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INTRA-ABDOMINAL PRESSURE MONITORING

Evidence Based Medicine Guideline

Primary Author: Michael D. Tichenor, MD
Co-Authors:
Editors: Michael L. Cheatham MD, Chadwick Smith MD
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SUMMARY
Elevated intra-abdominal pressure (IAP) is commonly encountered in the critically ill, has detrimental effects on all organ systems, and is associated with significant morbidity and mortality. Serial IAP measurements are essential to the diagnosis, management, and resuscitation of patients who develop intra-abdominal hypertension (IAH) and/or abdominal compartment syndrome (ACS). IAP is both a diagnostic measurement, given the inaccuracy of clinical examination in detecting the presence of IAH, and a therapeutic measurement, as IAP guided resuscitation correlates with improved survival. Intravesicular pressure (IVP) is easily measured and should be monitored in all patients believed to be at risk for significant elevations in IAP.

RECOMMENDATIONS

- **Level 1**
  - IAP should be measured in the same body position to allow consistent trending of IAP. The transducer should be set at a consistent reference point.
- **Level 2**
  - Patients should be screened for IAH/ACS risk factors upon ICU admission and in the presence of new or progressive organ failure.
  - If two or more risk factors for IAH/ACS are present, a baseline IAP measurement should be obtained.
  - If IAH is present on baseline assessment, serial IAP measurements should be performed throughout the patient's critical illness.
- **Level 3**
  - IAP should be monitored using a closed technique.
  - IAP should be measured in mmHg (1 mmHg = 1.36 cm H_2O).
  - IAP should be measured in the supine position, at end-expiration, with the transducer zeroed at the mid-axillary line, 30-60 seconds after instillation of 10-25 mL of priming fluid (to allow bladder detrusor muscle relaxation), and in the absence of abdominal muscle contractions.

INTRODUCTION

Elevated intra-abdominal pressure (IAP) is frequently encountered among a variety of patient populations and causes significant morbidity and mortality (1-16). Increased recognition of its prevalence among the critically ill, combined with advances in both the diagnosis and management of intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS), have resulted in significant improvements in patient survival (5). IAP measurements are essential to the diagnosis and management of IAH/ACS. The World Society of the Abdominal Compartment Syndrome (WSACS) has previously published evidence-based medicine consensus guidelines for the measurement of IAP and treatment of IAH/ACS, most recently updated in 2013 (1-3).

LEVEL OF RECOMMENDATION DEFINITIONS

- **Level 1:** Usually based on Class I data or strong Class II evidence if randomized testing is inappropriate. Conversely, low quality or contradictory Class I data may be insufficient to support a Level I recommendation.
- **Level 2:** Reasonably justifiable based on available scientific evidence and strongly supported by expert opinion. Usually supported by Class II data or a preponderance of Class III evidence.
- **Level 3:** Supported by available data, but scientific evidence is lacking. Generally supported by Class III data. Useful for educational purposes and in guiding future clinical research.

DEFINITIONS

Intra-abdominal pressure (IAP) is the pressure concealed within the abdominal cavity (1). IAP increases with inspiration and decreases with expiration (17). It is directly affected by the volume of the solid organs or hollow viscera (which may be either empty or filled with air, liquid or fecal matter), 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). Normal IAP is approximately 5-7 mmHg in the critically ill, but varies by disease severity with an IAP of 20-30 mmHg being common in patients with severe sepsis or an acute abdomen (1). An IAP in excess of 15 mmHg is associated with significant end-organ dysfunction and failure.

Analogous to the widely accepted concept of cerebral perfusion pressure, abdominal perfusion pressure (APP), calculated as mean arterial pressure (MAP) minus IAP, has been proposed as a more accurate predictor of visceral perfusion and an endpoint for resuscitation (1,2,18-20). APP, by considering both arterial inflow (MAP) and restrictions to venous outflow (IAP), has been demonstrated to be statistically superior to MAP or IAP alone as well as to other common resuscitation endpoints such as arterial pH, base deficit, arterial lactate, and hourly urinary output in predicting survival from IAH/ACS. A target APP of 60 mmHg has been demonstrated to correlate with improved survival from IAH/ACS (2,20).

Intra-abdominal hypertension (IAH) is defined as a sustained or repeated pathologic elevation of IAP $\geq$ 12 mmHg (1-3). IAH is graded as follows:

Grade I	IAP 12-15 mmHg
Grade II	IAP 16-20 mmHg
Grade III	IAP 21-25 mmHg
Grade IV	IAP > 25 mmHg.

Abdominal compartment syndrome (ACS) is defined as a sustained increase in IAP > 20 mmHg (with or without an APP < 60 mmHg) that is associated with new organ dysfunction / failure (1-3). The most common clinical findings are hypotension, refractory metabolic acidosis, persistent oliguria, elevated peak airway pressures, refractory hypercarbia, hypoxemia, and intracranial hypertension. ACS may be classified as primary (a condition associated with injury or disease in the abdomino-pelvic region that frequently requires early surgical or interventional radiological intervention), secondary (a condition that does not originate from the abdomino-pelvic region), or recurrent (a condition in which ACS redevelops following previous surgical or medical treatment of primary or secondary ACS) (1-3,9-13).

INCIDENCE

Originally thought to be a disease solely of the traumatically injured, IAH and ACS have now been recognized to occur in a wide variety of patient populations (1-7,16). Historically, the reported incidences of IAH and ACS have varied significantly due to differences in definition, however recent studies have reported the presence of IAH in 34-49% of all adult ICU patients (21). Unrecognized, the mortality of IAH and ACS has been reported to be as high as 100%.

RISK FACTORS

Numerous risk factors for the development of IAH/ACS have been suggested secondary to different pathophysiologic mechanisms such as diminished abdominal wall compliance, increased intra-luminal contents, increased abdominal contents, and capillary leak/fluid resuscitation (2,4,8,10,22-25).

1. Diminished abdominal wall compliance.
    a. Acute respiratory failure with elevated intrathoracic pressure
    b. Abdominal surgery with primary fascial closure
    c. Major trauma/burns
    d. Prone positioning
2. Increased intra-luminal contents.
    a. Gastroparesis
    b. Ileus
    c. Colonic pseudoobstruction

3. Increased abdominal contents
    a. Hemoperitoneum/pneumoperitoneum
    b. Liver dysfunction with ascites
4. Capillary leak/fluid resuscitation
    a. Acidosis
    b. Hypotension
    c. Hypothermia
    d. Polytransfusion (>10 units of blood in 24 hours)
    e. Coagulopathy
    f. Massive fluid resuscitation (> 5 L in 24 hours)
    g. Oliguria
    h. Sepsis
    i. Damage control laparotomy

Given the broad range of potential etiologic factors and the significant associated morbidity and mortality of IAH/ACS, a high index of suspicion and low threshold for IAP measurement appears appropriate in the patient possessing any of these risk factors. Figure 1 depicts an algorithm for the initial evaluation of patients at risk for IAH (2). The WSACS strongly recommends that patients should be screened for IAH/ACS risk factors upon ICU admission and in the presence of new or progressive organ failure (3).

IAP MEASUREMENT

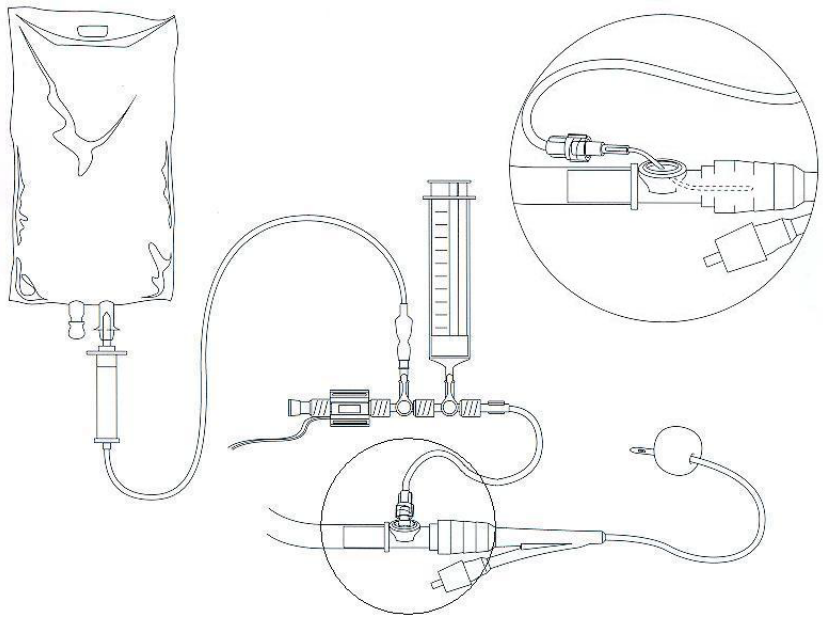
Physical examination is inaccurate in detecting elevated IAP with reported sensitivities of 40-60% (26,27). The diagnosis of IAH/ACS is therefore dependent upon the accurate and frequent measurement of IAP. IAP monitoring is a cost-effective, safe, and accurate tool for identifying the presence of IAH and guiding resuscitative therapy for ACS (2,3, 28-31). Given the favorable risk-benefit profile of IAP monitoring and the significant associated morbidity and mortality of IAH/ACS, the WSACS recommends that if two or more risk factors for IAH/ACS are present, a baseline IAP measurement should be obtained (2,3). Further, if IAH is detected, serial IAP measurements should be performed throughout the patient's critical illness.

The accuracy and reproducibility of IAP measurements are of paramount importance in the management of IAH/ACS (29,30,32). While direct intraperitoneal catheter determinations are ideal, a variety of less-invasive techniques for determining IAP have been devised including measurement of intravesicular (bladder), intragastric, intracolonic, and intrauterine pressure (28,29). Currently, over 90% of IAP measurements worldwide are performed using the intravesicular method as recommended by the WSACS (15). Continuous methods for monitoring IAP have been reported, but have not gained widespread implementation (28-30,33). While several studies have suggested that continuous femoral venous pressure measurements correlate with IAP, other more recent studies have not shown this to be a useful substitute for IVP in either adults or children (34-36).

Regardless of the technique utilized, several key principles must be followed to ensure accurate and reproducible measurements from patient to patient (2,29). IAP should be expressed in mmHg (1 mmHg = 1.36 cm H_2O) and measured at end-expiration after ensuring that abdominal muscle contractions are absent. Measurement of IAP should be accomplished using a consistent external reference point to zero the catheter (37,38). Head of bed elevation appears to significantly increase IAP measurements, the patient should be in the complete supine position with the transducer zeroed in the mid-axillary line at the level of the iliac crest (37-44). A maximal instillation volume of 10-25 mL of sterile saline (3 mL/kg for children) should be used for the intravesicular technique as several studies have demonstrated that larger volumes of fluid can lead to falsely elevated IAP measurements (45-50). Room temperature saline significantly increases IAP, presumably due to bladder detrusor contraction (47). As a result IAP determination should be performed 30-60 seconds after instillation of the priming fluid to allow bladder detrusor muscle relaxation (2,48).

While commercial IAP monitoring kits are widely available, these are unnecessary to measure IAP. IVP can be measured in virtually any patient with materials available in a typical ICU (Figure 1) (51).

Figure 1: Intra-vesicular Pressure Measurement (51)



Technique: A standard intravenous (IV) infusion set is connected to 500 mL of normal saline, two three-way stopcocks, a 20 mL Luer lock syringe, and a disposable pressure transducer. A short segment of arterial pressure tubing is used to connect the stopcocks to the sampling port of the patient's urinary drainage tubing. Alternatively, an 18-gauge plastic intravenous infusion catheter or needleless cannula can be inserted into the culture aspiration port of the urinary drainage tubing and the needle removed. The infusion catheter, cannula, or sampling port is attached to the first stopcock via pressure tubing. After being flushed with saline and zeroed at the level of the mid-axillary line (with the patient in the supine position), the urinary drainage tubing is clamped immediately distal to the catheter. The stopcocks are turned "off" to the patient and pressure transducer and 20 mL of saline is aspirated from the IV bag and instilled into the bladder. The stopcocks are turned "off" to the syringe and IV tubing. The clamp on the urinary drainage tubing is momentarily released to ensure that all air is flushed from the urinary catheter. After a stabilization period of 30-60 seconds to allow for bladder detrusor muscle relaxation, with the patient in the complete supine position and after ensuring that abdominal muscle contractions are absent, IAP is measured at end-expiration on the bedside monitor. The patient's IAP should be expressed in mmHg (1 mmHg = 1.36 cm H_2O). After IAP determination, the clamp is removed, the bladder allowed to drain, and the volume of saline utilized subtracted from the patient's urinary output for that hour.

Head of bed elevation is widely recommended to reduce the incidence of ventilator associated pneumonia. A number of studies have assessed the potential impact of such changes in body position on IAP measurements (39-44,52,53). These studies have routinely found that head of bed elevation significantly increases IAP compared to supine measurements. Such increases in IAP become clinically significant (increase > 2 mmHg) when the patient's head of bed exceeds 20 degrees elevation, well below that currently practiced in many intensive care units. As a result, supine IAP measurements may underestimate the patient's true IAP if the head of bed is being elevated between measurements. Prone positioning for acute lung injury has also been demonstrated to significantly increase IAP (54,55). Until further research is available to fully clarify this issue, the WSACS recommends that all IAP measurements be performed in the supine position and that the potential contribution of body position in elevating IAP should be considered in patients with moderate to severe IAH or ACS (2,3). Alternatively, the patient may be maintained in the reverse Trendelenberg position to maintain head of bed elevation while avoiding compression of the abdomen by the chest. This technique has the added benefit of utilizing gravity to decrease cephalad compression of the abdominal viscera upon the thoracic cavity, thereby reducing IAP. For serial measurements of IAP, a consistent body position should be utilized to promote consistency in readings (39,50).

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