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EDITORIAL

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The polycompartment syndrome: towards an understanding of the interactions between different compartments!

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Intensive Care Medicine presents the results of two clinical studies looking at the dynamic effects on central venous pressure (CVP), pulmonary artery occlusion pressure (PAOP), pleural pressure (Peso), and intra-abdominal pressure (IAP) caused by respiratory variations [1, 2]. The study by Bellemare et al. [1] examined simultaneous Peso, CVP, and PAOP tracings in 24 mechanically ventilated patients. The in- and expiratory changes in CVP and PAOP were found to be well correlated with changes in Peso. The bias was better for PAOP ($2.2 \pm 8.2\,cmH_2O$) than for CVP ($2.9 \pm 10.3\,cmH_2O$), but the limits of agreement were large, suggesting that they are not interchangeable. The lowest bias was observed with changes in PAOP with positive pressure ($-0.05 \pm 3.2\,cmH_2O$). The clinical importance of these results cannot be neglected. First, in analogy with functional hemodynamic monitoring, large respiratory swings in CVP or PAOP are correlated with fluid responsiveness [3, 4]. Second, the respiratory variations and especially the inspiratory fall in CVP or PAOP give an indication of the effort required to trigger the ventilator and are thus correlated with the work of breathing. Third, large inspiratory swings increase the afterload of the left ventricle which can result in pulmonary edema and respiratory failure postextubation.

The study by Leatherman et al. [2] analyzed simultaneous changes in IAP and CVP tracings also in 24 patients but this time during spontaneous breathing. A first group included 18 patients without and the second group included 6 patients with active expiration. The best CVP was defined as the end-expiratory CVP during relaxed breathing. The corrected CVP was calculated by subtracting the changes in IAP (ΔIAP) from the end-expiratory CVP during active expiration to correct for the effect of expiratory muscle activity (uncorrected CVP). The bias compared to the best CVP was lower for corrected CVP (2.3 ± 2 mmHg) than for uncorrected CVP (12.5 ± 4.7 mmHg). The most important clinical findings from this study are that, first, in the presence of active expiration a reasonable estimate of transmural CVP can be obtained by subtracting the expiratory increase in IAP from the end-expiratory CVP and, second, this approach may lessen the likelihood that fluid therapy would be withheld from hypovolemic patients.

Both studies have limitations: the first because simultaneous changes in IAP were not mentioned, the second because changes in Peso were not monitored. Further, in the first study it is not stated how deep the patients were

sedated, or whether they received curarization, and therefore we do not know about the use of accessory (abdominal) muscles, and in the second study it still remains unclear how one can objectively define "active expiration."

Poiseuille was the first to measure pressures in confined body regions or systems with mathematical accuracy [5]. After eighteenth century measurements of blood pressure in the horse by Stephen Hales (1677–1761) blood pressure measurement in the nineteenth century became successful in humans with a new sphygmomanometer, which Scipione Riva-Rocci (1863–1937) utilized from 1890 onwards. In France, in Claude Bernard's laboratory, Paul Bert (1833–1886) measured pressures through tubes inserted in the trachea and rectum. He ascribed elevation in the IAP during inspiration to diaphragmatic descent. Similar rectal pressure measurements were correlated by E.C. Wendt with urine production. Thus, everything was said two centuries ago, or not . . . Do we need to reinvent the future?

Only recently ICU clinicians have become aware of the effects of increased IAP on intra-abdominal and remote organ function [6]. Predictors for the development of intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS) are a high body mass index, capillary leak, and fluid resuscitation [7]. The combination of the latter two results in bowel edema, swelling, and ascites formation. The extravasation of fluids into the interstitium stiffens the abdominal wall, reducing its compliance, and further increases IAP at a given point for the same abdominal contents [6]. Increased IAP can influence respiratory function by aggravating ventilator induced lung injury, altering lung distention and recruitment and increasing pulmonary edema [8].

Many European studies have been published suggesting variable abdominothoracic transmission both in animal models and in humans. Most reports describe IAP transmissions between 35% and 60%, using *mean* pleural pressure, and between 55% and 70% using *end-inspiratory* pleural pressure assessment [9]. Quintel et al. [8] described no effect on *end-expiratory* pleural pressure in pigs, but IAP was increased only to 15 mmHg. When using mean or end-inspiratory pleural pressures, part of the pressure increment in pleural pressure may be attributable to changes occurring during insufflation of tidal volume, resulting in an overestimation of corresponding IAP transmission [9]. Based on these findings it seems reasonable to suggest that end-expiratory pleural pressures should be used for assessment of abdominothoracic pressure transmission and to estimate transmural CVP in clinical practice.

Changes in IAP are paralleled by changes in pleural pressures [10]. Changes in thoracic compliance are reflected by changes in abdominal compliance and vice versa. Hence increased IAP results in a low chest wall compliance. Even before any knowledge of measurable pressures, the Frenchman Etienne-Jules Marey (1830–1904) stated that effects produced in the thorax by respiration are inverse to those present in the abdomen [5].

A recent study by Valenza and colleagues [11] demonstrated that elevations in IAP significantly increase both Peso and traditional end-expiratory CVP. Transmural CVP was unaffected by IAH or positive end-expiratory pressure. Volumetric measurements of preload were also unaffected by IAH. The superiority of volumetric

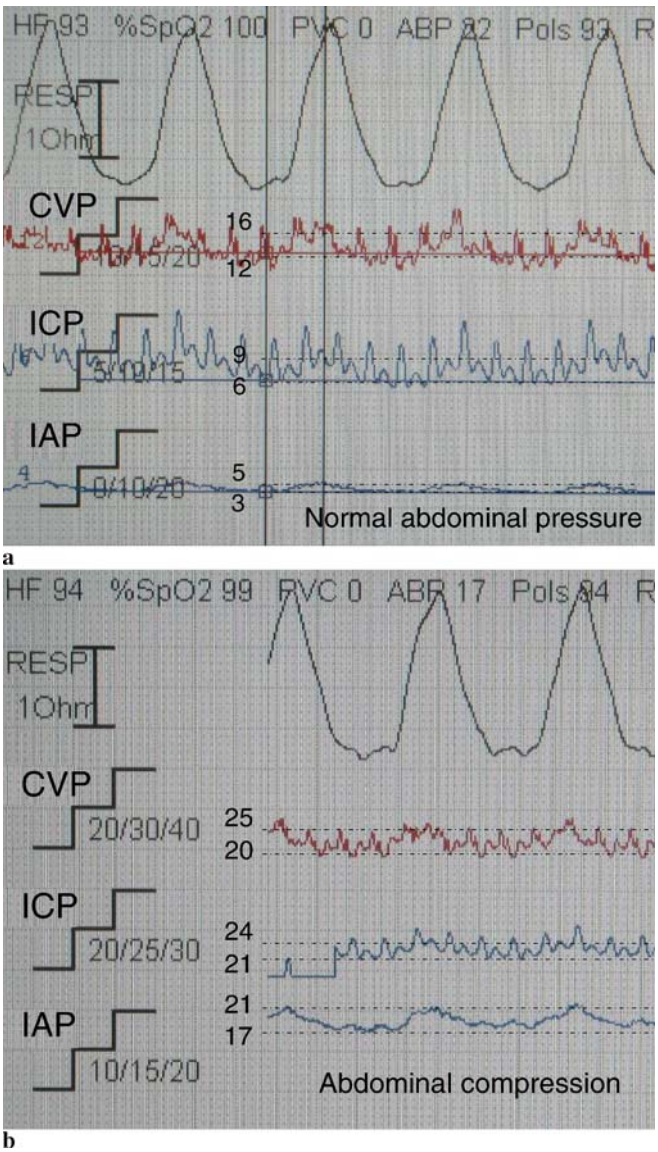

Fig. 1 Simultaneous tracings of respiration (*RESP*), central venous pressure (*CVP*), intracranial pressure (*ICP*), and intra-abdominal pressure (*IAP*) in a patient with combined head and abdominal trauma (see Table 1). The patient was mechanically ventilated via biphasic positive airway pressure mode with a RESP of 20 breaths per minutes, inspiratory pressure was set at 32 cmH_2O with a PEEP of 5 cmH_2O. Paper tracing speed at 6.25 mm/s. The respiratory in and end-expiratory variations in the pressure tracings can be observed. **a** During normal (baseline) IAP. **b** During increased IAP (abdominal compression with velcro belt for prevention of incisional hernia)

variables of resuscitation adequacy, including right ventricular end-diastolic volume, global end-diastolic volume, and intrathoracic blood volume, over intracardiac filling pressure measurements, such as end-expiratory CVP and end-expiratory PAOP, has previously been demonstrated in multiple human studies [12, 13].

The clinical relevance of these recent observations are, first, that they stress the importance of linking the different compartments while interpreting compartmental (intravascular) pressures. Second, they advocate the routine use of IAP in daily clinical practice while observing the dynamic changes in IAP and intracardiac filling pressures during respiration [14]. Third, they raise questions with regard to the standardization of IAP measurements in different head of bed positions or in awake patients. Fourth, abdominal compliance can be estimated by looking at the changes in IAP during respiration: A low abdominal compliance is characterized by large respiratory swings, and this could help to identify patients at risk for the detrimental effects associated with IAH or ACS. Fifth, the Surviving Sepsis Campaign guidelines emphasize the use of end-expiratory CVP as a resuscitation end-point, as suggested by the work of Rivers et al. [15]. The findings of the studies published now in *Intensive Care Medicine* and the study by Valenza et al. [11] should concern all critical care physicians who choose to use end-expiratory intracardiac filling pressures to guide the resuscitation of their patients. In the case of increased IAP or Peso these pressures cannot be used to accurately direct resuscitation, as was also suggested in a recent editorial by Cheatham [16]. Doing so places the patient at risk for under- or overresuscitation with resultant organ dysfunction, failure, and increased mortality. In the absence of Peso measurements transmural CVP can be estimated as end-expiratory CVP minus half the IAP given the typical transmission of IAP to the intrathoracic compartment [13].

The pressure tracings that can be obtained at the bedside in a clinical patient scenario are represented in Fig. 1 and Table 1. Abdominal compression with a Velcro belt (to prevent incisional hernia) not only increased the IAP but also the endexpiratory values for CVP and intracranial pressure (ICP).

Significant progress has been made over the past decade towards understanding the cause and pathophysiology surrounding IAH and ACS [7, 17]. In response to an outcry for consensus from clinicians worldwide the World Society on Abdominal Compartment Syndrome (www.wsacs.org) recently proposed state-of-the-art definitions and recommendations for the diagnosis, management, and prevention of IAH and ACS as well as recommendations for future clinical investigation [18–20]. We encourage all clinicians to review these guidelines and consider the impact of IAP and Peso in their daily patient care. Begin to measure IAP in patients who demonstrate risk factors for IAH and ACS, and, more importantly, try to transgress the boundaries of defined compartments to have a global, holistic view on the patient and the recognition of a multi- or polycompartment syndrome [21, 22].

Table 1 The different parameters at endexpiration and endinspiration at baseline conditions and after abdominal compression in a single patient (as an illustrative example; see Fig. 1). Abdominal compression resulted from the use of a Velcro belt. Average abdominothoracic transmission was around 60% while the abdominocranial transmission was almost 100% (*CVP*, central venous pressure; *ee*, end-expiratory; *ei*, end-inspiratory; *IAP*, intra-abdominal pressure; *ICP*, intracranial pressure; *ITee*, index of transmission during expiration; *ITei*, index of transmission during inspiration; *Δee*, difference between end-expiratory value during abdominal compression and baseline value; *Δei*, difference between end-inspiratory value during abdominal compression and baseline value; *ΔRES*, end-inspiratory minus end-expiratory value)

	Baseline			Compression			Index of transmission			
	ee	ei	ΔRES	ee	ei	ΔRES	Δee	Δei	ITee	ITei
IAP (mmHg)	3	5	2	17	21	4	14	16	–	–
ICP (mmHg)	6	9	3	21	24	3	15	15	107%	94%
CVP (mmHg)	12	16	4	20	25	5	8	9	57%	56%

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