

Validation of transurethral intra-abdominal pressure measurement in acute heart failure

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Introduction Intra-abdominal pressure (IAP) is the pressure within the abdominal cavity; its elevated values have been considered to be an important underlying mechanism of organ function deterioration in selected populations (described mainly in critical care patients). Interestingly, increased IAP was identified as one of the factors responsible for deteriorated renal function in patients with heart failure (HF) due to fluid overload.^{1,2} Venous congestion significantly decreases intra-abdominal venous and arterial blood flow, leading to multiorgan failure. In some cases, extravascular fluid (in the body cavities) require treatment modification, including evacuation to prevent further organ dysfunction.³⁻⁵ However, we should be aware that not only patients with evident signs of ascites present with abnormally increased IAP. Although fluid accumulation in this compartment is not evident in many cases, it may play an important role in organ perfusion, especially in HF with kidney deterioration or diuretic resistance. Moreover, a reduction of IAP through adequate treatment (optimal pharmacotherapy, ultrafiltration/dialysis, or, in some cases, paracentesis) could significantly improve organ function.¹⁻³ Therefore, this parameter could be useful in accurate patient profiling and providing adequate treatment strategies.

The direct IAP assessment is difficult because it involves an invasive penetration into the peritoneum. Therefore, many different (less invasive) methods have been proposed, with the transurethral approach being an interesting option. This technique assumes that pressure measured using a Foleys bladder catheter reflects the adequate IAP.³⁻⁷ However, there have been only limited reports showing the applicability of transurethral IAP measurement in selected populations (generally limited to critically ill patients under anesthesia or mechanically ventilated, individuals undergoing surgery, or patients with trauma).

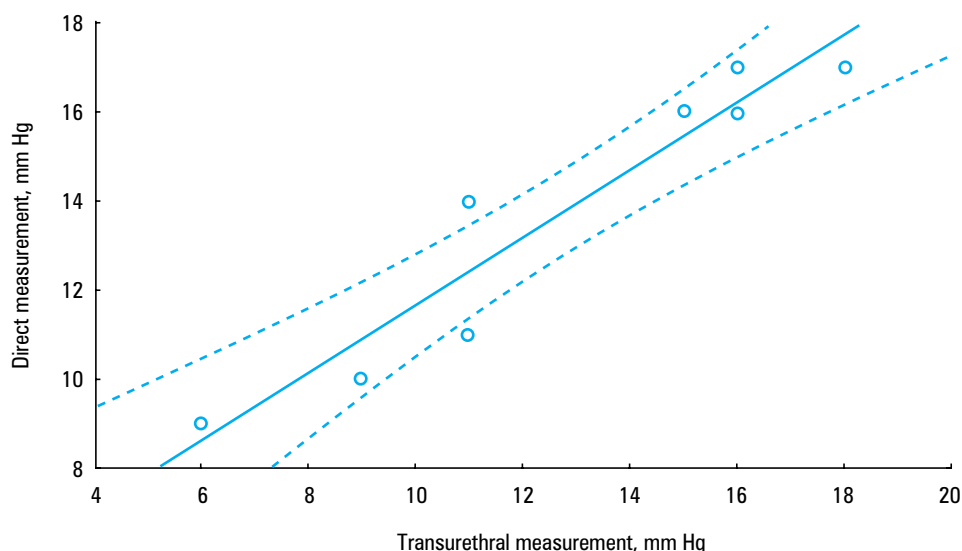
So far, the method has not been widely used in HF.¹⁻³ Hence, the aim of this study was to investigate whether this method could be also used in the general population of HF patients and to validate the transurethral method for IAP measurement in patients with acute HF.

Patients and methods The study population involved 8 prospectively enrolled patients with primary diagnosis of acute HF, who were hospitalized between 2010 and 2015 in the Centre for Heart Diseases at the 4th Military Hospital in Wrocław, Poland, and who underwent paracentesis due to ascites. All patients had Foley catheters inserted for clinical indications. Informed consent to participate in the study was obtained from all participants. The study protocol was approved by the local ethics committee, and the study was conducted in accordance with the Helsinki Declaration.

The IAP was measured using 2 simultaneous methods: direct and indirect (transurethral). The first technique involved intraperitoneal pressure measurement through the paracentesis. A needle was inserted into the abdominal cavity to evacuate the ascites (as a standard of care). The tip of the pressure tube line was placed in the peritoneal fluid through a paracentesis needle to prevent air from entering the line and the needle, and thus damping of the reading. The second method estimated the pressure in the urinary bladder using a Foley catheter after filling the bladder with saline, according to previous recommendations.^{2,3} The tip of the pressure tube was connected to the catheter to prevent air from entering the line and catheter, in order to avoid damping of the recording. All measurements were performed before the evacuation of ascites. Both pressure measurements were performed using the TruWave (3cc)/150 cm transducer sets (Edwards Lifesciences, Irvine, California,

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FIGURE 1 Correlation between direct (invasive) and transurethral intra-abdominal pressure measurements ($r = 0.95$, $P < 0.01$)



United States), which were first calibrated according to standard procedures.

Statistical analysis Continuous variables with normal distribution were presented as mean (SD); variables with skewed distribution were shown as medians with upper and lower quartiles; and categorical variables were presented as numbers and percentages. The significance of the differences between groups was assessed using the *t* test, and the relationship between variables was assessed using correlation rank coefficients. The concordance between the measurements obtained using the 2 methods was evaluated with the Bland–Altman test. A *P* value of less than 0.05 was considered significant. Statistical analyses were performed using Statistica 12 (StatSoft, Inc., Tulsa, Oklahoma, United States).

Results Characteristics of the study population

The study population predominantly consisted of men: 7 (87.5%) at a mean (SD) age of 62 (13) years. A total of 75% of these patients had an ischemic etiology of HF. All patients had decompensated chronic HF. Patients were examined within the first 48 hours of hospital stay. Mean (SD) systolic blood pressure was 94 (9) mm Hg, mean (SD) ejection fraction was 29% (10%), mean (SD) creatinine and serum sodium levels were 163.5 (70.7) $\mu\text{mol/l}$ and 136 (5) mmol/l, respectively. The mean (SD) volume of fluid aspirated from the peritoneum was 4.267 (1.850) ml.

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The mean (SD) IAP measured by the direct and transurethral methods was 13.75 (3) and 12.75 (4) mm Hg, respectively ($P > 0.05$). There was a correlation between the results obtained using the 2 methods (FIGURE 1). Moreover, the coefficient variability of both methods was 0.099 in the Bland–Altman test.

Discussion Undoubtedly, the novelty of the study is the first direct comparison of the 2 techniques

of IAP measurement, with further validation of the less invasive and easier-to-assess transurethral method in patients with acute HF. Our population differed from those described in previous reports. Our patients were not in a critical condition due to surgical problems; they were not intubated (breathed spontaneously) or sedated/under anesthesia. All these factors could have affected the measurements as the tension of the abdominal wall musculature as well as positive airway pressure (ventilation) might have an impact on IAP.^{4–7} We found a high concordance between the 2 methods for the measurement of abdominal pressure; thus, our data confirm the hypothesis that the pressure measured in the urinary bladder adequately reflects IAP and can be used as a surrogate of the direct measurement. This is an important finding as the transurethral assessment is less invasive and much safer than the direct method in selected cases. Moreover, as previously mentioned, these results may be applicable to other patients with or without ascites, treated in internal medicine wards and suspected of elevated IAP as a cause of diuretic resistance.

We believe that IAP may play an important role in the pathophysiology of organ function deterioration in patients with HF.^{1–5} Elevated abdominal pressure leads to increased venous pressure, which results in a lower perfusion gradient (the difference between mean arterial pressure and central venous pressure) with subsequent deterioration in the functioning of intra-abdominal organs. Finally, a low perfusion gradient decreases organ performance, which was also observed in our patients based on elevated creatinine levels (mean, 163.5 $\mu\text{mol/l}$).

Furthermore, our data support the hypothesis that elevated IAP is a common abnormality in acute HF, with a mean IAP in this population of 13 mm Hg, which is above the cutoff value of 12 mm Hg proposed to define intra-abdominal hypertension as well as above the values of normal IAP in critically ill patients (5–7 mm Hg).^{5–9} However, it should be noted that this study included

patients with acute HF and ascites. On the other hand, we believe that many patients with acute HF may present with elevated IAP even in the absence of ascites as a result of congestion and neurohormonal activation. Undoubtedly, this topic requires further research.

Our study was limited by the small number of patients. The population was unique as not all patients with acute HF developed ascites, which is considered to be a marker of advanced right-sided HF. We hope that our findings will be helpful in everyday practice also in patients with no symptomatic ascites, as well as will help determine the amount of fluid that can be safely evacuated.

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