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Effect of increased intra-abdominal pressure and decompressive laparotomy on aerated lung volume distribution

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Abstract: Increased intra-abdominal pressure (IAP) is common in intensive care patients, affecting aerated lung volume distribution. The current study deals with the effect of increased IAP and decompressive laparotomy on aerated lung volume distribution. The serial whole-lung computed tomography scans of 16 patients with increased IAP were retrospectively analyzed between July 2006 and July 2008 and compared to controls. The IAP increased from (12.1±2.3) mmHg on admission to (25.2±3.6) mmHg (P<0.01) before decompressive laparotomy and decreased to (14.7±2.8) mmHg after decompressive laparotomy. Mean time from admission to decompressive laparotomy and length of intensive-care unit (ICU) stay were 26 h and 16.2 d, respectively. The percentage of normally aerated lung volume on admission was significantly lower than that of controls (P<0.01). Prior to decompressive laparotomy, the total lung volume and percentage of normally aerated lung were significantly less in patients compared to controls (P<0.01), and the absolute volume of non-aerated lung and percentage of non-aerated lung were significantly higher in patients (P<0.01). Peak inspiratory pressure, partial pressure of carbon dioxide in arterial blood, and central venous pressure were higher in patients, while the ratio of partial pressure of arterial O_2 to the fraction of inspired O_2 (PaO_2/FIO_2) was decreased relative to controls prior to laparotomy. An approximately 1.8 cm greater cranial displacement of the diaphragm in patients versus controls was observed before laparotomy. The sagittal diameter of the lung at the T6 level was significantly increased compared to controls on admission (P<0.01). After laparotomy, the volume and percentage of non-aerated lung decreased significantly while the percentage of normally aerated lung volume increased significantly (P<0.01). In conclusion, increased IAP decreases total lung volume while increasing non-aerated lung volume. Decompressive laparotomy is associated with resolution of these effects on lung volumes.

Key words: Intra-abdominal pressure, Intra-abdominal hypertension, Lung volume, Decompressive laparotomy, Computed tomography

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1 Introduction

Intra-abdominal hypertension (IAH) and its effects on respiration and abdominal contents have been the subject of study for several decades (Kron *et al*., 1984; Oda *et al*., 2007). IAH has been recognized as a cause of organ dysfunction in critically ill patients, with respiratory and renal dysfunction being among the most prominent effects. Previous studies have shown that IAH increases the risk of lung edema, decreases total respiratory system compliance, and leads to pulmonary hypertension via increased intrathoracic pressure (Quintel *et al*., 2004; Malbrain, 2007). Increased intra-abdominal pressure (IAP) and abdominal expansion might induce compression of the basal lung regions and a cranial shift of the diaphragm (Andersson *et al*., 2005). The combination of capillary leaking, positive fluid balance, and raised IAP poses an important risk for lung edema. A progressive increase in IAP may cause abdominal compartment syndrome (ACS) with organ dysfunction,

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possibly requiring decompressive laparotomy to reverse respiratory failure and other organ dysfunction. Few data are available, however, concerning the extent to which the respiratory dysfunction caused by IAH may be reversed by decompressive laparotomy. The aim of the current study was to measure the effect of increased IAP and subsequent decompressive laparotomy on lung volume and the distribution of aerated lung volumes.

2 Materials and methods

2.1 Patients and experimental protocol

Between July 2006 and July 2008, patients who were admitted to our intensive-care unit (ICU) with suspected increased IAP after primary screen underwent further assessment of transvesicular pressures. Patients were excluded if they had any of the following conditions: (1) pneumonia; (2) chronic heart or lung diseases; (3) malignant carcinoma; (4) decompensated liver cirrhosis; (5) previous lung or abdominal surgery. Of the initial 164 patients with suspected increased IAP, 16 patients had available serial computed tomography (CT) imaging data, an IAP >20 mmHg, one or more organ dysfunction refractory to other medical treatments [defined as ACS by ACS consensus definitions conference (Malbrain *et al*., 2006)], and required decompressive laparotomy. These were enrolled into retrospective analysis. All enrolled cases had been ventilated via endotracheal intubation. Pleural effusions had been drained continuously with an Arrow catheter or chest tube under the guidance of a bedside ultrasound before CT scan. Serial thoracic CT scans had been performed on admission, before and after decompressive laparotomy. CT images of another subject without recorded pulmonary disease were adopted as control.

2.2 IAP measurement

Transvesicular pressure measurement was used for intermittent IAP assessment. After emptying the catheter tubing and bag, a clamp was applied to the catheter tubing just before its connection to the drainage bag. After 25 ml normal saline was injected into the bladder, the catheter tubing was raised vertically to the patient's pelvis with the one-way valve on the end of the drainage bag kept open. Then, the tubing was unclamped, and the height of the fluid column at end-expiration was recorded (1 mmHg= 1.36 cmH_2O) in the supine position with the midaxillary line at the level of the iliac crest used as the zero-reference point (Malbrain *et al*., 2006; Cheatham *et al*., 2007).

2.3 CT imaging and image analysis

Each patient had been transferred to the radiology department with a ward assistant, a nurse and a physician. Patients were sedated with propofol to a Ramsay Scale score of 2–4 throughout the experiment in supine position. In all patients, ventilation was set to a pressure controlled mode with a fraction of inspired oxygen (FIO_2) between 40% and 70% and frequency of 10 to 16 breaths/min. End-expiratory pressure was set at 9 to 15 cmH_2O. Imaging was performed at end-inspiration and whole lung scanning was undertaken by spiral-CT acquisition from the apex to the costophrenic sulcus. Scanning parameters were set to: tube voltage, 120 kV; tube current, 110 mA; and slice thickness, 7.0 mm (Sensation 16, Siemens, Germany). Images were reconstructed using a high-resolution reconstruction algorithm. In each CT image, the lung parenchyma was differentiated from nonpulmonary tissues (ribs, sternum, spine, heart, mediastinum and diaphragm) by a manually drawn circle (Fig. 1). To quantitatively measure the respective volumes of gas and tissue on the CT scans, the lung was divided into four functional compartments according to densitometry (David *et al*., 2005). A density range of −300 to 200 Hounsfield units (HU) was used to define non-aerated lung parenchyma, whereas a −600 to −300 HU reflected poorly aerated lung parenchyma. A density range of −900 to −600 HU was used to define normally aerated lung parenchyma, and the range of −1000 to −900 HU was used to quantify hyperinflated lung parenchyma. The four different lung volumes, i.e., hyperinflated, normally aerated, poorly aerated, and non-aerated, were calculated from all voxels with the Volume software in Siemens Volume Wizard Workstation. The following parameters were calculated: (1) total lung volume and the percentage of aerated lung volume, and (2) sagittal diameter of the lung at the sixth thoracic vertebral level (T6). The total lung volume was derived by manually circling the inner thoracic area, excluding hilar structures in all slices from the apex to the

costophrenic angles, containing pixels with values between −1000 and 1000 HU; and the percentage of specified aerated lung volume was calculated as: (specified aerated lung volume/total lung volume)× 100%. The sagittal diameter was the average of distances measured 5.0 cm from the lateral border of the sternum at both the left and the right sides of the lung at the T6 level (Andersson *et al*., 2005). The vertical distance between the right hemidiaphragm and the T6 level was used as a measure for the change of diaphragmatic position (Fig. 2).

2.4 Statistical analysis

Statistical analysis was performed using SPSS Version 11.0 for Windows (SPSS Inc., USA). Data were tested for normality using the Kolmogorov-Smirnov test and presented as mean±standard deviation (SD). Differences between various IAP values in patients and control were compared using one-way analysis of variance (ANOVA). In the case of significant results, a post hoc multiple comparison analysis was performed using Bonferroni's correction. A P value of less than 0.05 was deemed statistically significant.

3 Results

3.1 Clinical features

Sixteen patients were included in the analysis. Females were predominant (62.5%) in the study group, ranging in age from 20 to 70 years, with a mean age of 45 years. Primary hospital admission diagnoses were trauma in six patients, severe acute pancreatitis for six patients, and sepsis for four patients. The two males and four females in the control group ranged from 24 to 62 years in age, with a mean age of 43 years. The vast majority of patients (75%) had secondary ACS. All the patients were endotracheally intubated. The mean time from admission to decompressive laparotomy and length of ICU stay were 26 h and 16.2 d, respectively (Table 1).

Table 1 Descriptive characteristics of the patients*

Variable	Value
Age (year)	45±10.6
Female	10 (62.5%)
Mean body mass index (kg/m^2)	26.2±5.8
Hospital admission diagnosis	
Trauma	6 (37.5%)
Severe acute pancreatitis	6 (37.5%)
Sepsis	4 (25.0%)
Abdominal compartment syndrome	16 (100%)
Primary	4 (25%)
Secondary	12 (75%)
No. of intubation	16 (100%)
Admission to decompression time (h)	26±11.2
Length of ICU stay (d)	16.2±8.7

* n=16; Values are expressed as number (percentage) or mean±SD

3.2 IAP, hemodynamics and respiratory parameter measurements

Before decompressive laparotomy, there was a significant increase in IAP compared to the level on admission, from (12.1±2.3) to (25.2±3.6) mmHg (P<0.01). After decompression, IAP decreased to (14.7±2.8) mmHg. Fig. 3 shows the IAP before decompression and daily evolution during the first seven days after decompression. The ratio of partial pressure of arterial O_2 to the fraction of inspired O_2 (PaO_2/FIO_2) decreased from (364±25) mmHg to (272±32) mmHg before decompressive laparotomy, climbing significantly to (296±12) mmHg with surgical decompression (P<0.01). A significant increase in peak inspiratory pressure, partial pressure of carbon dioxide in arterial blood, and central venous pressure was noted before decompressive laparotomy, and this dropped remarkably after laparotomy. Mean arterial pressure and heart rate did not differ significantly before and after laparotomy (Table 2).

3.3 Measurements derived from the spiral CT

On admission, the percentage of normally aerated lung volume in the study group was significantly lower than that of controls (P<0.01; Table 3). Prior to decompressive laparotomy, the total lung volume and percentage of normally aerated lung volume were significantly lower compared to those on admission and in controls (P<0.01; Table 3). On the other hand, the absolute volume of non-aerated lung and percentage of non-aerated lung volume were significantly increased compared to those on admission and in controls (P<0.01; Table 3).

With surgical drainage and decompression, nearly 500 ml total lung volume was gained compared to that before decompressive laparotomy, although

Table 2 Effect of IAH on hemodynamic variables and respiratory parameters

Study group (n=16)	IAP (mmHg)	MAP (mmHg)	HR (beat/min)	PIP (cmH_2O)	PaO_2/FIO_2 (mmHg)	$PaCO_2$ (mmHg)	CVP (mmHg)	APACHE II score
On admission	12.1±2.3	76.5±5.8	98.2±21.4	31.7±4.2	364±25	28.0±8.4	11.9±2.8	20.2±3.4
Before DL	25.2±3.6$^{\Delta}$	79.0±4.5	105.9±14.7	42.8±4.0$^{\Delta}$	272±32$^{\Delta}$	52.2±16.8$^{\Delta}$	18.3±7.2$^{\Delta}$	26.4±6.7$^{\Delta}$
Post DL	14.7±2.8	73.0±4.6	99.1±12.3	33.3±4.6	296±12$^{\Delta}$	40.5±12.1	14.0±3.1	22.3±5.9

Data are expressed as mean±SD. IAP: intra-abdominal pressure; MAP: mean arterial pressure; HR: heart rate; PIP: peak inspiratory pressure; PaO_2: partial pressure of oxygen in arterial blood; FIO_2: fraction of inspired oxygen; $PaCO_2$: partial pressure of carbon dioxide in arterial blood; CVP: central venous pressure; APACHE II: acute physiology and chronic health evaluation II; DL: decompressive laparotomy. $^{\Delta}P$<0.01 vs. on admission

Table 3 Effect of IAH on total lung volumes and percentages of different aerated lung volumes

Group	Total lung volume (ml)	Non-aerated lung volume (ml)	Non-aerated lung volume (%)	Poorly aerated lung volume (%)	Normally aerated lung volume (%)	Hyper inflated lung volume (%)
Control (n=6)	3403±656	94±53	2.7±1.9	11.2±2.7	82.5±3.2	3.6±2.2
Study group (n=16)						
On admission	3187±395	361±43	11.3±2.9	15.7±1.8	70.1±6.1*	2.9±1.2
Before DL	2400±347$^{*\Delta\Delta}$	549±63$^{*\Delta\Delta}$	22.9±3.8$^{*\Delta\Delta}$	18.2±1.5$^{*\Delta\Delta}$	56.3±4.5$^{*\Delta\Delta}$	2.6±1.5
Post DL	2897±478	449±86$^{*\Delta\dagger}$	15.5±2.8$^{*\Delta\Delta\dagger}$	16.9±1.0$^{*\Delta\Delta\dagger}$	64.8±3.6$^{*\Delta\Delta\dagger}$	2.8±1.9

Data are expressed as mean±SD. DL: decompressive laparotomy. $^{*}P$<0.01 vs. control; $^{\Delta}P$<0.05, $^{\Delta\Delta}P$<0.01 vs. on admission; $^{\dagger}P$<0.01 vs. before DL

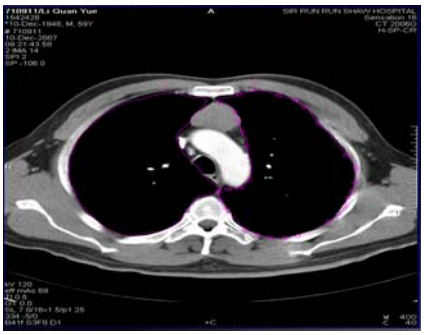

Fig. 1 Demonstration of lung parenchyma differentiated from non-pulmonary tissues by a manually drawn circle (red line)

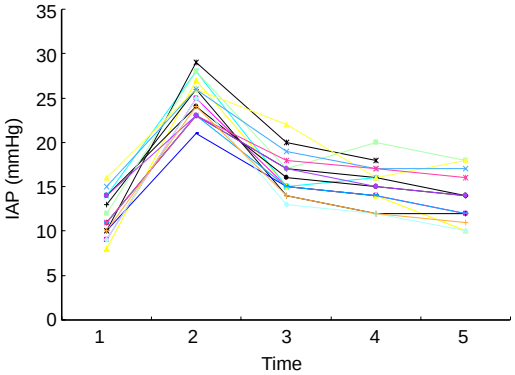

Fig. 3 IAP before decompression and daily evolution during the first seven days after decompression for sixteen patients

Time: 1, on admission; 2, before decompressive laparotomy (DL); 3, the 1st day post DL; 4, the 3rd day post DL; 5, the 7th day post DL

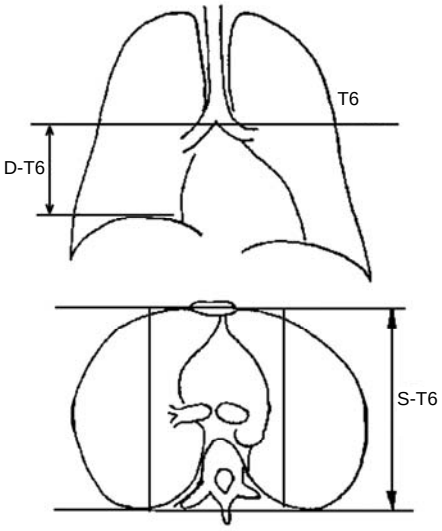

Fig. 2 Distance between right hemidiaphragm and T6 level (D-T6) and sagittal diameters of the lung at T6 level (S-T6)

this was not significant (P=0.162). The volume and percentage of non-aerated lung decreased significantly (P<0.01; Table 3) though these measures remained higher than controls and admission levels (P<0.05 and P<0.01, respectively; Table 3). The percentage of normally aerated lung volume increased significantly (P<0.01; Table 3) after decompression, though these remained lower than either controls or admission levels (P<0.01; Table 3). In all patients, the atelectasis was dorsally localized and the ratio of

abdominal anterior-posterior diameter to transverse diameter was increased prior to decompressive laparotomy, decreasing after decompression (Fig. 4).

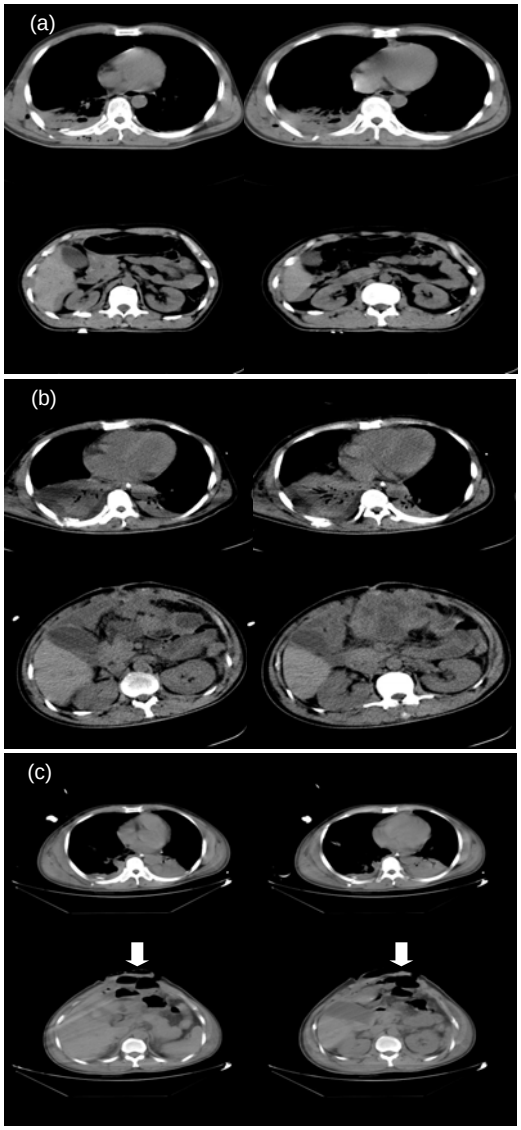

Fig. 4 Computed tomography scans showing atelectasis of lung and abdominal distension at the same level of the same patient on admission (a), and before (b) and after (c) decompressive laparotomy (DL), respectively. Unclosed abdomen incision (white arrows) was found after DL

With increased IAP, sagittal diameters were significantly increased at the T6 level (P<0.01; Table 4). A mean of 1.8 cm cranial displacement of the diaphragm was observed before decompressive laparotomy and the distance between right hemidiaphragm and the T6 level was decreased compared to admission levels or controls (P<0.01; Table 4). After decompressive laparotomy, sagittal diameters returned to the level on admission, while the right hemidiaphragm remained significantly higher than that in controls and as compared to admission levels (P<0.05 and P<0.01, respectively; Table 4).

Table 4 Effect of IAH on sagittal diameter of the lung at T6 level and the distance between right hemidiaphragm and T6 level

Group	S-T6 (cm)	D-T6 (cm)
Control (n=6)	14.1±1.2	8.0±1.8
Study group (n=16)		
On admission	15.1±1.2	7.6±0.6
Before DL	$17.3\pm1.1^{**\Delta}$	$5.8\pm0.5^{**\Delta}$
Post DL	15.4±0.6	$6.6\pm0.5^{*\Delta}$

Data are expressed as mean±SD. S-T6: sagittal diameter of the lung at T6 level; D-T6: distance between right hemidiaphragm and T6 level; DL: decompressive laparotomy. $^{*}P$<0.05, $^{**}P$<0.01 vs. control; $^{\Delta}P$<0.01 vs. on admission

4 Discussion

The current study shows a significant increase in non-aerated lung volumes and a cranial shift of the diaphragm in patients with increased IAP. Decompressive laparotomy provided relief of increased IAP and redistribution of aerated lung volumes toward control levels as measured by spiral CT.

IAH is defined by a sustained or repeated pathologic elevation of IAP ≥12 mmHg (Malbrain, 2004; Malbrain *et al*., 2006; Saggi *et al*., 1998). IAH is an important cause of morbidity and mortality in critically ill patients. It can result from multiple causes, such as abdominal or retroperitoneal bleeding, liver transplantation, massive fluid resuscitation for extra-abdominal trauma, and severe acute pancreatitis (de Waele *et al*., 2006). IAH occurs in up to 4%–15% patients in the ICU, and it is becoming increasingly crucial to measure IAP for high risk patients (Malbrain, 2004). The presence of IAH on admission to ICU has been associated with severe organ dysfunction during the ICU stay, and the development of IAH during an ICU stay is an independent predictor of mortality (Malbrain *et al*., 2005a). IAH is graded as follows: Grade I, IAP 12–15 mmHg; Grade II, IAP 16–20 mmHg; Grade III, IAP 21–25 mmHg; Grade IV, IAP >25 mmHg. ACS is defined as a sustained IAP >20 mmHg (with or without an abdominal

perfusion pressure <60 mmHg) associated with new organ dysfunction/failure (Malbrain *et al*., 2006).

It has been shown that IAH is transmitted to a large extent (25% to 80%) to the thoracic cavity, increasing peak inspiratory airway pressures, reducing functional residual capacity, and further exacerbating the increasing oxygen debt often observed in IAH (Malbrain *et al*., 2005b; Borges *et al*., 2006). Respiratory failure caused by IAH is characterized by a disparity between clinical manifestations and radiographic findings, i.e., severe hypoxemia in patients with only slightly elevated hemidiaphragm and mild basilar atelectasis on chest radiography (Andersson *et al*., 2005). We have postulated that the effect of elevated IAP on aerated lung distribution is similar to acute respiratory distress syndrome (ARDS) because progressive increased IAP might induce secondary ARDS (Malbrain, 2007). Clinical parameters, however, such as analysis of blood gas or lung compliance do not accurately assess this aerated lung volume redistribution. The demonstration of alveolar recruitment and collapse in ARDS has highlighted the value of CT in assessment of the underlying reasons for the changed respiratory mechanics in ARDS (Borges *et al*., 2006; Karmrodt *et al*., 2006). Therefore, in this study, we adopted CT to demonstrate the change of lung volume densitometry in order to define the change of respiratory mechanics in IAH.

In patients who undergo laparoscopic cholecystectomy, pneumoperitoneum at an IAP level of 11–13 mmHg displaces the diaphragm 1.9 cm cranially and total lung volume decreases by approximately 300 ml (Andersson *et al*., 2005). IAP in some critically ill patients or ACS patients is by far higher than the pressure of pneumoperitoneum. In the current study, prior to decompressive laparotomy, IAP averaged 25.2 mmHg. After decompressive laparotomy, it remained elevated at 14.7 mmHg, comparable to the pressure of pneumoperitoneum during laparoscopic surgery. In addition, prior to decompressive laparotomy, the 1.8 cm cranial displacement of the diaphragm was accompanied by a reduction of the total lung volume by approximately 1000 ml compared to the controls. In oleic acid-induced lung injury, beside the reduction of gas volumes, applying an IAP of 20 cmH_2O results in a more than two-fold increase in excess tissue mass, from (30±24)% to (103±37)% (Quintel *et al*., 2004). This means that in acute lung injury/ARDS, any increase in IAP would be dangerous, not only reducing the gas content of an already diseased lung, but also potentially inducing pulmonary edema.

There are some limitations to the present study. First, the enrolled patients were not randomly selected and the sample was small. Second, the underlying diseases leading to IAH were different between patients. Third, the intervals between CT scans were variable. Nevertheless, all enrolled patients had CT scans on admission, before and after decompressive laparotomy, and the level of IAP at each time point was comparable between patients.

Although our understanding of the pathophysiology of IAH has greatly improved, few advances have been made in the treatment of IAH with organ failure until very recently. Fortunately, animal and human studies have shown that abdominal decompressive laparotomy can reverse the pulmonary and abdominal detrimental effects of IAH (de Waele *et al*., 2006; Siebig *et al*., 2008). In the present study, with the relief of IAH after decompressive laparotomy, total lung volume and PaO_2/FIO_2 improved while the diaphragmatic displacement and the non-aerated lung volume decreased (Although it improved, the PaO_2/FIO_2 after decompressive laparotomy remained below the normal ranges). This is consistent with the fact that, although a number of physiological values improved, IAP remains moderately to severely increased in a number of patients after decompressive laparotomy (Balogh *et al*., 2003). Therefore careful ongoing observation is required and repeated decompressive laparotomy may be needed if the IAP remains >20 mmHg and organ dysfunction persists.

Surgical abdominal decompression has long been the standard treatment for the patient who develops ACS (de Waele *et al*., 2006). The timing of surgical intervention for IAH is controversial. It is difficult to specify which clinical condition should lead to the decision to undertake surgical decompression. The World Society of the Abdominal Compartment Syndrome (WSACS) (www.wsacs.org) has described a graded approach to the management of IAH/ACS which can be used to avoid the need for surgical decompression in many patients. In this algorithm, percutaneous drainage is considered as a potential therapeutic option for those IAH/ACS cases that are due to free intra-abdominal fluid, air, abscess,

or blood, before proceeding with surgical decompression (Cheatham *et al*., 2009). In patients with percutaneous drainage who fail to reduce IAP and restore adequate visceral perfusion or those with ACS that is refractory to other treatment options, however, urgent surgical intervention should be considered (Malbrain *et al*., 2009). In a recent review and analysis of 18 studies of the effect of surgical decompressive laparotomy on IAP and the outcomes, the mean interval from admission to the hospital or from the previous surgical intervention to decompressive laparotomy varied from 12 to 38 h (de Waele *et al*., 2006). Another problem is that although decompressive laparotomy can lead to recovery from respiratory failure, the risk of complications, such as massive intra-abdominal bleeding, persisting open abdomen and subsequent extensive abdominal wall reconstruction, needs to be taken into account.

In conclusion, the current study shows that increased IAP displaces the diaphragm cranially and increases non-aerated lung volumes. Decompressive laparotomy decreases IAP and redistributes aerated lung volumes toward control values.

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