondaryintra-abdominal hypertension because of the effects of fluid resuscitation, the use of inotropes and positive pressure ventilation. Recent work suggests that intensive care unit nurses are often unaware of the secondary effects of intra-abdominal pressure and therefore this is not monitored effectively. Therefore being aware of the cardio-respiratory effects may alert theintensive care nurse nurse to the development of intra-abdominal hypertension. The aim of this paper is to discuss the pathophysiology associated with the cardio-respiratory effects seen with intra-abdominal hypertension in the critically ill. In particular it will discuss how intra-abdominal hypertension can inadvertently be overlooked because of the low flow states that it produces which could be misconstrued as something else. It will also discuss how intra-abdominal hypertension impedes ventilation and respiratory mechanics which can often result in a non-cardiogenic pulmonary oedema. To close, the paper will offer some implications for critical care nursing practice.

Implications for clinical practice

- It is common for the overweight or obese patient to have undiagnosed chronic intra-abdominal hypertension.
- Secondary intra-abdominal hypertension has a high mortality rate, is insidious in its development and is often missed in routine nursing assessment especially in the over-weight and obese critically ill patient.
- The most common early sign of intra-abdominal hypertension is oliguria, despite mean arterial pressure being within normal limits or an elevated central venous pressure.
- The application of positive end expiratory pressure can raise intra-abdominal pressure by as much as 25% especially in those patients with a body mass index of greater than 30.
- Routine monitoring of intra-abdominal pressure in the overweight or obese patient should be considered as part of the normal routine nursing assessment.

Introduction

Sustained intra-abdominal hypertension (IAH) can lead to abdominal compartment syndrome, a condition which if left untreated can lead to renal and cardiorespiratory complications and iscahemic bowel. Defined as a steady-state pressure within the abdominal cavity, intra-abdominal pressure (IAP) is governed by the elasticity of the abdominal wall and by the characteristics of the abdominal contents (Malbrain et al., 2006). Whereas IAH is

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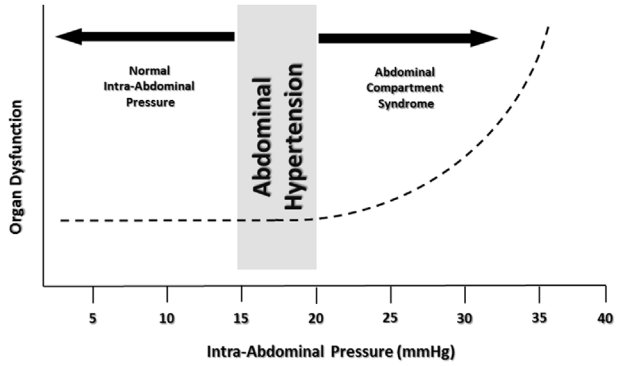

Fig. 1. Normal Intra-Abdominal pressure versus Abdominal Hypertension (Malbrain et al., 2006; 1777).

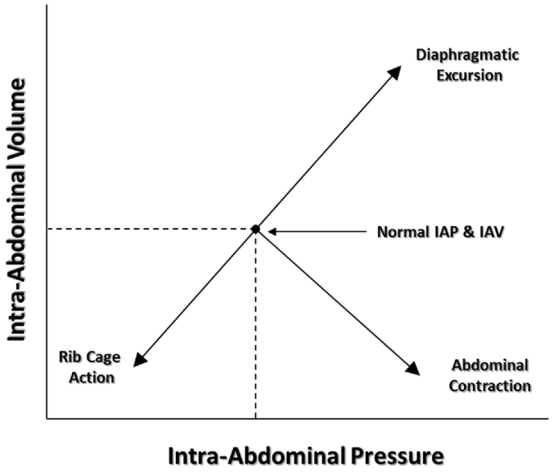

Fig. 2. Relationship between Intra-Abdominal Pressure and Volume (De Keulenaer et al., 2009).

defined as: "*a sustained or repeated pathophysiological elevation in IAP* ≥ 12 mmHg" (Malbrain et al., 2006; p1724). IAP can be classified according to the extent of intra-abdominal involvement and the degree of organ dysfunction ranging from mild IAH (Grade I: IAP 12–15 mmHg) to severe (Grade IV: IAP >25 mmHg) (Papavramidis et al., 2011). However, Kirkpatrick et al. (2013) suggests that this pressure can be as low as 12 mmHg. This differs in the critically ill were IAH has been found to be 5–7 mmHg (Fig. 1) (Malbrain et al., 2006).

Normal intra-abdominal pressure – fluid dynamics

Normal intra-abdominal pressure (IAP) is generally considered to be within the range of 0–7 mmHg (mean 5 mmHg ± 2.9; 0–9 cmH_2O) (Sanchez et al., 2001). What determines IAP has generated considerable debate, the most reasonable explanation is following Pascal's law of fluid mechanics in relation to the transmission of fluid-pressure. Pascal states that any change in pressure in an enclosed fluid is transmitted equally and undiminished to every part of the fluid (Walker, 2016). Therefore, with the abdomen considered an enclosed fluid filled container any change in pressure will be equally distributed through-out the abdomen (Malbrain and De Waele, 2013). However, earlier work reported that abdominal pressures fluctuate as a result of diaphragmatic flattening and gravitational forces suggesting that intra-abdominal pressure may be more dependent on the displacement of abdominal contents (Decramer et al., 1984) (Fig. 2). In measuring intra-abdominal pressure in the dog model Loring et al. (1994) found normal IAP is reliant on three factors: gravity, uniform compression and shear deformation.

Normally the abdominal cavity contains fluid that moves freely around and supports the viscera (Fig. 3a). But as the abdominal contents shift with patient positioning to the upright position, the weight of the contents significantly increases intra-abdominal pressure in the lower abdominal quadrants (Fig. 3b). Loring et al. (1994) demonstrated a positive correlation between the distance and gravitational pressure gradients measured at the xiphoid process and the bladder. The pressure gradients showed an average pressure increase of 0.90 cmH_2O/centimetre (cm) (0.66 mmHg/cm) away from the xiphoid process in the up-right position (0.90 ± 0.09 cmH_2O/cm; $R \geq 0.966$). The lateral, prone and supine positions did show some gravitational changes but not to the same extent.

Uniform compression which can be described as diaphragmatic and rib cage flattening as well as abdominal contraction for which the abdominal contents are compressed (Fig. 3c). Unlike pure gravitational pressures, uniform compression is spatially homogenous throughout the abdomen with fluctuations dependent on diaphragmatic and abdominal muscle contraction. An example of this is the initiation of the vomiting reflex where the abdominal viscera are compressed between the descending flattened diaphragm and the contracting abdominal muscles. However uniform compression can be superimposed on gravitational pressures and patient positioning. For example patient positioning can influence IAP from between 6 mmHg (lateral position) to 16 mmHg (semi-Fowlers position). In addition, a high body mass index (BMI) will result in abdominal compression with an increase in IAP by 8–13 mmHg respectively Sanchez et al. (2001). One possible explanation is the weight of the adipose tissue having a direct compression effect on intra-abdominal contents (Fig. 2) (Frezza et al., 2007).

Shear deformation differs again from gravitational forces and uniform compression in as much that this pressure is often associated with deformation of the tissue itself as would be seen with intestinal oedema for example. The effect of intestinal oedema is not uniform throughout the abdomen and as such there may well be variations in abdominal pressure gradients as oedematous tissue displaces or compresses less oedematous tissue surrounding it (Loring et al., 1994). This is different from conditions such as abdominal ascites where the pressures generated are as a result of the excess fluid in the abdomen acting more homogenously throughout (uniform compression) and as such the IAP can remain constant.

Pathophysiology of IAH

Blood supply to the abdominal organs is extremely complex. Approximately 25% of the systemic blood volume resides here and it receives almost 30% of the cardiac output (1500 mls/min), with 30% of this volume going directly to the liver (Gallacher et al., 1999). The three main arterial systems that supply blood to the gut are the coeliac (CA), the superior mesenteric (SMA) and the inferior mesenteric arteries (IMA). The CA supplies blood to the stomach, spleen and the pancreas, the SMA to the small intestine and IMA to the large intestine. Venous outflow from these areas are directed towards the portal vein, the liver, the hepatic vein and then onto the inferior vena cava (Craft et al., 2015).

The pathophysiology of IAH is the result of either a physiologic insult such as complex abdominal surgery to critical illness for example pancreatitis or sepsis (Cheatham, 2009). This results in a complex interplay of inflammatory mediators which increase capillary permeability leading to tissue engorgement, oedema and the formation of abdominal ascites (Spencer et al., 2008). There are a number of identified risk factors that often result in IAH (Table 1)

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