Effect of intra-abdominal pressure on respiratory function in patients undergoing ventral hernia repair

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IAP monitoring was performed via both a balloon-tipped nasogastric probe [intragastric pressure (IGP), CIMON, Pulsion Medical Systems, Munich, Germany] and a urinary catheter [intrabladder pressure (IBP), UnoMeterAbdo-Pressure Kit, Unomedical, Denmark] on five consecutive stages: (1) after tracheal intubation (AI); (2) after ventral hernia repair; (3) at the end of surgery; (4) during spontaneous breathing trial through the endotracheal tube; and (5) at 1 h after tracheal extubation. The patients were in the complete supine position during all study stages.

RESULTS: The IAP (measured via both techniques) increased on average by 12% during surgery compared to AI (P < 0.02) and by 43% during spontaneous breathing through the endotracheal tube (P < 0.01). In parallel, the gradient between PaCO2 and EtCO2 [P(a-et)CO2] rose significantly, reaching a maximum during the spontaneous breathing trial. The PaO2/FIO2 decreased by 30% one hour after tracheal extubation (P = 0.02). The dynamic compliance of respiratory system reduced intraoperatively by 15%-20% (P < 0.025). At all stages, we observed a significant correlation between IGP and IBP (r = 0.65-0.81, P < 0.01) with a mean bias varying from -0.19 mmHg (2SD 7.25 mmHg) to -1.06 mm Hg (2SD 8.04 mmHg) depending on the study stage. Taking all paired measurements together (n = 133), the median IGP was 8.0 (5.5-11.0) mmHg and the median IBP was 8.8 (5.8-13.1) mmHg. The overall r’ value (n = 30) was 0.76 (P < 0.0001). Bland and Altman analysis showed an overall bias for the mean values per patient of 0.6 mmHg (2SD 4.2 mmHg) with percentage error of 45.6%. Looking at changes in IAP between the different study stages, we found an excellent concordance coefficient of 94.9% comparing ∆IBP and ∆IGP (n = 117).

CONCLUSION: During ventral hernia repair, the IAP rise is accompanied by changes in P(a-et)CO2 and PaO2/FIO2-ratio. Estimation of IAP via IGP or IBP dem-

Abstract

AIM: To determine the influence of intra-abdominal pressure (IAP) on respiratory function after surgical repair of ventral hernia and to compare two different methods of IAP measurement during the perioperative period.

METHODS: Thirty adult patients after elective repair of ventral hernia were enrolled into this prospective study.
onstrated excellent concordance.

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Key words: Intra-abdominal pressure; Gastric pressure; Bladder pressure; Intra-abdominal hypertension; Hernia; Oxygenation; Respiratory function

Core tip: The surgical repair of ventral hernia is accompanied by a rise of intra-abdominal pressure, a deterioration of CO₂ elimination and a decrease in arterial oxygenation. The measurements of intra-abdominal pressure using nasogastric tube and urinary catheter demonstrate a close agreement between both methods; thus, both these methods can be used in clinical practice.

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INTRODUCTION

Abdominal wall reconstruction during ventral hernia repair can be associated with perioperative intra-abdominal hypertension (IAH), respiratory dysfunction and complications[1-3]. The relationship between intra-abdominal pressure (IAP) and respiratory function was demonstrated for the first time in 1863[4]. Today the negative effects of IAH on respiratory system have been investigated in a large number of studies[5-7]. Development of IAH decreases chest wall compliance and functional residual capacity, shifts the end-expiratory position of the diaphragm, and leads to development of atelectasis. Thus, it may affect blood oxygenation and carbon dioxide elimination[2,5]. The abdominal compartment syndrome (ACS) is defined as a sustained increase in IAP exceeding 20 mmHg with the presence of new organ dysfunctions that is associated with significant morbidity and mortality[6-8]. Therefore, in patients with risk factors for IAH it is necessary to measure IAP with simultaneous evaluation of respiratory function[6,8,13].

There are direct and indirect methods for the measurement of IAP[9]. The direct technique involves estimation of IAP through the placement of intraperitoneal catheter. This method however is invasive and thus not applicable in most clinical situations[9]. Many simple and less invasive indirect methods are most often used in routine clinical practice for IAP estimation. These methods include measurement of pressure in hollow organs of the abdomen or pelvis cavity such as bladder, stomach, intestine, and uterus[9-14]. Among them, the intrabladder technique using Foley catheter has been forwarded as the “gold” standard for IAP estimation in the consensus definitions report of the World Society on Abdominal Compartment Syndrome (WSACS, www.wscas.org)[6,7]. Another indirect technique is the measurement of IAP via nasal gastric probe[16,18,21].

The increase of the gradient between PaCO₂ and EtCO₂ [P(a-et)CO₂] can reflect changes in respiratory function. Moreover, the P(a-et)CO₂ value demonstrates an association with dead space volume and severity of pulmonary ventilation-perfusion mismatch due to IAH[19,22]. In 1984, Murray et al[23] suggested that P(a-et)CO₂ might be a more sensitive indicator in the search of the optimal positive end-expiratory pressure (PEEP) than changes in lung shunt or PaO₂. Later, it has been shown that monitoring of dead space and P(a-et)CO₂ was useful for detecting lung collapse[24]. In a porcine model of IAH, it has been demonstrated that assessment of P(a-et)CO₂ might help to evaluate the severity of atelectasis during laparoscopic surgery; however these findings still need to be validated in different clinical settings[25].

Today, there are a number of concerns regarding indirect evaluation of IAP. The intrabladder pressure measurements can be unreliable in case of low intrinsic bladder compliance (as in patients with chronic renal failure and anuria), and bladder trauma[9,14,17,28]. The intragastric estimation of IAP can be incorrect during intestinal obstruction with large volume gastric aspirate and partial or total gastric resection[29]. Therefore, these methods need validation in selected categories of patients at risk for IAH.

Thus, the goals of our study were to determine the influence of IAP on respiratory function after surgical repair of ventral hernia and to compare two different methods of IAP measurement during the perioperative period.

MATERIALS AND METHODS

The study was approved by the Medical Ethics Committee of Northern State Medical University, Arkhangelsk, Russian Federation. Written informed consent was obtained from every patient or next of kin.

This prospective study was performed in a 900-bed university hospital (City Hospital#1 of Arkhangelsk). From June 2011 to March 2012, we enrolled 30 adult (age > 18 years) patients (10 males and 20 females) after elective incisional ventral hernia repair, using an open technique. The patients were excluded from the study if they were above 75 years of age, were pregnant or required simultaneous operation, other than ventral hernia repair, or participated in other clinical investigations. Before the procedure all patients received standard premedication with diazepam and omeprazole according to a standard protocol.

All patients received monitoring of IAP via both intragastric pressure (IGP) with a balloon-tipped nasogastric probe (CIMON, Pulsion Medical Systems, Germany) and intrabladder pressure (IBP) with a urinary catheter system.
may be used interchangeably if $r^2$ (Pearson correlation coefficient) is $> 0.6$, if the differences between bias and limits of agreement (1.96 SD) are not clinically important and if the percentage error is less than 35%. Finally, the ability of IGP to track changes or trends in IBP was assessed by plotting ΔIBP against ΔIGP during the same time interval (four quadrants trend plot). The concordance coefficient is calculated as the percentage of pairs with the same direction of change. Based on clinical relevance, the concordance should be $> 90\%$ when pairs with both a ΔIBP and ΔIGP ≤ ± 2.5 mmHg are excluded for analysis.

**RESULTS**

The median age was 61 (53-69) years, weight 89 (73-103) kg, and body mass index (BMI) 31 (29-36) kg/m$^2$, respectively. The median size of incisional hernia was 244 (170-415) cm$^2$. The fluid balance for the first day after operation was 1700 (1325-2000) mL. According to both techniques of measurement, during surgery IAP increased on average by 12% from AI ($P = 0.013$ and $P = 0.002$ for IBP and IGP, respectively; Figure 1). The maximal increase of IAP by 43% was observed during spontaneous breathing through the endotracheal tube: up to 10 (9-15) mmHg for IBP and 10 (8-12) mmHg for IGP ($P = 0.001$). At the end of the investigation, IAP still exceeded the AI values ($P = 0.003$ and $P = 0.006$ for IBP and IGP, respectively). The abdominal perfusion pressure (APP), blood gases (PaCO$_2$ and EtCO$_2$) and arterial lactate concentrations are presented in Table 1. The values of APP rose significantly after transfer to spontaneous breathing ($P < 0.001$ for both IBP and IGP) in parallel with the increase in PaCO$_2$ and EtCO$_2$ ($P < 0.013$). The gradient between PaCO$_2$ and EtCO$_2$ also rose significantly reaching a peak during spontaneous breathing trial ($P = 0.02$) (Figure 2A). The mean arterial lactate concentration did not change significantly and did not exceed 1 mmol/L throughout the study.

The oxygenation index (PaO$_2$/FiO$_2$) was decreased by 30% one hour after tracheal extubation ($P = 0.02$ vs AI; Figure 2B). These changes were delayed as compared to the increase of IAP and P(a-et)CO$_2$.

Tidal volume during the study did not change significantly (not shown). However, the dynamic compliance of the respiratory system decreased with 15%-20% both after hernia repair and at the end of surgery ($P < 0.025$; Table 1).

At all stages, we found a significant correlation between the two methods of IAP measurement ($r = 0.65$-$0.81$, $P < 0.01$). The mean bias between gastric and urinary methods of IAP monitoring varied during the study from -0.19 mmHg (2SD 7.25 mmHg) to -1.06 mmHg (2SD 8.04 mmHg) (Table 2). Taking all paired measurements together ($n = 133$), the median IGP was 8.0 (5.5-11.0) mmHg and the median IBP was 8.8 (5.8-13.1) mmHg. In total, 4 outliers, related to measurement error, or abdominal muscle contraction or migrating motor complex activity (2 paired measurements each in 2
patients) were excluded from further analysis. Pearson correlation coefficient comparing mean IBP and IGP values \((n = 30)\) showed a \(r^2\) of 0.76 \((P < 0.0001)\). Figure 3 demonstrates the regression analysis between mean IBP and IGP values per patient. Bland-Altman analysis of all paired measurements \((n = 128)\) showed an overall bias of \(-0.7 ± 2.9\) mm Hg (with limits of agreement from -6.6 to 5.2 mmHg) with percentage error of 65.5\% (Figure 4A). When analyzing the mean values per patient \((n = 30)\), we found an overall bias of \(0.6 ± 2.1\) mm Hg (with limits of agreement from -3.7 to 4.8 mm Hg) with percentage error of 45.6\% (Figure 4B). Concordance correlation coefficients of the IAP measurements during the study are shown in Table 3. The precision and accuracy of IAP measurements during study stages varied within 0.63-0.85 and 0.95-0.98, respectively. The four quadrants trend plot is shown in Figure 5. From the 117 initial paired measurements, 55 pairs were excluded because either \(ΔIBP\) or \(ΔIGP\) were \(≤ ± 2.5\) mmHg or because \(ΔIBP\) or \(ΔIGP\) were equal to zero. The calculated level of concordance was 94.9\%.

**DISCUSSION**

This study demonstrates that the increase in IAP during surgical repair of ventral hernia and the early postoperative period is accompanied by deterioration of \(\text{CO}_2\) elimination followed by a decrease in arterial oxygenation. These
changes reflect the impairment of respiratory function after the procedure and could guide possible interventions.

The rise in IAP during abdominal surgery observed in our study can be explained by the stretch of abdominal wall following hernia repair. The peak of IAP increase was observed during spontaneous breathing after reversal of the effects of muscle relaxants and sedative agents. Despite the rise in IAP, APP remained above 60 mmHg during all study stages, and this was accompanied by normal arterial lactate concentrations, reflecting adequate organ perfusion. Previous studies considered an APP < 60 mmHg to be the indicator of abdominal hypoperfusion, moreover, APP has been shown to correlate well with survival from IAH and ACS.

The increase in IAP during and after repair of ventral hernia together with effects caused by general anesthesia can lead to deterioration of respiratory function. Thus, the rise in PaCO$_2$, EtCO$_2$ and P(a-et)CO$_2$ in parallel with reduction in respiratory compliance in our study may be caused by atelectasis formation in the basal lung areas, although we did not perform radiological imaging. The increase of the CO$_2$ gradient can occur when mixed venous blood passes the pulmonary circulation through shunt vessels without delivering CO$_2$ to alveolar air that is typical for atelectasis. Moreover, when ventilated lung areas are compromised by the cranial displacement of the diaphragm caused by IAH, a shift of ventilation can be anticipated so that regions ventilated normally before the insult are becoming over-ventilated in relation to their perfusion.

In addition, increased P(a-et)CO$_2$ can result from a decrease of cardiac output. The linear relationship between changes in EtCO$_2$ and cardiac output observed in animals has supported the necessity of clinical studies...
to determine whether a change in EtCO₂ would be useful as a noninvasive, continuous indicator of a change in cardiac output during anesthesia or intensive care[21]. In line with this hypothesis, McSwain et al[33] have shown that the P(a-et)CO₂ gradient increased in parallel with a rise in physiologic dead space (Vd). It is known that poor pulmonary perfusion from low cardiac output or hypotension can elevate Vd fraction[14] due to peripheral carbon dioxide production, which increases P(a-et)CO₂ in case of a persistent decreased blood flow[34].

In spite of decreased cardiac output as one of the reasons for the rise in P(a-et)CO₂, we suggest that the pivotal role in this process belongs to atelectasis formation. This speculation is confirmed by the delayed deterioration of arterial oxygenation in relation to the increase of IAP and P(a-et)CO₂. Similar findings were obtained by Strang et al[38]. During atelectasis formation, even a transient increase in PaO₂ might occur, due to a decreased intrapulmonary shunt (Qs/Qt). Consequently, oxygenation may not adequately reflect the severity of lung collapse during IAH[33], and hypoxemia usually develops later than changes in CO₂. The decrease in PaO₂/FiO₂ and atelectasis after the hernia repair may also be related to discontinuation of PEEP following extubation. Thus, Pelosi et al[32] recommended the application of PEEP to prevent atelectasis formation related to IAP in morbidly obese patients during general anesthesia.

The difference between gastric and urinary methods of IAP estimation observed in our study may be caused by the physical characteristics of the wall of bladder. This wall is not merely a membrane that transfers pressure from the intra-abdominal space to the bladder content[15,16,17]. Bladder wall compliance differs between patients and depends on several factors such as age, presence of chronic renal failure, BMI, filling status, fluid balance and bladder perfusion/ischemia. Moreover, several patients in our study had peritoneal adhesions, which might limit the transduction of abdominal pressure during measurement. Thus, IAP measured at one point cannot always be considered to be the pressure in the whole abdominal cavity[16,23,30]. Body anthropomorphic data may also have an impact on IAP measured at different sites[16,17]. We found that the measurement of IAP through the nasogastric probe correlates well with the results of the intrabladder measurement with mean difference between methods around -0.7 mmHg (with IGP being consistently lower than IBP). However, the mean percentage error of all measurements of IAP was 45.6%, thus in ventral hernia repair, both methods for the estimation of IAP can be used interchangeably keeping in mind the possibility of large data variations and the limitations of monitoring techniques. Moreover, both methods were able to keep track of changes in IAP during the different study stages as demonstrated by the concordance coefficient above 90%. In addition, Malbrain et al[31] concluded that in some patients, IAP estimation via nasogastric probe and IAP estimation via urinary catheter may differ significantly and this may have clinical implications. This situation can occur due to localized ACS, thus clinicians should be aware of this possibility. In order to identify risk factors and to recommend treatment for localized ACS, further studies of simultaneous intragastic and intrabladder IAP measurements are needed. In conclusion, this study fulfilled the minimal requirements for IAP measurement and validation studies as suggested by the “Recommendations for research by the International Conference of Experts on Intra-abdominal Hypertension and Abdominal Compartment Syndrome”[30]. More than 20 relevant patients were included with a broad range of IAP from normal to high. At least 50% of the measurements demonstrated IAP ≥ 12 mmHg and at least some measurements an IAP > 20 mmHg (5%). When looking at the mean values per patient, the bias was ≤ 1 mmHg with a precision close to 2 mmHg, good accuracy, reasonable limits of agreement and excellent concordance.

The surgical repair of ventral hernia is accompanied by a rise IAP and a parallel increase of PaCO₂, EtCO₂, and arterial to end-tidal gradient of CO₂, followed by a decrease in arterial oxygenation. The measurements of IAP using nasogastric tube and urinary catheter demonstrate a close agreement between both methods with excellent concordance, although the percentage error was quite high suggesting that the abdomen may not always act like a fluid filled compartment. Thus, both these methods can be used in clinical practice.

**COMMENTS**

**Background**

Abdominal wall reconstruction during ventral hernia repair can be associated with perioperative intra-abdominal hypertension (IAH), respiratory dysfunction and complications.

**Research frontiers**

The methods for measuring intra-abdominal pressure (IAP) are integrating in...
Intra-abdominal pressure during ventral hernia repair

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clinical practice but need validation in selected categories of patients at risk for IAH, including ventral hernia surgery.

**Innovations and breakthroughs**

The measurement of IAP through the nasogastric probe during hernia repair and postoperative period correlates well with the results of the intra-bladder measurement.

**Applications**

The study findings reflect the impairment of respiratory function after the surgery for ventral hernia that requires possible interventions, including measurement of IAP and correction of IAH.

**Terminology**

Intra-abdominal pressure: pressure in hollow organs of the abdomen or pelvic cavity such as bladder, stomach, intestine, and uterus. Intra-abdominal hypertension: intra-abdominal pressure exceeding 12 mmHg.

**Peer review**

The paper determined the influence of IAP on respiratory function after surgical repair of ventral hernia and compared two different methods of IAP measurement during the perioperative period. It’s very well done study.

**REFERENCES**

1. Reintam Blaser A, Parm P, Kitus R, Starkopf J. Risk factors for intra-abdominal hypertension in mechanically ventilated patients. *Acta Anaesthesiol Scand* 2011; 55: 607-614 [PMID: 21418151 DOI: 10.1111/j.1399-6576.2011.02415.x]

2. Pelosi P, Quintel M, Malbrain ML. Effect of intra-abdominal pressure on respiratory mechanics. *Acta Clin Belg Suppl 2007; (1): 78-88* [PMID: 17469705]

3. De Santis L, Frigo F, Bruttocao A, Terranova O. Pathophysiology of giant incisional hernias with loss of abdominal wall substance. *Acta Biomed 2003; 74 Suppl 2: 34-37* [PMID: 15050501]

4. Quintel M, Pelosi P, Caironi P, Meinhardt JP, Luette C, Herrmann P, Taccone P, Rylander C, Valenza F, Carlesso E, Gattinoni L. An increase of abdominal pressure increases pulmonary edema in oleic acid-induced lung injury. *Am J Respir Crit Care Med 2004; 169: 534-541* [PMID: 14670801 DOI: 10.1164/rccm.200209-1060OC]

5. Strang CM, Hachenberg T, Fredén F, Hedenstierna G. Development of atelectasis and arterial to end-tidal PCO2-difference in a porcine model of pneumoperitoneum. *Br J Anaesth 2009; 103: 298-303* [PMID: 19443420 DOI: 10.1093/bja/aep102]

6. Cheatham ML, Malbrain ML, Kirkpatrick A, Sugrue M, Parr M, De Waele J, Balogh Z, Leppäniemi A, Olive C, Ivatery R, D’Amours S, Wendon J, Hillman K, Wilmer A. Results from the International Conference of Experts on Intra-Abdominal Hypertension and Abdominal Compartment Syndrome. II. Recommendations. *Intensive Care Med 2007; 33: 951-962* [PMID: 17377769 DOI: 10.1007/s00134-007-0592-4]

7. Malbrain ML, Cheatham ML, Kirkpatrick A, Sugrue M, Parr M, De Waele J, Balogh Z, Leppäniemi A, Olive C, Ivatery R, D’Amours S, Wendon J, Hillman K, Johansson K, Kolkman K, Wilmer A. Results from the International Conference of Experts on Intra-Abdominal Hypertension and Abdominal Compartment Syndrome. I. Definitions. *Intensive Care Med 2006; 32: 1722-1732* [PMID: 16967294 DOI: 10.1007/s00134-006-0349-5]

8. Cheatham ML. Nonoperative management of intraabdominal hypertension and abdominal compartment syndrome. *World J Surg 2009; 33: 1116-1122* [PMID: 19363690 DOI: 10.1007/s00268-009-0003-9]

9. Gallagher JJ. Intra-abdominal hypertension: detecting and managing a lethal complication of critical illness. *AACC Adv Crit Care 2010; 21: 205-219* [PMID: 20431449 DOI: 10.1097/NCCI.0b013e3181d94f45]

10. Starkopf J, Tamke K, Blaser AR. Should we measure intra-abdominal pressures in every intensive care patient? *Ann Intensive Care 2012; 2 Suppl 1: S9* [PMID: 22873425 DOI: 10.1186/2110-5820-2-51-59]

11. De Keulenaer BL, De Waele JJ, Malbrain ML. Nonoperative management of intra-abdominal hypertension and abdominal compartment syndrome: evolving concepts. *Am Surg 2011; 77 Suppl 1: S34-51* [PMID: 21944450]

12. Björck M, Petersson U, Bjarnason T, Cheatham ML. Intra-abdominal hypertension and abdominal compartment syndrome in nontrauma surgical patients. *Am Surg 2011; 77 Suppl 1: S56-86* [PMID: 21944455]

13. Malbrain ML, Chiichelletto D, Pelosi P, Bihari D, Innes R, Ranieri VM, Del Turco M, Wilmer A, Brienda N, Malcangi V, Cohen J, Japiassu A, De Keulenaer BL, Daelemans R, Jiacquet L, Laterre PF, Frank G, de Souza P, Cesana B, Gattinoni L. Incidence and prognosis of intra-abdominal hypertension in a mixed population of critically ill patients: a multiple-center epidemiological study. *Crit Care Med 2005; 33: 315-322* [PMID: 15699833]

14. Malbrain ML. Different techniques to measure intra-abdominal pressure (IAP): time for a critical re-appraisal. *Intensive Care Med 2004; 30: 357-371* [PMID: 14730376 DOI: 10.1007/s00134-003-2107-2]

15. De Waele JJ, De laet I, Malbrain ML. Rational intraabdominal pressure monitoring: how to do it? *Acta Clin Belg Suppl 2007; (1): 16-25* [PMID: 17469688]

16. Malbrain ML, De laet IE, Willems A, Van Regenmortel N, Schoonheydt K, Dits H. Localised abdominal compartment syndrome: bladder-over-gastric pressure ratio (B/G ratio) as a clue to diagnosis. *Acta Clin Belg* 2010, 65: 96-106 [PMID: 20491359]

17. van Ramshorst GH, Salih M, Hop WC, van Waes OJ, Kleinrensink GJ, Goossens RH, Lange JF. Noninvasive assessment of intra-abdominal pressure by measurement of abdominal wall tension. *J Surg Res 2011; 171: 240-244* [PMID: 20462598 DOI: 10.1016/j.jss.2010.02.007]

18. Malbrain ML, De laet I, Vlaere D, Schoonheydt K, Dits H. In vitro validation of a novel method for continuous intraabdominal pressure monitoring. *Intensive Care Med 2008; 34: 740-745* [PMID: 18057530 DOI: 10.1007/s00134-007-0952-0]

19. De Potter TJ, Dits H, Malbrain ML. Intra- and interobserver variability during in vitro validation of two novel methods for intra-abdominal pressure monitoring. *Intensive Care Med 2005; 31: 747-751* [PMID: 15808751 DOI: 10.1007/s00134-005-2597-1]

20. Wouters J, Spinnicaille L, Dieudonne AS, Van Zwam K, Wilmer A, Malbrain ML. A Novel Method (CMON) for Continuous Intra-Abdominal Pressure Monitoring: Pilot Test in a Pig Model. *Crit Care Res Pract 2012; 2012: 181563* [PMID: 22454765 DOI: 10.1155/2012/181563]

21. Schachtrupp A, Tons C, Fackeldey V, Hoer J, Reinges M, Schumpelick V. Evaluation of two novel methods for the direct and continuous measurement of the intra-abdominal pressure in a porcine model. *Intensive Care Med 2003; 29: 1605-1608* [PMID: 12920511]

22. Yamanaka MK, Sue DY. Comparison of arterial-end-tidal PCO2 difference and dead space/tidal volume ratio in respiratory failure. *Chest 1987; 92: 832-835* [PMID: 3117500]

23. Murray IP, Modell JH, Gallagher TJ, Banner MJ. Titration of PEEP by the arterial minus end-tidal carbon dioxide gradient. *Chest 1984; 85: 100-104* [PMID: 6360567]

24. Tusman G, Suarez-Sipmann F, Böhm SH, Pech T, Reissmann H, Meschino G, Scandurra A, Hedenstierna G. Monitoring wall substance. *Acta Biomed* 2011; 82 (Suppl 1): 78-88 [PMID: 19444450]

25. Cresswell AB, Jassem W, Brittish R, Prachalias AA, Sizer E, Burnal W, Auinger G, Muiesan P, Rela M, Heaton ND, Bowles M, Wodon JA. The effect of body position on compartmental intra-abdominal pressure following liver transplantation. *Ann Intensive Care 2012; 2 Suppl 1: S12* [PMID: 23813906]
26 Luckianow GM, Ellis M, Governale D, Kaplan LJ. Abdominal compartment syndrome: risk factors, diagnosis, and current therapy. Crit Care Res Pract 2012; 2012: 908169 [PMID: 22720147 DOI: 10.1155/2012/908169]

27 De laet I, Malbrain ML. ICU management of the patient with intra-abdominal hypertension: what to do, when and to whom? Acta Clin Belg Suppl 2007; (1): 190-199 [PMID: 17469719]

28 Jones AE, Shapiro NI, Trzeciak S, Arnold RC, Claremont HA, Kline JA. Lactate clearance vs central venous oxygen saturation as goals of early sepsis therapy: a randomized clinical trial. JAMA 2010; 303: 739-746 [PMID: 2079286 DOI: 10.1001/jama.2010.158]

29 Cheatham ML, White MW, Sagraves SG, Johnson JL, Block EF. Abdominal perfusion pressure: a superior parameter in the assessment of intra-abdominal hypertension. J Trauma 2000; 49: 621-626; discussion 621-626; [PMID: 11038078]

30 Al-Dorzi HM, Tamim HM, Rishu AH, Aljumah A, Arabi YM. Intra-abdominal pressure and abdominal perfusion pressure in cirrhotic patients with septic shock. Ann Intensive Care 2012; 2 Suppl 1: S4 [PMID: 22873420 DOI: 10.1186/2110-5820-2-S1-S4]

31 Isserles SA, Breen PH. Can changes in end-tidal PCO2 measure changes in cardiac output? Anesth Analg 1991; 73: 888-894 [PMID: 1952183]

32 McSwain SD, Hamel DS, Smith PB, Gentile MA, Srinivasan S, Meliones JN, Cheifetz JM. End-tidal and arterial carbon dioxide measurements correlate across all levels of physiologic dead space. Respir Care 2010; 55: 288-293 [PMID: 20196877]

33 Ong T, Stuart-Killion RB, Daniel BM, Presnell LB, Zhuo H, Matthey MA, Liu KD. Higher pulmonary dead space may predict prolonged mechanical ventilation after cardiac surgery. Pediatr Pulmonol 2009; 44: 457-463 [PMID: 19382217]

34 Feng WC, Singh AK. Intraoperative use of end-tidal carbon dioxide tension to assess cardiac output. J Thorac Cardiovasc Surg 1994; 108: 991-992 [PMID: 7967688]

35 De Keulenaer BL, De Waele JJ, Powell B, Malbrain ML. What is normal intra-abdominal pressure and how is it affected by positioning, body mass and positive end-expiratory pressure? Intensive Care Med 2009; 35: 969-976 [PMID: 19242275 DOI: 10.1007/s00134-009-1445-0]

36 Malbrain ML, De laet I. Do we need to know body anthropomorphic data whilst measuring abdominal pressure? Intensive Care Med 2010; 36: 180-182; author reply 180-182; [PMID: 19841898 DOI: 10.1007/s00134-009-1685-z]

37 Malbrain ML, De laet I, Van Regenmortel N, Schoonheydt K, Dits H. Can the abdominal perimeter be used as an accurate estimation of intra-abdominal pressure? Crit Care Med 2009; 37: 316-319 [PMID: 19050639 DOI: 10.1097/CCM.0b013e318192678e]

38 De Waele JJ, Cheatham ML, Malbrain ML, Kirkpatrick AW, Sugrue M, Balogh Z, Ivatury R, De Keulenaer B, Kimball EJ. Recommendations for research from the International Conference of Experts on Intra-abdominal Hypertension and Abdominal Compartment Syndrome. Acta Clin Belg 2009; 64: 203-209 [PMID: 19670559]

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