

INTRODUCTION

Intra-abdominal hypertension (IAH) is defined as sustained or repeated pathological elevation in intra abdominal pressure (IAP \geq 12 mmHg) and abdominal compartment syndrome (ACS) sustained IAP \geq 20 mmHg associated with a new organ dysfunction or failure (*Scheppach, 2009*).

The mortality rate of intra-abdominal hypertension and abdominal compartment syndrome is high. Increase in intra-abdominal pressure causes significant impairment of almost all organ systems. Even slight increase in intra-abdominal pressure has negative influence on the respiratory, cardiovascular, cerebral, gastrointestinal, hepatic, and renal functions. Intra-abdominal hypertension causes visceral organ hypoperfusion, intestinal ischemia and may also lead to bacterial translocation, release of cytokines and production of free oxygen radicals. All these factors may contribute to the development of multiple organ failure in the critically ill patients (*Serpytis and Ivaskevicius, 2005*).

Abdominal compartment syndrome can be divided into the following three categories:

- Primary or acute abdominal compartment syndrome: this occurs when intra-abdominal pathology is directly responsible for the compartment syndrome.

- Secondary abdominal compartment syndrome: This occurs when no visible intra-abdominal injury is present but injuries outside the abdomen causing fluid accumulation.
- Chronic abdominal compartment syndrome: This occurs in the presence of cirrhosis and ascites, often in the later stage of the disease (*Paula, 2009*).

Increased intra-abdominal pressure (IAP) has received growing attention in critically ill patients. Pathophysiologically, it deranges cardiovascular haemodynamics, respiratory and renal functions and may eventually lead to multi-organ failure. It is primarily seen in surgical intensive care units and is frequently associated with abdominal trauma but also occurs after elective abdominal surgery. Non-surgical intensivists ought to be aware that the syndrome is also seen in a wide spectrum of medical conditions, e.g. acute pancreatitis (*Scheppach, 2009*).

The abdominal compartment should be suspected and sought for in any multiple trauma patient who has undergone a period of profound shock. Clinically it is characterized by a fall in urine output associated with an elevated central venous pressure. The diagnosis can be confirmed by the measurement of intra-abdominal pressure. This may be done either through a foley catheter in the bladder or a nasogastric tube in the stomach. Simple water-column manometry is used at 2 to 4 hourly intervals, although it is possible to connect a pressure transducer to a foley catheter (*Scheppach, 2009*).

Intravascular fluid replacement and abdominal decompression are the standards of treatment for abdominal compartment syndrome (*Serpytis and Ivaskevicius, 2005*).

The indications for surgical decompression of abdominal compartment syndrome (ACS) are not clearly defined, but undoubtedly some patients benefit from it. In patients without recent abdominal incisions, it can be achieved with full-thickness laparostomy (either midline, or transverse subcostal) or through a subcutaneous linea alba fasciotomy. In spite of the improvement in physiological variables and significant decrease in IAP, however, the effects of surgical decompression on organ function and outcome are less clear. Because of the significant morbidity associated with surgical decompression and the management of the ensuing open abdomen, more research is needed to better define the appropriate indications and techniques for surgical intervention (*Leppäniemi, 2009*).

AIM OF THE WORK

The aim of this work is to highlight the current and most recent trends in management of the abdominal compartment syndrome.

Chapter I

ANATOMY OF THE ABDOMINAL COMPARTMENTS AND SPACES

The abdominal compartment syndrome we should briefly review the anatomy to make it easy to understand the problem and how to deal with.

The abdomen is the largest cavity in the body. It is oval in shape; the extremities of the oval cavity are being directed upward and backward. The upper extremity is the diaphragm which extends as a dome over the abdomen, so that the cavity extends high into the bony thorax, reaching on the right side on the mammary line to the upper border of the 5th rib; on the left side however it falls below this level by about 2.5cm. The lower extremity is formed by structures which clothe the inner surface of the bony pelvis, principally the levatorani and the coccygeus on either side. These muscles are sometimes termed the diaphragm of the pelvis. The cavity is wider above than below, and measures more in vertical than in transverse diameter. In order to facilitate the description, it is artificially divided in two parts, the upper and larger part called the abdomen proper and the smaller part called pelvis. These two cavities are not separated from each other but the limit between them is the superior aperture of the lesser pelvis (*Dixon and Birmingham, 2007*).

The abdomen proper differs from the other great cavities of the body in being bounded for the most part by muscles and

fasciae, so that it can vary in capacity and shape according to the condition of the viscera which it contains. In addition to this, the abdomen varies in form and extent with age and sex. In the adult male, with moderate distension of the viscera, it is oval in shape, but at the same time flattened from before backward. In the adult female, with a fully developed pelvis, it is ovoid with the narrower pole upward, and in young children it is also ovoid but with the narrower pole downward (*Gray et al., 2000*).

Boundaries: it is bounded in front and at the sides by the abdominal muscles and the iliacus muscles, behind by the vertebral column and the Psoas and Quadratuslumborum muscles, above by the diaphragm and below by the plane of the superior aperture of the lesser pelvis. The muscles forming the boundaries of the cavity are lined upon their inner surfaces by a layer of fascia (*Gray et al., 2000*).

The abdomen contains the greater part of the digestive tube; some of the accessory organs to digestion such as the liver and pancreas. Other organs include the spleen, the kidneys, and the suprarenal glands. Most of these structures, as well as the wall of the cavity in which they are contained, are more or less covered by an extensive and complicated serous membrane, the peritoneum (*Gray et al., 2000*).

The apertures in the walls of the abdomen: the apertures in the walls of the abdomen, for the transmission of structures to or from it, are, in front, the umbilical (in the fetus), for the transmission of the umbilical vessels, the allantois, and vitelline

duct; above, the vena caval opening, for the transmission of the inferior vena cava, the aortic hiatus, for the passage of the aorta, azygos vein, and thoracic duct, and the esophageal hiatus, for the esophagus and vagi. Below, there are two apertures on either side: one for the passage of the femoral vessels and lumboinguinal nerve (illioinguinal nerve), and the other for the transmission of the spermatic cord in the male, and the round ligament of the uterus in the female (*Gray et al., 2000*).

ABDOMINAL VISCERA (ANTERIOR VIEW)

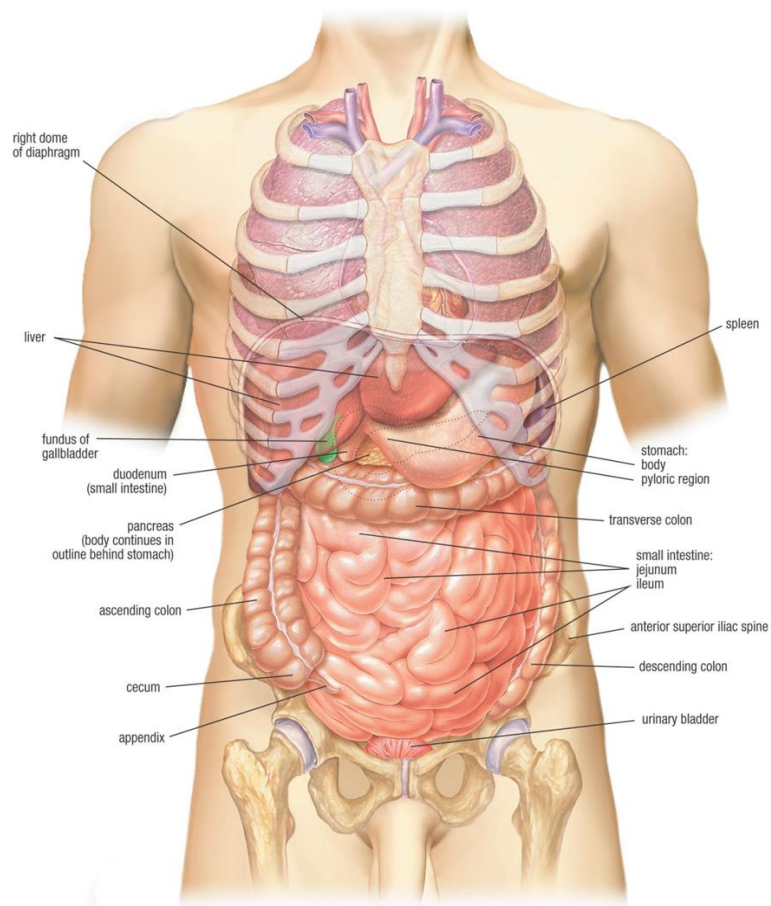


Figure (1): Abdominal viscera (*Gray et al., 2000*).

When the anterior abdominal wall is removed, the viscera are partly exposed as follows: above and to the right side is the liver, situated chiefly under the shelter of the right ribs and their cartilages, but extending across the middle line and reaching for some distance below the level of the xiphoid process. To the left of the liver is the stomach, from the lower border of which an apron-like fold of peritoneum, the greater omentum, descends for a varying distance, and obscures to a greater or lesser extent, the other viscera. Below it, however, some of the coils of the small intestine can generally be seen, while in the right and left iliac regions respectively the caecum and the iliac colon are partly exposed. The bladder occupies the anterior part of the pelvis, and, if distended, will project above the symphysis pubis; the rectum lies in the concavity of the sacrum, but is usually obscured by the coils of the small intestine. The sigmoid colon lies between the rectum and the bladder (*MacKenzie and Basmajian, 2004*).

When the stomach is followed from left to right it is seen to be continuous with the first part of the small intestine, or duodenum, the point of continuity being marked by a thickened ring which indicates the position of the pyloric valve. The duodenum passes toward the under surface of the liver, and then, curving downward, is lost to sight (*MacKenzie and Basmajian, 2004*).

If the greater omentum be thrown upward over the chest, the inferior part of the duodenum will be observed passing

across the vertebral column toward the left side, where it becomes continuous with the coils of the jejunum and ileum.

These measure some 6 meters in length, and if followed downward the ileum will be seen to end in the right iliac fossa by opening into the cecum, the commencement of the large intestine. From the cecum the large intestine takes an arched course, passing at first upward on the right side, then across the middle line and downward on the left side, and forming respectively the ascending transverse, and descending parts of the colon. In the pelvis it assumes the form of a loop, the sigmoid colon, and ends in the rectum.

The spleen lies behind the stomach in the left hypochondriac region, and may be in part exposed by pulling the stomach over toward the right side (*MacKenzie and Basmajian, 2004*).

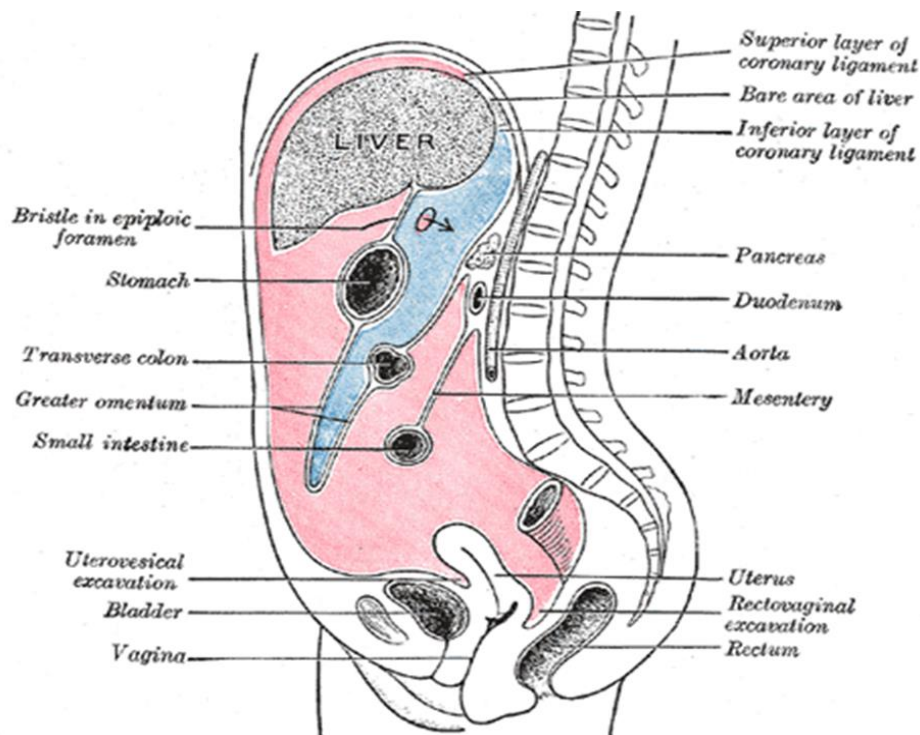


Figure (2): Peritoneal cavity (*Gray et al., 2000*).

The endothelial lining of the primitive coelomic cavity of the embryo becomes the thoracic pleura and the abdominal peritoneum. Each is invaginated by the ingrowing viscera which thus become covered by a serous membrane and to be packed snugly into a serous-lined cavity, the visceral and parietal layer respectively. In the male the peritoneal cavity is completely closed but in the female it is perforated by the openings of the uterine tubes which constitute a possible pathway of infection from the exterior. (*Gray et al., 2000*).

The glistening appearance of the deep surface of the abdominal wall and of the surfaces of the exposed viscera is

due to the fact that the former is lined and the latter are more or less completely covered by a serous membrane, the peritoneum.

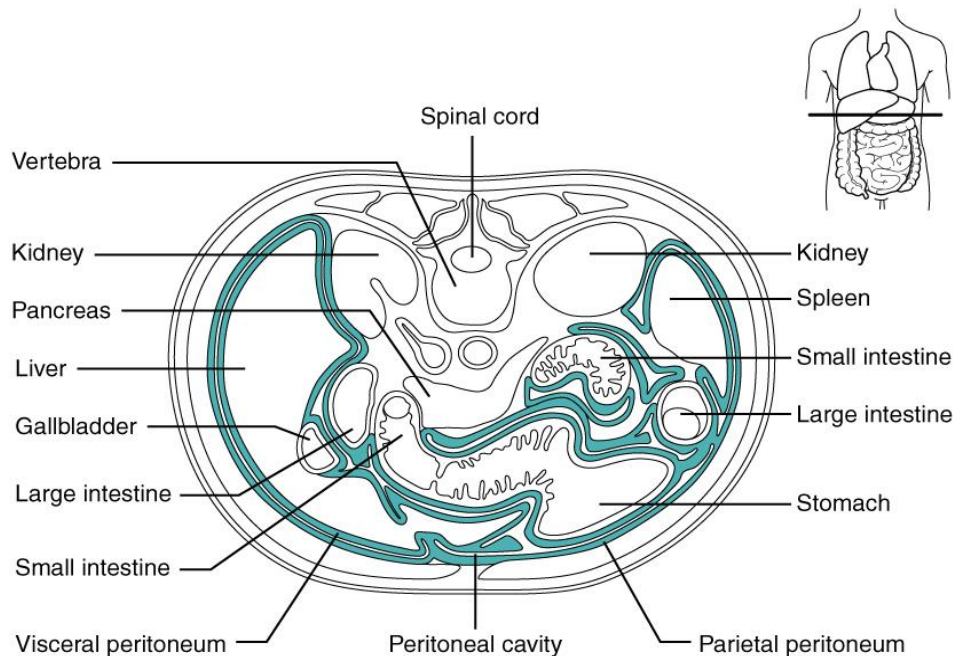


Figure (3): Peritoneal cavity (*Gray et al., 2000*).

The Peritoneum (Tunica Serosa) is the largest serous membrane in the body, and consists in the male of a closed sac, a part of which is applied against the abdominal parietes, while the remainder is reflected over the contained viscera. In the female the peritoneum is not a closed sac, since the free ends of the uterine tubes open directly into the peritoneal cavity. The part which lines the parietes is named the parietal portion of the peritoneum; that which is reflected over the contained viscera constitutes the visceral portion of the peritoneum. The free surface of the membrane is smooth, covered by a layer of flattened mesothelium, and lubricated by a small quantity of serous fluid. Hence the viscera can glide freely against the wall

of the cavity or upon one another with the least possible amount of friction. The attached surface is rough, being connected to the viscera and inner surface of the parietes by means of areolar tissue, termed the subserous areolar tissue. The parietal portion is loosely connected with the fascial lining of the abdomen and pelvis, but is more closely adherent to the under surface of the diaphragm, and also in the middle line of the abdomen (*Guyatt and Watters, 2005*).

The space between the parietal and visceral layers of the peritoneum is named the peritoneal cavity; but under normal conditions this cavity is merely a potential one, since the parietal and visceral layers are in contact. The peritoneal cavity gives off a large diverticulum, the omental bursa, which is situated behind the stomach and adjoining structures; the neck of communication between the cavity and the bursa is termed the epiploic foramen (foramen of Winslow). Formerly the main portion of the cavity was described as the greater sac, and the omental bursa as the lesser sac.

The peritoneum differs from the other serous membranes of the body in presenting a much more complex arrangement, and one that can be clearly understood only by following the changes which take place in the digestive tube during its development (*Guyatt and Watters, 2005*).

Chapter II

PATHOPHYSIOLOGY OF ABDOMINAL COMPARTMENT SYNDROME

Intra-abdominal hypertension (IAH) is defined as sustained or repeated pathological elevation in intra abdominal pressure (IAP \geq 12 mmHg) and abdominal compartment syndrome (ACS) sustained IAP $>$ 20 mmHg associated with a new organ dysfunction or failure (*Scheppach, 2009*)

Etiology:

Any abnormality that elevates the pressure within the abdominal Cavity can induce intra-abdominal hypertension.

1. Blunt abdominal trauma with intra-abdominal bleeding from splenic, hepatic, and mesenteric injuries is the most common cause of intra-abdominal hypertension
2. Bowel distention, as a consequence of hypovolemic shock and massive volume replacement, is an important cause of intra-abdominal hypertension, and subsequent ACS, in trauma patients.
3. Peritoneal tissue edema. Secondary to trauma or peritonitis
4. Retroperitoneal hematoma secondary to rupture aortic aneurysm
5. Tissue injury secondary to surgical procedure

6. Ileus, mechanical obstruction of the bowel, and abdominal growths
7. Surgical placement of abdominal packing to control hemorrhage may also increase pressure within the peritoneal space.
8. Abdominal closure under tension
9. Intra-abdominal tumors
10. Ascites and other forms of intra-abdominal fluid accumulation
11. Acute pancreatitis (*Schein and Ivatury, 2008*).

In shock states, vasoconstriction mediated by the sympathetic nervous system shunts blood away from the skin, muscles, kidneys, and gastrointestinal tract in favor of the heart and brain. Redistribution of blood from the gut produces cellular hypoxia in the tissues of the intestines. This hypoxia is associated with 3 sequels crucial to the development of the positive feedback loop that characterizes the pathogenesis of intra-abdominal hypertension and its progression to ACS;

1. Release of cytokines
2. Formation of oxygen free radicals, and
3. Decreased cellular production of adenosine triphosphate (*Ivatury et al., 2004*).

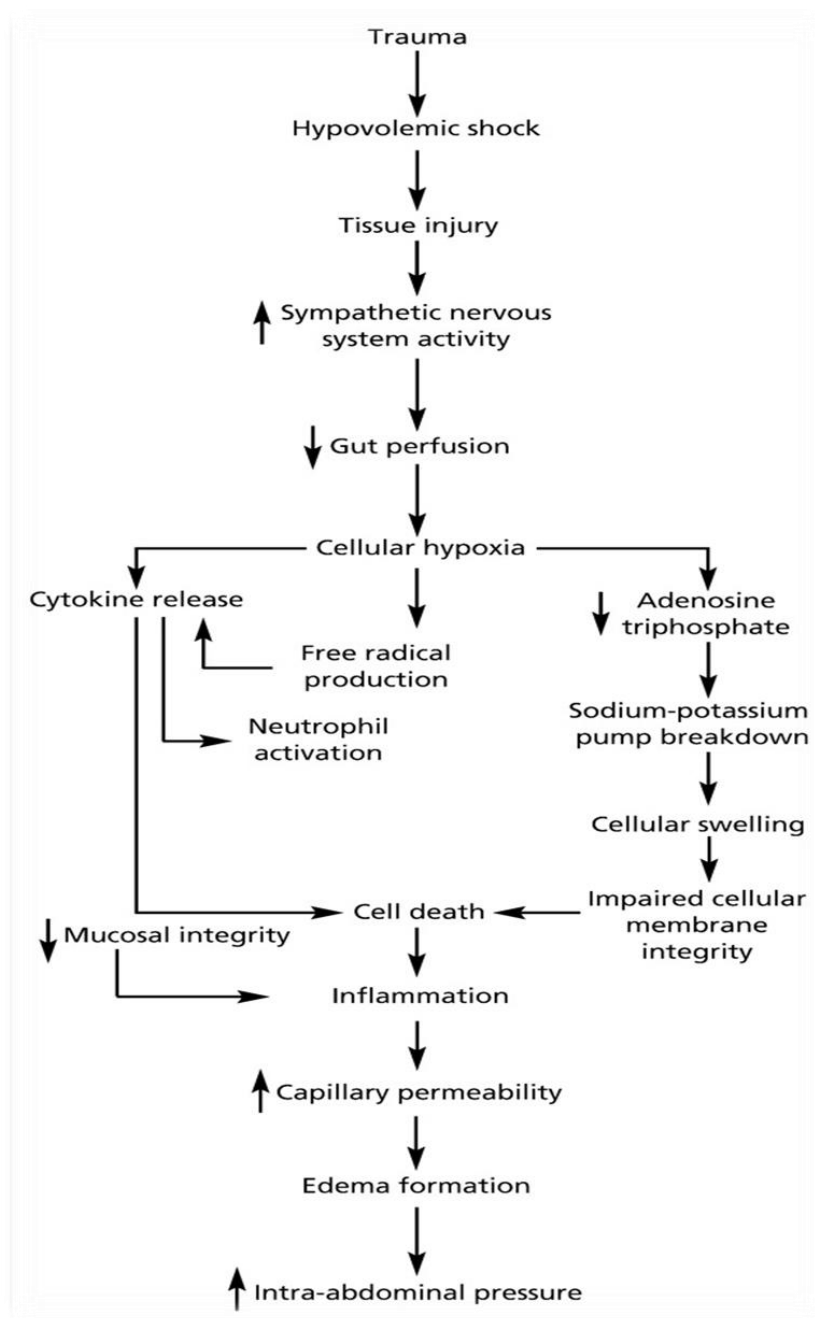


Figure (4): Pathophysiology of ACS (*Saleem and Ahmed, 2006*).