# *Definitions and Pathophysiological Implications of Intra-abdominal Hypertension and Abdominal Compartment Syndrome*

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**For any syndrome or disease process, uniform definitions are essential to facilitate effective clinical communication as well as evaluation of the scientific literature and standardization of research. The following consensus definitions for intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS) have been proposed by the World Society of the Abdominal Compartment Syndrome and are now widely accepted around the world. The use of these definitions, and their subsequent revisions as new evidence becomes published, will further improve communication and future research in this area. This review briefly addresses the present definitions as well as the pathophysiological effects of IAH/ACS on end-organ function.**

INTRA-ABDOMINAL HYPERTENSION (IAH) and abdominal compartment syndrome (ACS) have been increasingly recognized as causes of significant morbidity and mortality in critically ill medical and surgical patients.[1] Although IAH is a continuum of progressively worsening organ dysfunction, ACS is an "all or none" phenomenon that results when IAH remains either unrecognized or untreated. IAH and ACS impact end-organ function within not only the abdominal cavity (kidneys, liver, intestine), but also throughout the body (brain, lungs, heart). The last decade has witnessed an exponential increase in published literature on this topic.[2] Before 2006, there was little agreement regarding definitions for intra-abdominal pressure (IAP), IAH, or ACS, leading to confusion and an inability to compare the results of published clinical trials. The World Society of the Abdominal Compartment Syndrome (www.wsacs.org) has developed evidence-based consensus definitions outlining standards for IAP measurement as well as diagnostic criteria for IAH and ACS based on both the best available clinical evidence and expert opinion.[3–5] These definitions have become the standard nomenclature for IAH/ACS and are now widely accepted for scientific research and communication among clinicians worldwide. These definitions, together with the pathophysiological implications of IAH/ACS, are briefly discussed here.

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## Definitions

- **IAP is the steady-state pressure concealed within the abdominal cavity**
  The abdomen should be considered as a closed box with walls that are either rigid (costal arch, spine, and pelvis) or flexible (abdominal wall and diaphragm) (Table 1). IAP is directly affected by the volume of the solid organs or hollow viscera; the presence of ascites, blood, or other space-occupying lesions (such as tumors or a gravid uterus); and the presence of conditions that limit expansion of the abdominal wall (such as burn eschars or third-space edema).
- **APP = MAP – IAP**
  Analogous to cerebral perfusion pressure, abdominal perfusion pressure (APP) has been proposed as a more accurate predictor of visceral perfusion than arterial pH, base deficit, lactate, or hourly urinary output.[6, 7] APP, by considering both arterial inflow (mean arterial pressure) and restrictions to venous outflow (IAP), is statistically superior to either parameter alone in predicting patient survival from IAH/ACS and is a valuable end point for resuscitation. Failure to maintain APP above 60 mmHg has been demonstrated to correlate with increased patient mortality.[6]
- **FG = GFP – PTP = MAP – 2 * IAP**
  The filtration gradient (FG) is the mechanical force across the renal glomerulus and equals the difference between the glomerular filtration pressure (GFP) and

TABLE 1. *Consensus Definitions*

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| Definition 1 | IAP is the steady-state pressure concealed within the abdominal cavity. |
| Definition 2 | APP = MAP – IAP |
| Definition 3 | FG = GFP – PTP = MAP – 2 * IAP |
| Definition 4 | IAP should be expressed in mmHg and measured at end-expiration in the complete supine position after ensuring that abdominal muscle contractions are absent and with the transducer zeroed at the level of the midaxillary line. |
| Definition 5 | The reference standard for intermittent IAP measurement is through the bladder with a maximal instillation volume of 25 mL of sterile saline. |
| Definition 6 | Normal IAP is approximately 5 to 7 mmHg in critically ill adults. |
| Definition 7 | IAH is defined by a sustained or repeated pathologic elevation of IAP 12 mmHg or greater. |
| Definition 8 | IAH is graded as follows:<br>• Grade I: IAP 12 to 15 mmHg<br>• Grade II: IAP 16 to 20 mmHg<br>• Grade III: IAP 21 to 25 mmHg<br>• Grade IV: IAP greater than 25 mmHg |
| Definition 9 | ACS is defined as a sustained IAP greater than 20 mmHg (with or without an APP less than 60 mmHg) that is associated with new organ dysfunction/failure. |
| Definition 10 | Primary ACS is a condition associated with injury or disease in the abdominopelvic region that frequently requires early surgical or interventional radiological intervention. |
| Definition 11 | Secondary ACS refers to conditions that do not originate from the abdominopelvic region. |
| Definition 12 | Recurrent ACS refers to the condition in which ACS redevelops after previous surgical or medical treatment of primary or secondary ACS. |

IAP, intra-abdominal pressure; MAP, mean arterial pressure; APP, abdominal perfusion pressure; IAH, intra-abdominal hypertension; ACS, abdominal compartment syndrome; FG, filtration gradient; GFP, glomerular filtration pressure; PTP, proximal tubular pressure.

the proximal tubular pressure (PTP). In the presence of IAH, PTP may be assumed to equal IAP and GFP can be estimated as mean arterial pressure (MAP) minus IAP. Thus, changes in IAP will have a greater impact on renal function and urine production than will changes in MAP. This may explain why oliguria is one of the first signs of IAH and why the kidneys are usually the first organ that returns to normal function after decompression.[8]

- **IAP should be expressed in mmHg and measured at end-expiration in the complete supine position after ensuring that abdominal muscle contractions are absent and with the transducer zeroed at the level of the midaxillary line**

  IAP measurements are essential to the diagnosis of IAH/ACS because physical examination is notoriously poor in identifying elevated IAP. Several key principles must be followed to ensure accurate and reproducible measurements. Early studies using water manometers reported their results in cm $H_2O$, whereas subsequent studies using electronic pressure transducers reported IAP in mmHg (1 mmHg = 1.36 cm $H_2O$).[9] This led to confusion and difficulty in comparing studies. IAP varies with respiration and is most consistently measured at end-expiration. Changes in body position (i.e., supine, prone, head of bed elevated) and the presence of both abdominal and bladder detrusor muscle contractions have been demonstrated to impact on the accuracy of IAP measurements.[10, 11] Various transducer "zero reference" points have been suggested for IAP measurement, including the symphysis pubis, the phlebostatic axis, and the midaxillary line.[12] The application of positive pressure ventilation and positive end-expiratory pressure (PEEP) may also increase IAP.[10, 13] Each of these may result in different IAP values within the same patient.

- **The reference standard for intermittent intra-abdominal pressure measurement is through the bladder with a maximal instillation volume of 25 ml of sterile saline**

  Multiple studies have demonstrated that, for the bladder technique, instillation volumes in excess of 25 mL may artificially increase IAP, leading to potentially erroneous measurements and inappropriate therapy.[14, 15] Recent studies have suggested that volumes as low as 5 mL are sufficient.[16]

- **Normal IAP is approximately 5 to 7 mmHg in adults**

  Normal IAP in adults ranges from 0 to 5 mmHg. In critically ill patients, values of 10 to 40 mmHg have been reported. Morbid obesity or pregnancy may be associated with asymptomatic IAP elevations of up to 15 mmHg, which are generally well tolerated as a result of their chronic nature.[10] Recent abdominal surgery, sepsis, organ failure, and need for mechanical ventilation are all associated with elevated IAP, whereas children usually have lower IAP values.[17] The clinical importance of any IAP must always be assessed in view of the baseline IAP for the individual patient.

- **IAH is defined by a sustained or repeated pathologic elevation of intra-abdominal pressure of 12 mmHg or greater**

  Pathological IAP is a continuum ranging from mild, asymptomatic elevations in IAP to marked elevations in IAP with grave consequences on virtually all organ systems in the body. The exact IAP that defines IAH has long

been a subject of debate. The majority of studies show that visceral organ perfusion begins to decrease at an IAP of 10 to 15 mmHg.

- **IAH is graded as follows:**
  Grade I: IAP 12 to 15 mmHg
  Grade II: IAP 16 to 20 mmHg
  Grade III: IAP 21 to 25 mmHg
  Grade IV: IAP greater than 25 mmHg
  Patients with prolonged untreated elevations in IAP commonly manifest inadequate perfusion and subsequent organ failure. The more severe the degree of IAH, the more urgent the need to reduce the damaging elevation in pressure (either medically or surgically). Grading systems help to improve communication and improve clinical research.
- **ACS is defined as a sustained IAP greater than 20 mmHg (with or without an APP < 60 mmHg) that is associated with new organ dysfunction/failure**
  ACS is best remembered as the presence of significant IAH with organ failure. Failure to recognize and appropriately treat ACS is uniformly fatal, whereas prevention and timely intervention are associated with marked improvements in organ function and patient survival. In contrast to IAH, ACS is not graded, but rather considered an "all or none" phenomenon. There are three different types of ACS: 1) primary ACS is a condition associated with injury or disease in the abdominopelvic region that frequently requires early surgical or interventional radiological intervention; 2) secondary ACS refers to conditions that do not originate from the abdominopelvic region; and 3) recurrent ACS refers to the condition in which ACS redevelops after previous surgical or medical treatment of primary or secondary ACS

## Pathophysiological Implications

IAH has an impact on every organ system in the body. Any patient who develops one or more organ failures should be evaluated for possible IAH/ACS by measuring the patient's IAP.[1] A practical way to do so would be to daily monitor organ function by means of a scoring system such as the Multiple Organ Dysfunction Score or the Sequential Organ Failure Assessment score and to initiate IAP monitoring once organ failure becomes evident. IAH can affect multiple organ systems in a graded fashion. To better understand the clinical presentation and management of disorders of IAH, one must understand the physiological derangements within each organ system separately. It is beyond the scope of this review to give a concise and complete overview of the pathophysiological implications of raised IAP on end-organ function. We only discuss some key messages related to each organ that will affect daily clinical practice. Figure 1 summarizes these effects.

### *Cardiac Function*

Cardiovascular dysfunction and failure (low cardiac output) are commonly observed in patients with IAH or ACS and are aggravated by the presence of hypovolemia and the application of PEEP.[6] Accurate assessment and optimization of preload, contractility, and afterload are essential to restore end-organ perfusion and function. Our understanding of traditional hemodynamic monitoring techniques and parameters, however, must be re-evaluated in IAH/ACS because pressure-based estimates of intravascular volume because pulmonary artery occlusion pressure (PAOP) and central venous pressure (CVP) are readily misinterpreted. The clinician must be aware of the interactions among intrathoracic pressure (ITP), IAP, PEEP, and cardiac filling pressures (such as PAOP and CVP). Misinterpretation of the patient's minute-to-minute cardiac status may result in the institution of inappropriate and potentially detrimental therapy (typically inappropriate and commonly injurious excessive volume administration).

Transmural (tm) cardiac filling pressures, calculated as the end-expiration value (ee) minus the ITP, better reflect a patient's true preload status but require monitoring with an esophageal balloon device:

$$CVP_{tm} = CVP_{ee} - ITP$$
$$PAOP_{tm} = PAOP_{ee} - ITP$$

A quick estimate of transmural filling pressures can be obtained by subtracting half of the patient's IAP from the end-expiratory filling pressure because abdominothoracic pressure transmission has been estimated to be approximately 50 per cent.[6, 17]

$$CVP_{tm} = CVP_{ee} - IAP/2$$
$$PAOP_{tm} = PAOP_{ee} - IAP/2$$

The Surviving Sepsis Campaign guidelines target initial and ongoing resuscitation toward a CVP of 8 to 12 mmHg and a MAP of 65 mmHg.[18] As a result of the impact of elevated IAP on the accuracy of CVP measurements, such targets must be used with caution in the presence of IAH/ACS to avoid underresuscitation.

Volumetric estimates of preload status such as right ventricular end diastolic volume index or global end diastolic volume index may be especially useful in guiding fluid resuscitation because their accuracy is not affected by changing ventricular compliance and elevated ITP.[6, 19] Functional hemodynamic parameters such as pulse pressure variation and to a lesser extent stroke volume (but not systolic pressure variation) may also be useful in determining the need for volume expansion to support preload-responsive cardiac performance or the initiation of vasoactive medication

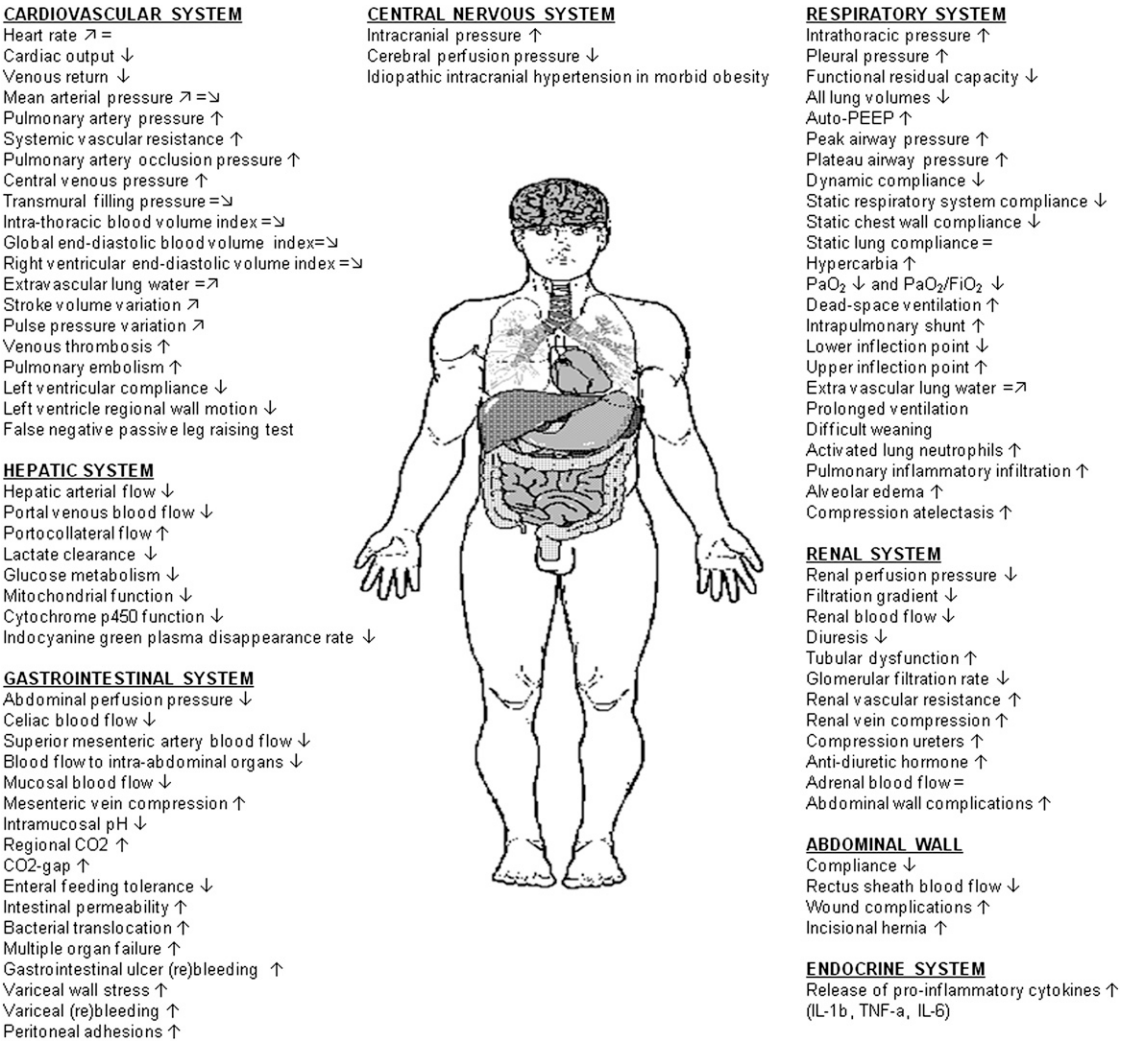


FIG. 1. Organ system effects of intra-abdominal hypertension: cardiovascular effects are exacerbated in case of hypovolemia, hemorrhage, ischemia and high positive end-expiratory pressure (PEEP) ventilation ↑, increased; ↓, decreased; =, unchanged; ↗, slightly increased; ↘, slightly decreased.

administration. Like with cardiac filling pressures, the usual target thresholds for resuscitation adequacy (10 to 12%) must be redefined in the patient with IAH/ACS to 20 to 25 per cent as a result of the impact of increased IAP and ITP on intrathoracic volumes.[20] For the same reasons, it is important to recognize that a passive leg raising test may by false-negative in patients with IAH/ACS.[21]

*Respiratory Function*

IAH decreases total respiratory system compliance by a decrease in chest wall compliance, whereas pulmonary parenchymal compliance remains unchanged.[22] IAH also leads to pulmonary hypertension through increased ITP with direct compression on lung parenchyma and vessels and through diminished left and right ventricular compliance. The effect of IAP on pulmonary parenchymal compression is exacerbated in cases of hypovolemia regardless of cause.

Alveolar atelectasis is common and PEEP should be set to counteract the effects of elevated IAP while at the same time avoiding overinflation of already well-aerated or normal lung regions. During lung protective ventilation in the setting of IAH, it is important to use the transpulmonary plateau pressure ($Pplat_{tm}$), which corrects for the negative impact of IAP, and maintain this pressure below 35 $cmH_2O$ to avoid overinflation of alveoli.

$$Pplatt_m = Pplat - IAP/2$$

As discussed, the PAOP resuscitation target in the ARDSnet consensus definitions is often not useful in patients with IAH and should be revised upward because most patients with IAH and secondary acute lung

injury/acute respiratory distress syndrome will have a PAOP above 18 mmHg. IAH increases extravascular lung water and thereby renders monitoring of extravascular lung water index a potentially useful parameter in guiding fluid balance.[23]

*Renal Function*

Decreased renal perfusion pressure (RPP) and renal FG have been proposed as key factors in the development of IAP-induced renal failure.[24]

$$\begin{aligned} \text{RPP} &= \text{MAP} - \text{IAP} \\ \text{FG} &= \text{GFP} - \text{PTP} = (\text{MAP} - \text{IAP}) - \text{IAP} \\ &= \text{MAP} - 2 * \text{IAP} \end{aligned}$$

The prerenal azotemia seen in IAH is typically unresponsive to both plasma volume expansion and diuretic therapy. In patients with secondary ACS, renal function may be improved by paracentesis of ascitic fluid and reduction in the patient's IAP. Prompt reduction of IAP has a dramatic beneficial effect on urinary output in patients with primary or secondary ACS after trauma provided that the genesis of the oliguria is IAH-associated decreased cardiac performance and decreased stroke volume and renal perfusion.[8] In patients with acute tubular necrosis, decreases in IAP are not likely to augment urine flow. Traumatic hemorrhage within the capsule of the kidney (subcapsular hematoma) may have an adverse affect on tissue perfusion causing a local renal compartment syndrome.[25–27]

*Hepatic Function*

With increasing IAP, there is decreased hepatic arterial flow, decreased portal venous flow, and an increase in the portocollateral circulation. As a result, decreased lactate clearance, altered glucose metabolism, and altered mitochondrial function are common. Close monitoring and early recognition of IAH followed by aggressive treatment may confer an outcome benefit in patients with liver disease. In this unique patient population, it may be useful to measure the plasma disappearance rate for indocyanine green because this correlates not only with liver function and perfusion, but also with IAP.[28] Because cytochrome $P_{450}$ function may be diminished in the presence of IAH/ACS, medication doses should be adjusted accordingly. Like with the kidney, subcapsular hematoma formation may have an adverse affect on tissue perfusion causing a local hepatic compartment syndrome.[25–27]

*Neurologic Function*

Because of the close interactions among IAP, ITP, and intracranial pressure, routine IAP monitoring in patients with traumatic brain injury and associated abdominal trauma should be strongly considered.[29] IAH can be an extracranial cause of intracranial hypertension in patients with abdominal trauma who are without overt craniocerebral lesions.[25–27] For similar reasons, recent head injury should be considered a contraindication for laparoscopic procedures.

TABLE 2. *Key Messages Regarding IAH/ACS*

- IAP is an important physiological measurement.
- Both IAP and APP should be followed serially during patient resuscitation.
- IAH = IAP 12 mmHg or greater
- ACS = IAP greater than 20 mmHg with organ failure
- IAH/ACS has an impact on end-organ function both within and outside the abdominal cavity.
- Early recognition and organ support are the key to improving patient outcome.

IAP, intra-abdominal pressure; APP, abdominal perfusion pressure; IAH, intra-abdominal hypertension; ACS, abdominal compartment syndrome.

## Conclusions

A common nomenclature of terms and definitions has greatly facilitated recent research and communication regarding IAH/ACS. Continued use of these definitions, with subsequent revision and improvement based on new scientific findings, will improve both the prevention and treatment of IAH/ACS. All clinicians are encouraged to adopt these definitions and use them in their clinical practice and research. IAH/ACS has a far-reaching impact on end-organ function. Early recognition and treatment of elevated IAP are key to improving patient outcome (Table 2). A common theme within all of the end-organ support measures advocated in IAH/ACS is improved perfusion and microcirculatory delivery of oxygen at the organ level. Further data are needed to enable the clinician to better support end-organ function and avoid the detrimental sequelae of IAH and ACS.

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