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Polycompartment syndrome – intra-abdominal pressure measurement

Zsolt Bodnar

Department of Surgery, Letterkenny University Hospital, Letterkenny, Ireland

Abstract

Intra-abdominal hypertension and the abdominal compartment syndrome are well-known, serious, life-threatening clinical entities in acute care surgery. A common characteristic of these syndromes is the permanent and irreversible damage that may affect the organs which can be found inside the given compartment if quick intervention cannot be provided. All factors which may and can lead to a sudden increase in the intra-abdominal pressure can be found among the triggering factors of abdominal compartment syndrome. Despite the modern and quick diagnostics, and the adequate surgical interventions performed in time, the mortality of this syndrome is extremely high (38–71%). It affects practically all vital organ systems: cardiovascular, respiratory, urinary and central nervous system. There are four major compartments in the human body: the head, the chest, the abdomen and the extremities. When two or more compartments have elevated pressures the name of the clinical entity is polycompartment syndrome, first described in 2007. The only possible way of establishing the diagnosis is to measure the intra-abdominal pressure, a widespread manner of which is the measurement through the bladder. Treatment of abdominal and polycompartment syndrome is nearly always surgical decompression with temporary abdominal wall closure or open abdominal treatment. Clinicians need to be aware of the real existence of polycompartment syndrome and the complex and constant interplay of raised pressure between compartments. This highlights the importance of research and development of new intra-abdominal pressure measurement techniques.

Key words: intra-abdominal pressure, abdominal compartment syndrome, polycompartment syndrome, intermittent pressure measurement, continuous intra-abdominal pressure measurement.

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CORRESPONDING AUTHOR:

Mr. Zsolt Bodnar, MD, PhD, FRCSI, FICS, FRCS, Letterkenny University Hospital, Letterkenny, Co. Donegal, Ireland, e-mail: drbozsolt@gmail.com

INTRODUCTION

The brief history of intra-abdominal hypertension and the abdominal compartment syndrome is typical of any medical innovation: described, forgotten, re-discovered, and faced with scepticism and ridicule. Eventually, after being scientifically proven and re-proven and supported by "clinical leaders" and widely published in reputable journals, it is accepted as "truth" [1]. Now is widely accepted that intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS) are frequent findings among severely ill surgical patients [2, 3]. Severe pancreatitis, inflammatory processes, retroperitoneal haemorrhage, bowel obstruction, ascites, over-resuscitation, blunt abdominal trauma, peritonitis, or even massive transfusion can be found among the triggering factors of IAH and ACS. Step by step it became clear that the IAH/ACS is not only a surgical but also a medical problem.

The human body has not only one compartment; it is subdivided into smaller or larger units by well-defined separating walls. The function of these compartments is to mechanically protect and separate the organs or organ systems situated inside them. The skull, the spinal canal, the orbit, the pericardium, the thoracic and abdominal cavities are well-known cavities of our body [4]. The elasticity of the tissues of the separating walls has a strong determining effect on the tolerance for volume or pressure changes exerted on the organs which can be found inside these compartments. Compartment syndrome defines those changes which occur in the given compartments due to the increased pressure. Scalea in 2007 suggested the complex and constant interplay of elevated pressure between the different compartments [5], but the "terminus technicus" of polycompartment syndrome (PCS) was first coined by Malbrain in the same year [6].

BRIEF HISTORY

ACS was first described in relation to abdominal traumatic injuries. Kron was the first, although he did not use the term itself, to describe compartment syndrome in 1984. It was again Kron who routinely used abdominal pressure measurement through urinary bladder catheterisation, which became widespread by 1989; however, the fundamentals of the method were described 100 years earlier by Oderbrecht. The first description of the effects of the intra-abdominal pressure (IAP) was published by Etienne-Jules Marey [1], but the first measurement was performed by Braune in Germany in 1865 [1]. Between 1870 and 1900 further developments were made in the understanding of IAP, including the fundamental works of Bert (1870), Schroeder (1886), Schatz (1872), Wendt (1873), Oderbrecht (1875), Wegner (1877), Quinke (1878), Mosso and Pellacani (1882), Senator (1883) and Heinricius (1890). In 1911 it was published by Emerson that elevated IAP decreases blood pressure because of diminished venous return to the heart as well as depressed cardiac contractility. He then provided a relevant clinical correlation, which subsequently has been totally ignored by many generations of surgeons [1, 7]. The creation of abdominal compartment syndrome as a technical term is associated with the work of Fietsam in 1989 [8]. The golden age of ACS was launched by two papers of Schein [9] and Burch [10] published in 1995 and 1996, respectively. Later on several research groups developed the method of modern IAP measurement (Iberti, Sugrue, Malbrain, Balogh). The World Society of Abdominal Compartment Syndrome (WSACS) was founded in 2004. This name was recently changed to the Abdominal Compartment Society.

DEFINITIONS

IAP: The steady-state pressure concealed within the abdominal cavity, which is approximately 5–7 mm Hg in critically ill adults [11].

IAH is defined by a sustained or repeated pathological elevation in IAP ≥ 12 mm Hg [11].

ACS is defined as a sustained IAP > 20 mm Hg (with or without an APP < 60 mm Hg) that is associated with new organ dysfunction/failure (abdominal perfusion pressure [APP] = mean arterial pressure [MAP] – IAP) [11].

PCS is a condition where two or more anatomical compartments have elevated compartmental pressures [11].

Primary IAH or ACS: This is a condition associated with injury or disease in the abdominopelvic region that frequently requires early surgical or interventional radiological intervention [11].

Secondary IAH or ACS: It refers to conditions that do not originate from the abdominopelvic region [11].

Recurrent IAH or ACS: It refers to the condition in which IAH or ACS redevelops following previous surgical or medical treatment of primary or secondary IAH or ACS [11].

GRADES OF INTRA-ABDOMINAL HYPERTENSION

Grade I: IAP 12–15 mm Hg
Grade II: IAP 16–20 mm Hg
Grade III: IAP 21–25 mm Hg
Grade IV: IAP > 25 mm Hg [11]

RISK FACTORS

Risk factors of intra-abdominal hypertension and abdominal compartment syndrome are presented in Table 1.

TABLE 1. Risk factors of intra-abdominal hypertension and abdominal compartment syndrome [11–14]

Decreased compliance of abdominal wall	Increased intra-abdominal content	Abdominal space occupation	Capillary leaking over resuscitation
Acute respiratory insufficiency (elevated intra-thoracic pressure: PEEP)	Dilatations in gastrointestinal tract	Haemoperitoneum	Acidosis (pH < 7.2)
Abdominal wall closed under tension	Gastric paresis	Pneumoperitoneum	Hypotension
Severe trauma	Gastric dilatation	Ascites	Hypothermia (core temperature < 33°C)
Severe burning	Volvulus	Liver insufficiency	Polytransfusion (< 10 U day^{-1})
Prone position	Bowel obstruction		Extreme fluid resuscitation (< 5 L day^{-1})
Head of bed elevated more than 30°			Pancreatitis
High BMI			Oliguria
Central obesity			Sepsis
			Severe trauma or burning
			"Damage control surgery"
			Coagulopathy (platelets < 55 G L^{-1}, PT < 15 s, PTT double normal value, INR < 1.5)

BMI – body mass index, PEEP – positive end expiratory pressure, PT – prothrombin time, PTT – partial thromboplastin time, INR – international normalised ratio

PATHOPHYSIOLOGICAL CHANGES CAUSED BY ELEVATED INTRA-ABDOMINAL PRESSURE

Pathophysiological changes caused by elevated intra-abdominal pressure are presented in Table 2.

INTRA-ABDOMINAL PRESSURE MEASUREMENT TECHNIQUES

The only possible way of establishing the diagnosis is to measure the intra-abdominal pressure, a widespread manner of which is the measurement through the bladder. The fundamental principle of the method is the law which says that if pressure is exerted on the surface of a compartment predominantly containing some kind of fluid, then this pressure imposed upon the practically incompressible fluid will be transmitted unaltered to each and every point of the affected compartment. Consequently the IAP and the intravesical pressure values are strictly identical. If the bladder is filled with 50 mL of physiological saline and the previously inserted catheter is closed, then the pressure predominating the bladder will be transmitted to the catheter and become easily measurable through a sterile needle inserted into the catheter. This procedure was simplified by the working group of Sugrue, who placed a "T-element" into the catheter, which rendered unnecessary the closure and insertion of it, also significantly reducing the prevalence of infections associated with this measurement. To surmount points of weakness (labour-intensive, intermittent) Balogh and his working group developed and validated the method of continuous intra-abdominal pressure monitoring (CIAPM) [15].

Intermittent pressure measurement technique

This technique is carried out using a simple bladder catheter (Foley balloon catheter, 16–20 Fr, latex or silicone). During the measurement the urine collection bag is removed and the bladder is filled with 25 mL of physiological saline through the lumen of the catheter. The next step is to connect the lumen of the catheter to a set traditionally designed and used for the measurement of the central venous pressure (B. BRAUN Medifix pressure measurement scale) with or without the insertion of a T-tap. The zero point of the scaled measurement tube is designated in the medioaxillary line corresponding to the anterior superior iliac crest. After waiting 1–2 minutes, at the end of exhalation the value of IAP could be read off the scale in units of cm H_2O. The values read off should be converted to mm Hg (1 mm Hg = 1.36 cm H_2O). Once the measurement is completed the system and the bladder catheter are disconnected and the latter is connected to a urine collection bag [16].

Continuous pressure measurement technique

The technique of continuous intra-abdominal pressure measurement was published by Balogh *et al.* in 2004 [17]. For this procedure the generally used catheter is an 18 Fr (or bigger) standard three-way bladder catheter (LubriSil All-Silicone Foley catheter, C.R. Bard, Inc., Covington, GA, U.S.A.). The catheter and the urine collecting bag remain connected all the time. In order to perform the pres-

TABLE 2. Pathophysiological changes caused by elevated intra-abdominal pressure

IAP = 0–9 mm Hg	IAP = 10–15 mm Hg	IAP = 16–25 mm Hg	IAP = 26–40 mm Hg
Cytokine release	Circulation of abdominal wall decreased by 42%	Significant decrease in parenchymal circulation and venous return	"Haemodynamic collapse"
Increased capillary permeability	Significant decrease in blood supply of intra-abdominal organs	Increased systemic vascular resistance, central venous pressure and respiratory peak flow	Fatal acidosis
Increased "third space" fluid content	Local acidosis	Decreased total respiratory and vital capacity	Hypoxia Hypercapnia
Decreased venous return Decreased preload	Free radical release	Hypoxia Hypercapnia	Anuria
Early central nervous system effects	Bacterial translocation through bowel wall	Circulation of bowel mucous decreases by 61%	Circulation in celiac trunk decreases by 58%
		Severe acidosis	Superior mesenteric artery circulation decreases to 39%
		Renal insufficiency: oliguria, anuria	Renal artery circulation decreases to 30%
		Central nervous system injuries	Circulation in abdominal wall muscles decreases by 80% (infection, abnormal wound healing)

sure measurement the so-called flushing port of the catheter is connected with the insertion of a transducer to a 24-hour bedside monitor. The connection of the flushing port and the transducer is effectuated with a triple tap. The collapse of the bladder is prevented with physiological saline continuously perfused with the speed of 4 mL day^{-1}. The zero point for the fixation of the transducer is established in the plane determined by the axillary median line and the anterior superior iliac crest. After the system is set to zero the measured data are continuously recorded; the data can be easily read off from the bedside monitor. The actual IAP value appears directly in mm Hg and requires no further conversion.

Comparative study of intermittent and continuous pressure measurement techniques

In order to determine the objectivity of the continuous intra-abdominal pressure measurement, we carried out measurements in patients with normal and elevated IAP. The results of this study were published in 2017 [15].

Significant difference could not be observed between the results of the two procedures. According to the statistical analysis, the concordance correlation coefficient was higher than 0.97 in all cases, which shows a strongly significant agreement between the two different techniques (Figures 1 and 2). The 95% limits of agreement of the Bland-Altman method were within the non-significant ± 2 mm Hg range (Figures 3 and 4).

According to our results, we can summarise that the continuous IAP-monitoring technique is a modern, safe and accurate method for IAP monitoring, which provides immediate results in millimetres of mercury without need of conversion.

SERUM ADENOSINE AND INTRA-ABDOMINAL PRESSURE

The technique of continuous intra-abdominal pressure monitoring (CIAPM) is accurate, precise, reproducible and cost-effective. However, laboratory measures for monitoring of IAH have not been defined. In one of our studies we investigated the linkage between the serum levels of adenosine (Ado) and interleukin 10 (IL-10) with IAP [2].

Significant correlations of IAP were found with serum levels of Ado and IL-10. In the sera of patients with IAP > 12 mm Hg (> 1.6 kPa), the levels

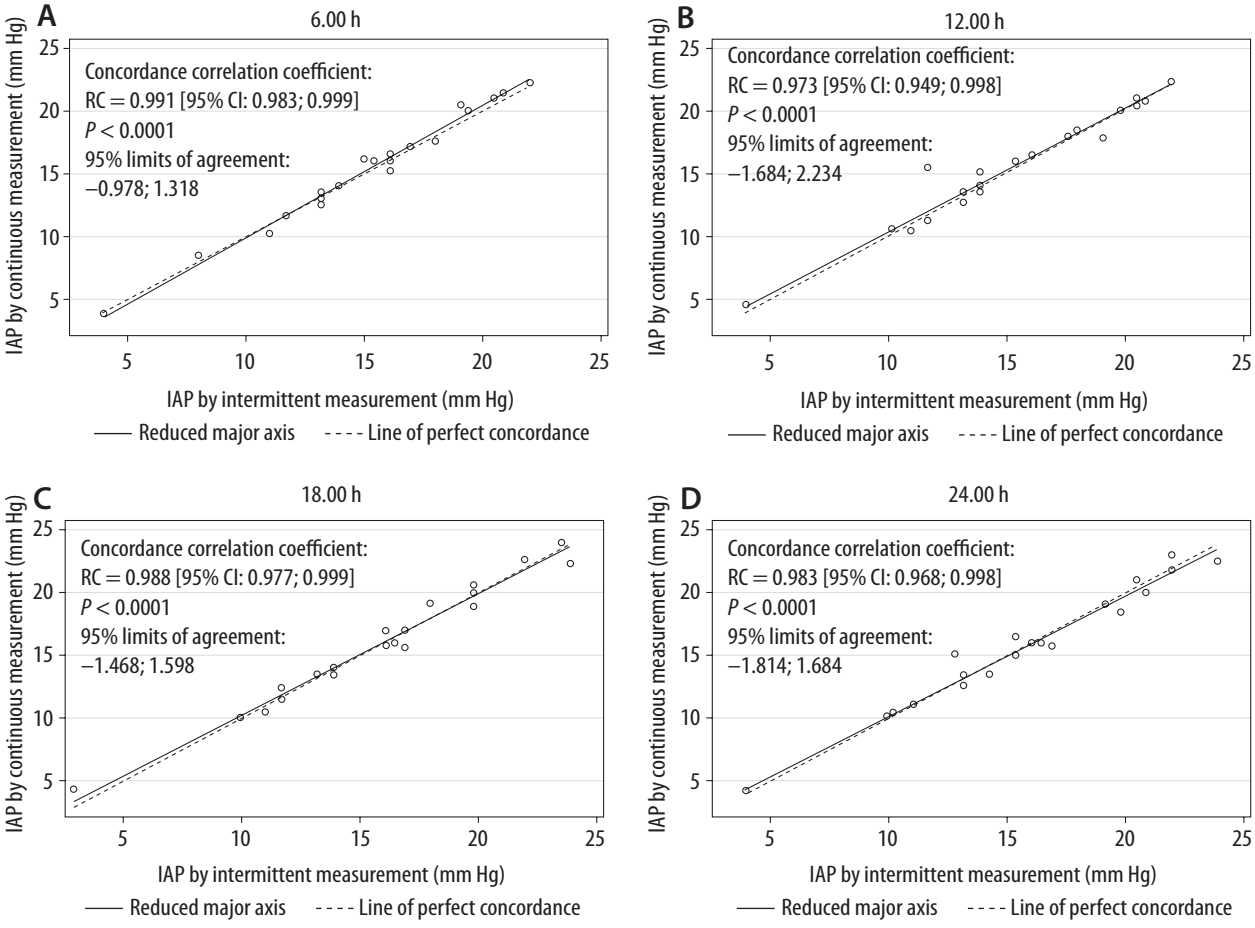

FIGURE 1. The concordance correlation coefficient was higher than 0.97 in all cases during the measurements carried out at 6.00 (**A**), at 12.00 (**B**), at 18.00 (**C**) and at 24.00 (**D**) hours

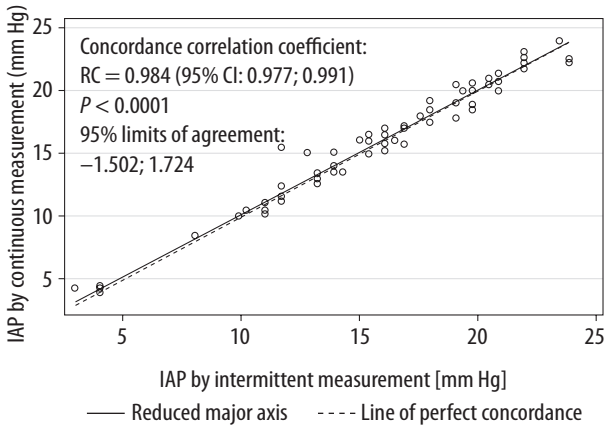

FIGURE 2. The concordance correlation coefficient was higher than 0.97 in all cases

of both Ado (1.61 vs. 0.06 μM, $P < 0.01$) and IL-10 (63.23 vs. 27.27 pg mL^{-1}, $P < 0.01$) were significantly higher than those in patients with IAP < 12 mm Hg (< 1.6 kPa). Moreover, significant correlations were found between individual patient IAP-Ado values ($r = 0.766$, $P < 0.001$), IAP-IL-10 values ($r = 0.792$, $P < 0.001$) and Ado-IL-10 values ($r = 0.888$, $P < 0.001$). A direct linear correlation between IAP-Ado and IAP-IL-10 values was only observed with IAP > 15 mm Hg (> 2 kPa) [2].

In conclusion, we reported that serum concentrations of adenosine and IL-10 are strongly and linearly correlated with the values of IAP > 15 mm Hg (> 2 kPa) in surgical patients. Thus, monitoring of serum adenosine and IL-10 concentrations may offer significant insights into the progression and treatment of IAP, particularly in patient populations at risk of IAH and ACS. The role of adenosine in the pathomechanism of IAH-ACS offers a new insight into this severe clinical syndrome [2, 3, 18, 19].

POLYCOMPARTMENT SYNDROME

The PCS is a rare, extremely serious, life-threatening clinical picture, when two or more compartments have elevated pressures at the same time and upon releasing one of the affected compartments the syndrome disappears [20, 21]. Due to its unusual nature it is frequently underdiagnosed and untreated. The real existence of this syndrome was first suggested by Scalea in 2007 [5]. For description of this clinical entity the "multiple compartment syndrome" was introduced by him. The term used by Scalea was modified by Malbrain in the same year and in the international literature the term "polycompartment syndrome" became widely used [22–26]. The diagnostic process is based on the compartment pressure

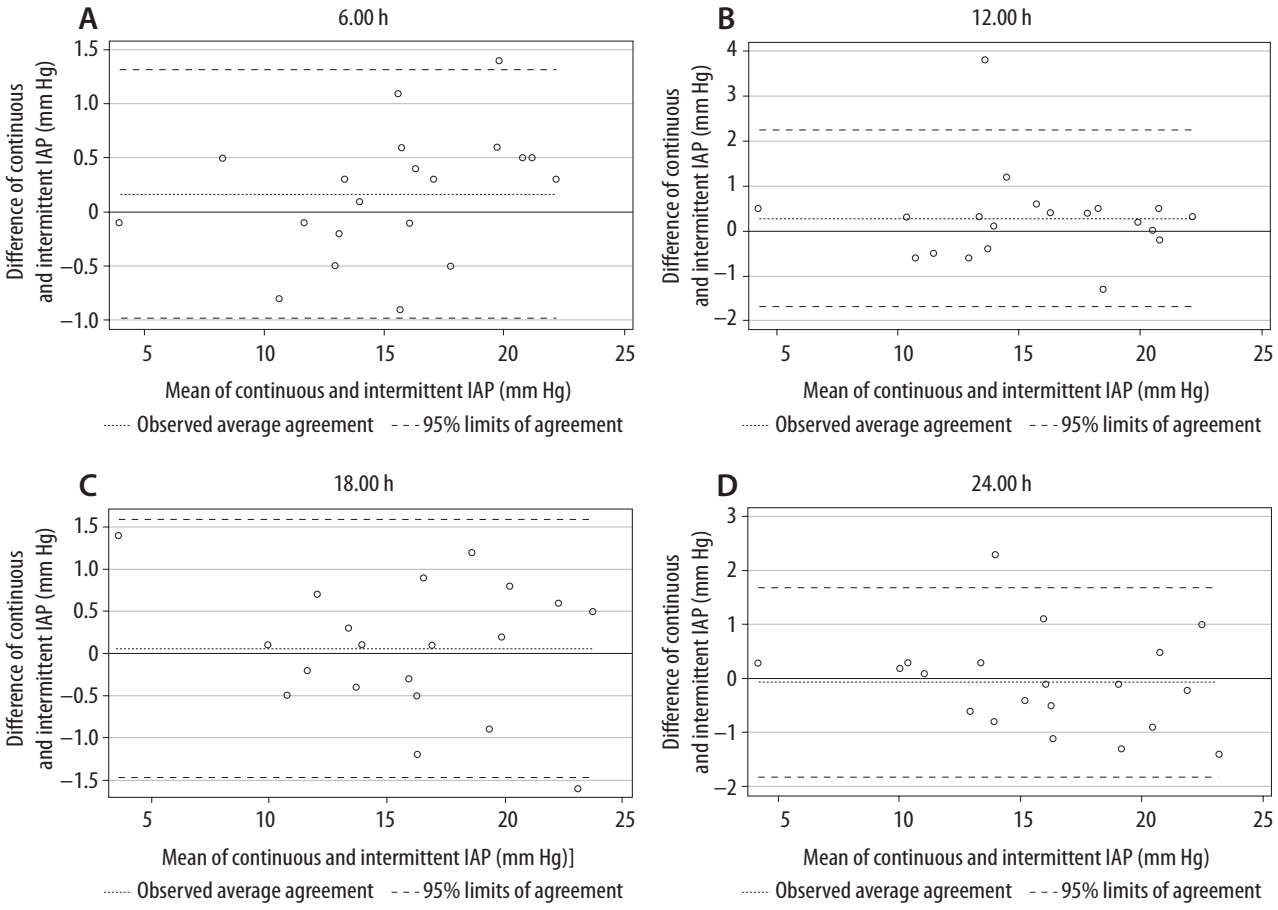

FIGURE 3. The 95% limits of agreement of the Bland-Altman method were within the non-significant ± 2 mm Hg range in all cases during the measurements carried out at 6.00 (**A**), at 12.00 (**B**), at 18.00 (**C**) and at 24.00 (**D**) hours

measurement. The abdominal compartment and the effect of the elevated IAP on different organs and/or organ systems play the central role. The goals of treatment are: to reduce the compartment pressures (to improve the compliance and/or to open the different compartments = decompressive surgery); patient tailored general and organ supportive therapy (be aware, fluid resuscitation is a double edged sword!); to avoid the adverse effects of ischaemia-reperfusion after performing surgical decompression [22–26].

DISCUSSION

Measurement of the IAP is essential in the differentiated diagnostics of acute abdominal pathologies, in the follow-up process of critically ill surgical patients, in the prevention of IAH/ACS, as well as in the monitoring of the already developed syndrome [15]. The IAP is never a constant value, but has an oscillatory nature even during 24 hours [27]. This nature was the main factor necessitating development of a continuous control providing measurement method [17]. In order to determine the objectivity of the continuous technique we carried out measurements in twenty patients and we verified that the intermittent and continuous measurements are trusty methods of intra-abdominal pressure monitoring without significant differences between them [15].

Besides the pathophysiological changes in each organ there is also a crucial role of cell abnormalities among which the most important is hypoxia due to the elevated IAP, leading to a significant increase of serum adenosine level [2, 3]. During the last few years it was confirmed by numerous studies that hypoxia, trauma and inflammation are trigger factors of adenosine production [2, 3, 28–31]. Above all, the idea to study the role that adenosine potentially plays in conditions of increased intra-abdominal pressure was provided by previous adenosine-related experience of the researchers of our working group [2, 3, 32].

In the development of ACS we assumed the central role of adenosine produced by the hypoxic tissues as an effect of elevated intra-abdominal pressure [2, 3].

Surgical decompression and open abdomen management are the definitive treatment options of IAH/ACS. However, the trend is more towards less invasive management, and in the future, medical treatment may play an increasingly important role in the prevention and management of IAH [3, 18, 19].

Based on the central role played by adenosine in the development of abdominal compartment syndrome and assuming that it has a central paper in the signal transfer processes of the human body, it seems to be logical that adenosine should have a crucial role in the pathophysiology of PCS as well. However, further studies are required to demonstrate this effect.

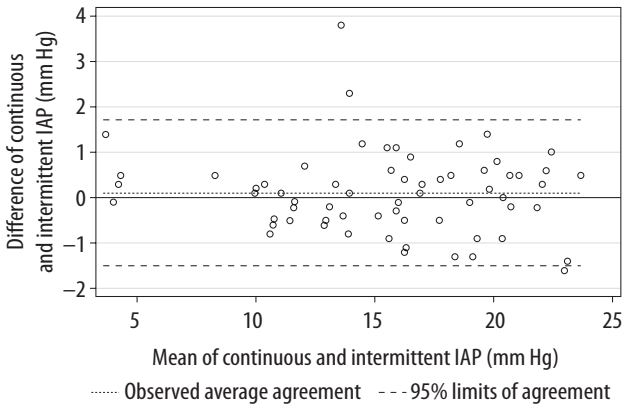

FIGURE 4. The 95% limits of agreement of the Bland-Altman method were within the non-significant ± 2 mm Hg range in all cases

CONCLUSIONS

The thoracic and/or cranial compartment syndrome results as an accumulation of air, fluid or blood in the chest and/or skull leading to secondary abdominal compartment syndrome. During the last 10 years many case studies have demonstrated that the urgent decompressive laparotomy was successful to decrease not only the IAP, but also the intra-thoracic and intra-cranial pressures, supporting the correlation of pressures and the existence of polycompartment syndrome [20, 21].

The measurement of IAP is essential in development of PCS. Clinicians need to be aware of the real existence of this life-threatening syndrome and the complex and constant interplay of raised pressure between different compartments [22–26].

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