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Abdominal pressure in the critically ill: measurement and clinical relevance

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Introduction

For years intensive care physicians have focused on the abdomen, and especially the gut, and bacterial translocation as the motor of the multiple organ dysfunction syndrome (MODS). Within this concept, intra-abdominal pressure (IAP) is an important parameter of underlying abdominal problems and may be a prognostic indicator of the patient's physiological status. It is easy to measure at the bedside with the standardised intravesical recording method and it has been used mainly by surgeons as an indicator for the abdominal compartment syndrome (ACS), or as a guide to perform a second look laparotomy or to leave the abdomen open after decompression. The measurement of IAP (normally subatmospheric to zero mmHg), hence, is not a new concept but it is only recently that its importance and therapeutic implications for the intensive care unit (ICU) physician have become apparent. It was probably Wendt, as early as 1876, who first described the association between intra-abdominal hypertension (IAH) and renal impairment. However, until the early 1920s there was a poor understanding of the concept of IAP. Only later, in the 1940s and more recently in the 1970s and 1980s, were numerous human and animal studies published in the surgical literature which showed the different deleterious effects of raised IAP on the cardiovascular and respiratory system and every organ concealed within the abdominal cavity. It was suggested that the abdomen and its contents should be considered as relatively non-compressive and primarily fluid in character behaving in accordance with Pascal's law. Although initially thought to affect primarily trauma and surgical patients, IAH and ACS, have since the late 1980's and 1990's, also been identified in medical patients. Only since then have ICU physicians become aware of the deleterious effects on organ function of only slight rises in IAP as low as 10 mmHg. In the years to follow, IAP will therefore be increasingly used as part of routine monitoring in the ICU, and maybe as a independent prognostic factor. Before adding IAP to our armamentarium of monitoring variables, we have to understand better the measurement technique, the incidence of IAH in different ICU patient populations, the pathophysiological implications on cardiovascular, cerebral, respiratory, renal and visceral function, and the possible therapeutic options. This article on current topics in intensive care will take us from the basics of measurement to animal studies, human studies and finally to some interesting options for the future. It was beyond the scope of the paper to discuss ACS also: for further reading on this topic there is an excellent review by **Cheatham ML (1999 New Horizons 7:96–115**).

From the basics of measurement . . .

Cheatham ML, Safcsak K (1998) Intraabdominal pressure: a revised method for measurement. J Am Coll Surg 186:594–595

As originally described, the Kron technique (**1984 Ann Surg 199:28–30**) disrupts for each IAP measurement what is normally a closed sterile system placing the patient at increased risk of urinary tract infection or sepsis and subjecting healthcare providers to the risk of needle stick injuries and exposure to blood and body fluids. Iberti and collegues (1989 Anesthesiology 70:47–50) re-

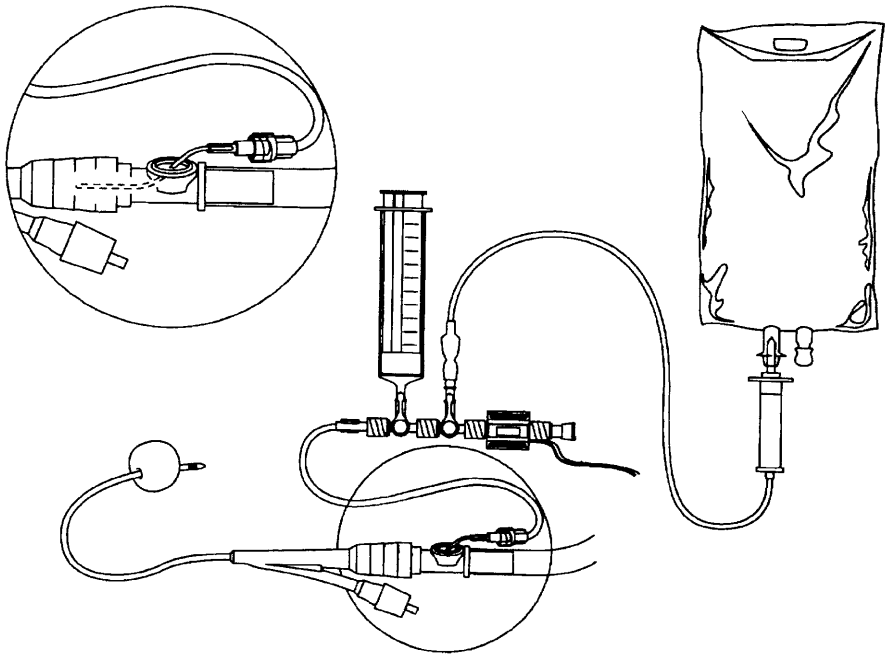

Fig. 1. Revision of the original Kron method for intravesicular pressure measurement by Cheatham and Safcsak. Reprinted with permission from the American College of Surgeons (Journal of the American College of Surgeons, 1998, 186, 594–595)

ported the use of a closed system drain and transurethral bladder pressure monitoring technique, using a 20-gauge needle for each IAP measurement, hence also subjecting healthcare workers to needle stick injuries. **Cheatham and Safcsak** report a revision of Kron's original technique, which is safer, less invasive, more efficient (repeated measurements possible) and cost-effective (Fig. 1). A standard intravenous infusion set is connected to 1000 ml of normal saline, two stop-cocks, a 60 ml Luer lock syringe and a disposable pressure transducer. An 18-gauche plastic intravenous infusion catheter is inserted into the culture aspiration port of the Foley catheter and the needle is removed. The infusion catheter is attached to the first stop-cock via arterial pressure tubing. After being flushed with saline and "zeroed" at the level of the symphysis pubis (or the midaxillary line when the patient is in complete supine position), the Foley catheter is clamped immediately distal to the culture aspiration port. The stop-cocks are turned "off" to the patient and pressure transducer and 50 ml of saline is aspirated from the intravenous bag. The first stop-cock is turned "on" to the patient and the 50 ml of saline is instilled into the bladder. The stop-cocks are turned "off" to the syringe and the intravenous tubing. After equilibration, the patient's IAP is then measured at end-expiration on the bedside monitor. To verify correct measurement, gentle compression of the abdomen should give instant variations on the IAP reading in form of oscillations, if a damped signal is noted then the clamp on the Foley catheter is momentarily released in order to ensure that all air is flushed and IAP is measured again. After correct reading the clamp is removed, the bladder allowed to drain, and the volume of saline utilised is substracted from the patient's urine output for that hour. The authors claim that using this revised technique the cost of performing intravesicular pressure measurements is reduced to < US $10 per day and required nursing time is reduced to < 1 min per measurement. Most importantly, the risk of urinary tract infection and sepsis as well as needle stick injury is minimised; furthermore, this technique allows repeated IAP measurements with the same equipment. Intravesical pressures are not reliable in cases with low intrinsic bladder compliance, bladder trauma or a pelvic haematoma compressing the bladder, since bladder pressure may then overestimate IAP: in those cases the above described procedure can be applied via a nasogastric or gastrostomy tube as studied by **Collee et al. (1993 Intensive Care Med 19: 478–480)**.

. . . to animal studies . . .

Diebel LN, Dulchavsky SA, Wilson RF (1992) Effect of increased intra-abdominal pressure on mesenteric arterial and intestinal mucosal blood flow. J Trauma 33:45–49

The effects of serially increasing IAP on intestinal blood flow were studied in eight anaesthetized pigs. The authors assessed blood flow to the small bowel using three techniques: ultrasonic flow probe (to measure mesenteric artery blood flow), laser Doppler flow probe (to measure direct intestinal mucosal blood flow) and determi-

nation of intramucosal pH (to measure regional intestinal mucosal perfusion). While systemic perfusion indices [mean arterial pressure, pulmonary capillary wedge pressure (PCWP) and cardiac output] were maintained during the study period with the infusion of lactated Ringer's solution, the authors observed a severe progressive decrease in mesenteric and mucosal blood flow as IAP was increased, particularly at levels above 20 mmHg (30–40 % decrease at IAP levels of 10–20 mmHg and 40–70 % at levels of 20–40 mmHg). Although actual correlation coefficients were not reported, it appears that the three techniques are similar. The authors did not include a control group but each pig served as its own control since at the end of the experiment IAP was returned to baseline (decompression) and with this there was a prompt restoration of mucosal and mesenteric blood flow. One concern is that the authors reported a rise in PCWP with increasing IAP – this can only be correct when PCWP is measured against atmospheric pressure, but it should be measured against intrathoracic pressure. Right ventricular end-diastolic volume index should be a better preload parameter in this condition. In the discussion, the authors elaborate on the different pathophysiological implications of raised IAP and postulate that when intestinal mucosal ischaemia is allowed to develop and progress it may lead to translocation of bacteria into the portal venous blood and intestinal lymphatics, leading to sepsis and multiple organ failure. They advocate the use of laser Doppler probes and tonometric techniques in combination with IAP measurement in the clinical setting in guiding abdominal decompression.

Diebel LN, Dulchavsky SA, Wilson RF (1992) Effect of increased intra-abdominal pressure on hepatic arterial, portal venous, and hepatic microcirculatory blood flow. J Trauma 33:279–283

In a comparable animal model with five domestic pigs, the same authors document in this well designed study that although systemic perfusion parameters were again maintained, slight increases in IAP (10 mmHg) resulted in dramatic reductions in hepatic arterial blood flow (39 %). It was only at IAP levels of 20 mmHg that decreases in portal venous flow (34 %) and microcirculatory blood flow (29 %) became apparent. The vascular resistance of the intestine, by regulating the inflow of blood into the portal circulation, and of the hepatic arterial system is the major determinant of liver blood flow. In IAH, however, IAP can become the major factor for determining mesenteric vascular resistance, and thus hepatic blood flow can be reduced on a purely mechanical basis. Impairment in visceral perfusion with increased IAP may be more likely in the hypovolaemic patient. This study clearly demonstrates that the deleterious effects of IAH can occur at relatively low levels of IAP of about 10 mmHg.

Diebel LN, Dulchavsky SA, Brown WJ (1997) Splanchnic ischemia and bacterial translocation in the abdominal compartment syndrome. J Trauma 43:852–855

The same group continued their initial observations in another animal model (23 rodents) and demonstrated the occurrence of decreased mesenteric blood flow and loss of intestinal barrier function (as indexed by bacterial translocation) in the presence of increased IAP at levels of 20–25 mmHg for 60 min. Regardless of the relative importance of bacterial translocation in the subsequent development of multiple organ failure, this study demonstrates that the deleterious effects of IAH can occur in a relatively short time.

. . . and human studies . . .

Sugrue M, Jones F, Lee A et al. (1996) Intraabdominal pressure and gastric intramucosal pH: is there an association? World J Surg 20:988–991

A prospective study of 73 consecutive patients undergoing major abdominal surgery: all underwent gastric tonometry and IAP measurement three times daily. Mean Acute Physiology and Chronic Health Evaluation (APACHE) II admission score was 16 ± 9. Abnormal gastric intramucosal (pHi) readings (< 7.32) were present in 49.3 % of patients (39.7 % on ICU admission). Raised IAP (> 20 mmHg) was present in 38.4 % (28.8 % on admission), IAPs above 15 mmHg were present in 42.5 %. Compared to patients with normal pHi, patients with abnormal pHi were 11.3 [95 % confidence interval (CI) 3.2 to 43.5] times more likely to have increased IAP (> 20 mmHg), with an adjusted odds ratio (OR) of 1.4 (95 % CI 0.4 to 5.1). With a cutoff at 15 mmHg, the crude OR for abnormal pHi was 14.7 (95 % CI 4 to 57.7). Both abnormal pHi and raised IAP predict the same adverse outcome with increased risk for hypotension (OR 6.4; 95 % CI 1.5 to 32.2), intra-abdominal sepsis (OR 8.7, 95 % CI 0.97 to 199), renal impairment (OR 63.7; 95 % CI 7.6 to 1397), need for relaparotomy (OR 5.8; 95 % CI 1 to 43) and death (OR 18; 95 % CI 2.2 to 395). In their stratified analysis, the authors took into account only the values on admission for IAP, pHi and APACHE II score. Since about 70 % of all abnormal pHi and IAP readings were obtained on admission, there seems to be a need for more refined management of these patients in the operating room before they arrive in the ICU. Global indices of tissue perfusion, such as blood pressure, pulse rate, arterial pH and lactate, may not reflect aberrations of regional perfusion. Both IAP and pHi are strongly correlated and

both may better reflect early problems in regional perfusion of intra-abdominal organs.

Sugrue M, Buist MD, Hourihan F et al. (1995) Prospective study of intra-abdominal hypertension and renal function after laparotomy. Br J Surg 82:235–238

A prospective study of 88 consecutive patients after laparotomy by the same authors. The incidence of raised IAP (> 20 mmHg) was 33 %, as was the incidence of renal failure, and 69 % of the patients with renal failure had raised IAP. The authors conclude that there is a strong correlation between increased IAP and the development of renal failure (OR 12.4; (95 % CI 3.8 to 41.7) and subsequent death (OR 11.2; 95 % CI 2.8 to 47.9). Unfortunately, the authors did not mention if they used the same patient population as in the abovementioned study, which could cause some bias in interpreting the results.

Ivatury RR, Porter JM, Simon RJ et al. (1998) Intra-abdominal hypertension after life-threathening penetrating abdominal trauma: prophylaxis, incidence, and clinical relevance to gastric mucosal pH and abdominal compartment syndrome. J Trauma 44:1016–1023

A retrospective study of 70 patients admitted to a level I trauma centre with life-threatening abdominal trauma all had IAP measurements. Patients with IAH (IAP > 25 mmHg) were treated with bedside or operating room laparotomy. Injury severity was similar between patients who had mesh closure (n = 45) and those who had fascial suture (n = 25). The overall incidence of IAH was 32.9 %, 22.2 % in the mesh group versus 52 % in the fascial suture group. Mortality, MODS points, lactate and base deficit were significantly worse in the IAH group. Tonometry was performed in 42 patients (60 %) and, of these, 11 (18.3 %) also had IAH, of whom 8 (72.7 %) had abnormal pHi. In patients with IAH, pHi improved after abdominal decompression in 75 %. The authors conclude that IAH is frequent after major abdominal trauma and it may cause gut mucosal acidosis long before the onset of clinical ACS. Uncorrected, it may lead to splanchnic hypoperfusion, ACS, distant organ failure and death. Since it was a retrospective study that only examined patients with arbitrary "life-threatening abdominal trauma," the incidence of 32.9 % lacks meaning. The authors did not demonstrate whether IAH was an independent risk factor for multiple organ failure or death (no multivariate analysis done), neither did they indicate the level of IAH which requires decompression, since all patients with IAP > 25 mmHg had "prophylactic" decompression.

. . . creating options for the future

Sugrue M, Buist MD, Lee A et al. (1994) Intra-abdominal pressure measurement using a modified nasogastric tube: description and validation of a new technique Int Care Med 20:588–590

This prospective study assessed the accuracy of an intragastric method of measuring IAP via the balloon of a gastric tonometer and compared the results with simultaneous intravesical pressure recordings in nine patients who underwent laparoscopic cholecystectomy. The authors found a very good correlation between both IAP measurement methods. IAP measured via a tonometer balloon allows a continuous trend to be obtained without interfering with urinary output estimation. Unfortunately, simultaneous pHi and IAP measurements were not (yet) possible. A possible disadvantage of intragastric recording is the effect on interpretation of IAP values by the Migrating Motor Complex; however, these can easily be identified. Recording the "diastolic" value of IAP at end-expiration can resolve this problem. A fancy new monitoring device capable of measuring IAP and pHi simultaneously and showing their evolution over time as a trend could be the necessary push that IAP and pHi need before being generally accepted as part of routine monitoring in every ICU. In view of the numerous data obtained from the literature supporting the prognostic and therapeutic value, as well as the clinical relevance, of IAP and pHi, this is not merely a vague futuristic idea but it seems only a small step away from reality . . .

Discussion

The data obtained from the surgical literature show us the following:

1. IAP can easily be measured at the bedside with the standardised intravesical pressure recording method.
2. The exact level of IAH that defines „critical IAP" remains subject to debate, but there is consensus that decompression should be performed at levels of IAP above 20–25 mmHg; recent reviews, however, suggest that levels of IAP as low as 10 mmHg can cause organ dysfunction.
3. The incidence of IAH (IAP above 12–15 mmHg) varies according to underlying pathology but seems to be around 30 % in the surgical ICU population and is even higher in emergency surgery patients.
4. The abdomen acts as a fluid compartment and follows the laws of Pascal.
5. Normal parameters of preload (central venous pressure and PCWP) are unreliable in IAH, in this condition right ventricular end-diastolic volume index seems to be a better preload parameter.

6. IAH has deleterious effects on all organs concealed within the abdomen and outside, and these effects can occur at IAP values as low as 10 mmHg and in a relatively short time.
7. There seems to be a strong correlation between IAP, pHi and increased gut permeability (as demonstrated by bacterial translocation). The association between increased gut permeability and the subsequent development of multiple organ failure and death has been recently demonstrated by **Doig CJ et al.** (**1998 Am J Respir Crit Care Med 158:444–451**).
8. IAP and pHi predict the same adverse outcomes (shock, renal failure, sepsis, need for re-laparotomy and death).
9. IAP acts as a guide for re-laparotomy or decompression.

Since most of the data is obtained from anecdotal reports, animal studies and retrospective or small prospective human studies from the surgical literature, and since until now there have been no data available from large multicentre randomised trials in mixed ICUs, it is clear that the clinical relevance of IAP in the general ICU population seems controversial. However, in the last decade more and more clinical studies have been performed on mixed ICU populations. In a prospective pilot study of 405 ICU patients, we found that the overall incidence of IAH (> 12 mmHg) was 17.5 %. The mean IAP value in 71 patients with IAH was 15.8 ± 3.6 vs 6.4 ± 2.6 mmHg in the patients without IAH. The incidence in emergency surgery patients was 39.4 % versus 19.8 % in medical patients versus 6.1 % in scheduled surgical patients (**Malbrain 1999 Crit Care 3[Suppl 1]:20**). Seventy four patients (18.3 % of the study population) died in the ICU following the index admission. Twenty eight of 334 patients (8.4 %) without IAH died versus 46 of 71 patients (64.8 %) with IAH (crude OR 20.9; 95 % CI 11.2 to 39; $p < 0.0001$). The IAP was significantly higher in patients who died in the ICU: 13.2 ± 5.2 versus 7 ± 3.6 ($p < 0.0001$) as well as in patients who died in the hospital: 11.5 ± 5.3 versus 6.9 ± 3.6 ($p < 0.0001$). With a cutoff at 12, IAP had 62.2 % sensitivity, 91.6 % specificity, 86.9 % accuracy, 64.8 % positive predictive value and 91.6 % negative predictive value for ICU mortality, respectively, 45, 81.7, 79.8, 70.4 and 81.7 % for hospital mortality, and the higher the IAP value the poorer the survival.

Multivariate analysis showed that IAP is an independent risk factor for ICU and hospital mortality ($p < 0.0005$), with an adjusted OR of 1.5 (1.3 to 1.6) for ICU mortality and 1.4 (1.2–1.5) for hospital mortality. Beside death, the presence of IAH was also predictive for the need for and the duration of mechanical ventilation (suggesting difficult weaning), the occurrence of renal failure and the need for renal replacement therapy, the cost of ICU and hospital stay and the length of ICU stay. To conclude, this pilot study shows that the incidence of IAH in the general ICU population is quite high – about 17.5 % – and the presence of only a slight rise of IAP at 12 mmHg can cause significant organ dysfunction and is an independent predictor for mortality and morbidity. In another study we demonstrated the beneficial effects of very high positive end-expiratory pressure (PEEP) levels adjusted for IAP on respiratory function in patients with acute lung injury (ALI) (**Malbrain 1998 Int Care Med 24 [Suppl 1]:S125**). In this study of seven patients with ALI and IAH, we were confronted with the fact that the lung protective mechanisms suggested in the literature were difficult to achieve or to apply. After PEEP adaptation according to IAP, oxygenation parameters dramatically improved but at the expense of raised peak, plateau and mean alveolar pressures. In some patients peak pressures were as high as 57 and plateau pressures as high as 40–45 cmH_2O, without an increased risk for early barotrauma (one patient developed a pneumotharox on day 20). Instead of talking about surgical versus medical, pulmonary versus extrapulmonary, primary versus secondary acute respiratory distress syndrom (ARDS), we introduced the term ARDS with or without an abdominal component (increased IAP) that might influence chest wall mechanics. Our data as well as those obtained from **Gattinoni** (**1997 Am J Respir Crit Care Med 158:3–11**) and **Ranieri** (**1997 Am J Respir Crit Care Med 156:1082–1091**) clearly demonstrated that all ARDS patients are not the same in terms of lung mechanics, that underlying aetiologies and co-morbidities are important, that determination of upper and lower inflection points as settings for peak inspiratory pressure (and correlated maximal tidal volume) and PEEP is not as simple as it seems at the bedside, that IAP may affect chest wall mechanics and that abdominal decompression has beneficial effects on respiratory mechanics and oxygenation and that there might be a good correlation between Pflex and IAP. The correlation between IAP and the lower inflection point was studied in a prospective study of 11 ALI patients (**Malbrain 1999 Crit Care 3[Suppl 1]: 20–21**): over a 2-month period, 115 measurements were performed in 11 patients (10.5 ± 7.5 measurements in each patient). The values for IAP (mmHg), IAP (cmH_2O) and Pflex (cmH_2O) were 14.9 ± 6.8, 19.4 ± 8.9 and 13.3 ± 5.5, respectively, for the whole group of patients; 15.8 ± 7.6, 20.6 ± 9.8 and 13.2 ± 6 respectively in secondary ALI/ARDS and 12.6 ± 3.4, 16.4 ± 4.4 and 13.6 ± 3.9, respectively, in primary ARDS. There was a very good correlation between IAP (cmH_2O) and Pflex (cmH_2O) for the whole group of patients: $Pflex = 0.5552 \times IAP + 2.5146$ ($R^2 = 0.808$, $p < 0.0001$) and this correlation was even better in the patients with secondary ALI/ARDS: $Pflex = 0.5745 \times IAP + 1.3227$ ($R^2 = 0.888$, $p < 0.0001$). As suspected, the correlation was worse in patients

with primary ARDS: Pflex = 0.7622 × IAP + 1.1624 ($R^2 = 0.7428$, $p < 0.0001$).

On the basis of the literature review and the preliminary results of our own investigations, we believe that monitoring of IAP is of the utmost importance for the ICU physician; however, a lot of research for the general ICU population still needs to be done and the pilot studies need to be validated. The dilemma between "dry lungs are happy lungs" and "keep them dry watch them die" with regard to IAP also remains to be solved. Animal data showed interesting results regarding fluid balance and IAP, suggesting that large positive fluid balances result in increased IAP and lowered chest wall compliance (**Mutoh T et al. 1991 J Appl Physiol 70:2611–2618**). This is an interesting twist to the "dry versus wet" debate with respect to fluid therapy in ARDS. The truth may be somewhere in between, since other studies have shown that the deleterious effects of IAH occur at a lower level of IAP in hypovolaemia or haemorrhagic shock (**Simon RJ et al. 1997 J Trauma 42:398–405**) and may even be triggered by high PEEP levels, but in view of the changes in chest wall mechanics by increased IAP it may be preferable not to fluid overload our ARDS patients (**Schuller D et al. 1991 Chest 100:1068–1075**). Volume overload may contribute to abdominal distention in different ways: (a) dilating and engorging abdominal vessels, (b) generating ascites, (c) causing intestinal swelling with elongation of mesenteric veins causing outflow obstruction and tissue hypoxia and more intestinal swelling, (d) causing tissue and abdominal wall edema.