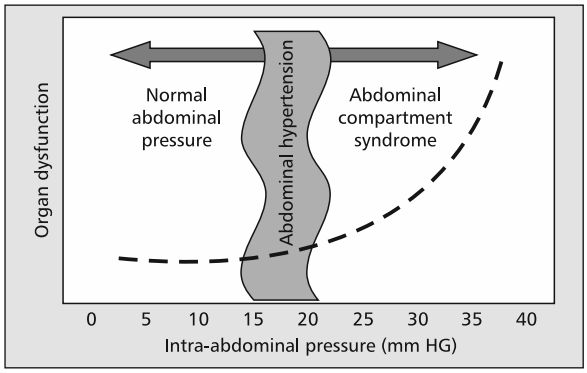


Figure 6. Distinctions between normal intra-abdominal pressure, intra-abdominal hypertension (IAH), and abdominal compartment syndrome (ACS). The shaded area illustrating IAH may undergo shifts to the right or left depending on the clinical scenario. Courtesy of David J.J. Muckart, MD, University of Natal Medical School, Republic of South Africa, and Rao Ivatury, MD, PhD, Virginia Commonwealth University, Virginia, USA, and adapted from Malbrain *et al.* [46]

proposed as a more accurate marker of critical illness and endpoint for resuscitation in patients with IAH, although a large prospective multicentric validation of this parameter is still pending.

$$APP = MAP - IAP$$

Target APP values can be achieved through a balance of judicious fluid resuscitation and the application of vasoactive medications. Maintaining an APP of 50 to 60 mm Hg may appear to be a better resuscitation endpoint compared to other macro and microcirculatory parameters. Multiple regression analysis has demonstrated that APP is superior as a resuscitation endpoint parameter compared to arterial pH, base deficit, arterial lactate, and hourly urinary output in surgical patients [47]. The authors concluded that using APP can allow one to predict survival from IAH or ACS that is not identified by IAP alone [47]. However, studies with regard to APP are scarce, often retrospective and include only small patient numbers. Therefore, APP as a resuscitation parameter cannot be recommended based on the current literature.

USE OF TRANSPULMONARY THERMODILUTION IN IAH

The continuous CO measured by TD or pulse contour with different devices (Pulsioflex, PiCCO, Swan Ganz) showed good agreement with TPTD in 10 pigs without IAH, and reflected an increase in CO following fluid loading [48]. Induction of IAH via the pneumoperitoneum did not significantly influence the CO measured by all devices. However, in IAH, an increase in CO following fluid loading was indicated by calibrated CO but not by uncalibrated CCO methods using arterial waveform analysis. In the critically ill patient, recalibration of continuous arterial waveform CO methods should be performed after fluid loading or before a major change in treatment is initiated [49]. The relative position of the catheters used for TPTD measurements may also be relevant. Huber *et al.* found that performing TPTD via a femoral central venous line (CVL) had a significant impact on the calculated variables [50]. Thus, they found that CI measured with TPTD via a jugular CVL can be calculated with the following formula:

$$0.931 \times CI_{fem} + 1.45 - 0.00042 \times GEDVI_{fem} + 0.028 \times CVP_{fem} - 0.009 \times \text{height}$$

(with CI_{fem}, $GEDVI_{fem}$ and CVP_{fem} respectively the CI, GEDVI and CI when measured with TPTD via a femoral CVL).

Since IAP and CVP_{fem} are closely related especially from grade III to IV IAH, this formula suggests that IAP may have an impact on TPTD CI when bolus injection is performed via a femoral CVL [18, 51]. The GEDVI and EVLWI values were also affected when performing TPTD via a femoral CVL. This phenomenon can be explained by the fact that the calculation of these volumetric parameters is based upon the MTT and DST, and since venous return is diminished in IAH, transit times may be affected when the thermodilution bolus is injected via the femoral CVL.

EFFECT OF INTRA-ABDOMINAL HYPERTENSION ON AFTERLOAD

Elevated ITP and IAP can cause increased systemic vascular resistance (SVR) through direct compressive effects on the aorta and systemic vasculature and increased PVR through compression of the pulmonary parenchyma. Organ compression may also result in alterations in the renin-angiotensin-aldosterone mechanism [11, 52]. More commonly, however, increased SVR occurs as compensation for the reduced venous return and falling stroke volume outlined above. As a result of this physiologic compensation, MAP typically remains stable in the early stages of IAH/ACS despite reductions in venous return and cardiac output. These increases in afterload may be poorly tolerated by patients with impaired cardiac contractility or inadequate intravascular volume [27, 53–55].

The concept of abdominal vascular zones may be present in the patient with IAH, analogous to the pulmonary vascular zone conditions described by West. In this concept, an increased IAP increases venous return when the transmural IVC pressure (defined as IVC pressure minus IAP) at the thoracic inlet significantly exceeds the critical closing transmural pressure (=zone 3 abdomen) [15]. This is most often the case in hypervolemic patients with a high IVC pressure. In zone 3 conditions, the abdominal venous compartment functions as a capacitor. In contrast, when

the transmural IVC pressure at the thoracic inlet is below the critical closure transmural pressure (=zone 2 abdomen), venous return is significantly decreased. This is most often the case in hypovolemic patients and by extension in most non-cardiogenic shock patients. In zone 2 conditions, the abdominal venous compartment functions as a collapsible starling transistor [56]. This model clearly illustrates why hypovolemia (and especially in combination with positive pressure ventilation and high levels of PEEP) predisposes patients to lower CO in response to elevated IAP than does normovolemia [57].

For the same reasons mean systemic filling pressure may also increase during IAH as was found in pregnant woman with pre-eclampsia [58]. This may explain the marked susceptibility to pulmonary edema seen with even minimal volume administration.

USE OF FUNCTIONAL HEMODYNAMICS IN IAH

IMPACT OF IAP ON FLUID RESPONSIVENESS

An increase in IAP will result in a concomitant increase in ITP and, as such, also in an increase in stroke volume variation (SVV) and pulse pressure variation (PPV) [59, 60]. Other studies have also shown increases in systolic pressure variation (SPV) that were mainly related to the Δup component and not to a Δdown phenomenon. As different mechanisms have been suggested, the increases seen in functional hemodynamic parameters may not univocally correspond to fluid responsiveness [61]. These include the following firstly, a change in aortic compliance and an increase in aortic transmural pressure induced by increased IAP (either via direct compression or increased vasomotor tone); secondly, errors in the measurement of dynamic indices in conditions of increased IAP, or, if we assume that no measurement errors are induced by IAP, then this implies that these indices do not perform well during IAH (since SVV and SPV no longer predict fluid responsiveness); and thirdly, changes in extramural pressure, ITP or chest wall compliance. Although in several animal studies, PPV (but not SVV) maintained its ability to predict fluid responsiveness even at IAP levels of 25 mm Hg, a receiver operating characteristics (ROC) curve analysis identified 20.5% as the best threshold for fluid responsiveness (instead of the classical 12% and the 9.5% identified in this study at baseline) [62–64]. This means that we cannot use the same thresholds for different conditions. The threshold value will depend on the amount of tidal volume, PEEP application or increased ITP and consequent changes in pleural pressure and chest wall compliance, the presence of obesity, heart failure with changes in right and left ventricular preload and afterload, pulmonary hypertension, the use of a pneumoperitoneum or increased IAP and may also differ in children or neonates [65]. Moreover, PPV has been shown to be superior to SVV in order to predict fluid responsiveness in the patients. This is somewhat surprising since PPV is a surrogate of SVV (derived from a pulse contour analysis based on a complex algorithm) and the latter should be less influenced by changes in vasomotor tone. Changes in pulse contour due to increased ITP may be more complex than previously thought. A recent animal study on severe pancreatitis showed that goal-directed hemodynamic management guided by SVV led to improved survival, tissue oxygenation, and microcirculatory perfusion, as well as less histopathologic damage [66]. Although the authors did not measure actual IAP, the model was interesting and can provide useful information for patients with IAH [67].

VALIDITY OF THE PASSIVE LEG RAISING TEST IN IAH

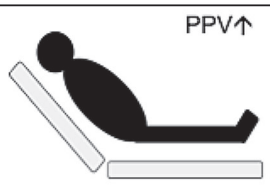
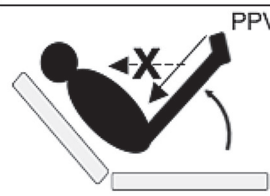
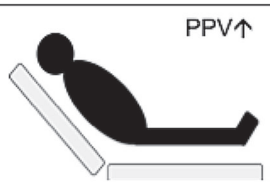
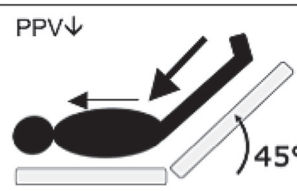
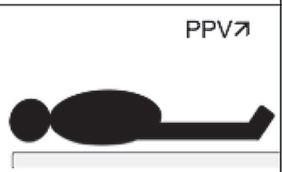
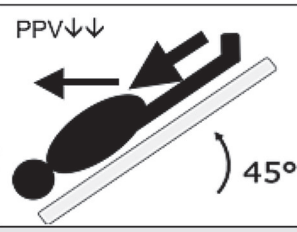
Recent data have shown that about 25% of critically ill patients with a PPV above 12% are not fluid responsive, suggesting different thresholds for different conditions [68]. Similar false positive PPV values have been reported previously and were related to right ventricular dysfunction. This also shows that the PLR test can be a false negative in responders to fluid administration and this can be related to increased IAP and diminished venous return from the legs and mesenteric veins. Care should be taken when a PLR test is performed, and an IAP measurement is needed whilst interpreting the result of a PLR test. The PLR test is difficult to standardise since it does not provide information on the exact amount of endogenous transfusion and there is some debate whether the starting position should be supine or upright (HOB) or whether the Trendelenburg position should be used. In fact, depending on patient anthropomorphism, the amount of fluid loading with a PLR may vary. During IAH one can expect an increase in baseline PPV especially in the 45° HOB position (Fig. 7). Performing a PLR maneuver from HOB (with the least risk for ventilator associated pneumonia) will further increase IAP and will only result in a marginal venous return from the legs but not from the mesenteric veins. Performing a PLR maneuver from the supine position will have a neutral effect on IAP and result in a better venous return from the legs but not from the mesenteric veins. At the same time, the Trendelenburg position will have a beneficial effect on IAP and, depending on body anthropomorphism, will result in a more pronounced venous return from the legs, as well as from the mesenteric veins [69].

CARDIOVASCULAR OPTIMIZATION IN IAH

IMPROVEMENT OF PRELOAD

Although initial fluid loading may improve venous return and restore CO, fluid overload must be avoided, especially in the setting of a capillary leak [70, 71]. To solve this

Panel A. Schematic overview comparing the possible effects and (dis)advantages of different PLR tests during normal IAP

TYPE	STARTING POSITION	POSITION DURING PLR	ADVANTAGES	DISADVANTAGES
HOB-PLR			No risk for VAP	Laborious Only small endogenous transfusion from legs
SUP-PLR			Not labour intensive Combination of endogenous transfusion from legs and mesenteric veins	Risk for VAP Risk for ICP increase
TREND ELENB URG			Not labour intensive Biggest endogenous transfusion from legs and mesenteric veins	Biggest risk for VAP Biggest increase in ICP

Panel B. Schematic overview comparing the possible effects and (dis)advantages of different PLR tests during increased IAP

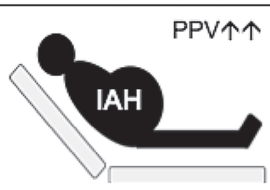
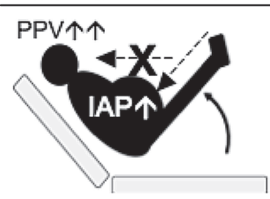
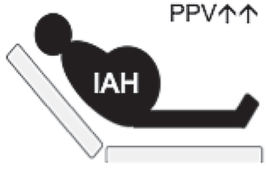
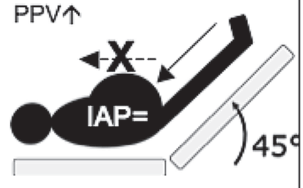
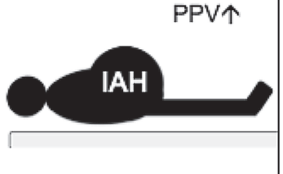
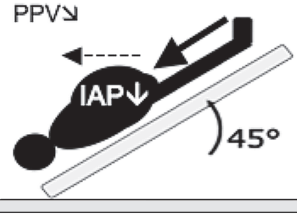
TYPE	STARTING POSITION	POSITION DURING PLR	ADVANTAGES	DISADVANTAGES
HOB-PLR			No risk for VAP	Labour intensive Increases IAP (lung compression) No endogenous transfusion
SUP-PLR			No increase in IAP (no lung compression) Not labour intensive	Risk for VAP Risk for ICP increase Only small endogenous transfusion (from legs)
TREND ELENB URG			Decrease in IAP (effects on lungs unclear) Not labour intensive Best endogenous transfusion in case of IAH	Biggest risk for VAP Biggest increase in ICP

Figure 7. Comparison of different types of passive leg raising tests. The PLR can be performed from HOB (first row) or supine (second row) position or putting the patient in the Trendelenburg position (third row). Panel A shows the effects of different PLR tests in patients with normal IAP while Panel B hypothesizes on the effects in patients with IAH. Endogenous fluid resuscitation comes from venous return from the legs (oblique arrow) and the mesenteric veins (horizontal arrow). The amount endogenous fluid resuscitation is indicated by the thickness of the arrow (dotted line is smallest while 3mm line is largest amount), a dotted line marked with a "X" indicates the absence of endogenous transfusion (adapted from Malbrain *et al*. [69]); HOB — head of bed; IAH — intra-abdominal hypertension; IAP — intra-abdominal pressure; ICP — intra cranial pressure; PLR — passive leg raising; PPV — pulse pressure variation; SUP — supine; VAP —ventilator associated pneumonia; ↑ — increase; ↑↑ — huge increase; ↗ — small increase; ↘ — small decrease; ↓ — decrease; ↓↓ — huge decrease

problem, the use of colloids or hyperoncotic solutions like hypertonic lactated saline or albumin 20% have been shown to reduce fluid intake whilst at the same time preserving urine output in severely burnt patients [72, 73]. The net result was a lower IAP and higher APP [72]. An important question in these patients is whether they are truly volume depleted,

and the use of parameters such as those mentioned above should always be correlated with clinical parameters of volume depletion. As fluids are the major contributor to secondary ACS, the judicious use of fluids is recommended [74]. As for all organ dysfunctions, decreasing IAP is another effective way of decreasing the negative cardiac effects described above.

IMPROVEMENT OF CONTRACTILITY

Non-operative measures to decrease IAP will result in a caudal movement of the diaphragm and decrease of cardiac compression, while the concomitant drop in ITP will improve cardiac contractility. After the initial resuscitation, a positive inotrope like dobutamine, levosimendan or milrinone can be considered if the patient has a low CO, increased lactate or low $S_{cv}O_2$. Although dobutamine infusion reverses the decrease in CO, it cannot restore superior mesenteric artery blood flow; however, intestinal mucosal blood flow returns to baseline levels [75]. Dopamine should not be used, since studies have shown no beneficial effect on splanchnic hemodynamic variables [75].

IMPROVEMENT OF AFTERLOAD

Preload augmentation through volume administration appears to ameliorate, at least partially, the injurious effects of IAH-induced increases in afterload. It has also been proposed, that the use of a moderate level of PEEP might efficiently reduce the increase in ventricular afterload [37, 40, 76, 77].

CONCLUSIONS AND KEY MESSAGES

- Cardiovascular dysfunction and failure (low CO, low contractility, high SVR) are common in IAH or ACS.
- Clinical evaluation of a patient is essential when interpreting the hemodynamic parameters obtained.
- Before administering fluids to patients with IAH or ACS, carefully check whether the patient is truly intravascular fluid depleted – do not act solely on preload parameters.
- Accurate assessment and optimization of preload, contractility, and afterload is essential to restore end-organ perfusion and function.
- Traditional hemodynamic monitoring techniques must be re-evaluated in IAH/ACS since pressure-based estimates of intravascular volume as PAOP and CVP can be erroneously increased.
  - The clinician must be aware of the interactions between ITP, IAP, PEEP, and intracardiac filling pressures.
  - Misinterpretation of the patient's minute-to-minute cardiac status may result in the administration of inappropriate and potentially detrimental treatment.
  - Transmural filling pressures may better reflect preload in the setting of increased IAP.
  - The mean systemic filling pressure may increase in IAH.
  - The Surviving Sepsis Campaign guidelines targeting initial and ongoing resuscitation towards a CVP of 8 to 12 mm Hg and other studies targeting a MAP of 65 mm Hg should be interpreted with caution in cases of IAH/ACS to avoid unnecessary over- and under resuscitation.
  - There is insufficient data to recommend resuscitation towards an APP > 60 mm Hg.
- Volumetric estimates of preload status such as right ventricular end-diastolic volume index (RVEDVI), global end-diastolic volume index (GEDVI) or left ventricular end-diastolic area (LVEDA), can be useful because of the changing ventricular compliance with elevated ITP.
- Clinicians must be aware of abdominal West abdominal zones.
- Although functional hemodynamic parameters such as PPV (but not SVV nor SPV) should be used to assess volume responsiveness, the traditional thresholds need to revised.
  - About 25–35% of patients with IAH and a PPV > 12% are non-responders to fluids.
  - The best threshold to predict fluid responsiveness in grade II IAH (15 to 20 mm Hg) is a PPV > 20%.
- IAH can be a cause of a false negative passive leg raising test.
- IAH causes pulmonary hypertension via increased ITP with direct compression on lung parenchyma and vessels and via the diminished left and right ventricular compliance.
- Transpulmonary thermodilution CO measurements are validated in the setting of IAH.
- Cardiovascular effects are aggravated by hypovolemia and the application of PEEP, whereas hypervolemia has a temporary protective effect.

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