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جامعة عين شمس
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ABDOMINAL COMPARTMENT SYNDROME

Essay
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بِسْمِ اللَّهِ الرَّحْمَٰنِ الرَّحِيمِ

﴿ وَالرَّاسِخُونَ فِي الْعِلْمِ يَقُولُونَ آمَنَّا بِهِ كُلٌّ مِّنْ عِندِ رَبِّنَا وَمَا يَذَّكَّرُ إِلَّا أُولُو الْأَلْبَابِ ﴾

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(آل عمران ٧)

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Waleed Elkafrawy

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LIST OF ABBREVIATIONS

Abbrev.	*MEANING*
ACS	Abdominal Compartment Syndrome
AIDS	Acute Intestinal Distress Syndrome
AP	Anteroposterior
APP	Abdominal Perfusion Pressure
BMI	Body Mass Index
CSF	Cerebrospinal Fluid
CT	Computed Tomographic
CVP	Central Venous Pressure
FIO2	Fractional Inspired Oxygen
GI	Gastrointestinal
IAH	Intra-Abdominal Hypertension
IAP	Intra-Abdominal Pressure
ICP	Intra-Cranial Pressure
ICU	Intensive Care Unit
INR	International Normalized Ratio
ITP	Intra-Thoracic Pressure
IV	Intra-Venous
IVC	Inferior Vena Cava
MAP	Mean Arterial Pressure
NMB	Neuromuscular blockade
NSAID	Nonsteroidal Anti-Inflammatory Drug

OA	Open Abdomen
PaO2	Arterial Oxygen Partial Pressure
PCD	Percutaneous Catheter Drainage
PCWP	Pulmonary Capillary Wedge Pressure
PEEP	Positive End Expiratory Pressure
PH	Power of Hydrogen
PT	Prothrombin Time
PTT	Partial Thromboplastin Time
SLAF	Subcutaneous Linea Alba Fasciotomy
STSG	Spilt-Thickness Skin Graft
VAC	Vacuum Assisted Closure
WSACS	World Society of the Abdominal Compartment Syndrome

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INTRODUCTION

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Intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS) are distinct clinical entities and should not be used interchangeably. Intra-abdominal pressure (IAP) is the steady state pressure concealed within the abdominal cavity. Intra-abdominal hypertension (IAH) is defined by a sustained or repeated pathological elevation in IAP≥12mmHg and abdominal Compartment Syndrome (ACS) is sustained IAP > 20 mmHg associated with organ dysfunction.

The normal range described above is not applicable for all patients. Patients with increased abdominal girth that developed slowly may have higher baseline intra-abdominal pressures. As an example, morbidly obese and pregnant individuals can have chronically elevated intra-abdominal pressure (as high as 10 to 15 mmHg) without adverse squeal **(Malbrain and Cheatham, 2011).**

An increase in intra-abdominal pressure leads to multisystem dysfunction. Pressure within the abdominal cavity is directly transmitted to the abdominal vasculature. Compression of the venous system leads to venous occlusion and a reduction in preload, while arterial compression leads to reduced arterial compliance and an increased after load. This combination leads to a decrease in cardiac output and often, abdominal perfusion pressure **(Pelosi and Vargas, 2012).**

Although initially considered a disease of the traumatically injured, IAH/ACS is now recognized as a cause of significant organ failure, morbidity and mortality in all critically ill patient populations. Given the broad multitude of predisposing conditions that may lead to the development of IAH/ACS.

We believe it is useful to classify ACS as either: primary, secondary, or recurrent according to the duration and cause of the patient's IAH. The duration of IAH, in conjunction with the acuity of onset as described above, is commonly of greater prognostic value than the absolute increase in IAP. Patients with prolonged untreated elevations in IAP commonly manifest inadequate perfusion and subsequent organ failure **(Macedo et al, 2015).**

Intra-abdominal pressure measuring in the past was through nasogastric catheters, inferior vena cava vein or directly intraperitoneally. Recently, the method of choice is used through urinary bladder by sterile procedure **(Rosemary, 2012).**

Clinically, abdominal hypertension with abdominal distention and a tense abdominal wall presents with shallow respiration with an increased respiration rate, high diaphragms on percussion and auscultation, poor urinary output, and increased central venous pressure. Intubated patients require increased ventilator pressure **(Harrell and Melander, 2012).**

Radiological findings done by CT scan were : 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, direct renal compression or displacement, bowel wall thickening with enhancement **(Aashish et al, 2007).**

One of the key aims in management is to optimize cardiac output. Cardiovascular compromise due to the increased intra-abdominal pressure is much more marked in the hypovolemic under-resuscitated patient. Initial fluid resuscitation should be aimed at restoring normovolemia.

Central venous pressure measurement and pulmonary artery wedge pressures may be misleading as a guide to fluid replacement as they may be falsely elevated due to the intra-thoracic pressure increase; however, trends and response to fluid challenges may be of benefit. Markers such as stroke volume variation that are independent of intra-thoracic pressure may be useful guides to fluid resuscitation **(Rubenstein et al, 2015).**

Surgical intervention, the decompression of the abdomen by way of laparotomy has been shown to improve mortality in patients with abdominal compartment syndrome. After laparotomy, the abdominal cavity is simply a larger closed compartment and further increases in volume may still result in an