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What is normal intra-abdominal pressure and how is it affected by positioning, body mass and positive end-expiratory pressure?

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Abstract Purpose: To describe what is defined as normal intra-abdominal pressure (IAP) and how body positioning, body mass index (BMI) and positive end-expiratory pressure (PEEP) affect IAP monitoring. **Methods:** A review of different databases was made (Pubmed, MEDLINE (January 1966–June 2007) and EMBASE.com (January 1966–June 2007)) using the search terms of “IAP”, “intra-abdominal hypertension” (IAH), “abdominal compartment syndrome” (ACS), “body positioning”, “prone positioning”, “PEEP” and “acute respiratory distress syndrome” (ARDS). Prior to 1966, we selected older articles by looking at the reference lists displayed in the more recent papers. **Results:** This review focuses on the concept that the abdomen truly behaves as a hydraulic system. The definitions of a normal IAP in the general patient population and morbidly obese patients are reviewed. Subsequently, factors that affect the accuracy of IAP monitoring, i.e., body position (head of bed elevation, lateral decubitus and prone position)

and PEEP, are explored. **Conclusion:** The abdomen behaves as a hydraulic system with a normal IAP of about 5–7 mmHg, and with higher baseline levels in morbidly obese patients of about 9–14 mmHg. Measuring IAP via the bladder in the supine position is still the accepted standard method, but in patients in the semi-recumbent position (head of the bed elevated to 30° and 45°), the IAP on average is 4 and 9 mmHg, respectively, higher. Future research should be focused on developing and validating predictive equations to correct for supine IAP towards the semi-recumbent position. Small increases in IAP in stable patients without IAH, turned prone, have no detrimental effects. The role of prone positioning in the unstable patient with or without IAH still needs to be established.

Keywords Intra-abdominal hypertension · Abdominal compartment syndrome · Body positioning · Prone positioning · PEEP and ARDS

Introduction

Intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS) are associated with significant morbidity and mortality [1, 2]. Risk factors for developing IAH and ACS have gradually been

established, culminating in the publications of the World Society of Abdominal Compartment Syndrome (WSACS, www.wsacs.org) recommendations relating to the assessment and management of patients with IAH and ACS [3–6]. It is clear that clinical assessment of the abdomen for elevated intra-abdominal pressure (IAP) is

neither sensitive nor specific and therefore it is important to measure the IAP intermittently [4, 7, 8]. Direct IAP measurement is impractical in most situations and the most common method is an indirect measurement via the bladder, a technique that has been validated [9–12] and which correlates well with IAP [3, 4]. This review will only focus on bladder pressure measurements in different body positions. Other routes for IAP measurement, e.g., intragastric, rectal, intravaginal and inferior vena cava pressure monitoring, are described elsewhere in the literature [13].

Ideally, the IAP should be expressed in mmHg and measured at end-expiration in a completely supine position, ensuring that abdominal muscle contractions are absent and with the transducer zeroed at the level of the mid-axillary line [3, 4]. However, most patients in the ICU are nursed with the head of the bed (HOB) elevated to 30° or 45°. This is because there is evidence that patients in a semi-recumbent position (HOB elevation) have a decreased incidence of ventilator-associated pneumonia (VAP) [14, 15] and that this position, in combination with pressure reducing devices, reduces the incidence of pressure ulcers and decubitus [16–18].

This review will focus on the effect different body positions can have on the accuracy and reproducibility of the measured IAP via the standard bladder technique recommended by the WSACS. Further, we will discuss what is considered a normal IAP in the general patient population and the morbidly obese and if the abdomen truly behaves as a hydraulic system. Finally, we will look at the effects of applied PEEP on IAP values.

Methods

A review of different databases was made (Pubmed, MEDLINE (January 1966–June 2007) and EMBASE.com (January 1966–June 2007)) using the search terms of “IAP”, “intra-abdominal hypertension” (IAH), “abdominal compartment syndrome” (ACS), “body positioning”, “prone positioning”, “PEEP” and “acute respiratory distress syndrome” (ARDS). Prior to 1966, we selected older articles by looking at the reference lists displayed in the more recent papers. The search was limited to English, Dutch and French language publications.

Does the abdomen behave as a hydraulic system?

Pressures in the abdomen were recognized to be atmospheric or positive when Rushmer showed that the magnitude of pressure at various levels in the abdomen were related to the height of the hydrostatic column of the abdominal contents above the point of measurement [19].

Prior to 1911, the IAP was considered to be positive or negative depending on the method used to measure it and there was no consensus about this discrepancy [20–22]. In other words, it was recognized that the abdomen behaved as a hydraulic system and the pressures within were hydrostatic in nature.

Rushmer’s findings were later challenged by Decramer et al. [23] who reported that gastric pressure swings in dogs were not simply hydrostatic. This study found that the abdomen does not behave as a hydraulic system or a liquid-filled container. Interestingly, the IAP differences observed in the dogs disappeared when the abdominal cavity was filled with 2 L of saline. These rather conflicting results were re-explored by Loring et al. [24], who measured pressures in the abdomen near the ventral abdominal wall and within the stomach of anesthetized dogs. This study concluded that there were three factors that affected IAP: gravity, uniform compression and shear deformation. Uniform compression, i.e., abdominal contraction, diaphragmatic contraction, mechanical ventilation, rib cage excursions and abdominal binding, result in spatially homogeneous changes in pressure that can be superimposed on the gravitational gradients (Fig. 1). Shear deformation, however, which is dependent on the shape stability of the tissue and the degree of deformation, is associated with spatially diverse pressure gradients. It is the relative importance of these individual factors that will determine ultimately if the abdomen behaves as a liquid-filled container. Therefore, the authors concluded that at times the abdomen behaves as a hydraulic system when the viscera are not subjected to shearing forces. All these data, however, are based on animal studies. In 1996, Tzelepis et al. [25] re-established the concept that the

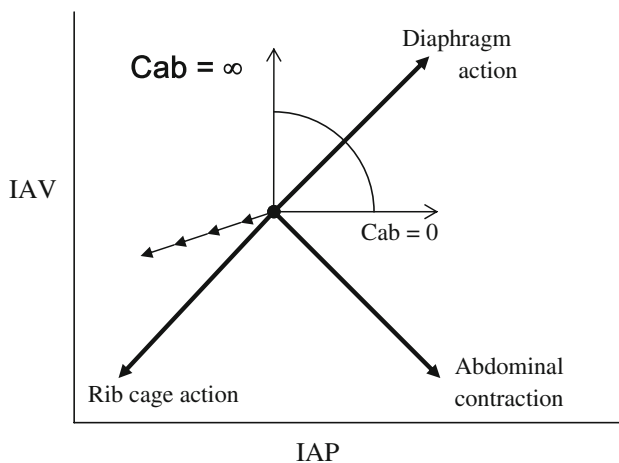


Fig. 1 Relationship between intra-abdominal volume, abdominal wall compliance and intra-abdominal pressure. Intra-abdominal volume (IAV) versus intra-abdominal pressure (IAP). The direction of the movement associated with the sole action of the rib cage inspiratory muscles, abdominal expiratory muscles and the diaphragm is shown. The direction of the latter depends on abdominal compliance (C_{ab}) but is constrained within the sector shown

Table 1 Elective intra-abdominal pressure measurement in the general non-obese patient population

	Patient population	Number	IAP (mean \pm SD)
Sugerman [31]	UC	5	5.1 \pm 1.2
Sanchez [30]	S-NS	27	5.0 \pm 2.9
Chionh [29] ^a	S-NS	58	7.0 (0.7–13.2)
Lambert [32]	ELS	4	0.0 \pm 1.5
Arfvidsson [33]	GS	4	6.2 \pm 1.2

^a Data expressed as median \pm range

UC ulcerative colitis, ELS elective laparoscopic surgery, GS groin surgery, S-NS surgical-non-surgical

abdomen behaves as a hydraulic system. Based on the available data, we hypothesize that the impact of shear deformation on the measurement of IAP is probably not significant in the fully sedated, mechanically ventilated patient with sepsis, capillary leak and a positive fluid balance, with or without neuromuscular blocking agents.

What is normal intra-abdominal pressure?

As mentioned before, IAP was originally considered to be positive or negative, based on the methodology used to measure it. In animal studies, sub-atmospheric pressures were often reported [26, 27]. In 1984, Kron et al. [28] published their data suggesting that IAP after abdominal surgery varied between 3 and 15 mmHg, indicating that it was more likely that IAP in humans was positive and not sub-atmospheric. To determine normal values for IAP in the general patient population, several trials measured and compared IAP in non-obese surgical or non-surgical patients [29–33](Table 1).

Based on this data, the WSACS defined that a normal IAP in the general patient population lies between 5 and 7 mmHg. However, in an obese patient the baseline value can be significantly higher (Table 2). Sugerman et al. [31,

34, 35] reported a positive correlation between bladder pressures and the sagittal abdominal diameter (SAD) and found that surgical patients with a mean BMI of 52 \pm 1 kg/m² had an IAP of 13.2 \pm 0.5 mmHg versus surgical patients with a BMI of 24 \pm 2 kg/m², where the IAP was significantly lower at 5.1 \pm 1.2 mmHg. The close correlation between IAP and SAD suggests a link between IAP and visceral fat. Hence, they postulated that increased IAP can contribute to the health risks associated with severe obesity such as pulmonary or arterial hypertension, proteinuria, idiopathic intracranial hypertension, type 2 diabetes, increased cardiac filling and hepatic venous pressures, reflux oesophagitis, hypoventilation and venous stasis [36]. Similar results of elevated IAP have been reported by Sanchez [30] where IAP was higher in patients with a high BMI. The mean IAP for patients with a BMI of 25.0–29.9 kg/m² and 30–39.9 kg/m² was 6.3 \pm 2.9 and 8.9 \pm 3.5 mmHg, respectively. A recent multicenter study also identified BMI as an independent predictor of intra-abdominal hypertension (IAH) [2]. Some have even suggested that chronic IAH in patients with central obesity could be responsible for syndrome X [31]. Although it is difficult to draw any conclusions based on the above data, due to differences in measurement techniques and the amount of volume instilled into the bladder during the trials, we suggest that the normal values of IAP in the obese patients should be considered as between 7 and 14 mmHg. A possible explanation for higher pressures in the obese is that there could be a direct effect from the intra-abdominal adipose tissue itself on the measurement of IAP.

In literature, there is also some controversy with regard to the effect of mechanical ventilation and the use of PEEP on IAP. Sussman [37] was the first to look at the effects of PEEP on IAP and showed in their experiment that increasing PEEP to 15 cm of H₂O did not affect the IAP. This was confirmed by Guimaraes and animal data [38–40]. However, on increasing PEEP to 15 cm of H₂O, others have found only a mild increase in IAP in patients with a baseline IAP below 12 mmHg [41–43]. Further, in

Table 2 Comparison of IAP among different weight groups

	Study characteristics N (observations)	IAP (mean + SD) according to BMI			
		Normal	Overweight ^a	Obese ^b	Morbidly obese ^c
Sanchez [30]	77 (231) ^d	5.0 \pm 2.9	6.3 \pm 2.9	8.9 \pm 3.5	8.4 \pm 2.5
Sugerman [31]	84 (84)	5.1 \pm 1.2	NA	NA	13.2 \pm 0.5
Sugerman [59]	15 (15)	NA	NA	7.4 \pm 0.7	NA
Lambert [32]	45 (45)	0 \pm 1.5	NA	NA	8.8 \pm 0.6
Vasquez [47] ^e	45 (675)	6.5 (4.5–8.5)	11.2 (7.7–14.6)	13.7 (11.4–16.0)	NA
Arfvidsson [33]	15 (15)	6.2 \pm 1.2	NA	NA	14 \pm 3
Sugerman [34]	6 (6)	NA	NA	12.5	16.2 \pm 2.2

^a BMI 25.0–29.9 kg/m²

^b BMI 30.0–39.9 kg/m²

^c BMI > 40

^d IAP observations

^e Data expressed as mean with 95% CI

Table 3 Effect of PEEP on IAP

	N	PEEPBL	PEEPset	DeltaPEEPa	DeltaPEEPb	IAP BL	IAP PEEP	Delta PEEP	TAI (%)
Sussman [37]	15	0	15	15	11	10.8	11.7	0.9	8.1
Gattinoni [44]	21	0	15	15	11	10.6 ± 6.0	11.8 ± 6.3	1.4 ± 1.3	12.7
Malbrain [42]	27	15	0	15	11	11.3 ± 4.3	10.3 ± 2.9	1.5 ± 1.2	13.6
Malbrain [42]	27	0	15	15	11	17 ± 3.9	18.3 ± 4.1	1.3 ± 1.1	11.8
Jaber ^a	30	0	12	12	8.8	11.7 ± 4.5	15.2 ± 5.8	3.5 ± 1.7	39.6
Ferrer [43]	12	5	15	15	11	9.5 ± 2.7	11.1 ± 2.8	1.6 ± 0.9	14.5

PEEPBL PEEP baseline, *DeltaPEEPa* PEEP expressed in cm H₂O, *deltaPEEPb* PEEP expressed in mmHg, TAI (thoraco-abdominal index) deltaIAP/deltaPEEPb, MA midaxillary, *delta IAP* IAP PEEP minus IAP BL

^a Personal communication

patients with a baseline IAP above 12 mmHg, the effect of PEEP seems to be more pronounced [44]. Table 3 shows the effects of PEEP on IAP.

Head of bed elevation/body positioning

Most patients in the ICU are nursed with an HOB elevation of 30–45° to reduce the risk of VAP and pressure ulcers [14, 16–18]. IAH and ACS, as mentioned before, are associated with increased morbidity and mortality. Therefore, patients at risk of developing IAH or ACS should have their IAP measured every 4 h [4]. It is a common practice to measure IAP in the supine position via the bladder after a maximum of 20 ml of normal saline had been instilled into the bladder with the mid-axillary line at the level of the superior iliac crest as the zero reference point [3, 4].

McBeth et al. [45] showed that IAP varied significantly between different HOB elevations. A total of 37 patients with an 18-Fr triple lumen bladder catheter had their IAP measured intermittently and continuously with the pressure transducer zeroed at the level where the mid-axillary line crossed the iliac crest. Pressures were measured in the supine position and at different HOB elevations. A total of 300 measurements were done showing an IAP difference of 1.2, 2.9, 5.0 and 7.4 mmHg at 10°, 20°, 30° and 45°, respectively. Based on the recent grading classifications of IAH [3, 4], this means that IAH would be over-diagnosed when the HOB is elevated (IAP at 45° would overestimate the pressure by two full grades). They also found in addition to HOB elevation that BMI, PEEP and body temperature correlated with the increase in IAP.

Cheatham et al. [46] conducted a similar, but larger, prospective multi-center trial looking at the effect of different body positions on IAP. A total of 132 patients were included in the trial. IAP was measured via the intermittent transvesical technique with only 20 ml of normal saline instilled and using the same zero reference point as in the previous study. They found similar effects

with a mean IAP difference of 1.5 mmHg at 15° and 3.6 mmHg at 30°. Interestingly, this pressure difference was less impressive with IAP equal or above 20 mmHg (0.2 mmHg at 15° and 2.7 mmHg at 30°). This suggests that in patients with impending ACS, IAP could be measured in the semi-recumbent position at 30°. This finding is yet to be confirmed in a larger multi-center trial.

Vasquez et al. [47] measured IAP in 45 trauma patients, comparing the supine position with different HOB elevations as well as the reverse trendelenburg position. The zero reference point was not defined and the authors used 50 ml of normal saline to measure IAP. There was no information about the reverse trendelenburg position and the way the transducer was zero referenced. Nevertheless, overall there was a significant increase in the mean IAP in patients in the supine position compared with those at HOB 45° (10.2 versus 16.7 mmHg). Although the difference in IAP measured in the supine and at 30° with 15° HOB was the highest, a systematic error could have occurred on tilting the bed and potentially changing the zero reference point. Vasquez et al. [47] also found that the BMI was responsible for 25–36% of the total variance in IAP. Similar results were reported by Malbrain where the effect of the upright position (HOB 45°) versus the supine was more significant in patients with higher BMI (10.1 ± 4.4 versus 6.8 ± 3.3 mmHg). The reversed trendelenburg position of the patients increased the IAP by 4.5 mmHg (8.8 ± 3.9 versus 13.3 ± 4.8) [48].

Chionh et al. [29] studied IAP values in three different positions (supine, 30° and 45°) in 58 awake patients who were not receiving mechanical ventilation. They used a portable water manometry, infusing 50–100 ml of normal saline into the bladder with the symphysis pubis as the zero reference point. The median IAP difference in supine versus both upright positions was statistically significant (7.0 versus 8.5 and 10.3 mmHg) and there was a trend toward higher IAP in men versus women. Finally, Cobb et al. [49] measured the IAP in healthy volunteers in different positions and after performing several tasks such as coughing, Valsalva maneuver, standing, jumping or other exercises. They found an average pressure of 16.7 in

Table 4 Effect of body positioning on intra-abdominal pressure measurements

	N (observations)	Supine	HOB 15	HOB 30	HOB 45	Lateral	Reverse trendelenberg	Prone
Malbrain [13] ^a	37 (79)	8.8 ± 3.9	NA	NA	17.1 ± 6.1	6.6 ± 2.9	13.3 ± 4.8	
Chionh [29] ^b	58 (174)	7 (0.7–13.2)	NA	8.5 (2.2–14)	10.3 (2.9–16.2)	NA	NA	
McBeth [45] ^a	37 (300)	13.4 ± 4.2	NA	18.4 ± 4.8	21.5 ± 5.0	NA	NA	
Vasquez [47] ^c	45 (675)	10.2 (8.7– 11.8)	12.4 (10.7– 14.1)	14.0 (12.3– 15.8)	16.7 (14.8– 18.5)	NA	19.3 (16.8–21.8)	
Cheatham [46] ^d	132 (396)	11.8 (11.4– 12.2)	13.3 (12.8– 13.8)	15.4 (14.9– 15.9)	NA	NA	NA	
De Keulenaer [58] ^a	10 (60)	6.6 ± 2.9	NA	NA	NA	11.2 ± 3.3	NA	
Cobb [49] ^a	20 (180)	1.8 ± 2.0	NA	NA	16.7	NA	NA	
Chiomello [55] ^a	11 (60)	8.8 ± 2.1	NA	NA	NA	NA	NA	10.7 ± 2.5
Hering [53] ^a	12 (24)	10 ± 3	NA	NA	NA	NA	NA	13 ± 4 12 (15–7)

^a Data are expressed as means ± SD

^b Data expressed as median ± range

^c Data expressed as means with 95% CI

^d Data expressed as means ± range

the sitting position with a significant correlation between increasing IAP and BMI.

These data, summarized in Table 4, confirm that variation in bladder pressure is a function of body position and that HOB elevation significantly increases IAP. This effect is more pronounced in patients with higher BMI. If we accept that the abdomen behaves as a hydraulic system, then perhaps the descent of intra-abdominal contents by HOB elevation may exert external pressure on the bladder, hence increasing the IAP indirectly when measured via the bladder. Although it is generally accepted that IAP should be measured in the supine position throughout the day, the patient in the ICU will be nursed predominantly in the semi-recumbent position [3, 4]. Thus, patients who have a high risk of developing IAH and ACS could in theory already experience a grade 3 or 4 IAH or even ACS in the elevated position. Whether the increased pressure in that position is an accurate pressure measurement still remains subject to debate. If the abdomen behaves as a liquid-filled container, the pressure we measure (Pascal's law) is a true pressure and therefore should be interpreted according to the above data. We have published a case report of a patient on non-invasive positive pressure ventilation experiencing a cardiac arrest while he was placed in the semi-recumbent position, which instantly increased his IAP and led to ACS [50]. This is an important observation as most ICU patients will be nursed in a semi-recumbent to lateral position, rather than supine, causing an underestimation of the measured IAP that could be significant in the critically ill patient.

It is difficult to make any recommendations with regard to the semi-recumbent position due to differences in methodology, data expression, patient population studied and differences in the degree of the head of bed elevation in all the trials presented here. But based on the available trials [45–48], we conclude that the IAP in the semi-recumbent position at HOB 30° and 45° on average

is 4 and 9 mmHg, respectively, higher than the standard bladder pressure measurement in the supine position and that in patients with impending ACS or grade 3–4 IAH this should be taken into account.

Prone positioning

Turning patients to the prone position to improve gas exchange is still a common practice in many ICUs for patients with acute respiratory distress syndrome (ARDS) and acute lung injury (ALI). Yet, the restriction of the abdomen during that position is associated with an increase in IAP in both humans and animals [51, 52]. Hering [53] showed that the IAP increased in patients with ALI, who were mechanically ventilated in the prone position, but there was no effect on systemic blood flow, renal perfusion, hepato-splanchnic function or gastric mucosal-arterial PCO₂ gradients. Kiefer et al. [54] demonstrated that in nearly 50% of their prone patients with abdominal sepsis, there was an increase in IAP (intra-gastric) by more than 3 mmHg and simultaneously an increase in the gastric mucosal-arterial PCO₂ gradient. Overall, there was no significant change in the mean intra-gastric pressure. Finally, Chiumello et al. [55] found an increase in both gastric and bladder pressures in patients with ALI/ARDS when turned to the prone position with or without thoraco-pelvic support. This data contrasted with others where the prone position did not result in a change in IAP. Martejovic et al. [56] studied the effects of proning on hepato-splanchnic hemodynamics in acute lung injury patients and found no change in IAP, hepato-splanchnic blood flow or gastric mucosal-arterial PCO₂ gradient. Michelet [57] did not find an increase in IAP in patients who were in the prone position on an air-cushioned mattress. However, the IAP was increased and

the plasma disappearance rate of indocyanine green was reduced when foam mattresses were used, probably due to restriction of abdominal movement. Therefore, the type of mattress used is important, but most trials do not specify which device is used. Although this was an interesting finding, it did not influence extravascular lung water and cardiovascular parameters such as cardiac index and mean arterial pressure or oxygenation. Most of these trials, however, did not include patients who were hemodynamically compromised or who at baseline had IAH. Based on the current literature, prone positioning is not significantly detrimental to hepato-splanchnic perfusion or gut mucosal energy balance in patients who are cardiovascularly stable without IAH; however, special precautions should be taken allowing the abdomen to hang free. The relevance of increased IAP in prone positioning still needs to be established.

Lateral position

It is still a common practice in many ICUs to nurse patients every 2–4 h in the left or right lateral semi-recumbent position (interval turning). Turning is generally considered to be another important and effective way of preventing pressure ulcers. There is little evidence describing how the lateral position affects IAP. We conducted a small trial [58] designed to evaluate if the IAP

was influenced by lateral decubitus compared to the standard supine position. Ten patients were included and the mean IAP at different time intervals (morning, afternoon and evening) in the lateral and supine positions were 10.9 ± 2.0 versus 6.6 ± 3.2 mmHg (SD with $P < 0.001$), 11.0 ± 4.0 versus 5.4 ± 2.2 ($P < 0.0005$) and 11.6 ± 3.8 versus 7.8 ± 3.0 . From these data, we concluded that measuring the IAP in the lateral position can lead to falsely elevated readings (depending on the position of the zero reference) and therefore cannot be recommended for current practice.

Conclusion

The abdomen behaves as a hydraulic system with normal IAP of about 5–7 mmHg and with higher baseline levels in morbidly obese patients of about 9–14 mmHg. Measuring IAP via the bladder in the supine position is still the accepted standard method, but in patients in the semi-recumbent position (HOB 30° and 45°) the IAP on average is 4 and 9 mmHg higher. Future research should be focused on developing and validating predictive equations to correct for supine IAP towards the semi-recumbent position. Small increases in IAP in stable patients without IAH, turned prone, have no detrimental effects.

References

1. Malbrain ML, Chiumello D, Pelosi P, Bihari D, Innes R, Ranieri VM, Del Turco M, Wilmer A, Brienza N, Malcangi V, Cohen J, Japiassu A, De Keulenaer BL, Daelemans R, Jacquet L, Laterre PF, Frank G, de Souza P, Cesana B, Gattinoni L, Malbrain MLNG, Chiumello D, Pelosi P, Bihari D, Innes R, Ranieri VM, Del Turco M, Wilmer A, Brienza N, Malcangi V, Cohen J, Japiassu A, De Keulenaer BL, Daelemans R, Jacquet L, Laterre P-F, Frank G, de Souza P, Cesana B, Gattinoni L (2005) Incidence and prognosis of intraabdominal hypertension in a mixed population of critically ill patients: a multiple-center epidemiological study. *Crit Care Med* 33:315–322 Comment in: *Crit Care Med* 2005 Sep;33(9):2150; author reply 2150–2153, *Crit Care Med* 2005 Feb;33(2):447–449
2. Malbrain ML, Chiumello D, Pelosi P, Wilmer A, Brienza N, Malcangi V, Bihari D, Innes R, Cohen J, Singer P, Japiassu A, Kurtop E, De Keulenaer BL, Daelemans R, Del Turco M, Cosimini P, Ranieri M, Jacquet L, Laterre PF, Gattinoni L, Malbrain MLNG, Chiumello D, Pelosi P, Wilmer A, Brienza N, Malcangi V, Bihari D, Innes R, Cohen J, Singer P, Japiassu A, Kurtop E, De Keulenaer BL, Daelemans R, Del Turco M, Ranieri M, Jacquet L, Laterre P-F, Gattinoni L (2004) Prevalence of intra-abdominal hypertension in critically ill patients: a multicentre epidemiological study. *Intensive Care Med* 30:822–829
3. Malbrain ML, Cheatham ML, Kirkpatrick A, Sugrue M, Parr M, De Waele J, Balogh Z, Leppaniemi A, Olvera C, Ivatury R, D'Amours S, Wendon J, Hillman K, Johansson K, Kolkman K, Wilmer A, Malbrain MLNG, Cheatham ML, Kirkpatrick A, Sugrue M, Parr M, De Waele J, Balogh Z, Leppaniemi A, Olvera C, Ivatury R, D'Amours S, Wendon J, Hillman K, Johansson K, Kolkman K, Wilmer A (2006) Results from the International Conference of Experts on Intra-abdominal Hypertension and Abdominal Compartment Syndrome. I. Definitions. *Intensive Care Med* 32:1722–1732
4. Cheatham ML, Malbrain ML, Kirkpatrick A, Sugrue M, Parr M, De Waele J, Balogh Z, Leppaniemi A, Olvera C, Ivatury R, D'Amours S, Wendon J, Hillman K, Wilmer A (2007) Results from the International Conference of Experts on Intra-abdominal Hypertension and Abdominal Compartment Syndrome. II. Recommendations. *Intensive Care Med* 33:951–962

5. Malbrain ML, De Laet I, Cheatham M (2007) Consensus conference definitions and recommendations on intra-abdominal hypertension (IAH) and the abdominal compartment syndrome (ACS): the long road to the final publications, how did we get there? *Acta Clin Belg Suppl* 62:44–59
6. Malbrain ML, Vidts W, Ravvyts M, De Laet I (2008) Acute intensitinal distress syndrome: the importance of intra-abdominal pressure. *Minerva Anesthesiol* 74:657–673
7. Kirkpatrick AW, Brenneman FD, McLean RF, Rapanos T, Boulanger BR (2000) Is clinical examination an accurate indicator of raised intra-abdominal pressure in critically injured patients? *Can J Surg* 43:207–211 [see comment]
8. Sugrue M, Bauman A, Jones F, Bishop G, Flabouris A, Parr M, Stewart A, Hillman K, Deane SA (2002) Clinical examination is an inaccurate predictor of intraabdominal pressure. *World J Surg* 26:1428–1431
9. Fusco MA, Martin RS, Chang MC (2001) Estimation of intra-abdominal pressure by bladder pressure measurement: validity and methodology. *J Trauma* 50:297–302
10. Iberti TJ, Lieber CE, Benjamin E (1989) Determination of intra-abdominal pressure using a transurethral bladder catheter: clinical validation of the technique. *Anesthesiology* 70:47–50
11. Yol S, Kartal A, Tavli S, Tatkan Y (1998) Is urinary bladder pressure a sensitive indicator of intra-abdominal pressure? *Endoscopy* 30:778–780
12. Iberti TJ, Kelly KM, Gentili DR, Hirsch S, Benjamin E (1987) A simple technique to accurately determine intra-abdominal pressure. *Crit Care Med* 15:1140–1142
13. Malbrain ML (2004) Different techniques to measure intra-abdominal pressure (IAP): time for a critical re-appraisal. *Intensive Care Med* 30:357–371 [see comment]
14. Chastre J, Fagon JY (2002) Ventilator-associated pneumonia. *Am J Respir Crit Care Med* 165:867–903
15. Bonten MJ, Kollef MH, Hall JB (2004) Risk factors for ventilator-associated pneumonia: from epidemiology to patient management. *Clin Infect Dis* 38:1141–1149
16. Fowler G (1900) Diffuse septic peritonitis, with special reference to a new method of treatment, namely, the elevated head and trunk posture, to facilitate drainage into the pelvis, with a report of nine consecutive cases of recovery. *The Medical Record, New York*, pp 617–623
17. Defloor T, De Bacquer D, Grypdonck MH (2005) The effect of various combinations of turning and pressure reducing devices on the incidence of pressure ulcers. *Int J Nurs Stud* 42:37–46
18. Knox DM, Anderson TM, Anderson PS (1994) Effects of different turn intervals on skin of healthy older adults. *Adv Wound Care* 7(1):48–52, 54–56
19. Rushmer R (1946) The nature of intraperitoneal and intrarectal pressure. *Am J Physiol*:242–249
20. Duomarco JLRR (1947) La pression intra-abdominal en el hombre. *El Ateneo, Buenos Aires*
21. Campbell EJ, Green JH (1953) The variations in intra-abdominal pressure and the activity of the abdominal muscles during breathing: a study in man. *J Physiol (Lond)* 122:282–290
22. Mills JN (1950) The pressures developed in abdomen and thorax during the Flack tests. *J Physiol (Lond)* 111:368–375
23. Decramer M, De Troyer A, Kelly S, Zocchi L, Macklem PT (1984) Regional differences in abdominal pressure swings in dogs. *J Appl Physiol* 57:1682–1687
24. Loring SH, Yoshino K, Kimball WR, Barnas GM (1994) Gravitational and shear-associated pressure gradients in the abdomen. *J Appl Physiol* 77:1375–1382
25. Tzelepis GE, Nasiff L, McCool FD, Hammond J (1996) Transmission of pressure within the abdomen. *J Appl Physiol* 81:1111–1114
26. Wagoner G (1926) Studies on intra-abdominal pressure. *Am J Med Sci* 171:697–707
27. Overholt R (1931) Intraperitoneal pressure. *Arch Surg* 22:691–700
28. Kron IL, Harman PK, Nolan SP (1984) The measurement of intra-abdominal pressure as a criterion for abdominal re-exploration. *Ann Surg* 199:28–30
29. Chionh JJ, Wei BP, Martin JA, Opdam HI (2006) Determining normal values for intra-abdominal pressure. *ANZ J Surg* 76:1106–1109
30. Sanchez NC, Tenofsky PL, Dort JM, Shen LY, Helmer SD, Smith RS (2001) What is normal intra-abdominal pressure? *Am Surg* 67:243–248
31. Sugerman H, Windsor A, Bessos M, Wolfe L (1997) Intra-abdominal pressure, sagittal abdominal diameter and obesity comorbidity. *J Intern Med* 241:71–79
32. Lambert DM, Marceau S, Forse RA, Lambert DM, Marceau S, Forse RA (2005) Intra-abdominal pressure in the morbidly obese. *Obes Surg* 15:1225–1232
33. Arfvidsson B, Eklof B, Balfour J (2005) Iliofemoral venous pressure correlates with intraabdominal pressure in morbidly obese patients. *Vasc Endovascular Surg* 39:505–509
34. Sugerman HJ, DeMaria EJ, Felton WL 3rd, Nakatsuka M, Sismanis A (1997) Increased intra-abdominal pressure and cardiac filling pressures in obesity-associated pseudotumor cerebri. *Neurology* 49:507–511
35. Sugerman HJ Effects of increased intra-abdominal pressure in severe obesity. *Surg Clin North Am* 81:1063–1075
36. Mullens W, Abrahams Z, Skouri HN, Francis GS, Taylor DO, Starling RC, Paganini E, Tang WH (2008) Elevated intra-abdominal pressure in acute decompensated heart failure: a potential contributor to worsening renal function? *J Am Coll Cardiol* 51:300–306
37. Sussman AM, Boyd CR, Williams JS, DiBenedetto RJ (1991) Effect of positive end-expiratory pressure on intra-abdominal pressure. *South Med J* 84:697–700
38. Hazebroek EJ, Haitisma JJ, Lachmann B, Steyerberg EW, de Bruin RW, Bouvy ND, Bonjer HJ (2002) Impact of carbon dioxide and helium insufflation on cardiorespiratory function during prolonged pneumoperitoneum in an experimental rat model 16:1073–1078
39. Kotzampassi K, Paramythiotis D, Eleftheriadis E (2000) Deterioration of visceral perfusion caused by intra-abdominal hypertension in pigs ventilated with positive end-expiratory pressure. *Surg Today* 30:987–992
40. Guimaraes HPSA, Leal PHR, Barcelos GK, Resque Ap, Amaral JL (2003) Influence of the increase of positive end-expiratory pressure (PEEP) on the intra-abdominal pressure. *Intensive Care Med* 29:S55
41. Ashraf A, Conil JM, Georges B et al (2008) Relation between ventilatory pressures and intra-abdominal pressure. *Crit Care* 12:P324
42. Malbrain MDY, Bertieaux S (2000) Effect of abdominal compression and decompression on cardiorespiratory function and regional perfusion. *Intensive Care Med* 26:S264
43. Ferrer CPE, Molina E (2008) Higher PEEP levels result in small increases in intraabdominal pressure in critical care patients. *Intensive Care Med* 34:S140
44. Gattinoni L, Pelosi P, Suter PM, Pedoto A, Vercesi P, Lissoni A (1998) Acute respiratory distress syndrome caused by pulmonary and extrapulmonary disease. Different syndromes? *Am J Respir Crit Care Med* 158:3–11 [see comment]

45. McBeth PBZD, Widder S, Cheatham M, Zengerink I, Glowa J, Kirkpatrick AW (2007) Effect of patient positioning on intraabdominal pressure monitoring. *Am J Surg* 193:644–647
46. Cheatham MLDWJ, De Keulenaer B, Widder S, Kirkpatrick A, Creswell B, Malbrain M, Bodnar Z, Meija J, Reis R, Parr M, Schulze R, Companso S, the WSACS Clinical Trials Group (2007) Effect of body position on intra-abdominal pressure measurement: a multicenter analysis. *Acta Clin Belgica* 62:246
47. Vasquez DG, Berg-Copas GM, Wetta-Hall R, Vasquez DG, Berg-Copas GM, Wetta-Hall R (2007) Influence of semi-recumbent position on intra-abdominal pressure as measured by bladder pressure. *J Surg Res* 139:280–285
48. Malbrain MVMN, Verbrugghe W, Daelemans R, Lins R (2003) Effects of different body positions on intra-abdominal pressure and dynamic respiratory compliance. *Crit Care* 29:1177–1181
49. Cobb WS, Burns JM, Kercher KW, Matthews BD, James Norton H, Todd Heniford B (2005) Normal intraabdominal pressure in healthy adults. *J Surg Res* 129:231–235
50. De Keulenaer BL, De Backer A, Schepens DR, Daelemans R, Wilmer A, Malbrain ML (2003) Abdominal compartment syndrome related to noninvasive ventilation. *Intensive Care Med* 29:1177–1181
51. Douglas WWRK, Froukje MB, Sessler AD, Marsh HM (1973) Improved oxygenation in patients with acute respiratory distress syndrome. *Am Rev Respir Dis* 115:559–566
52. Takata M, Wise RA, Robotham JL (1990) Effects of abdominal pressure on venous return: abdominal vascular zone conditions. *J Appl Physiol* 69:1961–1972
53. Hering R, Vorwerk R, Wrigge H, Zinslerling J, Schroder S, von Spiegel T, Hoeft A, Putensen C (2002) Prone positioning, systemic hemodynamics, hepatic indocyanine green kinetics, and gastric intramucosal energy balance in patients with acute lung injury. *Intensive Care Med* 28:53–58
54. Kiefer P, Morin A, Putzke C, Wiedeck H, Georgieff M, Radermacher P (2001) Influence of prone position on gastric mucosal–arterial PCO₂ gradients. *Intensive Care Med* 27:1227–1230
55. Chiumello D, Cressoni M, Racagni M, Landi L, Li Bassi G, Polli F, Carlesso E, Gattinoni L (2006) Effects of thoraco-pelvic supports during prone position in patients with acute lung injury/acute respiratory distress syndrome: a physiological study. *Crit Care* 10:R87
56. Matejovic M, Rokyta R Jr, Radermacher P, Krouzecky A, Sramek V, Novak I (2002) Effect of prone position on hepato-splanchnic hemodynamics in acute lung injury. *Intensive Care Med* 28:1750–1755
57. Michelet P, Roch A, Gainnier M, Sainty JM, Auffray JP, Papazian L (2005) Influence of support on intra-abdominal pressure, hepatic kinetics of indocyanine green, extravascular lung water during prone positioning in patients with ARDS: a randomized crossover study. *Crit Care* 9:R251–R257 [see comment; erratum appears in *Crit Care*. 2005 Aug;9(4):308]
58. De Keulenaer BLCG, Maddox I, Powell B, Jenkins I (2007) Intraabdominal pressure measurements in lateral decubitus and supine position. *Acta Clin Belg Suppl* 62:269
59. Sugerman H, Windsor A, Bessos M, Kellum J, Reines H, DeMaria E (1998) Effects of surgically induced weight loss on urinary bladder pressure, sagittal abdominal diameter and obesity comorbidity. *Int J Obes Relat Metab Disord* 22:230–235