Abdominal compliance: A bench-to-bedside review

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ABSTRACT: Abdominal compliance (AC) is an important determinant and predictor of available workspace during laparoscopic surgery. Furthermore, critically ill patients with a reduced AC are at an increased risk of developing intra-abdominal hypertension and abdominal compartment syndrome, both of which are associated with high morbidity and mortality. Despite this, AC is a concept that has been neglected in the past.
AC is defined as a measure of the ease of abdominal expansion, expressed as a change in intra-abdominal volume (IAV) per change in intra-abdominal pressure (IAP):
AC = ΔIAV / ΔIAP
AC is a dynamic variable dependent on baseline IAV and IAP as well as abdominal reshaping and stretching capacity. Whereas AC itself can only rarely be measured, it always needs to be considered an important component of IAP. Patients with decreased AC are prone to fulminant development of abdominal compartment syndrome when concomitant risk factors for intra-abdominal hypertension are present.
This review aims to clarify the pressure-volume relationship within the abdominal cavity. It highlights how different conditions and pathologies can affect AC and which management strategies could be applied to avoid serious consequences of decreased AC.
We have pooled all available human data to calculate AC values in patients acutely and chronically exposed to intra-abdominal hypertension and demonstrated an exponential abdominal pressure-volume relationship. Most importantly, patients with high level of IAP have a reduced AC. In these patients, only small reduction in IAV can significantly increase AC and reduce IAPs.
A greater knowledge on AC may help in selecting a better surgical approach and in reducing complications related to intra-abdominal hypertension. (*J Trauma Acute Care Surg*. 2015;78: 1044–1053. Copyright © 2015 Wolters Kluwer Health, Inc. All rights reserved.)

Abdominal compliance (AC) together with the intra-abdominal volume (IAV) will determine the intra-abdominal pressure (IAP). Consequently, reduced AC together with increased IAV can increase IAP and lead to intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS). IAH and ACS are defined as a sustained IAP equal to or greater than 12 mm Hg and as a sustained IAP greater than 20 mm Hg associated with new organ dysfunction/failure, respectively.[1] The incidence of IAH is high in the critically ill patient and is associated with adverse outcome.[2] ACS is a life-threatening condition with high mortality.[2]

Moreover, AC will, for a given intra-abdominal laparoscopic working pressure, determine the resulting IAV and thus the available workspace to perform laparoscopic surgery.[3]

Correct estimation of AC might help avoid complications related to IAH and ACS, by identifying the patient with decreased AC, who is at increased risk of developing IAH and ACS.

Measuring AC is complicated and often not feasible in the clinical setting. However, understanding theoretical concepts and practical aspects of its assessment and management may help clinicians provide optimal health care for critically ill patients as well as patients undergoing laparoscopic surgery.

This review aims to clarify the pressure-volume relationship within the abdominal cavity, the mechanisms influencing AC, and the pathophysiologic effects of reduced AC. We will also discuss treatment options when caring for patients with reduced AC.

PATIENTS AND METHODS

The search of different databases (PubMed, MEDLINE, and EMBASE) with unlimited start date until September 2014 was performed using the search terms *intra-abdominal pressure*, *abdominal pressure*, *abdominal volume*, and *abdominal compliance*.

Articles were also selected from the reference lists. We limited the languages to English, German, and French.

For the creation of abdominal pressure-volume curves, we included all available articles with at least two available human intra-abdominal pressure-volume measurements.

RESULTS

Definition of AC, Abdominal Wall Compliance, and Abdominal Elastance

The World Society of Abdominal Compartment Syndrome (www.wsacs.org) provides the updated consensus definition of *abdominal compliance* as a measure of the ease of abdominal expansion, determined by the elasticity of the abdominal wall and diaphragm and expressed as a change in IAV per change in IAP (L/mm Hg).[1]

$$\text{AC} = \Delta\text{IAV} / \Delta\text{IAP}$$

When describing the abdominal pressure-volume relationship, the term *abdominal compliance* is better suited than abdominal wall compliance because both the abdominal wall and diaphragm are distensible.

Initial increases in IAV lead to a reshaping of the abdominal wall and the diaphragm, only minimally increasing IAP. Further increases in IAV however will lead to stretching and pressurization of the abdomen (see the section on *Reshaping, Stretching, and Pressurization of the Abdomen*). The term *abdominal wall compliance* is reserved to describe the elastic tissue properties of the abdominal wall.

$$\text{Abdominal elastance} = \Delta\text{IAP} / \Delta\text{IAV} = 1 / \text{abdominal compliance}$$

AC is often preferred over the use of abdominal elastance because of the familiarities of clinicians with the concept of respiratory compliance. However, abdominal elastance might be easier to directly derive in clinical practice as the slope (gradient) on an abdominal pressure-volume curve (Fig. 1).

Anatomy of the Abdominal Cavity Enclosure

The anatomy of the abdominal cavity restricts the possibilities of volume expansion. The posterior wall is rigid because of the spine and the retroperitoneal organs; the lower abdominal wall is restricted by the pelvic bones. The upper abdominal wall constitutes the diaphragm, which can, if IAP increases, expand into the chest with negative respiratory effects.[4–7]

The elasticity of the anterior and lateral abdominal wall and, to a lesser extent, the diaphragm determines the AC.[1,8,9] The anterolateral abdominal wall consists of skin, superficial fascia, fat, muscles with their aponeuroses, transverse fascia, and the parietal peritoneum.

The rectus abdominis muscle and its associated fascia are the principal muscle of the anterior, whereas the external oblique, internal oblique, and transverse abdominis muscles form the lateral abdominal wall.

It is thought that the anterior abdominal aponeurosis and, to a lesser degree, the abdominal muscles are the main structural components determining abdominal wall compliance.[10] The abdominal muscles have a composite-laminate structure, the extracellular matrix playing a key role in determining their nonlinear stretch characteristics.[10] Transverse fascial fibers are responsible for the transverse stiffness of the abdominal wall, whereas the rectus abdominis muscle in the sagittal plane is less stiff.[4,5]

Reshaping, Stretching, and Pressurization of the Abdomen

When IAV is added to the abdominal cavity, three different phases can be distinguished as follows: (*a*) the reshaping phase with configuration changes and minimal change in IAP (small slope on the abdominal pressure-volume curve), (*b*) stretching phase through elastic elongation of the abdominal wall and diaphragmatic tissue (medium slope), and (*c*) pressure phase with the characteristic pressure-volume relationship found in a confined space (large slope). All three phases occur in parallel and overlap (Fig. 1).

These dynamic changes are partially dependent on resting (baseline) values of IAV and IAP. Resting IAV is different in each patient; there is no IAV defined to be normal or increased. In 12 healthy adult subjects, total IAV, assessed by computer tomography, was estimated to be approximately 13 L.[11]

Resting IAP (baseline IAP) will depend on the amount of abdominal cavity "prefilling" or the resting IAV in proportion to the reshaping capacity of the abdominal wall and diaphragm. Normal IAP ranges between 5 mm Hg and 7 mm Hg.[8] Assessment of IAP is described elsewhere.[1]

In contrast to the intracranial compartment, adding volume to the abdominal cavity reshapes the abdominal wall and diaphragm.[6,9,12] This reshaping capacity can be described as the difference between "resting/baseline IAV" and the maximum IAV reached without increasing IAP (the "maximal unstressed internal abdominal cavity surface area" before stretching of the abdominal wall occurs). Reshaping capacity depends on age, sex, height, weight, and comorbidities.

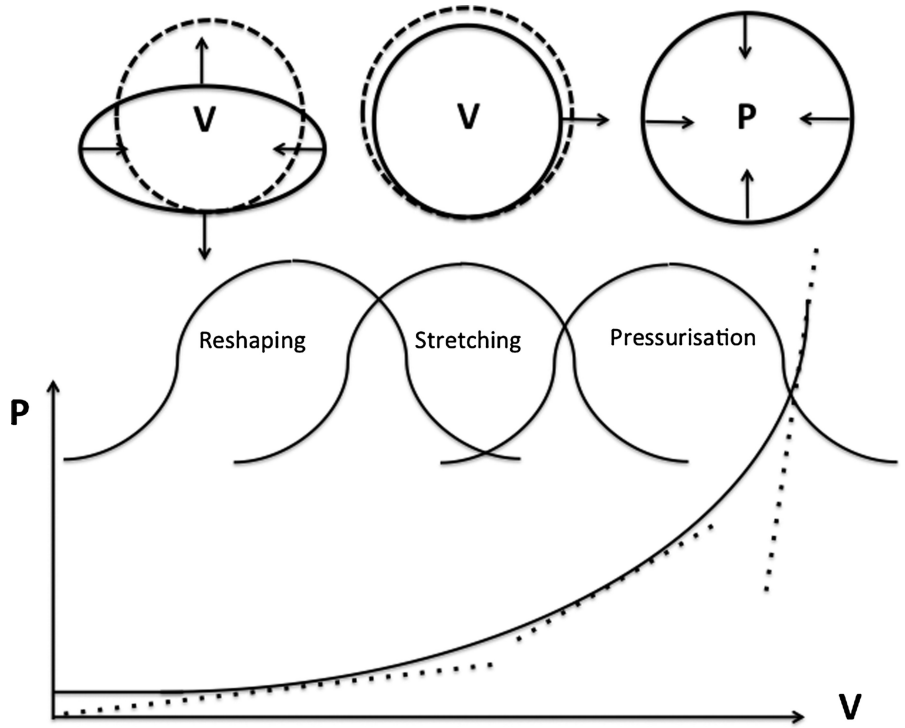

Figure 1. Theoretical abdominal pressure-volume curve showing different abdominal adaptation phases occurring in response to increasing additional IAVs. Theoretical abdominal pressure-volume curve with *P* depicting IAPs on the y-axis and *V* depicting additional IAV on the x-axis. With increasing IAV, three abdominal adaptation phases can be distinguished as follows: initial ''reshaping'' followed by ''stretching'' and finally ''pressurization'' in response to increasing additional IAV. These abdominal adaptation phases most likely occur to some degree in parallel. Abdominal wall circumference is shown in transverse abdominal plane: anteroposterior dimension increases during the reshaping phase, anteroposterior and lateral-lateral dimension increase during the stretching phase, and no further increase in dimensions is expected during the pressurization phase. *Dotted lines* represent the slope or the ΔIAP / ΔIAV or the abdominal elastance being small, moderate, and large during the reshaping, stretching, and pressurization phases.

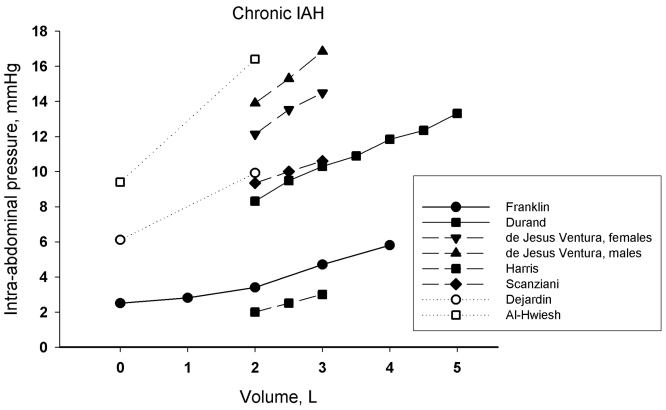

Figure 2. Abdominal pressure-volume relationships in patients receiving peritoneal dialysis (chronic exposure to IAH).

Usually, reshaping continues until the abdominal wall develops a circular shape;[13] additional IAV results in stretching only[14] (Fig. 1).

During the stretching phase, IAV increases in parallel with IAP, with the magnitude of changes depending on compliance of the abdominal wall and diaphragm. A relatively large increase in IAV results in a minor increase in IAP in a person with highly compliant abdominal wall/diaphragm, whereas the same additional IAV results in remarkable increase in IAP in case of a stiff abdominal wall/diaphragm (Fig. 1). Laplace's law has been used to describe the stress forces that occur within the abdominal wall.[5] The stress force in the transverse plane is thought to be nearly double that found in the sagittal plane.[5] At the end of the stretching phase, the maximal stressed internal abdominal cavity surface area is reached. During the pressurization phase, IAP increases exponentially, whereas no further increase in dimensions is expected.

A decreased abdominal wall compliance does not necessarily lead to decreased AC and vice versa. In case of previous overdistension (e.g., after relevant weight loss, pregnancies), the abdominal wall itself may be less distensible because of tissue damage through previous overdistension. However, the "reshaping capacity" is increased, and larger IAV can be accommodated before IAP increases. Hence, the abdominal wall compliance is less important in determining the effective AC.

In summary, AC is dynamic, depending on baseline IAV and IAP as well as reshaping and stretching capacity. The latter is dependent not only on abdominal wall structure and compliance but also on the shape, elasticity, and function of the diaphragm. Mechanisms of thoracoabdominal interactions are described in detail elsewhere.[15,16]

Abdominal Pressure-Volume Relationship

Similar to the intracranial or intrathoracic (respiratory) pressure-volume curves, an abdominal pressure-volume curve can be constructed by plotting resulting IAP values taken at different IAVs (Fig. 1).

Abdominal pressure-volume curves derived from all available data of patients chronically or acutely exposed to IAH are depicted in Figures 2 and 3, respectively.

Abdominal pressure-volume curve has often been described as following a linear relationship,[17–20] but the studied IAPs were mostly less than 15 mm Hg, and/or few IAP/IAV pairs were measured.

Abdominal pressure-volume curve more likely follows an exponential function as recently demonstrated experimentally.[21] Human data derived from laparoscopy and peritoneal dialysis support an exponential abdominal volume-pressure curve.[22,23]

This exponential abdominal pressure-volume relationship (Fig. 1) has fundamental clinical consequences, as the actual AC will depend on its position on the abdominal pressure-volume curve.

Thus, during normal physiologic conditions, an additional predefined IAV only minimally increases IAP. However, when the resting IAV and resting IAP are already elevated (presence of IAH), adding the same IAV will significantly further increase IAP. Categorizing AC values derived from different studies in relation to IAP demonstrates that AC decreases with increasing IAP in a nonlinear AC-IAP manner (Table 3).

Consequently, in a patient experiencing IAH/ACS, removing only small IAVs may dramatically improve the patient's condition. The exponential abdominal pressure-volume relationship also explains why a linear function has been described for laparoscopic workspace. With an IAP of up to 12 mm Hg, the pressure-volume relationship is on the lower end of the exponential curve, showing pseudolinear characteristics.[23]

Indeed, with pressures of up to 15 mm Hg, the pressure-volume relationship seems to be linear[17–19,23] but curves up exponentially when higher IAPs are examined[21–23] (Fig. 1).

Individual pressure-volume curves cannot be predicted, but patients in whom reshaping capacity of abdominal wall (e.g., so-called central, abdominal, or apple-shaped obesity) or diaphragm is limited (e.g., chronic obstructive pulmonary disease [COPD]) are likely to have an unfavorable pressure-volume relationship. Such patients, when undergoing abdominal surgery or being admitted to the intensive care unit (ICU), are at greater risk of IAH/ACS.

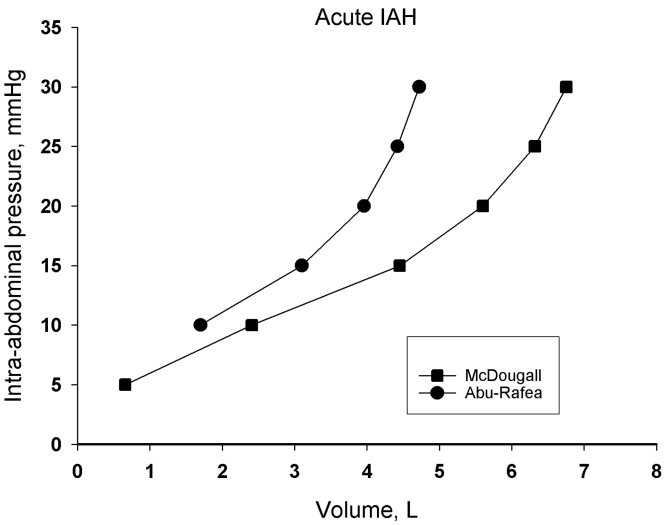

Figure 3. Abdominal pressure-volume relationships in patients with a pneumoperitoneum (acute exposure to IAH).

TABLE 1. AC Studies in Adults

Author	Year	n	Acute vs. Chronic	Procedure	Patient Characteristics	IAP Range, mm Hg	ΔIAV Range (L)	AC (L/mm Hg)	Comments
McDougall et al.[22]	1994	41	Acute	Pneumoperitoneum	Healthy male	5–25	0.43–1.75	0.09–0.41	Exponential
Abu-Rafea et al.[23]	2006	100	Acute	Pneumoperitoneum	Healthy female	10–30	0.30–1.40	0.06–0.28	Exponential
Reed et al.[24]	2006	4	Acute	Abdominal drainage	Abdominal hematoma	12–21	2.22	0.23	Two-point measurement
Reed et al.[24]	2006	4	Acute	Abdominal drainage	Burns	20–27	0.16	0.02	Two-point measurement
Reed et al.[24]	2006	4	Acute	Abdominal drainage	Peritoneal edema	23–30	0.61	0.08	Two-point measurement
Papavramidis et al.[25]	2009	9	Acute	Abdominal drainage	Pancreatic pseudocyst	5–9	2.31	0.55	Two-point measurement
Horer et al.[26]	2012	13	Acute	Abdominal drainage	Abdominal hematoma	16–24	1.52	0.20	Two-point measurement
Franklin et al.[27]	1988	8	Chronic	Peritoneal dialysis		3–6	1.00	0.77–3.33	Exponential character
Durand et al.[28]	1994	20	Chronic	Peritoneal dialysis	Male and female	8–13	0.51–1.18	0.52–0.85	Three-point measurement
de Jesus Ventura et al.[29]	2000	42	Chronic	Peritoneal dialysis	Male	14–17	0.50	0.32–0.36	Three-point measurement
de Jesus Ventura et al.[29]	2000	39	Chronic	Peritoneal dialysis	Female	12–15	0.50	0.36–0.52	Three-point measurement
Harris et al.[30]	2001	12	Chronic	Peritoneal dialysis	Male and female	9–14	0.50	0.19–0.26	Three-point measurement
Scanziani et al.[31]	2003	34	Chronic	Peritoneal dialysis	Male and female	9–11	0.16–0.43	0.26–0.64	Three-point measurement
Paniagua et al.[32]	2004	13	Chronic	Peritoneal dialysis	Male and female	11–15	0.50	0.23–0.26	Three-point measurement
Dejardin et al.[17]	2007	61	Chronic	Peritoneal dialysis	Male and female	6–10	2.0	0.52	Two-point measurement
Al-Hwiesh et al.[33]	2011	25	Chronic	Peritoneal dialysis	Male and female	9–16	2.0	0.29	Two-point measurement
Papavramidis et al.[18]	2011	15	Chronic	Abdominal drainage	Ascites	15–18	1.62	0.43	Two-point measurement

Assessment of AC

AC measurements were performed in humans by assessing IAP at least at two different IAV levels before and after either gas insufflation during laparoscopy,[4,22,23,34,35] intra-abdominal fluid addition (peritoneal dialysis)[17,23] or drainage (ascites, pancreatic fluid, or serous fluid in trauma patients),[18,24,25,36] sometimes in an experimental setting.[21,37]

The derived AC in adult humans ranges between 0.06 mm Hg and 1.92 L/mm Hg (Table 1).[17,18,22–33] AC decreases with increasing IAP levels and is reduced in patients who have not been chronically exposed to high IAP levels.

It was suggested that AC could be estimated by respiratory variation of IAP by calculating ΔIAP (difference between end-inspiratory and end-expiratory IAP) and that if all other parameters remain constant, then a rise in ΔIAP could reflect a decrease in AC.[38]

Laparoscopic Workspace

During laparoscopic surgery, filling the peritoneal cavity with gas lifts the abdominal wall.[4,9,39] The increase of IAV achieved is called the laparoscopic workspace.[3] Recent data suggest that in most patients with low anesthetic risk, laparoscopic cholecystectomy can be successfully performed with peritoneal insufflation pressures of less than 12 mm Hg.[40] There is no comparable data in high-risk patients, however. Preoperative estimation of AC as a tool to identify high-risk patients would be desirable in planning alternative surgical approaches.

In case of a high resting IAP and/or a noncompliant abdominal wall, the workspace is limited. Such insufficient workspace predicts a more difficult operation.[41] In morbidly obese patients, high resting IAP might be a more limiting factor than decreased elasticity of the abdominal wall. The minimum increase of IAV for a successful operation was not defined, but the greater is the laparoscopic workspace, the easier it is to perform laparoscopic manipulations.[42] Therefore, it is important to know that some conditions (previous pregnancy or laparoscopic surgery) may be rather protective, whereas others make the patient prone to insufficient laparoscopic workspace.

Factors and Conditions Influencing AC

Age

Decreased AC in elderly has been reported,[20] probably explained by reduced elastic properties of abdominal wall. Theoretically, decreased AC should be expected also in young athletic patients with strong abdominal muscles (Table 2). This would correspond to personal experience of the authors, but to our knowledge, this has never been studied.

History of Abdominal Surgery or Pregnancy

Previous abdominal surgery or pregnancy has been shown to increase AC.[43,44] This can be explained by a gradual prestretching of the abdominal wall when exposed to higher IAP levels. Even a short period of prestretching (20 minutes) is sufficient to increase AC in pigs.[9] A gradual increase in IAV when maintaining target pneumoperitonal pressures was observed in patients undergoing gynecologic or bariatric laparoscopic surgery.[44] The AC changed less when pneumoperitoneum was applied for a very short time.[44] Patients with a history of laparotomy, laparoscopy, or multiple pregnancies had greater AC at the start but showed smaller increase in AC throughout the procedure. This finding suggests increased reshaping capacity but decreased abdominal wall compliance, that is, a decreased stretching capacity in patients with previous temporary distension of the abdominal wall. Therefore, prestretching even with relatively low IAP applied during laparoscopy seems to cause permanent changes in abdominal wall structure, most likely lengthened fibers with diminished elastic retraction capability.

TABLE 2. Conditions and Pathologies Associated with Changed AC

Condition	AC	Main Pathophysiologic Mechanism	Changes in AC: Resting IAV	Resting IAP	Reshaping Capacity	Abdominal Wall Compliance	Management
Previous pregnancy Multiple	Increased More pronounced	Chronic distension of abdominal wall	≈	≈?	↑	↓	Laparoscopic workspace may be sufficient.
Previous laparoscopy Multiple	Increased More pronounced	Temporary distension of abdominal wall	≈	≈?	↑	↓	Laparoscopic workspace may be sufficient.
Previous open abdominal surgery	Decreased if scarring Increased if hernia	Scarring. Adhesions between the abdominal wall and the intestines. Hernia	≈/↑/↓	≈?	≈/↑/↓	↓	Not predictable. Laparoscopic workspace may be limited.
External bandage, abdominal wall pathology, tight surgical closure	Decreased	Mechanical limitation	≈/↓	≈/↑	↓	↓	Avoid tight closure in risk patients. Measure IAP.
Obesity Abdominal	Decreased More pronounced	Weight/gravity. Increased intra-abdominal fat mass	↑	↑	↓	≈/↓/↑	Consider weight loss before elective laparoscopic surgery.
Athlete	Possibly decreased	Strong/thick abdominal muscles with reduced distendability	≈	≈?	↓	↓	Laparoscopic workspace may be insufficient. Cave IAH in ICU patient
ICU pathology/treatment							
Mechanical ventilation	Decreased	Increased intrathoracic pressure	≈	↑	↓	≈	Limited ventilator pressures. Consider muscle relaxation if ACS.
Intra-abdominal or retroperitoneal fluid collections	Decreased	Fluid collections, tissue edema.	↑	↑	↓	≈	Risk of IAH increased. Measure IAP Consider removal of fluid.
Capillary leak syndrome	Decreased	Tissue edema	↑	↑	↓	↓	Risk of IAH increased. Measure IAP. Consider negative fluid balance.

As a result, maximal internal abdominal cavity surface area increases, and larger IAVs are accommodated at equal pneumoperitoneal working pressures. After reaching maximum reshaping capacity, these previously overstretched fascia and muscular fibers may seem more rigid compared with undamaged fibers.

Two possible mechanisms reducing AC in patients with previous laparotomies are scarring of the abdominal wall, which may result in decreased distendability and adhesions between the abdominal wall and the intestines causing decreased mobility. Why reshaping capacity might be increased in this patient group[44] is not clear. Possibly a reduction in intra-abdominal mass (e.g., following bowel resection) or in abdominal wall muscles or subcutaneous tissue mass following perioperative immobilization contributes.

Obesity

Morbidly obese patients have higher resting IAP between 9 mm Hg and 14 mm Hg,[8,45,46] and central obesity seems to correlate with increased IAP.[45] Morbidly obese patients with predominant abdominal obesity (sometimes referred to as apple-shaped obesity) accordingly have only limited reserve to accommodate more IAV as they start with a higher "resting IAV" and have already reshaped their abdomen into a more spheric shape, resulting in a decreased AC.[8,47]

The effect of the increase in fat in the subcutaneous tissue of obese patients is thought to have a negative effect on the elastic properties of the abdominal wall. At the same time, thin muscular layer might rather increase the abdominal wall compliance. Therefore, the abdominal wall compliance is not directly related to the extent of obesity but is rather individual. The mechanisms for decreased AC in obesity are (1) increased IAV resulting in decreased reshaping capacity (with adipose tissue being an important factor) and (2) gravitational weight of the abdominal wall resulting in increased resting IAP.

No correlation between the thickness of the rectus abdominis muscle and AC in morbidly obese patients has been found.[48] In contrast, it has not been ruled out that well-trained abdominal muscles in the absence of obesity might lead to reduced abdominal wall compliance.

In case of relevant weight loss in obese patients, similarly to women after giving birth, the baseline IAV decreases, whereas the maximal internal abdominal cavity surface area stays relatively unchanged, and therefore, reshaping capacity is increased.

Chronic Medical Conditions

In medical conditions with chronic exposure to higher IAP/IAV (e.g., ascites, peritoneal dialysis), the reshaping capacity (maximal internal abdominal cavity surface area) seems to increase when compared with acute conditions (Figs. 2 and 3, Table 3).

In contrast, COPD is associated with decreased AC because of reduced reshaping capacity of the diaphragm.[49] Moreover, fast increase in IAP leads to respiratory decompensation in patients with severe COPD.

TABLE 3. AC in Relation to the IAP

Acute vs. Chronic	Range of Highest Measured IAP, mm Hg	Mean (95% CI) of AC, L/mm Hg
Acute	0–5	0.45 (0.17 to 0.73)
	5–10	0.27 (0.08 to 0.47)
	10–15	0.17 (0.04 to 0.29)
	15–20	0.06 (0.01 to 0.12)
Chronic	0–5	1.92 (−0.63 to 4.47)
	5–10	0.62 (0.12 to 1.12)
	10–15	0.48 (−0.01 to 0.96)
	15–20	0.35 (0.23 to 0.47)

Data derived from references in Table 1.

Acute Changes in Elastic Properties of the Abdominal Wall

Structural changes of the abdominal wall occur in patients with abdominal wall burns eschars or following surgery.[4,50] Mesh repair for hernia induces abdominal wall stiffness and thereby decreases AC.[51,52] The application of adhesive drapes can change AC without influencing the abdominal wall structure.[53]

Critical Illness

IAH occurs in approximately one third of critically ill patients. Although AC is not directly measured, we know that when IAP increases, AC decreases. The mechanisms of IAH in critically ill patients are multiple such as a large positive cumulative fluid balance, bowel distension, and mechanical ventilation. When a critically ill patient already has a high grade of IAH, small amounts of extra IAV will significantly increase IAP. Vice versa, reducing IAV even in small amounts can dramatically reduce IAP in such patients.

Increased intrathoracic pressure in mechanically ventilated patients with reduced lung compliance (e.g., adult respiratory distress syndrome) or reduced chest wall compliance (e.g., thoracic burn eschars) limits the diaphragmatic reshaping capacity and thereby impairs AC. So far, the influence of raised intrathoracic pressures to further worsen IAH has been shown to be small.[8,54]

Possible Consequences of Decreased AC

The same increase in IAV may have minimal effect on IAP or can cause IAH and ACS in patients with normal versus decreased AC, respectively. IAH may lead to serious cardiovascular, respiratory, abdominal, neurologic, and other adverse effects.[1,16] Increased IAP leads to reduced venous return and thereby necessitates increased fluid loading, starting a vicious circle with further increase in IAP. The most severe form of IAH, ACS, is a situation where very high IAP is a main factor directly leading to hypoperfusion and organ failure. Such situation needs to be prevented, anticipated, and/or avoided whenever possible or, if not, then immediately recognized and managed accordingly. In simple terms, either IAV has to be removed (e.g., fluid removal via renal replacement therapy, ascites drainage, laparotomy with evacuation of a hematoma) or the maximum internal abdominal cavity surface area has to be increased (e.g., by performing a decompressive laparotomy and creating a laparostoma).

Management of Abdominal Surgical Patients With Decreased AC

Optimization of Laparoscopic Workspace

In patients with predicted insufficient laparoscopic workspace, open surgery or weight loss before elective laparoscopy should be considered. In bariatric surgery, which is becoming the most common laparoscopic procedure in most countries in Europe and North America, that may be quite difficult to achieve, however. It has been suggested that in morbidly obese patients with severe cardiac or respiratory dysfunction, decision against laparoscopic surgery could be the best option because these patients are at high risk for intraoperative and postoperative complications related to pneumoperitoneum.[46] Such decisions need to be made on an individual basis.

In addition, during laparoscopy, the body position might help to optimize the laparoscopic workspace. Mulier et al.[55] suggest that the straight Trendelenburg position with 20 degrees results in optimal workspace for lower abdominal laparoscopic surgery in obese patients. At the same time, flexing the legs in reverse Trendelenburg position (resulting in a "beach-chair position") effectively improved workspace for upper abdominal laparoscopic surgery.[55]

Higher working pressures could improve laparoscopic workspace but cannot be recommended because of multiple adverse effects. Laparoscopic pressures greater than 15 mm Hg can be used only for limited time and under cautious monitoring of vital organ functions. If higher working pressures are needed, intermittent desufflation should be considered to limit the negative effects of IAP on organ function. Higher working pressures cannot be routinely recommended for obese patients with high resting IAP because reduction of complications emerging from high IAP has not been confirmed in this patient group; cardiovascular and respiratory comorbidities might even further complicate the situation.

Closure of the Abdomen

In case of open surgery, AC becomes important with closure of the abdomen. Decreased AC can often be recognized only when it is difficult to close the abdomen. Patients with decreased AC are at increased risk of developing IAH and ACS and wound dehiscence postoperatively. Monitoring of IAP at the time of abdominal closure and in the first days after abdominal closure is advisable in patients with decreased AC. If IAP and/or airway plateau pressure remain unacceptably high, abdominal closure may need to be postponed until medical optimization of AC. The risk of open abdomen becomes justified when weighed against the development of ACS or wound dehiscence, especially if early closure is aimed and achieved.

Anesthetic Management

Anesthetic management in patients with decreased AC includes deep muscle relaxation as neuromuscular blocking agents (NMBAs) can improve AC by reducing resting IAP.[56] However, no additional increase in abdominal wall compliance after muscle contractions are fully blocked according to

train-of-four has been shown.[56] The risk of atelectasis and hypoventilation versus high ventilatory pressures needs to be carefully weighed in each individual case.

Management of Critically Ill Patients With Decreased AC

Monitoring of IAP is of utmost importance in critically ill patients[57] especially in patients with reduced AC. It is not clear how moderately increased IAP influences outcome in an individual patient. One should be aware of unpredictable dynamics of IAP dependent on AC, however. To avoid excessive fluid overload and abdominal wall edema after the initial period of resuscitation in the critically ill, a rather restrictive fluid management plan is important because there is evidence that a cumulative positive fluid balance by Day 3 is associated with increased morbidity and mortality.[58] Apart from the judicious use of fluid, fluid removal can be achieved by a furosemide infusion and or via renal replacement therapy.[58]

Percutaneous catheters are increasingly used to drain intra-abdominal fluids and have shown to successfully reduce IAP levels in patients with secondary ACS caused by pancreatitis, liver cirrhosis with ascites, and after massive fluid resuscitation in patients with burns and sepsis.[59,60]

Different modes and ventilatory pressures may have different impacts, but patient-ventilator asynchrony has probably the most negative effect on AC. Breathing against the ventilator always involves contraction of abdominal muscles and leads to increase in IAP.[20] Therefore, sufficient ventilatory pressures should be used to achieve optimal synchrony with pressure support mode.[61] In cases where adequate synchronization is difficult to achieve, the temporary use of NMBA with controlled mode should be considered. Identifying optimal positive end-expiratory pressure level in patients with low AC and already elevated IAP still needs to be clarified.

Avoidance of ACS in patients with decreased AC is a real challenge because the possibilities to acutely increase AC are limited and carry risks. Aggressive medical management can be trialed for a short period. Negative fluid balance may reduce IAV and possibly decrease AC but is suitable and effective only in patients with fluid overload. NMBA can improve AC by reducing resting IAP[56] and possibly slightly increase AC via progressive stretching over time. NMBA should be considered as a temporary measure until other treatment strategies have been implemented.

Verbeke et al.[44] showed that progressive stretching with improvements in AC may take place in relatively short time (during elective laparoscopic procedure), making short-term use of NMBA in the acute setting encouraging.

The last resort treatment of ACS is surgically creating an open abdomen[1] (laparostoma) because the only way to achieve a significant expansion of the IAV is to open the anterior abdominal wall.

CONCLUSION

AC is a measure of the ease of abdominal expansion expressed as change in IAV per change in IAP (L/mm Hg) and is to be distinguished from the abdominal wall compliance. AC can be assessed by measuring the difference in IAP caused after the removal or addition of IAV but is not assessable in patients without these interventions. Available data derived from multiple IAP/IAV measurements suggest that abdominal pressure-volume curve has a linear characteristic in lower but changes to exponential in higher IAP range. Therefore, AC changes the dynamics of IAP and vice versa, making systematic monitoring and interpretation of dynamics of IAP essential.

AC is reduced in different conditions and pathologies.

Future research to address bedside assessment of AC and to refine respective management strategies for different patient groups is warranted.

AUTHORSHIP

All authors equally participated in the acquisition, analysis, and interpretation of data. All authors were actively involved in the drafting and revising of the manuscript. All authors have read and approved the final version of the manuscript.

DISCLOSURE

The authors declare no conflicts of interest.

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