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The role of abdominal compliance, the neglected parameter in critically ill patients — a consensus review of 16. Part 1: definitions and pathophysiology

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Abstract

Over the last few decades, increasing attention has been paid to understanding the pathophysiology, aetiology, prognosis, and treatment of elevated intra-abdominal pressure (IAP) in trauma, surgical, and medical patients. However, there is presently a relatively poor understanding of intra-abdominal volume (IAV) and the relationship between IAV and IAP (i.e. abdominal compliance). Consensus definitions on C_{ab} were discussed during the 5th World Congress on Abdominal Compartment Syndrome and a writing committee was formed to develop this article. During the writing process, a systematic and structured Medline and PubMed search was conducted to identify relevant studies relating to the topic. According to the recently updated consensus definitions of the World Society on Abdominal Compartment Syndrome (WSACS), abdominal compliance (C_{ab}) is defined as 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 IAV per change in IAP (mL [mm Hg]$^{-1}$). Importantly, C_{ab} is measured differently than IAP and the abdominal wall (and its compliance) is only a part of the total abdominal pressure-volume (PV) relationship. During an increase in IAV, different phases are encountered: the reshaping, stretching, and pressurisation phases.

The first part of this review article starts with a comprehensive list of the different definitions related to IAP (at baseline, during respiratory variations, at maximal IAV), IAV (at baseline, additional volume, abdominal workspace, maximal and unadapted volume), and abdominal compliance and elastance (i.e. the relationship between IAV and IAP). An historical background on the pathophysiology related to IAP, IAV and C_{ab} follows this. Measurement of C_{ab} is difficult at the bedside and can only be done in a case of change (removal or addition) in IAV. The C_{ab} is one of the most neglected parameters in critically ill patients, although it plays a key role in understanding the deleterious effects of unadapted IAV on IAP and end-organ perfusion. The definitions presented herein will help to understand the key mechanisms in relation to C_{ab} and clinical conditions and should be used for future clinical and basic science research. Specific measurement methods, guidelines and recommendations for clinical management of patients with low C_{ab} are published in a separate review.

Key words: abdominal pressure, abdominal volume, abdominal compliance, abdominal wall, pressure volume relation, diagnosis, treatment, abdominal hypertension, abdominal compartment, laparoscopy, risk factors

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Over the last few decades, increasing attention has been paid to understanding the importance of intra-abdominal pressure (IAP), intra-abdominal hypertension (IAH), and the abdominal compartment syndrome (ACS) [1–6]. However, little attention has been devoted to the potential importance of the structure of the abdominal compartment and its compliance [(i.e. the relationship between IAP and intra-abdominal volume (IAV)]. According to the World Society on Abdominal Compartment Syndrome (WSACS, www.wsacs.org) consensus definitions, abdominal compliance (C_{ab}) is defined as a measure of the ease of abdominal expansion, which is determined by the elasticity of the abdominal wall and diaphragm [3]. It should be expressed as the change in IAV per change in IAP (mL [mm Hg]$^{-1}$). The given C_{ab} (albeit rarely measured) at a certain point, together with the corresponding actual IAV, will determine the resulting IAP. As such, the C_{ab} plays a key role in understanding the deleterious effects of unadapted IAV on IAP and end-organ perfusion, although at present it is one of the most neglected parameters in critically ill patients.

This narrative review article will describe the anatomic features of the abdominal compartment and wall, followed by definitions of IAP, IAV and C_{ab}. In a separate review, we will also look in detail at the different methods for the measurement of abdominal wall compliance and suggest some therapeutic options in patients with a low C_{ab} [7]. It must be said that measurement or estimation of C_{ab} is difficult at the bedside and can only be done in a case of change (removal or addition) in IAV.

METHODS

While preparing for the fifth World Congress on ACS (WCACS), several international surgical, trauma, and medical critical care specialists recognised the lack of existence and uniformity among current definitions for abdominal compliance. The 5th WCACS meeting was held 10–13 August 2011, in Orlando, Florida, USA. Consensus definitions on C_{ab} were extensively discussed during the conference and a writing committee was formed to develop this article. Afterwards, the present co-authors corresponded, providing feedback to questions and issues raised. During the whole writing process, a systematic or structured Medline and PubMed search was conducted to identify relevant studies relating to the topic using the search term 'abdominal compliance'. This search yielded a total of 1,890 references, most of which were not relevant to the subject of this narrative review paper. The remaining abstracts were screened and selected on the basis of relevance, methodology and number of cases included. Full text articles of the selected abstracts were used to supplement the authors' expert opinion and experience. The content of this paper will focus on the definitions and pathophysiology associated with IAP, IAV and C_{ab}. Specific measurement methods, guidelines and recommendations for clinical management of patients with low C_{ab} are published in a separate review [7]. The reader must take into account that, as pointed out in the title, this manuscript is the reflection of the consensus of 16 experts in the field; therefore some of the statements are based on expertise and clinical judgement only. The updated consensus definitions and recommendations on IAH and ACS were recently published elsewhere [3].

DEFINITIONS

THE ABDOMINAL COMPARTMENT

The abdominal compartment is a technical miracle, as the small human abdominal cavity houses 8.5 metres of intestine. Analogous to the head, the abdomen may be considered a closed box. This box has an anchorage above (costal arch) and rigid (spine and pelvis) or partially flexible sides (abdominal wall and diaphragm) filled with organs (Fig. 1) [8]. These organs are perfused by the mesenteric arteries (which have a mesenteric and venous capacitance

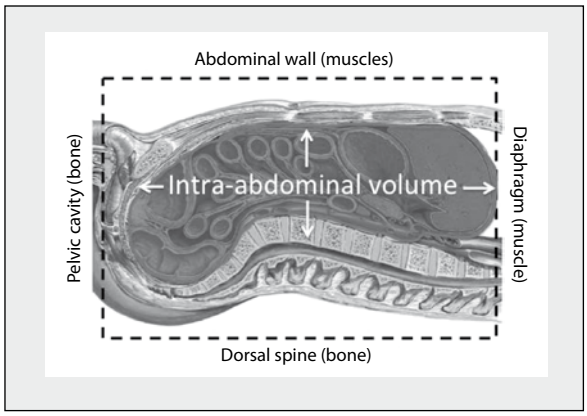

Figure 1. The abdominal cavity borders. The borders of the abdominal cavity are partially rigid bone structures (pelvis and dorsal spine) and partially flexible muscles (abdominal wall and diaphragm)

blood volume) and are surrounded by an intra-abdominal third space filled with peritoneal fluid. The size and/or volume of the abdomen may be affected by the varying location of the diaphragm, the shifting position of the costal arch, the contractions of the abdominal wall, and the amount of contents (air, liquid, or faeces) contained within the intestines [9, 10]. The anatomy of the abdominal cavity affects the possibilities of adaptation to increases in IAV. The caudal and dorsal parts of the abdomen are rigid structures formed by the pelvic bone and dorsal spine. Only the ventral (abdominal wall and muscles) and cranial (diaphragm) parts of the abdominal cavity are flexible [11, 12]. The diaphragm can expand cranially with concomitant negative effects on respiratory function [13–16].

THE ABDOMINAL WALL

The abdominal wall represents the boundaries of the abdominal cavity between the xyphoid bone and costal margins cranially and the iliac and pubis bones of the pelvis caudally [17]. The abdominal wall is split into: the posterior (back; often considered to be the portion posterior or dorsal to the posterior axillary lines); lateral (sides or flanks; often considered to be the portion between the anterior and posterior axillary lines); and anterior (front; often considered to be the portion anterior or ventral to the anterior axillary lines) walls. In this review, only the anterior abdominal wall and its muscles will be discussed [18]. There is a common set of layers covering and forming the abdominal wall: the deepest are the extraperitoneal fat and peritoneal peritoneum (Fig. 2). Superficial to these is the rectus abdominis muscle and its associated fascia. The *m. rectus abdominis* is the central muscle of the anterior abdominal wall. Laterally are three layers of muscle and fascia: the transverse abdominal muscle (lateral *m. transversus abdominis*), the internal (posterolateral *m. obliquus internus*) oblique, and the external oblique (posterolateral *m. obliquus externus*). As the bone structures of spine and pelvis and the posterior muscles (e.g. *m. psoas*) with their fascias are rigid, they cannot be modulated and as such they don't play a relevant role in determining C_{ab}, which is mainly defined by the elasticity of the abdominal wall (anterior and lateral parts) and to a lesser extent the diaphragm muscle [3, 19, 20]. The abdominal muscles have a composite-laminated structure determining their non-linear stretch capacities [21]. The *m. transversus abdominis* fascial fibres are responsible for the transverse stiffness of the abdominal wall, whereas the *m. rectus abdominis* is much more compliant in the sagittal plane [11].

INTRA-ABDOMINAL PRESSURE

Intra-abdominal pressure: The IAP is the steady-state pressure concealed within the abdominal cavity [3].

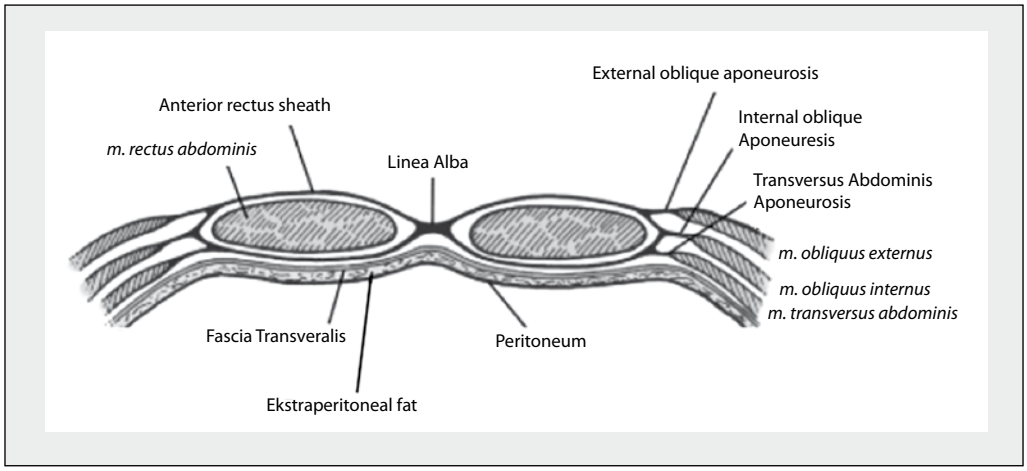

Figure 2. The anatomy of the anterior abdominal wall

IAP measurement: The reference standard for intermittent IAP measurements is via the bladder with a maximal instillation volume of 25 mL of sterile saline. IAP should be expressed in mm Hg and measured at end-expiration in the supine position after ensuring that abdominal muscle contractions are absent and with the transducer zeroed at the level of the midaxillary line.

Baseline IAP: also called resting, starting, static or opening IAP during laparoscopy is the IAP obtained at normal resting conditions. The baseline IAP depends on the amount of 'prefilling' of the abdominal cavity or thus the baseline IAV in proportion to the compliance and reshaping capacity (see further) of the abdominal wall and diaphragm. Normal IAP is approximately 5–7 mm Hg in healthy individuals, and around 10 mm Hg in critically ill adults.

Intra-abdominal hypertension: IAH is defined by a sustained or repeated pathological elevation in IAP ≥ 12 mm Hg. IAH is graded as follows: Grade I, IAP 12–15 mm Hg; Grade II, IAP 16–20 mm Hg; Grade III, IAP 21–25 mm Hg; and Grade IV, IAP > 25 mm Hg.

Abdominal compartment syndrome: ACS is defined as a sustained IAP > 20 mm Hg (with or without an APP < 60 mm Hg) that is associated with new organ dysfunction/failure. In contrast to IAH, ACS is an all-or-nothing phenomenon.

Delta IAP: ΔIAP is calculated as the difference between the end-inspiratory (IAP_{ei}) and the end-expiratory IAP (IAP_{ee}) value:

$$\Delta IAP = IAP_{ei} - IAP_{ee}$$

Abdominal pressure variation: APV is calculated as the difference between the IAP_{ei} and the IAP_{ee} value, or thus ΔIAP divided by the mean IAP (MIAP) and expressed as a percentage:

$$APV = \Delta IAP/MIAP = (IAP_{ei} - IAP_{ee})/MIAP$$

INTRA-ABDOMINAL VOLUME (IAV)

Baseline IAV: also called resting, starting or static IAV which is the IAV at baseline conditions without additional pathologic volume increase or C_{ab} decrease, with corresponding baseline IAP. The baseline IAV in healthy individuals has been found to be around 13 L [22].

Abdominal distension: is defined as a sagittal abdominal diameter (approximately at the level of the umbilicus) higher than the virtual line between xiphoid and symphysis pubis. According to Accarino, abdominal distension is caused by an increase in IAV or abdomino-phrenic displacement and ventro-caudal redistribution of contents [22].

Abdominal workspace: this is the additional IAV that can be added upon the baseline IAV when IAP is limited to a certain pressure (e.g. 14 mm Hg during laparoscopic surgery). The normal workspace during laparoscopy ranges between 3 and 6 litres [23, 24].

Maximal stretched volume: the maximal volume is calculated as the baseline IAV plus the maximal workspace resulting in maximal stretch of the abdominal cavity (from ellipse to sphere on transverse plane). The maximal stretched volume depends on baseline IAP and C_{ab}.

ABDOMINAL COMPLIANCE (C_{ab})

Abdominal compliance: abdominal compliance (C_{ab}) is defined as a measure of the ease of abdominal expansion, which is determined by the elasticity of the abdominal wall and diaphragm [3]. An increased compliance indicates a loss of elastic recoil of the abdominal wall. A decreased compliance means that the same change in IAV will result in a greater change in IAP. It should be expressed as the change in intra-abdominal volume (IAV) per change in IAP (mL [mm Hg]$^{-1}$). Normal C_{ab} is around 250 to 450 mL (mm Hg)$^{-1}$.

Abdominal PV relationship: Importantly, C_{ab} is measured differently than IAP, and the abdominal wall (and its compliance) is only a part of the total abdominal pressure-volume (PV) relationship. The relation between pressure and volume can be expressed by the analysis of PV curves (plotting the resulting changes in IAP values with corresponding increasing/changing IAV, e.g. during laparoscopy), similar to the intracranial and intrathoracic compartment. Analogous to the respiratory system, the abdominal compliance is calculated by the change in volume over the change in pressure or thus [25]:

$$C = \frac{\Delta V}{\Delta P} \quad or\ thus\ C_{ab} = \frac{\Delta IAV}{\Delta IAP}$$

The elastance (E) is

$$E = \frac{\Delta V}{\Delta P} = \frac{1}{C}$$

The relation between abdominal volume and abdominal pressure is curvilinear with an initial linear part followed by an exponential increase once a critical volume is reached. This is illustrated in Figures 3, 4.

PATHOPHYSIOLOGY

INTRA-ABDOMINAL PRESSURE

HISTORICAL BACKGROUND

Poiseuille was first to measure pressures in confined body regions with mathematical accuracy [26]. In France, in Claude Bernard's laboratory, Paul Bert (1833–1886) measured pressures through tubes inserted in the trachea and rectum. He ascribed elevation of the intra-abdominal pressure (IAP) during inspiration to diaphragmatic descent. Similar rectal

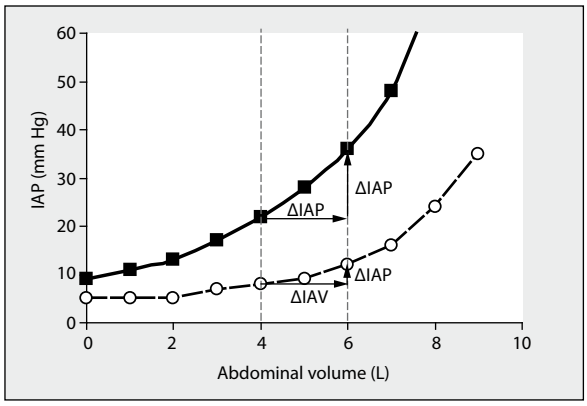

Figure 3. Pressure-volume curve in the abdominal compartment. Abdominal pressure volume curves in a patient with low abdominal compliance (closed squares) and normal compliance (open circles). At a baseline IAV of 4 L, the same 2 L increase in IAV will only lead to a small increase in IAP (5 mm Hg) in a patient with good C_{ab} versus a high increase in IAP (15 mm Hg) in a case of a stiff abdominal wall and diaphragm. The compliance is respectively 133 mL (mm Hg)$^{-1}$ [2,000/(37–22)] versus 400 mL (mm Hg)$^{-1}$ [2,000/(12–7)] for the same change in IAV from 4 to 6 L

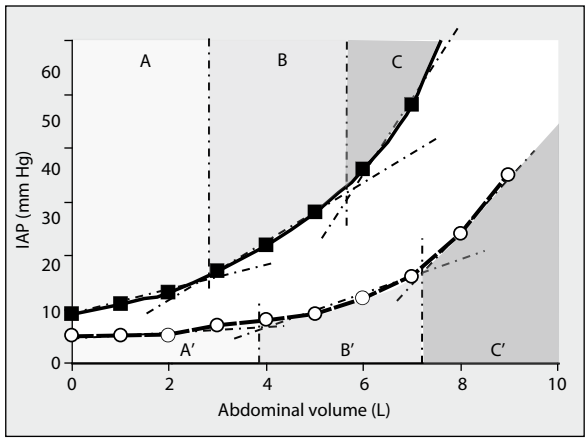

Figure 4. Pressure-volume curve in the abdominal compartment. Schematic representation of different phases during increasing intra-abdominal volume (IAV) in two patients undergoing laparoscopy (CO_2^- insufflation). Shaded areas represent in light grey the reshaping phase (A and A'), in mid-grey the stretching phase (B and B'), and in dark grey the pressurisation phase (C and C'). The accent (') indicates the patient with good abdominal compliance. In the patient with poor compliance, the reshaping phase went from IAV of 0 to 2.8 L (vs 0 to 3.8 L when compliance was normal), the stretching phase from IAV of 2.8 to 5.6 L (vs 3.8 to 7.2 L respectively), and the pressurisation phase from IAV beyond 5.6 L in the patient with low vs 7.2 L in the patient with normal compliance

pressure measurements were performed by Christian Wilhelm Braune (1831–1892), which were subsequently correlated with urine production by E.C. Wendt [27]. Other intravisceral pressure measurements succeeded each other very rapidly, namely in the bladder by Ernst Odebrecht and Mosso and Pellacani and in the uterus by Friedrich Schatz (1841–1920) [26]. These measurements were correlated with absorption of intra-abdominal fluid by Wegner of Germany in 1877 [26]. Direct puncture of the peritoneal cavity is another and direct method of IAP measurement [28]. Once the 20th century era of pressure measurements came along, IAP was evaluated in different physiological and pathological circumstances. Haven Emerson (1874–1957) published his epoch-making results of IAP measurements in 1911 [29]. In the abdominal cavity as a whole, there are no waves travelling through a tube-like system like blood running through vessels. However, there can be dynamic pressure volume changes or the effect of inertia and friction. Again, these factors are very important in the lung and the thorax but less so in the abdomen. This makes the abdomen more accessible.

FLUID MECHANICS

Fluid pressure is the pressure at some point within a fluid, and can occur in an open or closed situation. Pressure in open conditions usually can be approximated as the pressure in 'static' or non-moving conditions. Such conditions conform to principles of fluid statics, with the pressure at any given point of a non-moving (static) fluid being called hydrostatic pressure. Closed bodies of fluid are either 'static,' when the fluid is not moving, or 'dynamic,' when the fluid can move. Fluid dynamics can mean two things, and these are most often not properly distinguished: 1) fluid dynamics can be related to changes over time of pressure, volume and/or wall characteristics; and 2) fluid dynamics can refer to the movement of a fluid, either inside a large recipient (e.g. ascites) or through a recipient with a tube like structure (e.g. blood vessel). If this tube is elastic, specific effects can be observed according to the wall compliance, the fluid density, and the velocity. It is even so that fluid movement itself is less important than the pressure wave propagation when pressure or volume changes rapidly in an elastic tube generating waves that can be described by the linear wave propagation theory. Again these phenomena are not important in the abdominal cavity as it is not a tube-like structure and pressure or volume changes occur slowly, except with positive pressure ventilation at high frequency affecting the abdomen [30].

The pressure in closed conditions conforms to the principles of fluid statics and dynamics. The concepts of static fluids are predominantly attributed to the discoveries of Blaise Pascal and the dynamics to Daniel Bernoulli, while the wave concepts are attributed to Westerhof [31, 32].

Emerson developed an apparatus for direct intraperitoneal pressure measurements and found the pressure to be equal in different parts of the abdomen [29]. Hence the abdomen was considered primarily fluid in character, following Pascal's law [19]. He correlated signs of cardiovascular collapse with elevated IAP and concluded that ascites evacuation may be therapeutic in this setting. This induced Helen Coombs to refine the ideas about the mechanisms of regulation of IAP [33].

In physical sciences, Pascal's law or Pascal's principle states that fluid pressure is the same at all points in a confined space of an incompressible fluid at rest, given that they are positioned at the same absolute height. This also applies if additional pressure is applied on the fluid at any location. The difference of pressure (ΔP) between two different points P, is given by:

$$\Delta P = P_2 - P_1 = -\rho \times g \times (h_2 - h_1)$$

where ρ (rho)=the density of the fluid, g=the acceleration due to the earth's gravity (which varies depending on the elevation of the object relative to sea level), and h_1 and h_2 represent the heights of the two points. The intuitive explanation of this formula is that the change in pressure between two elevations is due to the weight of the fluid between the two heights. Note that the pressure difference does not depend on any additional pressures. Therefore Pascal's law can be interpreted as stating that any change in pressure applied at any given point of the fluid is transmitted undiminished throughout the fluid to all points.

ZERO REFERENCE POINT AND THE INFLUENCE OF BODY POSITION

With regard to IAP measurement, the fact that the abdomen is fluid in character implies that IAP can be measured in any part of the abdomen, but it also raises the important issue of the zero reference point (Fig. 5). The zero reference point is theoretically the middle of the abdominal cavity (as a sphere). Hence, the impact of a non-supine body position becomes important when the measurement point is at a different level (e.g. the midaxillary line or the symphysis) than the theoretical central abdominal reference point.

When IAP is measured via the bladder in a non supine position (i.e. as with a greater than zero degree head of bed (HOB) elevation), the IAP supine can be calculated with the following formula: (in which IAP_α is the IAP measured via the bladder with the HOB at an angle of α°, IAP_{supine} is the IAP obtained in the supine position, SAD the sagittal abdominal diameter, and [C] a correction factor based on α) (Fig. 6):

$$IAP_\alpha = IAP_{supine} + [C]$$

From a theoretical point of view, the correction factor is based on the height difference between the theoretical zero point and the relative position of the bladder and can be estimated as follows:

$$[C] = cos_\alpha \times [SAD]/2$$

As an example, if the IAP_{supine} in a patient with a SAD of 20 cm was 13 mm Hg, the IAP at HOB 45° can be calculated as follows (1 mm Hg = 1.36 cm H_2O):

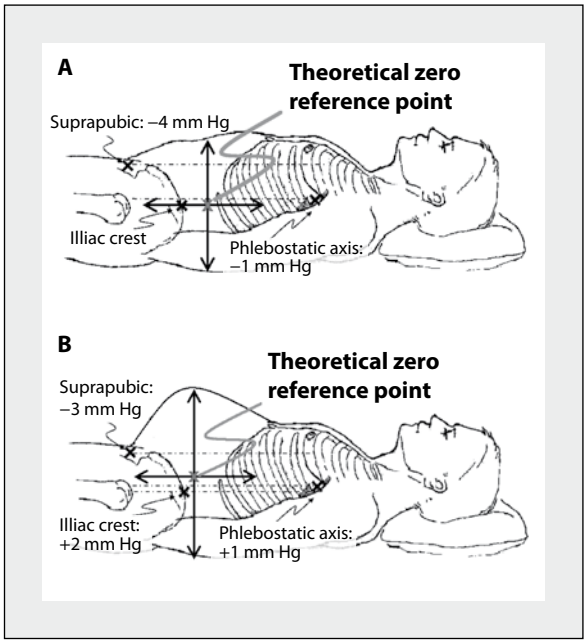

Figure 5. Anatomical location of the different zero reference points in relation to the theoretical zero reference (= the midabdominal position); **A** — location of different zero reference positions (suprapubic, iliac crest, and phlebostatic axis) and their relative position to the theoretical zero reference point (= mid abdominal position) in a patient with normal anthropomorphy and normal IAP. The iliac crest appears to be closest to the theoretical zero reference in this patient, while the IAP is underestimated when the phlebostatic axis (–1 mm Hg) or the symphysis (–4 mm Hg) are used; **B** — location of different zero reference positions in a patient with a distended abdomen and increased IAP, the phlebostatic axis may be closest to the theoretical zero reference in supine position with an overestimation of 1 mm Hg (zero reference via midaxillary line at iliac crest overestimates with 2 mm Hg while the symphysis underestimates true IAP with 3 mm Hg). Adapted from Malbrain et al. [35]

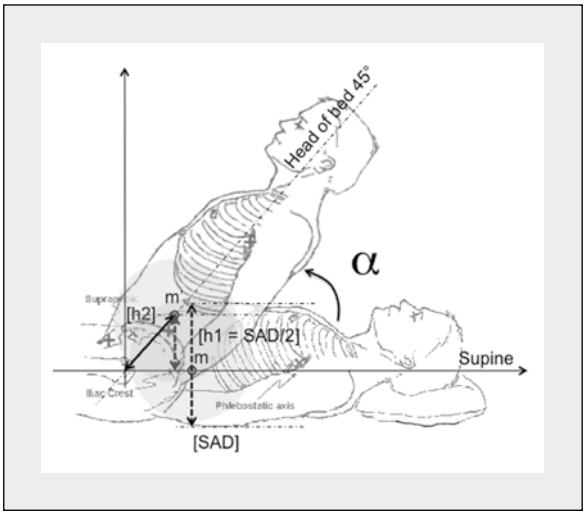

Figure 6. Effect of body position on reference point and baseline intra-abdominal pressure. Schematic drawing of the effect of head of bed (HOB) position on IAP measured via the bladder. The X-axis runs through the anatomical position of the bladder, (m) indicates the middle of the abdomen as the theoretical zero reference point, (m') shows the midabdomen position at HOB 45°, where [h_1] equals half of the sagittal abdominal diameter (SAD) of the patient or thus the distance from the dorsal or ventral side of the abdominal cavity to the middle (m). The height above the X-axis of the new position (m') can then be calculated as follows [h_2] = cos 45° × [h_1] (on the assumption that the abdomen is sphere-shaped, represented by the grey shaded circles). See text for explanation

$$IAP_{45°}\ (cm\ H_2O) = 17.68 + [(cos45°) \times 20/2] =$$
$$= 17.68 + (0.7) \times 10 =$$
$$= 24.68\ cm\ H_2O\ or\ thus\ 18.14\ mm\ Hg$$

However, studies in real life have shown that the 'real' IAP_{α} is higher. McBeth found at HOB 45° an increase in IAP from 13.4 to 21.5 mm Hg [34] and on average the increase in IAP from supine to HOB 45° is around 6 to 8 mm Hg [19]. As such, another coefficient, termed κ, must be accounted for:

$$[C] = \kappa \times [(cosa) \times [SAD]/2]$$

The magnitude of the coefficient κ is related to body anthropomorphy (BMI, body mass index, SAD), the abdominal compliance (C_{ab}), the amount and uniformity of compression, the congestion of the abdominal organs (shear stress), the use of medication (sedation, analgesia, muscle relaxants), and the presence of a positive cumulative fluid balance or ascites [19, 35].

The above-mentioned assumptions depend on the zero reference position and body anthropomorphy. Previous studies looking at increases in IAP with HOB elevation speculated that there was an additional effect of bending the torso and compressing the viscera beyond adding a hydrostatic fluid column. Figure 7 schematically represents the different effects of body position on baseline IAP.

IS THE ABDOMEN A HYDRAULIC SYSTEM?

Pressures in the abdomen were recognised to be atmospheric or positive when Rushmer showed that IAP was related to the height of the hydrostatic column of abdominal contents above the point of measurement, suggesting that the abdomen behaved as a hydraulic system and the pressures within were hydrostatic in nature [36]. Decramer suggested that the abdomen does not behave as a hydraulic system or liquid-filled container because he found that gastric pressure swings in dogs were not simply hydrostatic [37]. Interestingly, the IAP differences observed in the dogs disappeared when the abdominal cavity was filled with 2 L of saline. Loring concluded that there were three factors affecting IAP: gravity, uniform compression, and shear deformation [38]. Uniform compression (e.g. anterior abdominal wall contraction, diaphragmatic contraction, mechanical ventilation, ribcage excursions, and abdominal binding) of

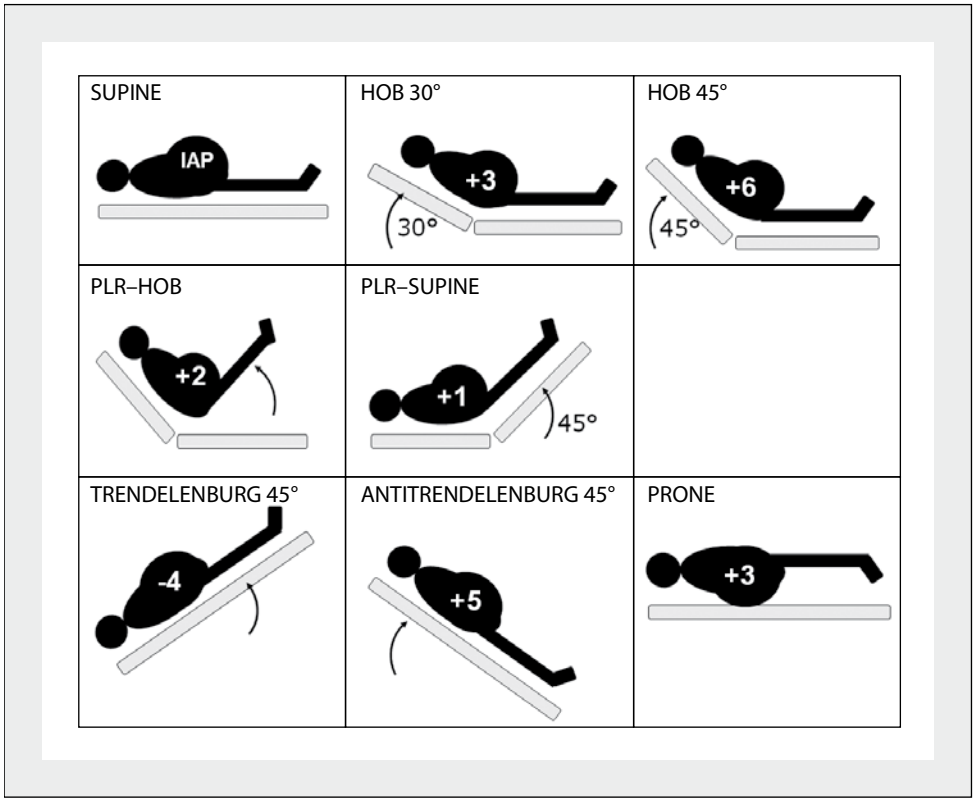

Figure 7. Summary of effects of different body positions on IAP compared to the supine position. Different body positions will have an impact on IAP when compared to IAP obtained in the supine position. The HOB will increase IAP by 3–8 mm Hg. Performing a passive leg raising manoeuvre will increase IAP with 1–2 mm Hg while the Trendelenburg position will lower IAP, and the anti-Trendelenburg and prone positions will increase IAP. The observed effects will be dependent on body anthropomorphy, baseline IAP, and the compliance of the abdominal wall. HOB — head of bed, PLR — passive leg raising

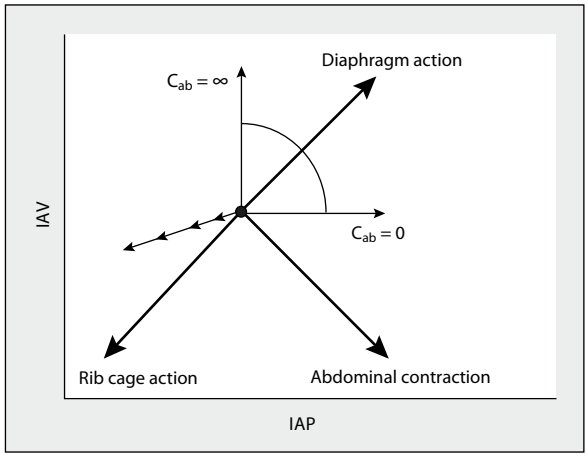

Figure 8. Relationship between intra-abdominal volume, abdominal wall compliance and intra-abdominal pressure. Intra-abdominal pressure (IAP) versus intra-abdominal volume (IAV). The direction of the movement associated with the sole action of the ribcage inspiratory muscles, the abdominal expiratory muscles and the diaphragm are shown. The direction of the latter depends on abdominal compliance (C_{ab}) but is constrained within the sector shown. When the diaphragm contracts, it moves downwards into the abdominal cavity and this displacement will increase IAV with a resulting increase in IAP (depending on C_{ab}). Reductions in IAV will result in a decrease in IAP (small arrows). Adapted from De Keulenaer et al. [19]. See text for explanation

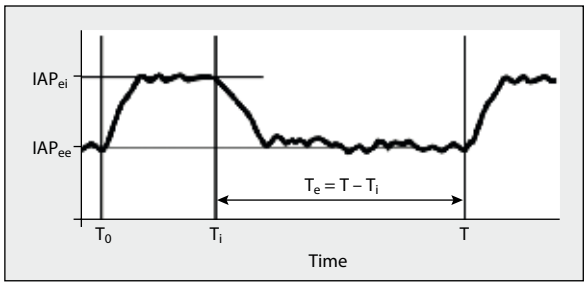

Figure 9. Respiratory variations on IAP curve. The IAP increases with inspiration (IAP_{ei}) and decreases with expiration (IAP_{ee}) before returning to baseline. The slope on in- and expiration correlates with C_{ab} as well as the magnitude of IAP_{ei} and the ΔIAP (= $IAP_{ei} - IAP_{ee}$). The slope (α°) correlates with C_{ab} for the same tidal volume given at a certain flow (L min^{-1}), if the angle α is close to 90°, compliance is close to zero whereas an angle of 0° corresponds to an infinite C_{ab}. Adapted from Ahmadi-Noorbakhsh and Malbrain [46]; T_o — start of inspiration, T_i — inspiratory time, T_e — expiratory time, T — total time of respiratory cycle ($T = T_i + T_e$)

the abdominal contents results in spatially homogeneous changes in IAP that can be superimposed on the gravitational gradient. This is illustrated in Figure 8.

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 ultimately determines if the abdomen behaves as a liquid-filled container. After these animal findings, it was Tzelepis who re-established the concept that the abdomen behaves as a hydraulic system [39]. Based on the available data, we hypothesise 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, a positive fluid balance, and possibly a certain amount of ascites, in the completely supine position.

DYNAMIC PROPERTIES AND CALCULATION OF MEAN IAP

Assuming no respiratory movement, the intra-abdominal compartment is under a constant (static) IAP. This static IAP is determined by two main factors: body posture and body anthropomorphy (e.g. the body mass index of the patient) [40–42]. Other factors contributing to a relatively permanent change in static or baseline IAP are related to changes in IAV: the volume of the solid and hollow viscera, presence of ascites, fluid balance, intravascular blood volume, tumours, pregnancy, peritoneal dialysis, sepsis with capillary leak, intra-abdominal haemorrhage or haematoma. Changes in baseline IAP are also related to changes in C_{ab}, which may occur with burn eschars on the abdominal wall, third space oedema, previous abdominal surgery or pregnancy, the tone of the abdominal wall musculature, or with physical activity [1, 43–45]. Besides this static IAP, frequent movements of diaphragm (as contraction and flattening) pose an additional force on the abdominal contents. Depending on the compliance of the abdominal cavity, of which the abdominal wall is only one part, this force is transferred to pressure waves inside the abdomen [46]. Since the thorax and abdomen are linked compartments, they must be considered as a single unit, as will be discussed separately [7, 47]. Therefore respiratory fluctuations in IAP cannot be ignored [48].

Instead of measuring IAP at end-expiration and in analogy to the calculation of the geometric mean of blood pressure, one must extrapolate this concept to IAP, mimicking systolic arterial pressure by the positive slope of the IAP curve from IAP_{ee} to IAP_{ei} (Fig. 9). Diastolic pressure is related to the negative slope at the end of inspiration; the faster the slope returns to baseline, the better the C_{ab}.

The steady state pressure inside the abdominal cavity is defined as mean IAP (MIAP) and is calculated by dividing the area under the curve by the time of measurement. Therefore, the approximate calculation of the MIAP is proposed as follows:

$$MIAP = \frac{T_i \times IAP_{ei} + T_e \times IAP_{ee}}{T}$$

$$MIAP = \frac{T_i \times IAP_{ei} + (T - T_i) \times IAP_{ee}}{T}$$

$$MIAP = IAP_{ee} + \frac{T_i \times (IAP_{ei} - IAP_{ee})}{T}$$

$$MIAP = IAP_{ee} + \frac{T_i \times (\Delta IAP)}{T}$$

In this formula, T_i is the inspiratory time, T_e is the expiratory time ($T - T_i$), T is the total time of a respiratory cycle ($T_i + T_e$), IAP_{ee} is the end expiratory IAP, and IAP_{ei} is the end inspiratory IAP. The effect of respiration on IAP may be different depending on the method of IAP measurement (i.e. the IAP measured via the stomach is more affected by respiratory movements than the IAP measured via the bladder). Therefore, the effect of respiratory movements on the abdomen can be seen as the distribution of a pressure wave inside the body, which may be attenuated while moving through tissues. Thus, MIAP may not be uniformly equal within the abdominal cavity. As such, it is possible to select the most appropriate IAP measurement with regard to underlying problems in an individual patient. For example, in a trauma patient with a major liver injury, MIAP measurement via the stomach may be more appropriate than via the bladder. In support of this argument, Wendon et al. recently reported that the IAP in the upper and lower abdominal compartments are not identical [49]. Further, in some clinical situations there may be a difference between gastric and bladder pressures and the bladder-to-gastric pressure gradient may give a clue to the diagnosis [50]. Another factor that needs to be taken into account when calculating MIAP is the respiratory rate and the speed of diaphragmatic descent. In the ideal abdomen (in which all of the intra-abdominal contents are non-compressible), the inspiratory IAP (IAP_{ei}) is defined by the following formula:

$$IAP_{ei} = IAP_{stat} + P_{dia}$$

where IAP_{stat} is the static abdominal pressure (i.e. the constant pressure inside the abdomen, without any respiratory movements), and P_{dia} is the pressure caused by diaphragmatic movement.

Diaphragmatic pressure is defined as the force (F) divided by the area bearing the force (the diaphragmatic surface; A):

$$IAP_{ei} = IAP_{stat} + \frac{F}{A}$$

The exerted force to the abdominal viscera is mainly caused by the acceleration (a) of the mass of abdominal organs (m) and the force needed to stretch the abdominal wall (F_{str})

$$IAP_{ei} = IAP_{stat} + \frac{(m \times a) + F_{str}}{A}$$

The acceleration is calculated as the amount of diaphragmatic (or visceral) dislocation (x) divided by the second power of the dislocation time (i.e. the inspiratory time; T_i):

$$IAP_{ei} = IAP_{stat} + \frac{\left(m \times \frac{x}{(T_i)^2}\right) + F_{str}}{A}$$

And, by replacing the pressure resulting from the stretching of the abdominal wall (P_{str}):

$$IAP_{ei} = IAP_{stat} + P_{str} + \frac{m \times x}{(T_i)^2 \times A}$$

The above implies that in the ideal situation, mainly rapid and powerful contractions of the diaphragm (e.g. increased respiratory rate and shorter inspiratory time) will result in a higher inspiratory IAP. The effect of diaphragmatic descent on IAP_{ei} can be used for estimation of C_{ab}, as will be discussed elsewhere [7].

ABDOMINAL COMPLIANCE

ACCOMMODATION OF THE ABDOMINAL CAVITY

In contrast to the intracranial compartment that is confined within a rigid bony structure, the abdominal compartment can change shape during increasing IAV. As explained above, only the abdominal wall and the diaphragm are flexible [11, 20]. During the initial phase of increasing IAV (e.g. laparoscopic insufflation) to the abdominal cavity, IAP only rises minimally (linear ***reshaping*** *phase* from sphere to circle), this is followed by a ***stretching*** *phase* of the rectus abdominis muscle (curvilinear phase) and finally when further IAV is added only small increases in IAV will result in a dramatic increase in IAP (exponential ***pressurisation*** *phase*) (Figs 3, 4) [25, 51]. During the stretching phase, the shape of the abdomen will change from an ellipse to a sphere (this is illustrated in Fig. 10). This change in shape is mainly due to an increase in the antero-posterior and a decrease in the transverse diameter (transverse plane) of the internal abdominal perimeter [11, 20, 22, 52, 53]. To a lesser extent, the cranio-caudal abdominal distance of the rectus abdominis sheath in the sagittal plane increases while the transverse diameter does not [11, 20]. This reshaping capacity results in the difference between the baseline IAV and the maximal stretched IAV, corresponding to the maximal internal abdominal cavity perimeter and surface area and continues until the internal abdominal perimeter develops a sphere-like shape (Fig. 11) [54]. Further addition of IAV will result in stretching only and a dramatic increase in IAP [52].

PREDICTORS FOR STRETCHING AND RESHAPING CAPACITY

The factors determining the reshaping properties of the abdominal wall and diaphragm are not well understood, but the mechanical properties are related to C_{ab}. The stress force on the transverse plane during increas-

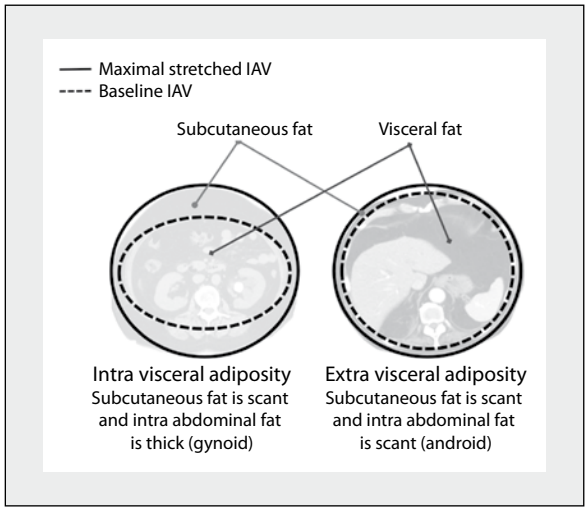

Figure 10. Schematic drawing of android versus gynoid obesity. For the same external abdominal perimeter and body mass index (full line), a different internal abdominal cavity perimeter is observed (dotted line). The external perimeter corresponds to the maximal possible stretched intra-abdominal volume (IAV), while the internal abdominal perimeter represents the resting or baseline IAV

ing IAV is thought to be nearly double that of the sagittal forces [12]. The stretching capacity is influenced by body anthropomorphy (weight, height, BMI), age, gender and visceral versus subcutaneous fat distribution [52]. Comorbidities like chronic obstructive lung disease COPD with emphysema (flattening of diaphragm), fluid overload (tissue and interstitial oedema) or burn injury (with circular eschars) all have negative effects on stretching capacity. Android obesity usually results in increased visceral fat and a sphere-like baseline shape of the abdominal cavity with poor stretching capacity, whereas gynoid obesity presents with more subcutaneous fat for the same BMI or abdominal perimeter (Fig. 11). In a case of gynoid obesity, the internal abdominal perimeter is shaped as an ellipse. Patients with an ellipse-shaped internal perimeter have a huge stretching capacity (and thus a very good abdominal compliance). It is noteworthy that the presence of decreased abdominal wall compliance does not necessarily imply a decreased C_{ab} or vice versa. In patients with previous overdistension of the internal abdominal perimeter (during pregnancy, laparoscopy), the abdominal wall itself becomes less flexible (due to tissue damage and fibrosis) but the reshaping capability is increased and a larger additional workspace IAV can be accommodated before IAP will increase.

ABDOMINAL PRESSURE VOLUME RELATIONSHIP

A linear abdominal PV relationship has been described previously. However, this was mainly in studies where the observed IAP values were below 15 mm Hg and only a few data points were measured [55–59]. During laparoscopy with limitation of insufflation pressures at 12 to 15 mm Hg, the insufflated IAV does not reach a critical point at which an exponential increase in IAP occurs [60]. As discussed above, the initial phase of the PV curve may indeed be linear (as observed during laparoscopy) but the remaining part is curvilinear or rather exponential, as was recently demonstrated in a nice animal experimental study [61] confirming previous animal data in pigs [62] (Figs 3, 4). Human data obtained from studies in patients undergoing laparoscopy or peritoneal dialysis also supports an exponential shaped abdominal PV relationship [60, 63, 64]. Because of this exponential relationship, it is important to know both the shape and the position on the curve, as the actual position

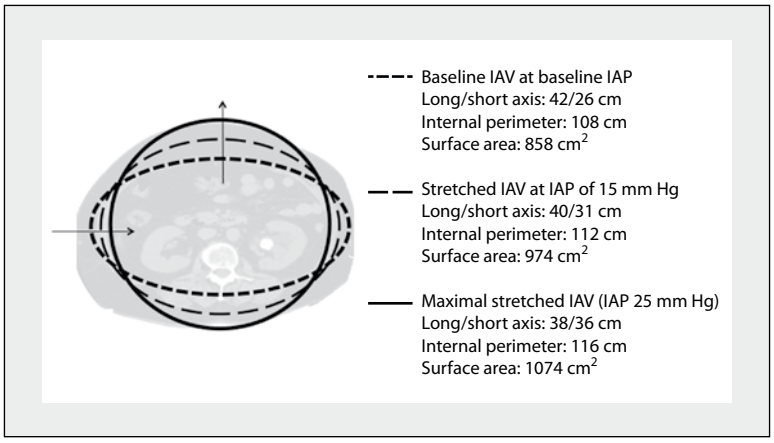

Figure 11. Evolution of internal abdominal cavity perimeter during increase in volume. In a case of gynoid obesity, the internal abdominal perimeter is shaped as an ellipse. Patients with an ellipse-shaped internal perimeter have a huge stretching capacity (and thus a very good abdominal compliance); this is illustrated with the progression of the shape from ellipse (dotted line) at baseline to a sphere (full line) at very high intra-abdominal pressures (IAP), obtained during laparoscopy. The arrows show the centripetal movement of the lateral edges of the ellipse and a centrifugal movement of the cranio-caudal edges. During increase in IAV from baseline to stretched and maximal stretched IAV, the difference between the long and short axis of the ellipse decreases, while the internal perimeter and surface area increase. At maximal stretch, the external and internal abdominal perimeters are equal. Patients with android obesity don't have this reshaping and stretching capability

will determine the corresponding C_{ab}. In patients with IAH, a small increase in IAV may push them into ACS (especially if C_{ab} is low) and vice versa, in patients with ACS a small decrease of IAV (with paracentesis) may result in a dramatic improvement in IAP.

DISCUSSION

Based on the foregoing, we can state that with regard to the basic principles of IAP, IAV and C_{ab}, a number of questions and issues still need to be addressed:

— What are the determinants of IAP, IAV and C_{ab}? Both IAV and IAP and their relation C_{ab} are determined by body anthropomorphy and posture (body position) and they are affected by uniformity of compression, shear deformation and gravity [35, 37, 38]. They depend on the actions of the diaphragm, the ribcage and the abdominal wall muscles [19].

— Is the intraperitoneal pressure the average of the individual intravisceral pressures (intragastric, intrarectal, intrabladder)? Difficult to tell, but different pressures in different parts of the abdomen have been described [49, 50]. This led to the concept of the polycompartment model and syndrome [65–69].

— Is the IAV the average of the individual intravisceral volumes? No, it is the sum of the visceral parenchymateus volume, the blood volume and the peritoneal fluid [70].

— Is the abdomen primary fluid in character or a mixture of tissues with different weights, volumes and densities? From a theoretical point of view, one could define a model of an 'ideal abdomen' (generally considered as fluid in nature without significant gaseous contents, which acts following Pascal's law) and validate the mathematical laws for this model. However, this model then needs to be extended to a more realistic 'clinical abdomen' which is what ICU practitioners encounter [28, 29, 71].

— Can there be a 'local' increase in IAP? A localised compartment syndrome, as seen in patients with pelvic trauma, has been described; this can lead to increased bladder pressures while gastric pressures remain normal [49, 50, 65, 72].

— Can Pascal's law be applied to the abdominal contents? Probably not for 100%, as stated above. One could suggest a grading system for each of the main parameters of the ideal abdomen. For example: a parameter as 'hollow organ gaseous content' should be close to zero for an ideal abdomen. Similarly one could define a parameter as 'abdominal wall elasticity' for the ideal abdomen.

— Is the IAP, IAV or C_{ab} static or dynamic? Depending on the interactions with other organs (like the heart beating in proximity to the stomach) and respiratory movements they can either be static or dynamic or a combination of both [30].

— What is the natural history of IAP, IAV or C_{ab}? This depends on the underlying causes, aetiologic factors and predisposing conditions. With massive fluid loading, C_{ab} will decrease over time. However, one single stretch of the fascia (e.g. a 40 mm Hg applied pressure for four minutes) will rupture collagen fibres and act as a protective mechanism by increasing internal abdominal perimeter and surface area and maximal stretched volume. This explains why previous childbirth or laparoscopy increase C_{ab} [25].

— What is the critical IAP, IAV or C_{ab}? There is no single threshold that defines a pathological state, but it is rather the evolution over time that will define outcome. Therefore continuous monitoring and looking at trends over a certain period to assess the effects of treatment will become more and more important in the near future [73, 74].

— Can the IAV be modulated by medications or other interventions? A positive fluid balance with capillary leak and interstitial oedema will decrease C_{ab}. Paracenthesis will increase C_{ab}, as does previous gravidity and previous abdominal surgery [25, 75]. Increasing age will decrease C_{ab}.

— Are there diurnal and nocturnal variations in IAP, IAV or C_{ab}? Preliminary data (on file) shows that IAP may be lower during the night because of less abdominal muscle contractions. This is especially true in patients with COPD and forced expiration (in this case IAP_{ee} may be higher than IAP_{ei})

— How can anthropomorphic parameters be linked to IAP, IAV or C_{ab}? The presence of abdominal muscles decreases C_{ab}, so that for the same IAV, the IAP will be higher. The BMI and weight are related to baseline IAP while the use of neuromuscular blockers will not change C_{ab} but will lower resting IAP.

— How does BMI affect IAP, IAV or C_{ab}? BMI is related to baseline IAP and central obesity (apple, sphere shape) is related to increased IAV and low C_{ab}.

— What are the determinants of isolated organ compliance (like bladder, stomach)? At the moment we don't have a clue, although there are some conditions with low bladder compliance like chronic renal failure, neurogenic bladder or bladder/pelvic trauma. The instillation volume and temperature may also exert an effect on the tone of the detrusor muscle [76–79].

— Can the data obtained during laparoscopy with regard to C_{ab} be extrapolated to the critically ill? So far we can only conclude that up to an IAP of 10–15 mm Hg, the relation between IAP and IAV is linear. On the other hand, laparoscopy is probably not the ideal model to simulate the effects of increased IAP on end-organ function.

— Is the initial relationship between IAV and IAP always linear? There may be patients and situations where this will not be the case.
— What are the determinants of the curvilinear or exponential shape? The current literature data does not allow providing a fair answer.
— Does gender have an impact on C_{ab}? Only if the patient is a woman who has given birth will the C_{ab} be higher. When the patient is a (male) body builder with hypertrophy of the rectus muscles, the C_{ab} will be lower, but of course there can also be female body builders [80].

CONCLUSIONS

Abdominal compliance is defined as 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 (IAV) per change in IAP (mL [mm Hg]$^{-1}$). The abdominal PV relation is believed to be linear up to pressures of 12–15 mm Hg and increases exponentially afterwards. The C_{ab} in resting conditions at baseline is defined by the baseline IAP and IAV, the external and internal abdominal cavity perimeter and surface area and shape, the additional and maximal stretched volume, the presence of predisposing conditions and comorbidities, as well as tissue properties of the fascia, abdominal wall and diaphragm. As such, the C_{ab} is different and should be seen separately from the abdominal wall and diaphragm compliance with its specific elastic properties. The C_{ab} is one of the most neglected parameters in critically ill patients, although it plays a key role in understanding the deleterious effects of unadapted IAV on IAP and end-organ perfusion.

State-of-the-art definitions for IAP, IAV and C_{ab} are proposed based upon current medical evidence as well as expert opinion. These definitions will help to understand the key mechanisms in relation to C_{ab} and should be used for future clinical and basic science research. Specific measurement methods, guidelines and recommendations for clinical management of patients with low C_{ab} are published in a separate review [7].

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