Value of measuring esophageal pressure to evaluate heart-lung interactions—applications for invasive hemodynamic monitoring

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Abstract: Evaluation of intrathoracic pressure is the cornerstone of the understanding of heart-lung interactions, but is not easily feasible at the bedside. Esophageal pressure (P₀ₑₑ) has been shown to be a good surrogate for intrathoracic pressure and can be more easily measured using a small esophageal catheter, but is not routinely employed. It can provide crucial information for the study of heart-lung interactions in both controlled and spontaneous ventilation. This review presents the physiological basis, the technical aspects and the value in clinical practice of the measurement of P₀ₑₑ.

Keywords: Heart-lung interactions; esophageal pressure; pulsus paradoxus; reverse pulsus paradoxus

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Since the first description of heart-lung interactions by Stephen Hales in 1733 (1), the evaluation of the intrathoracic/pleural pressure at the bedside has always been an issue. Esophageal pressure (P₀ₑₑ) has been shown to be a good surrogate for intrathoracic pressure and can be more easily measured using a small esophageal catheter, but is not routinely employed. It can provide crucial information for the study of heart-lung interactions in both controlled and spontaneous ventilation. This review presents the physiological basis, the technical aspects and the value in clinical practice of the measurement of P₀ₑₑ.

The transmural pressure: back to physiology and definitions

Heart-lung interactions can be defined as the effects of pressure variations of the respiratory system on the circulatory system. Two mains pressures are responsible for such effects: pleural pressure per se and transpulmonary pressure (Pₜₚ). The transmural pressure of a cavity is defined as the pressure measured inside minus the surrounding pressure. The transmural pressure then represents the distending pressure of the cavity and can be considered as a surrogate of its volume. The surrounding pressure of the heart is the pleural pressure, approximated by P₀ₑₑ, except when the pericardial pressure is significantly increased (Figure 1).
Right atrial transmural pressure (\(\text{RAP}_{\text{TM}}\)) is defined by the intravascular RAP, measured invasively by a pulmonary artery catheter or a central venous line, minus the surrounding pressure of the atrium. The latter can be approximated by the measurement of pericardial pressure (10). In the absence of pericardial effusion, which is responsible for significant increase in pericardial pressure (11,12), the difference between pleural and pericardial pressure is low and well correlated with \(P_{es}\) (13). \(\text{RAP}_{\text{TM}}\) differs from intravascular RAP since an increase in the latter will decrease venous return according to Guyton’s law (14,15), while an increase of the former will increase cardiac output according to the Frank-Starling law (16,17). Then, except when the chest is opened, as reported by Guyton in his landmark study (18), the venous return to the intravascular RAP curve and the cardiac output to the \(\text{RAP}_{\text{TM}}\) cannot be superimposed, as frequently proposed.

At the interstitial level, the impact of \(P_{in}\) modification can also be very marked. Indeed, the transvascular pressure, i.e., luminal microvascular pressure minus interstitial pressure assumed to be equivalent to pleural pressure, can differ greatly between controlled breathing and spontaneously breathing patients. In the case of spontaneous breathing, the negative pleural pressure generated by the forceful inspiratory effort can be responsible for very high fluid-filtering transvascular pressure, which could enhance the pulmonary edema of such patients (19,20). This was one of the mechanisms proposed to promote ventilator-induced lung injury and was called lung interdependence (21).

Applying a positive (or less negative) pleural pressure by means of application of positive end-expiratory pressure (PEEP) could in part explain the protective effect of PEEP on the generation of pulmonary edema (Figure 2).

**\(P_{es}\) in usual practice**

The first measurement of \(P_{es}\) in healthy humans goes back to the end of the 19th century (22) and the first measurement of \(P_{es}\) was made in 1900 (23), whereas the classic monograph on \(P_{es}\) dates from 1949 and describes the insertion of a balloon into the esophagus (2). Initially based on a large inflation of the balloon, the method of measuring \(P_{es}\) was improved during the second part of the twentieth century. The most recent advances in the field were the modification of the amount of gas used to inflate the balloon (24,25) and the evaluation of the impact of the position of the balloon in the esophagus (26).
Effect of PEEP on transvascular pressure [adapted from (19)]. The left panel represents the example of a mechanically ventilated ARDS patient with 10 cmH₂O PEEP. Assuming that the transmission of PEEP to the pleural/interstitial pressure is about one-third, the pleural pressure measured by Pₚᵥ is 4 cmH₂O and the transvascular pressure 11 cmH₂O for an intravascular pressure of 15 mmHg. In contrast, ventilated in ZEEP conditions, this patient would have a Pₑₛ of −3 cmH₂O and then an increase in the transvascular pressure to 18 cmH₂O for the same intravascular pressure, exposing him to the risk of pulmonary edema by fluid leakage. PEEP, positive end-expiratory pressure; Pₑₛ, esophageal pressure; Pᵥₜ, transvascular pressure.

**How to measure Pₑₛ in 2018**

The first generation of custom-made esophageal balloons was exclusively used for experimental research. Such devices have been replaced by second-generation air- or liquid-filled (mainly in neonates) balloons, developed for bedside clinical use thanks to the connection to different monitoring devices (7). Each type of esophageal balloon has technical properties according to its composition, diameter, or length, but usually requires the injection of 0.5 to 4 mL of gas (27). The longer and the more compliant the balloon, the more accurate the measurement of Pₑₛ (28). Some devices are equipped with two balloons for measurement of both esophageal and gastric pressures. New devices are incorporated in naso- or orogastric tubes and allow continuous measurements for a long period (29).

After placing the patient in a semirecumbent position and deflating the esophageal balloon, the catheter is transorally or transnasally introduced in the upper esophagus and then pushed into the stomach. It should be noted that the semirecumbent position should be preferred to the supine position, because in the latter Pₑₛ is more altered by cardiac artifacts (30) and also tends to overestimate the value of Pₑₛ (26). Once in a gastric position, the balloon is inflated according to the recommendations and the distal part of the catheter is connected to the pressure transducer of a specific device or the ventilator. The gastric position of the balloon can be easily verified by the presence of a positive pressure deflection during a manual epigastric compression (7). The balloon is then progressively withdrawn into the lower two-thirds of the intrathoracic esophagus to obtain the most accurate values (25,30). In spontaneously breathing patients, a Mueller maneuver (inspiration with a closed glottis) can be used to verify that inspiratory effort-generated change in Pₑₛ is equal to the change in mouth pressure, in what is called the Baydur test (30). In controlled breathing patients, an external compression of the rib cage can be applied during an expiratory pause to observe a concomitant shift of the Pᵥₑₛ and the Pₑₛ (28).

**What are the limits?**

Due to the esophagus wall elastance, the filling volume can be responsible for an overestimation of Pₑₛ, even if the balloon is partially inflated (31). In contrast, it has been suggested that Pₑₛ could be underestimated if the balloon is unfilled (25,27,32). This might suggest that only respiratory-related changes of Pₑₛ (and note absolute values) should be studied with such methods (33). Recently, Mojoli et al. have suggested determining the best filling volume using the volume providing the maximum difference between end-inspiratory and end-expiratory Pₑₛ (i.e., maximizing ΔPₑₛ) (34). The best filling volume was higher than the factory-recommended inflating volume and was then associated with a significant esophageal wall pressure. A simple calibration procedure defined by PₑₛCAL = Pₑₛ – Pᵥₑₛ (where Pew defines the pressure generated by the esophageal wall and is calculated according to the esophageal elastance described in human beings), allowed improvement of the assessment of both relative changes and absolute values of Pₑₛ (34).

Each commercially available balloon catheter system has its own properties (material, compliance, recommended filling volume...), which exposes Pₑₛ measurements to the risk of artifacts. Users of such devices should be aware of the limitations of the corresponding balloon catheter system (35).

**The use of Pₑₛ for heart-lung interaction evaluation and hemodynamic monitoring**

As already said, heart-lung interactions rely on the pleural and transpulmonary pressures, while their evaluation in usual practice by Pₑₛ measurement is rarely performed.
In the last part of this review, we will describe how the use of P \(_e\) measurement could yield crucial information for the understanding of cardiorespiratory interactions, leading to very practical information for appropriate patient management. For educational purposes, we will dichotomize the value of P \(_e\) for heart-lung interactions into positive pressure ventilation (Figure 3) and spontaneous breathing.

**Reversed pulsus paradoxus**

Reversed *pulsus paradoxus* designates pulse pressure variation during mechanical ventilation (36). It has been described in strict cyclic opposition to the effect of spontaneous breathing on the circulatory system, the inspiration being associated with increased left ventricle (LV) ejection and the expiration with decreased LV ejection. RV and LV stroke volume are 180° out of phase. From an invasive point of view, P \(_e\) is mandatory to understand the mechanism of tidal ventilation, either a decrease in RV preload or an increase in RV afterload (Figure 4), while doing an echocardiography permits a simplified approach (19). It is crucial to differentiate patients for whom the main effect of tidal ventilation will be related to increased P \(_p\) (preload effect) from those for whom the main effect of lung inflation will be related to increased P \(_p\) (afterload effect). Indeed, management will be dramatically different. In both mechanisms, intravascular RAP is increased due to the transmission of P \(_p\) to the right atrium. But in the case of a decrease in systemic venous return driven by P \(_p\), RAP \(_{TM}\) is decreased (Figure 4) (37,38), while it is increased in the case of increased RV afterload driven by P \(_p\) (Figure 4). Increased resistance to venous return is a key factor in the decrease in systemic venous pressure driven by P \(_p\) (39). It was suggested to occur at least in the superior vena cava (SVC), using transesophageal
echocardiography (40), and was recently demonstrated by Lansdorp et al. in patients after cardiac surgery in whom, by measuring \( P_{es} \), the authors reported a decrease in the SVC transmural pressure during tidal ventilation (10). A similar observation was made in animals by Berger et al. (41). The pressure transmitted to RAP is probably one of the reasons why RAP is so limited in evaluating RV preload and in predicting the response to fluids, especially in patients with high pericardial pressure, as in cardiac tamponade, or with high \( P_{pl} \), as observed in the case of high therapeutic PEEP or dynamic hyperinflation. Scharf et al. clearly reported that when a high PEEP was applied in animals, RAP increased dramatically, while RAP\( _{TM} \) did not change or even decreased (38). It has been suggested that pulse pressure variation more accurately predicts fluid responsiveness when corrected by changes in \( P_{es} \) (42). In the case of dilated cardiomyopathy, \( R \) increases and \( t \) decreases, leading to a huge rise in \( \sigma \). One factor on which physicians may easily act is the LVP\( _{TM} \). It is defined as the intraventricular pressure minus the \( P_{pl} \) evaluated by \( P_{es} \) in the absence of pericardial effusion. Under continuous positive pressure ventilation (CPPV) or intermittent positive pressure ventilation, the increase in \( P_{es} \) is responsible for a fall in LVP\( _{TM} \) and then in LV afterload. However, just looking at the pulmonary capillary wedge pressure (PCWP) induces a dramatic overestimation of LV end-diastolic pressure when PEEP is above 10 cmH\( _2 \)O, reflecting the West zone 1 or 2 condition induced by PEEP (44). In the absence of an esophageal balloon, an elegant method has been proposed to measure transmural PCWP (45). The positive effect of \( P_{pl} \) on LV function has been nicely illustrated by McGregor (46). In a canine model of acute LV failure, Pinsky et al. also demonstrated that abdominal and chest wall binding during positive-pressure ventilation was responsible for a significant increase in \( P_{es} \), which translated into a significant increase in cardiac output secondary to the decrease in LV afterload (47). Finally, it has been experimentally reported that intermittent increased \( P_{pl} \) (10 to 15 mmHg) is a strong determinant of the dUp component (increased LV stroke volume and systolic blood pressure compared with baseline) of blood pressure variations, by inducing a decrease in LV afterload, especially in acute ventricular failure (48).

Figure 4 Effects of tidal inflation on right atrial intravascular and transmural pressures. This figure represents the measurements of esophageal pressure (E) and right atrial intravascular pressure (RA) in two different patients under positive pressure ventilation. The transmural pressure of the right atrium defined as esophageal pressure minus RA pressure is materialized by the red arrows. In the first patient (panel A), right atrial transmural pressure decreases during tidal inflation, which illustrates the decrease in venous return due to increased intrathoracic pressure. This effect of tidal inflation is responsible for a fall in right ventricle ejection, leading to a decrease in pulse pressure, named delta-down, because of a preload effect of mechanical ventilation. Conversely, in the second patient (panel B), right atrial transmural pressure increases during tidal inflation, illustrating the systemic congestion secondary to the obstacle to RV ejection. This effect is also responsible for pulse pressure variation (delta-down), but is due to an afterload effect of mechanical ventilation. a, end-expiration; b, end-inspiration. E, esophageal pressure; RA, right atrial intravascular pressure.
Pulsus paradoxus

Pronounced respiratory variation in pulse amplitude was first described in 1850 (49) and named nearly 20 years later pulsus paradoxus (50). In 1924, Gauchat and Katz specified that such a rhythmic pulse on palpation of accessible arteries in natural breathing could be encountered in two kinds of patients, those with enhanced intrathoracic pressure variations and those with cardiac tamponade (51). Measurement of $P_{es}$ could be of a great interest in patients with deep intrathoracic pressure variations.

In acute asthma, pulsus paradoxus reflects deep $P_{pl}$ variations and was first recognized as a marker of severity (52,53). Actually, measuring $P_{es}$ is the only way to really understand heart-lung interactions in this situation. $P_{es}$ varies from a markedly negative level during inspiration (around −25 mmHg) to a positive level during expiration (around 8 mmHg) (54). If $P_{es}$ is not measured, physicians could consider that RV preload falls during inspiration, as RAP hugely decreases, and becomes negative; in fact, $P_{TM}$ increases from 4 to 21 mmHg (54), leading to a completely different interpretation. In this study by Jardin et al., the same observation was made for PCWP (54). These data were confirmed by an echocardiographic study which reported enlargement of the RV at inspiration (55).

In the case of acute airway obstruction, vigorous inspiratory efforts against a totally obstructed upper airway, mimicking a Mueller maneuver, are associated with $P_{pl}$ and higher transvascular pressure of small interstitial vessels (56,57). Both pulmonary capillary lesion and leakage associated with such conditions are responsible for acute negative-pressure pulmonary edema (58). Very negative $P_{pl}$ also leads to an increase in systemic venous return to the right heart (59) and then to an overflow into the pulmonary circulation and to an increase in LV afterload as explained above. Managing $P_{es}$, if possible, could help to determine the mechanism of such pulmonary edema.

Conclusions

Heart-lung interactions are the effects on the circulatory system of changes in pleural and transpulmonary pressures. In many common clinical situations, such as PEEP application, positive pressure ventilation, acute
asthma and cardiac tamponade, the only way to evaluate heart-lung interactions invasively and to determine the mechanism of shock or respiratory failure accurately is to measure $P_{es}$. This allows calculation of transmural vascular pressures, which are quite different in these situations from intravascular pressures, and of $P_{tp}$, which is a key factor in hemodynamic alterations in ARDS. However, many technical limitations remain when recommending routine $P_{es}$ measurement at the bedside, even though development of new devices should help physicians to consider it more in the future for evaluation of heart-lung interactions and hemodynamic monitoring. Another approach is to visualize the size of cardiac chambers directly by echocardiography.

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**Footnote**

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