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Association for  
Acute Cardiovascular Care

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# Handbook of Acute CardioVascular Monitoring

**Quick access to most relevant practical  
information on haemodynamic  
and general monitoring in acute  
cardiovascular care**

Sample chapter on  
**Intra-abdominal hypertension**



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## 8 - INTRA-ABDOMINAL HYPERTENSION

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### Key messages

- ✓ IAP is the steady-state pressure concealed within the abdominal cavity expressed in mmHg
- ✓ The gold standard IAP measurement technique is via the bladder
- ✓ IAH is defined as a sustained pathological elevation in IAP  $\geq 12$  mmHg and it is graded in four categories
- ✓ AbCS is a sustained IAP  $> 20$  mmHg that is associated with new onset organ failure (assessed with sequential organ failure assessment, SOFA score)
- ✓ Elevated IAP plays a major role in the development of multiple system organ dysfunction and failure as a result of significant hypoperfusion and the effects are not limited to the intra-abdominal organs alone
- ✓ Primary IAH/AbCS is a condition associated with injury or disease in the abdominopelvic region
- ✓ Secondary IAH or AbCS refers to conditions that do not originate from the abdominopelvic region, and are frequently associated with damage of the glycocalyx and/or tight junctions following massive fluid resuscitation

- ✓ Basic monitoring includes an accurate IAP measurement every 4 - 6 h in patients with at least 2 or more risk factors for IAH or AbCS
- ✓ Other important clinical parameters are gastric residual volume, urinary output, abdominal perfusion pressure calculated as MAP minus IAP, body weight, daily and cumulative fluid balance
- ✓ Advanced haemodynamic monitoring is required in severe IAH
- ✓ Medical management comes first and decompressive laparotomy should be used as last resort in cases other treatment options fail

## Introduction

Intra-abdominal hypertension is a relatively frequent medical situation in intensive care units which can trigger important complications because it plays a major role in the development of multiple system organ dysfunction. Therefore, intra-abdominal pressure should be considered another first line parameter to be monitored routinely.

## 8.1 - IAH and AbCS definitions

The 2013 the World Society of Abdominal Compartment Syndrome (WSACS, [www.wsacs.org](http://www.wsacs.org)) changed its name to The Abdominal Compartment Society and updated consensus definitions and recommendations on this topic were published.

### 8.1.1 - Intra-abdominal pressure (IAP)

IAP is the steady-state pressure concealed within the abdominal cavity and the reference standard for intermittent IAP measurements is via bladder with a maximal instillation volume of 25 mL of sterile saline (**Figure 1, 2**). IAP should be expressed in mmHg and measured at end-expiration in the supine position, after ensuring that abdominal muscle contractions are absent and with transducer zeroed at the level of midaxillary line. Normal IAP ranges from sub-atmospheric to zero mmHg, however, IAP is commonly elevated to a range of 5-7 mmHg in critically ill adults.

**Figure 1 - IAP measurement**

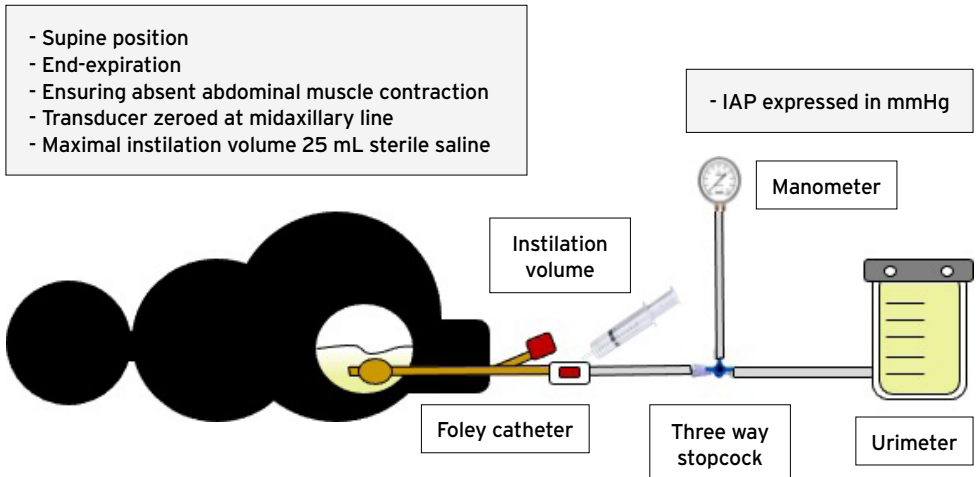
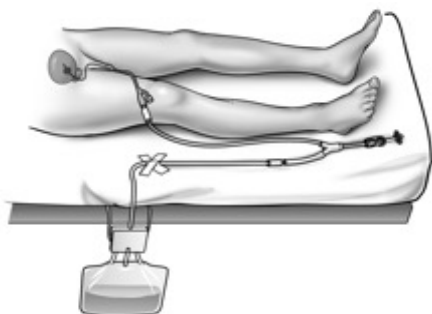
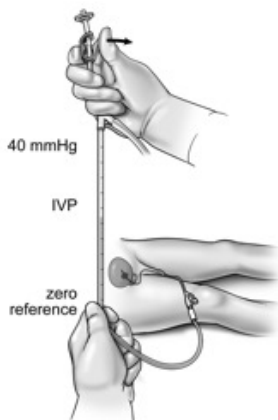


Figure 2 - IAP measurement with the Foley Manometer LV



- Open the Foley Manometer LV (Holtech Medical, Denmark) pouch and close the tube clamp
- Place the urine collection device under the patient's bladder
- Insert the Foley Manometer between catheter and drainage device
- Prime the Foley Manometer with 20ml of sterile saline through its needle-free injection/sampling port (only once i.e. at initial set-up)

- Urine sampling from the needle-free port is facilitated by temporarily opening the red clamp
- Replace the Foley Manometer whenever the Foley catheter or the urine collection device is replaced, or at least every 7 days



- Place the "0 mmHg" mark of the manometer tube at the midaxillary line at the level of the iliac crest and elevate the filter vertically above the patient
- Open the bio-filter clamp, and read IVP when the meniscus has stabilized after about 10 seconds
- Close clamp after IVP measurement and place the Foley Manometer in its drainage position

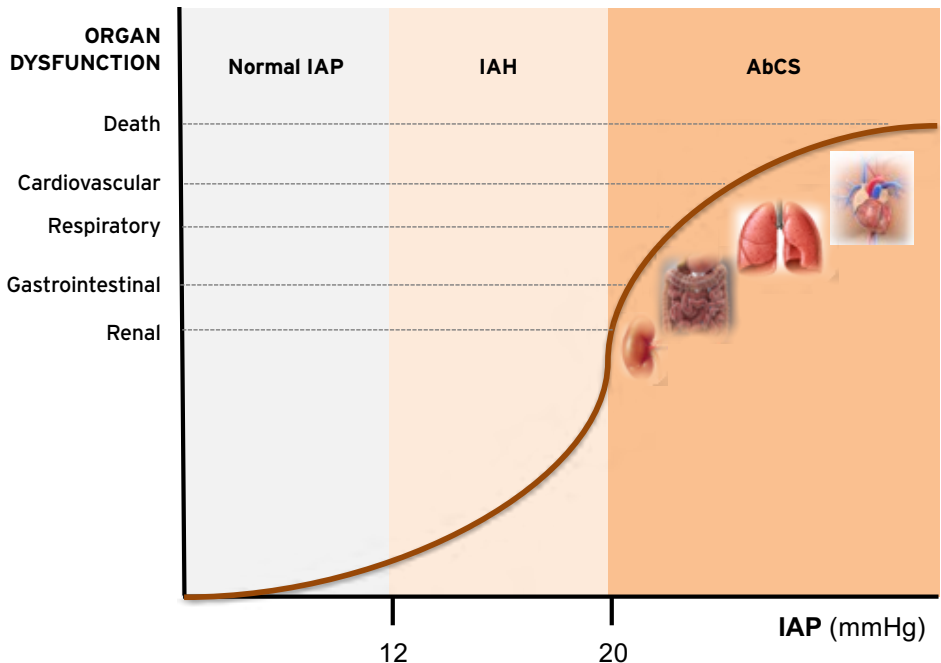
### 8.1.2 - Intra-abdominal hypertension

IAH is a sustained or repeated pathological elevation in IAP  $\geq 12$  mmHg and it's graded in four categories (**Table 1**). AbCS is a sustained increased IAP  $> 20$  mmHg that is associated with new onset organ failure (**Figure 3**). IAH is a graded phenomenon while AbCS is an all-or-nothing condition. AbCS is a syndrome and not a disease and as such it can have many causes or it can be associated with many disease processes. A polycompartment syndrome is a condition where two or more anatomical compartments have elevated compartmental pressures.

**Table 1 - WSACS grading of IAH**

IAH grade	IAP (mmHg)
Grade I	12-15
Grade II	16-20
Grade III	21-25
Grade IV	25

**Figure 3 - Natural course of IAH/AbCS**



### 8.1.3 - Abdominal compliance

Abdominal compliance is a measure of the ease of abdominal expansion, which is determined by the elasticity of the abdominal wall and diaphragm. It should be expressed as the change in intra-abdominal volume per change in IAP.

## 8.2 - Primary and secondary IAH

Primary IAH or AbCS is a condition associated with injury or disease in the abdominopelvic region that frequently requires early surgical or interventional radiological intervention. However, secondary IAH or AbCS refers to conditions that do not originate from the abdominopelvic region, frequently associated with massive fluid resuscitation in patients with sepsis, capillary leak and other pathological states (*Figure 4*). This condition could damage the endothelial glycocalyx layer (negatively charged luminal protein surface of vascular endothelium) and the tight junction complexes, whose general function is to prevent leakage of transported solutes and water, so they seal the paracellular pathway. If they are damaged, they contribute to interstitial oedema and then to IAH. This has been referred to as the GIPS. This mechanism of injury (i.e. increased vascular permeability) is widely recognized and accepted in the lung and kidneys, where it is classified as ALI and AKI. The same pathological process occurs in the gut, but this concept is much slower to seep through. However, the role of the gut as the motor of organ dysfunction syndrome cannot be denied and difficulties in assessing gut function should not deter us from recognizing that concept.

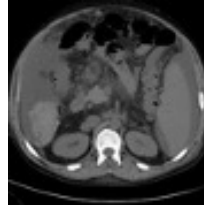
## 8.3 - Pathophysiology of IAH

Although the pathophysiologic implications of an elevated IAP have already been presented in the last century, they have been re-appreciated during the last two decades after scientific investigation and clinical experience confirmed the detrimental impact of IAH on end-organ function. Elevated IAP plays a major role in the development of MOF as a consequence of significant hypoperfusion (*Figure 5*). The effects of IAH are not limited to the intra-abdominal organs alone but have a direct or indirect impact on every organ system of the body, causing significant morbidity and mortality.

Figure 4 - Primary and secondary IAH/ACS

**PRIMARY**

- Abdominal surgery
- Hemoperitoneum
- Gastroparesis/ ileus
- Colonic obstruction
- Acute pancreatitis
- Intra-abdominal tumors/ collections



**SECONDARY**

- Sepsis
- Polytransfusion
- Massive fluid resuscitation

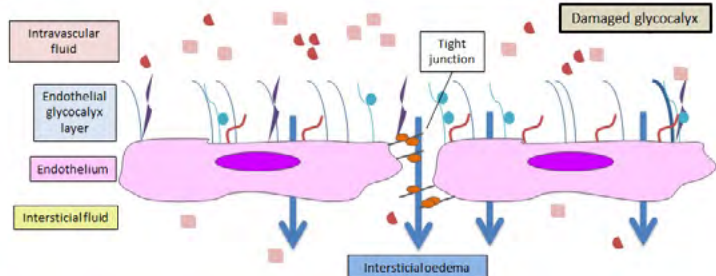
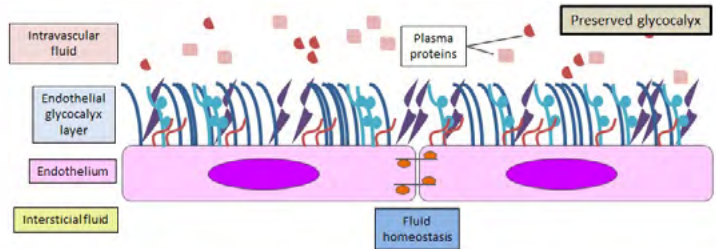
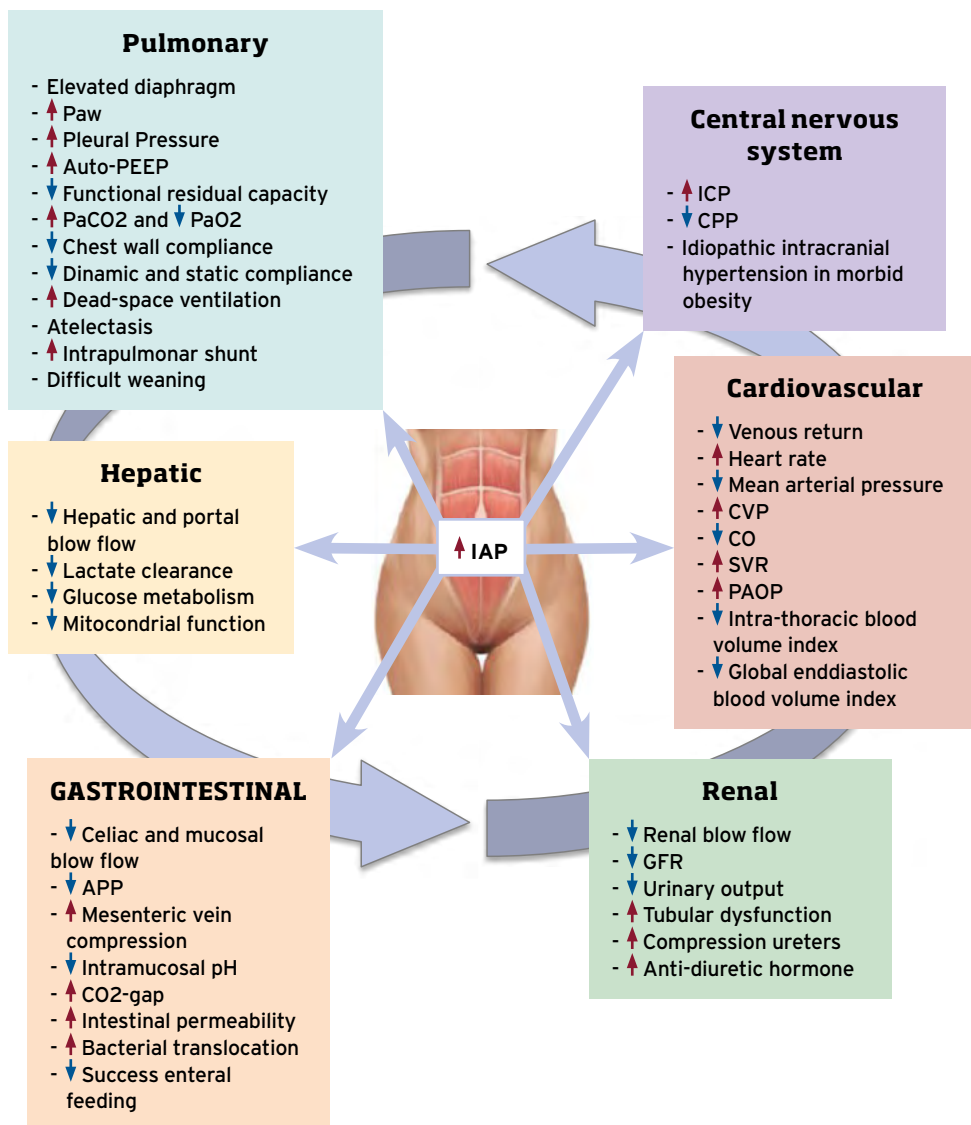




Figure 5 - Pathophysiologic implications of IAH



## 8.4 - Monitoring IAH and AbCS

Prevention of IAH is essential in patients with risk factors for IAH or AbCS. When two or more risk factors are present, baseline IAP monitoring is mandatory followed by sequential assessment in case of IAH grade I. The main monitoring parameter is the IAP, that can be obtained via trans-bladder measurement (**Figure 1, 2**) (**Table 2**). In order to perform a good measurement, pain and anxiety must be excluded, since voluntary contraction of the abdominal muscles can interfere with interpretation of the results. Other basic monitoring tools are gastric residual volume via nasogastric tube or antral cross-sectional area (ultrasound) and urinary output. Overzealous administration of fluids can cause secondary IAH/AbCS (endothelial dysfunction), so it is important to measure body weight and cumulative fluid balance. Abdominal perfusion pressure is the difference between mean systemic arterial pressure and IAP. It more accurately reflects visceral perfusion.

More advanced monitoring is needed in case of IAH grade II or higher. In the presence of an arterial line and a central venous catheter, the use of transpulmonary thermodilution (PiCCO, Getinge or EV1000, Edwards) can provide additional information on preload, contractility, afterload and pulmonary edema (**Table 2**): oxygenation ratio ( $\text{PaO}_2/\text{FiO}_2$ ),  $\text{ScvO}_2$ , CO (also through echocardiography), SVR, as well as fluid management status in the different phases of shock (PPV, GEDVI and EVLWI). Sequential arterial or venous lactate levels may be helpful. Barometric preload parameters like CVP or PCWP are erroneously increased in case of IAH and cannot be used.

### Patients with risk factors for IAH and AbCS

- Diminished abdominal wall compliance
- Increased intra-abdominal contents
- Increased intra-luminal contents
- Capillary leak/fluid resuscitation

### Table 2 - Main parameters to monitoring in IAH/ACS

	Monitoring	Technique	Normal values
<b>Main parameters</b>	IAP/4h	Trans-bladder measurement	$\leq 12$ mmHg
	APP	AMP - IAP	$\geq 60$ mmHg
	Gastric residual volume	Nasogastric tube	$< 500\text{cc}/6\text{h}$ or $< 1000\text{cc}/24\text{h}$
	Urinary output	Foley catheter	$> 0,5-1$ mL/Kg/h
	Glomerular filtration rate Body weight Cumulative balance fluid		$60-90$ mL/min/ $1,73\text{m}^2$
<b>Advanced parameters</b>	Continuous AP	A-line	
	Lactate	Blood analysis	$< 2$ mmol/L
	$\text{PaO}_2/\text{FiO}_2$	Arterial gases	$> 300$ mmHg
	$\text{ScvO}_2$	Central venous catheter	$> 70\%$
	CO	Transpulmonary thermodilution Echocardiography	$4-8$ L/min
	SVR	Transpulmonary thermodilution	$800-1200$ $\text{din}\cdot\text{s}\cdot\text{cm}^{-5}$
	PPV		$< 13\%$
	GEDVI		$600-800$ mL/ $\text{m}^2$
	EVLWI		$3-7$ mL/Kg



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