

Role of intra abdominal pressure in predicting outcome in cases of acute abdomen

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Abstract

Aim and objectives: The aim of this study was to know the role of intra-abdominal pressure in predicting outcome in cases of acute abdomen To determine the prognostic value of IAP measurements with respect to the association between IAH and development of new organ failure. To study the association between IAH and duration of ICU stay. **Methodology:** This was a prospective study of 50 patients of either sex and any age of acute abdomen admitted in surgical ICU of our hospital. To determine IAP we had used bladder pressure measurement method and IAP was measured every 6 hourly. **Results and Observations:** 30 patient developed IAH (60%) when IAP max was considered. Out of these 30 patients, 6(12%) developed ACS and 12% patients succumbed. With IAP mean, IAH was 48% i.e. (24/50). And out of these 24 cases 4 developed ACS. When extrapolated to the total no. of cases 8% (n=4/50) developed ACS. 28% of patients had failure of the cardiovascular system, 28% had renal failure and 20% patients had respiratory failure The increase in IAP was significantly associated with organ failure. ICU stay was maximum 35 days where pressure was maximum i.e.25mmHg if IAP max and 24 mmHg if IAP mean was considered. And ICU stay was minimum 1day where minimum pressure was 4 mmHg when IAP max and 3.5 mmHg when IAP mean was considered. **Conclusion:** The development of IAH and ACS is of extreme importance in surgical practice and in care of critically ill patients; because of the impact of increased IAP on end-organ function.IAP is significantly linked to the patient's outcome and significantly associated with prolonged ICU stay. **Key Words:** IAP- intra-abdominal pressure, IAH- intra-abdominal hypertension, ACS-abdominal compartment syndrome.

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Received Date: 22/03/2017 Revised Date: 10/04/2017 Accepted Date: 20/05/2017

Access this article online

Quick Response Code:	Website: www.medpulse.in
	DOI: 24 May 2017

INTRODUCTION

A compartment syndrome (CS) exists when increased pressure in a closed anatomical space threatens the viability of enclosed tissue.¹ Within our body there are four types of compartments head, chest, abdomen, and extremities, but when a compartment syndrome occurs in the abdominal cavity, the impact on end-organ function both within and outside of the cavity can be devastating.

Intra-abdominal hypertension (IAH) is a graded phenomenon and can evolve to the end-stage abdominal compartment syndrome (ACS), which is an all or none phenomenon.² IAH (3) is a clearly defined cause of organ dysfunction in patients after emergency abdominal surgery, a disease process like intestinal obstruction, pancreatitis, peritonitis or blunt abdominal trauma. It is also recognized in patients in intensive care units after elective surgical procedures, liver transplantation, massive fluid resuscitation for extra abdominal trauma and severe burns.ACS is defined as an “Acute increase in the intra-abdominal pressure (IAP) resulting in alteration in respiratory mechanism, hemodynamic parameters and renal and cerebral perfusions.”³ ACS occurs when IAP is abnormally raised in association with increasing organ dysfunction.⁴ The ACS is not a disease and as such it can have many causes and it can develop within many disease processes. Manifestation of ACS includes progressively marching events in the form of abdominal distension, followed by an increase in the peak airway pressure

leading to dyspnoea, oliguria followed by anuria and development of intracranial hypertension which turns fatal. The development of IAH and ACS is of extreme importance in surgical practice and care of critically ill patients, because of the impact of increased IAP on end-organ function. This study endeavors this often-neglected phenomenon. The aim of this study was to know the role of intraabdominal pressure in predicting outcome in cases of acute abdomen. To determine the prognostic value of IAP measurements with respect to the association between IAH and development of new organ failure. To study the association between IAH and duration of ICU stay. The permission and approval to perform this study was obtained from the Ethical and scientific Committee of hospital.

MATERIALS AND METHOD

This was a prospective study of 50 consecutive cases of acute abdomen admitted in a Surgical ICU and who needed indwelling bladder catheterisation over an eighteen month period. The detailed clinical history was recorded as per the proforma followed by clinical examination n IAP was measured every 6 hourly. All the patient of Acute abdomen with perforation, peritonitis, bowel gangrene, or acute pancreatitis, Patients with blunt abdominal trauma, patients with a distended abdomen and signs and symptoms consistent with Abdominal Compartment Syndrome (ACS) example: a. Oliguria b. Hypoxia c. Hypotension d. Unexplained acidosis were included in study. Patient with Bladder dysfunction in old age patients, Bladder surgery, Pregnancy, Neurogenic bladder were excluded from study.

Method of intra-abdominal pressure measurement

Equipment Needed

1. Indwelling urinary catheter with urine drainage bag and specimen port
2. Disinfectant (povidone -iodine solution, chlorhexidine, or alcohol)
3. Standard intravenous set with 500 ml of normal saline
4. 20-mL Luer lock syringe
5. Transducer and pressure tubing
6. 18 no. intravenous cannula
7. 2 three-way stopcocks

Under all aseptic precautions, a standard intravenous (IV) infusion set is connected to 500 ml of normal saline, two three-way stopcocks, a 20 ml Luer lock syringe, and a disposable pressure transducer. 1) An 18 gauge plastic intravenous infusion catheter or needle-less cannula is inserted into the culture aspiration port of the urinary drainage tubing and needle removed. 2) The infusion catheter, cannula, or sampling port is attached to the first stopcock via pressure tubing. 3) After being flushed with

saline and “zeroed” at the level of the mid-axillary line at level of iliac crest with patient in the supine position,^{5,6} the urinary drainage tubing is clamped immediately distal to catheter. 4) The stopcocks are turned “off” to the patient and pressure transducer and 25ml of saline is aspirated from the IV bag and instilled into the bladder⁷ after the turned off the stopcock 1 to syringe and stopcock 2 to IV tubing.5) The clamp on the urinary drainage tubing is momentarily released to ensure that all air is flushed from the urinary catheter. 6) After a stabilization period of 30-60 seconds to allow for bladder detrusor muscle relaxation, with the patient in the complete supine position and after ensuring that abdominal muscle contractions are absent, IAP is measured at end-expiration on the bedside monitor.7) The patient IAP should be expressed in mmHg (1 mmHg= 1.36 cm H₂O). After determination of IAP, the clamp is removed, the bladder allowed to drain, and the volume of saline utilized subtracted from the patient’s urinary output for that hour

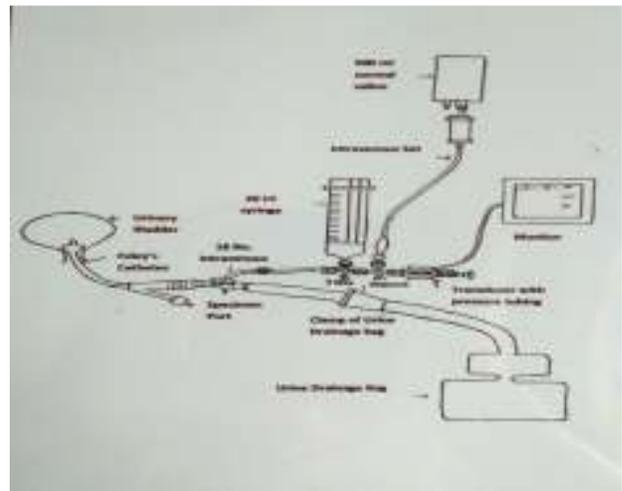


Figure 1: Schematic Diagram of Measurement of intra abdominal pressure with specimen port of urine drainage bag.⁸

Data collection

A data collection sheet was used to record patient demographics, IAP measurements, organ failure by Modified Marshall Scoring System for organ dysfunction, and ICU parameters relevant to study for each patient.

Data Acquisition

Intra-abdominal pressure (IAP) measurement was obtained in all patients of acute abdomen admitted in Surgical ICU. The readings were being taken at 6 hourly intervals. IAP max (the highest daily value) was considered for main analysis. IAP mean (mean of the four daily values) was also calculated and employed in analysis. The occurrence rate of organ dysfunction (cardiovascular, renal, and pulmonary) during ICU stay

was recorded. Organ dysfunction was defined according to the Modified Marshall Scoring System Cardiovascular: systolic BP < 90 mm Hg not fluid responsive; Renal: serum creatinine >1.9 mg/dl; Pulmonary, the need for mechanical ventilation or PaO₂/FiO₂ ratio < 300.⁹ The IAP measurements were discontinued when patient had recovered or expired. The observations were tabulated and results studied.

Data analysis

Data analysis was done using the SPSS (Statistical Package for Social Science) Version 11 for Windows. The demographic variables, organ failure, grades of IAP max and IAP mean and outcome were calculated with number and percentage. The t-test was used to find significance in difference of IAP according to organ failure and chi-square test was used to find the association between surgery done with outcome. Correlation and test for correlation was used to find the significant correlation between ICU stay and IAP. A probability value of ≤ 0.05 was accepted as the level of statistical significance.

RESULTS

Our study group consisted of 38 (76%) male patients and 12(24%) female patients.

Table 1: IAP Max and IAP Mean wise distribution of cases in study group

Grade	Pressure in mmHg	IAP Max (%)	IAP Mean (%)
0	<12	20 (40)	26 (52)
I	12-15	16 (32)	13 (26)
II	16-20	8 (16)	7 (14)
III	21-25	4 (8)	3 (6)
IV	>25	2 (4)	1 (2)
Total		50 (100)	50 (100)

When to measure intra-abdominal pressure, IAP max was considered, 30 patient had IAH out of which 16 patient had IAP of grade I, 8 patient of grade II, 4 of grade III, and 2 of grade IV. When IAP mean considered, then result showed that 24 patient had IAH out of which 13 patient had IAP of grade I, 7 patients of grade II, 3 patient of grade III and 1 patient of grade IV. The incidence of IAH (defined as IAP > 12mmHg) was 48% (24 patients) with 4 patient having IAP > 20 when IAP mean was used. However when IAP max was used, the incidence of IAH was 60% (30 patients) with 2 patients showed IAP > 20. The incidence of IAH differed with respect to whether mean or maximal IAP values were used. With the use of IAP max incidence of IAH was greater as compared to IAP mean.

Table 2: Association between IAP max grade and incidence of organ failure (Cardiovascular) in study group

Grade	Pressure (mmHg)	Organ failure CVS		Total
		Present	Absent	
0	<12	2	18	20
I	12-15	2	14	16
II	16-20	4	4	8
III	21-25	4	0	4
IV	> 25	2	0	2
Total		14	36	50

Chi-square = 22.47, P<0.0001

Table 3: Association between IAP max grade and incidence of organ failure (Respiratory system) in study group

Grade	Pressure (mmHg)	Organ failure RS		Total
		Present	Absent	
0	<12	2	18	20
I	12-15	1	15	16
II	16-20	2	6	8
III	21-25	3	1	4
IV	> 25	2	0	2
Total		10	40	50

Chi-square = 18.83, P<0.0001

Table 4: Association between IAP max grade and incidence of organ failure (Renal system) in study group

Grade	Pressure (mmHg)	Organ failure Renal		Total
		Present	Absent	
0	<12	3	17	20
I	12-15	3	13	16
II	16-20	3	5	8
III	21-25	3	1	4
IV	>25	2	0	2
Total		14	36	50

Chi-square = 12.24, P<0.05

In this study 28% of patients had cardiovascular system failure, 20% had respiratory failure and 28% of patients had renal failure. The above table no.2,3 and 4 shows that increase in IAP was significantly associated with development of new organ failure.

Table 5: Association between IAP max and survival outcome in study group

IAP max (mmHg)	Outcome		Total
	Survived	Death	
<12 (Grade 0)	20	0	20
12 – 20(Grade I, II)	21	3	24
>20 (Grade III, IV)	3	3	6
Total	44	6	50

Chi-square = 10.94, P<0.005

When association between IAP max and survival outcome studied, we found 3 patients with IAP max 12-20 out of 24, and 3 cases of IAP > 20 out of 6 had poor outcome. So this shows that the association between IAP max and outcome was statistically significant.

Table 6: Association between IAP mean and survival outcome in study group

IAP mean (mmHg)	Outcome		Total
	Survived	Death	
<12 (Grade 0)	26	0	26
12 – 20 (Grade I, II)	17	3	20
>20 (Grade III, IV)	1	3	4
Total	44	6	50

Chi-square = 18.75, P<0.0001

This table shows that 3 patients with IAP mean 12-20 out of 20 and 3 cases of IAP > 20 out of 4 had poor outcome. So this shows that the association between IAP mean and outcome was statistically significant.

Table 7: Correlation between ICU stay and IAP Max, IAP Mean in study group

Correlation between ICU stay and	r Value	P Value
IAP Max	0.567	<0.0001
IAP Mean	0.57	<0.0001

Table 8: Survival outcome analysis in surgical and Non-surgical group based on IAP max

IAP max (mmHg)	Outcome				Total
	Survived		Death		
	Surgery D	Surgery ND	Surgery D	Surgery ND	
<12	12	8	0	0	20
12 – 20	16	5	2	1	24
>20	3	0	2	1	6
Total	31	13	4	2	50

D = done, ND= not done

This table and graph shows that total 31(62%) of patient survived after surgery out of which 12 had IAP <12, 16 had IAP 12-20 and 3 had IAP of >20 mm Hg. And 4 (8%) patients had mortality in which surgery was done.

DISCUSSION

Intra-abdominal pressure (IAP) is the steady-state pressure within the abdominal cavity. So sustained increase in intra-abdominal pressure lead to intra-abdominal hypertension. Intra-abdominal hypertension (IAH) can affect all body systems and IAH has been proposed as the initial fall of the dominoes on the pathway of multisystem organ failure. Intra-abdominal hypertension (IAH) is a graded phenomenon and can evolve to the end-stage Abdominal compartment syndrome (ACS), which is an all or none phenomenon.² Abdominal compartment syndrome occurs when intra-abdominal pressure is abnormally raised in association with increasing organ dysfunction.⁴ The development of intra-abdominal hypertension and abdominal compartment syndrome is of extreme importance in surgical practice and care of critically ill patients, because of the impact of increased IAP on end-organ function. In this study, 60% (30/50) patient developed IAH and 12 %

(6/50) patient developed ACS when IAP max was considered. If IAP mean is considered the incidence of IAH was 48% i.e. 24 out of 50. And 8% (n=4/50) developed abdominal compartment syndrome.(Table no. 1) Multiple experimental and clinical reports have demonstrated that the presence of intra-abdominal hypertension affects vital organs leading to organ failure and eventually death, sometimes in as many as 80% of patients.^{12,19-23} The Modified Marshall Scoring System⁽⁹⁾ for organ dysfunction was used to assess organ failure in this study because of its simplicity and ease of use compared to other physiological scoring systems like the APACHE II OR III. It is a well accepted method used for defining organ failure and has been employed in other studies too.^{24,25} Walter LB *et al*²⁶ said in their study that the organ systems that appear most affected are the cardiovascular, pulmonary and renal systems. Cardiovascular effects of increased IAP include decreased Cardiac Index owing to diminished venous return; therefore, adequate volume resuscitation is a key feature of management of ACS. A markedly increased vascular resistance has also been recognized. Increased IAP pushes upon the diaphragm, decreasing pulmonary compliance and creating high airway pressures. Resultant hypoventilation leads to hypoxia and hypercarbia. As shown in Table No.2,3,4 in our study 28% of patients had failure of the cardiovascular system, 28% had renal failure and 20% patients had respiratory failure. De Waele *et al*²⁷ in their study observed that IAH is associated with a high occurrence of organ failure. The organ dysfunction in their study was cardiovascular system in 19%, respiratory system failure in 20% and renal failure in 18% patients. In our study as seen in Table No.2,3,4 the increase in IAP was significantly associated with organ failure of cardiovascular, pulmonary, and renal systems respectively. Malbrain *et al*¹² in 2004 conducted a study of 97 critically ill patients with IAH/ACS in which they demonstrated that respiratory, renal and coagulation systems were significantly more impaired in patients with IAH. Standl,²⁸ Bhandari *et al*²⁵ and Vidal *et al*¹⁰ also observed that the severity of IAH was significantly associated with more severe organ failure. In this study, 6(12%) patients out of the 50 did not survive. ACS developed in the 6 (12%) according to IAP max and of these only 3 survived i.e. 50% mortality (Table no.5). But if IAP mean was considered, ACS developed in 4(8%) and of these only 1 survived i.e.75% mortality (Table no.6). So according to IAP max 12% patients developed ACS and 12% patients succumbed and this result was similar to the study of Kim *et al*²⁹ who reported a mortality of 16%. and according to IAP mean only 8% patients developed ACS but we know that 12% patients succumbed. Therefore IAP max was

better than IAP mean for predicting outcome. The association between IAP max and IAP mean with survival outcome were statistically significant with p value <0.005 and <0.0001 using chi square test. This points towards the fact that non-survivors had higher pressures than survivors. Malbrain *et al*¹¹ in 2005 conducted a prospective multicentre study of 265 mixed ICU patients in 14 ICUs of six countries and demonstrated IAH as an independent risk factor for mortality. Vidal *et al*¹⁰ also had similar results. Carrillo-Esper R *et al*³⁰ and Bhandari *et al*²⁵ in their study concluded that IAH/ACS significantly increases the morbidity and mortality. But Patricia Santa-Teresa³¹ in their study showed that IAH was a non-independent predictor of mortality, and they said that IAH was a marker of mortality in association with other clinical factors like high severity score and number of risk factors during ICU stay. The correlation between IAP max, IAP mean with ICU stay was quite significant in our study i.e. increase in intra-abdominal pressure is associated with an increase in ICU stay. (Table no.7) ICU stay was maximum 35 days in patient where pressure was maximum i.e. 25 mmHg if IAP max was considered and 24 mmHg if IAP mean was considered. And ICU stay was minimum 1 day in patient where minimum pressure was 4 mmHg when IAP max was considered and 3.5 mmHg when IAP mean was considered. The mean ICU stay was 4.92 days when mean IAP was 12.8 mmHg according to IAP max and 11.65 mmHg according to IAP mean. Vidal *et al*,¹⁰ Bhandari *et al*²⁵ and Patricia Santa-Teresa *et al*³¹ in their studies also showed that IAH was significantly associated with a prolonged ICU stay. In our study, as shown in Table No. 8, out of 6 patients who had an IAP max of >20, 5 patients underwent surgical intervention and out of these 2 expired. The indication of surgery in all patients was not ACS. Patient with IAH >20 with severe organ failure were considered for a decompressive laparotomy. We performed a decompressive laparotomy in 3 patients. Two patients out of these improved well but one patient expired. The cause of mortality was multiorgan failure in this patient. Out of the 3 patients who underwent a decompressive laparotomy, 1 patient had perforative peritonitis due to abdominal Koch's. On admission his intra-abdominal pressure was 26 mm Hg. He underwent exploratory laparotomy with ileostomy with decompressive laparotomy. As there was bowel edema it was not possible to do a primary closure of his abdomen, so a Surgisis bioabsorbable mesh was used to close the open abdomen. The patient's outcome was very good in this case. The second patient who underwent a decompressive laparotomy had acute severe necrotising pancreatitis. On admission, his IAP was in the range of 16-19 mm Hg, initially to reduce IAP we performed

percutaneous drainage of infective necrosis. But even after this his IAP kept raising and reached up to 25 mm Hg with development of new organ failure we performed an open pancreatic necrosectomy with a decompressive laparotomy. We used vacuum assisted closure to close the open abdomen. His pressure was reduced but he did not survive due to multiorgan failure. The third patient who underwent a decompressive laparotomy was a patient with abdominal trauma with hemoperitoneum due to retroperitoneal haemorrhage and his pressure was 21 mmHg. As the patient was hemodynamically unstable, we performed a damage control surgery. Due to fluid volume resuscitation there was significant bowel edema so we kept the abdomen open. And after 48 hrs re-surgery was done with a primary closure of the abdomen. Patient outcome was also good in this case. In our study, the patients who had their abdomen kept open for decompression survived. So although it does *prima facie* suggest that surgical decompression is advantageous, and can play a significant role in salvaging patients of ACS, literature has not yet authenticated its use completely. Controversial reports do exist. This result differs from the study of De Waele *et al*²⁷ who in their study concluded that whether surgical decompression in their patients was advantageous or not was not very clear. But Bertram *et al*³² and Cirocchi R *et al*³³ stated that decompressive laparotomy is the therapy of choice for ACS. Our study would also suggest that decompressive laparotomy should be considered in those patients whose IAP becomes more than 20 and in whom organ failure has set in. This is the same logic as applicable in performing fasciotomy in a limb developing compartment syndrome.

CONCLUSION

The study has highlighted the adverse effects of raised intra-abdominal pressure on organ function. The worst affected organ systems were cardiovascular (28%), renal (28%), followed by respiratory system (20%) Intra-abdominal pressure is significantly linked to the patient's outcome. 12% of patients died; all of them had intra-abdominal hypertension and half of them developed abdominal compartment syndrome. An increased intra-abdominal pressure was significantly associated with prolonged ICU stay.

RECOMMENDATION

The occurrence of intra-abdominal hypertension and the risk factors for it should be appreciated by both surgeons and intensivists caring for patients after abdominal surgical operations. It would be advisable to adopt IAP measurement as a routine measure for all critically ill patients in the ICU as it can be extremely useful in identification and/or treating cases of ACS. Intervention

and treatment guidelines should be formulated for the management of patients who are found to have intra-abdominal hypertension or abdominal compartment syndrome. Decompressive laparotomy is considered in patients with abdominal compartment syndrome

REFERENCES

1. Malbrain ML, Cheatham ML, Kirkpatrick A, et al. Results from the International Conference of experts on intra-abdominal hypertension and abdominal compartment syndrome. I. Definitions. *Intensive Care Med* 2006; 32: 1722–32.
2. Manu L.N.G. Malbrain, Inneke E. De laet. Intra-abdominal Hypertension: Evolving Concepts. *Clin Chest Med* 2009; 30: 45-70.
3. Burch JM, Ernest E, Frederick A, Moore, Reginald F. The Abdominal Compartment Syndrome. *Surg Clin N Am* 1996; 76: 833-842.
4. Richard HT, Benjamin DLL, McDonald JC. Abdominal wall, umbilicus, peritoneum, mesenteries, omentum and retroperitoneum. In: Courtney MT, Beauchamp DR, Evers BM, editors. Sabiston textbook of surgery the biological basis of modern surgical practice. 17th ed. Pennsylvania: Elsevier Saunders; 2004.p.1180-1182.
5. De Waele J, Cheatham ML, De Keulenaer B, Widder S, Kirkpatrick A, et al. The optimal zero reference transducer position for intra-abdominal pressure measurement: A multicenter analysis. *Acta Clinica Belgica* 2007; 62(Supplement 1): 247.
6. McBeth PB, Zygun DA, Widder S, Cheatham M, Zengerink I, et al. Effect of patient positioning on intra-abdominal pressure monitoring. *Am J Surgery* 2007; 193:644–647.
7. Malbrain ML, Deeren DH. Effect of bladder volume on measured intravesical pressure: a prospective cohort study. *Crit Care*.2006; 10(4): 98.
8. Malbrain ML:Different techniques to measure intra-abdominal pressure (IAP):time for a critical re-appraisal.*Intensive Care Med*.2004 Mar;30(3):351-71.
9. Marshall JC, Cook DJ, Christou NV, et al. multiple organ dysfunction score: a reliable descriptor of a complex clinical outcome. *Crit Care Med* 1995; 23: 1638–52.
10. Vidal MG, Ruiz Weisser J, Gonzalez F, et al. Incidence and clinical effects of intra-abdominal hypertension in critically ill patients. *Crit Care Med* 2008; 36: 1823–1831.
11. Malbrain ML, Chiumello D,Pelosi P ,et al. Incidence and progression of intraabdominal hypertension in mixed population of critically ill patients:a multiple-center epidemiological study. *Crit Care Med* 2005; 33(2): 315-322.
12. Malbrain ML, Chiumello D, Pelosi P, et al. Prevalence of intra-Abdominal hypertension in critically ill patients: a multicentre epidemiological study. *Intensive Care Med* 2004; 30(50): 822-829.
13. Malbrain M, Deeren D, De Potter T. Intra-abdominal hypertension in the critically ill: it is time to pay attention? *Curr Opin Crit Care*. 2005; 11(2):156-171.
14. Sugrue M, Buist MD, Hourihan F, Deane S, Bauman A, Hillman K. Prospective study of intra-abdominal hypertension and renal function after laparotomy. *Br J Surg* 1995; 82(2): 235-238.
15. Balogh Z, McKinley BA, Cocanour CS, Kozar RA, Valdivia A, Sailors RM et al. Supranormal trauma resuscitation causes more cases of abdominal compartment syndrome. *Arch Surg* 2003; 138(6): 637-642.
16. Vallee F, Dupas C, Feuvrier V, Mebazaa A, Ferre F, Mari A et al. Intra-abdominal pressure measurement method via the urinary-tube: bedside validation of a biomechanical model integrating urine column height and bladder urinary volume. *Ann Surg* 2010; 251(1): 127-132.
17. Cheatham ML, De Waele JJ, De Laet I, De Keulenaer B, Widder S, Kirkpatrick AW et al. The impact of body position on intra-abdominal pressure measurement: a multicenter analysis. *Crit Care Med* 2009; 37: 2187-2190.
18. Reintam A, Parm P, Kitus R, Kern H, Starkopf J. Primary and secondary intra-abdominal hypertension – different impact on ICU outcome.*Intensive Care Med* 2008; 34(9): 1624-1631.
19. Sugrue M, Buhkari Y. Intra-abdominal pressure and abdominal compartment syndrome in acute general surgery. *World J Surg* 2009; 33(6): 1123-1127.
20. Fietsam R Jr, Villalba M, Glover JL, et al. Intra-abdominal compartment syndrome as a complication of ruptured abdominal aortic aneurysm repair. *Am Surg* 1989; 55(6): 396–402.
21. Bishara B, Karram T, Khatib S et al. Impact of pneumoperitoneum on renal perfusion and excretory function: beneficial effect of nitroglycerine. *Surg Endosc* 2007; 23: 568-576.
22. Kovac N, Sirnovic M, Mazul-Sunko B.Clinical significance of intra-abdominal pressure and abdominal perfusion pressure in patients with acute abdominal syndrome.*Signa Vitae* 2007; 2(2): 14-17.
23. Biffl WL, Moore EE, Burch JM, Offner PJ, Franciose RJ.Secondary abdominal compartment syndrome is a highly lethal event. *Am J Surg* 2001; 182: 645-648.
24. Chen H, Li F, Sun JB, Jia JG. Abdominal compartment syndrome in patients with severe acute pancreatitis in early stage. *World J Gastroenterol* 2008; 14: 3541–3548.
25. Vimal Bhandari, Jiten Jaipuri, Mohit Singh, Avneet Singh Chawla. Intra-Abdominal Pressure in the early phase of severe acute pancreatitis: canary in a coal mine? Results from a rigorous validation protocol.*Gut and Liver* 2013; 7(6): 731-738.
26. Walter LB, Tomomi O, William GC. Surgical Critical Care. In: Courtney MT, Beauchamp DR, Evers BM, editors. Sabiston textbook of surgery the biological basis of modern surgical practice. 17th ed. Pennsylvania: Elsevier Saunders; 2004. p. 627.
27. Jan J De Waele, Eric Hoste, Stijn I Blot, Johan Decruyenaere and Francis Colardyn: IAH in patients with severe acute pancreatitis. *Crit Care*.2005; 9(4): R452-R457.
28. Standl T. Abdominal compartment syndrome. A still underestimated problem? *Anesthesiol Intensivmed Notfallmed Schmerzther*.2007 Jul; 42(7): 500-503.
29. Kim IB, Prowle J,Baldwin I,Bellomo R, et al;. Incidence, risk factors and outcome associations of intra-abdominal

- hypertension in critically ill patients. *Anaesthesia Intensive Care*. 2012 Jan; 40(1): 79-89.
30. Carrillo-Espinoza R, Sosa-García JO, Carrillo-Córdova JR, Leyva-Mondragón C. Syndrome of abdominal compartment in trauma. *Cir Cir*. 2012 Nov-Dec; 80(6):550-5.
 31. Patricia Santa-Teresa, et al: Incidence and prognosis of intra-abdominal hypertension in critically ill medical patients: a prospective epidemiological study. *Annals of Intensive Care* 2012, 2(Suppl 1):S3.
 32. Bertram P, Schachtrupp A, Rosch R, Schumacher O, Schumpelick V. Abdominal Compartment syndrome. *Der Chirurg*. 2006; 77(7): 573-579.
 33. Cirocchi R, Barillaro I, Boselli C et al: The abdominal compartment syndrome and the importance of decompressive re-laparotomy. *G Chir*. 2010 Nov-Dec; 31(11-12):560-74.

Source of Support: None Declared
Conflict of Interest: None Declared