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 (P_{es}) 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 P_{es}.

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

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 (P_{es}) has been shown to be a good surrogate of pleural pressure, since its variations are similar despite small differences in absolute values (2-4). Thus, P_{es} has been considered as a major parameter in the evaluation of heart-lung interactions, but is scarcely used in daily practice for numerous reasons, including technical issues and difficulties in interpretation.

This article aims to show why and how P_{es} should be measured in interpreting heart-lung interactions. After physiological reminders about the concept of transmural pressure, we will describe the technical aspects and issues of P_{es} measurement and its interest in two different types of clinical situations: positive pressure breathing-related heart-lung interactions on the one hand, and negative pressure breathing-related heart-lung interactions on the other.

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 (P_{tp}). 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 P_{es}, except when the pericardial pressure is significantly increased (*Figure 1*).
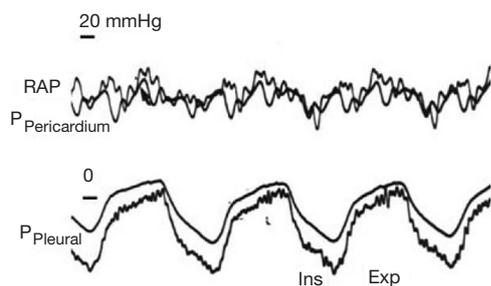
Figure 1 Right atrial transmural pressure during cardiac tamponade. During cardiac tamponade, the pressure surrounding the right atrium is no more the pleural pressure, but the pericardial pressure. While the pleural pressure, i.e., P_{es}, remains low, the pericardial pressure increases and the RAP_{tm}, i.e., RAP minus pericardial pressure, falls, meaning that the right atrial volume is very low. Exp, expiration; Ins, inspiration; P_{es}, esophageal pressure; RAP, right atrial pressure; RAP, right atrial transmural pressure.

In the lungs

P_{tp} can be defined as the pressure that counteracts all the inward-acting tissue forces distributed at the pleural surface. P_{tp} can be calculated by subtracting pleural pressure (P_{pl}) from alveolar pressure (P_{aw}) (5). It reflects the mechanical properties of the lungs and may be relevant in the management of intensive care unit (ICU) patients with increased elastance of the respiratory system. In particular, P_{tp} could help to overcome the great heterogeneity of ICU patients suffering from acute respiratory distress syndrome (ARDS). Indeed, the transmission of P_{aw} to the thoracic cavity varies as a function of the relative contribution of chest wall elastance to the total elastance of the respiratory system (E_{rs}) (6). P_{pl} can thus be estimated using the following formula: $\Delta P_{pl} = \Delta P_{aw} \times (E_L/E_{rs})$, where E_L defines lung elastance (7). In normal conditions, chest wall and lung elastances are similar and P_{pl} and P_{tp} contribute equally to P_{aw}. Conversely, the more E_L is increased, the less P_{pl} contributes to P_{aw}. In ARDS patients, Jardin *et al.* demonstrated that P_{pl} contributed 37% and 24% of P_{aw} in patients with respiratory compliance greater than 45 mL/cmH$_2$O (and a normal lung elastance) or less than 30 mL/cmH$_2$O (and a significantly increased lung elastance), respectively (8). For the same plateau pressure, P_{tp} may differ greatly according to E_L and E_{CW}: some patients will then be exposed to the consequences of a large increase in P_{pl}, while others will be exposed to the consequences of an increase in P_{tp} (see part III) (9).
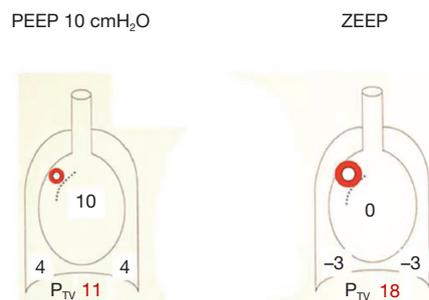
In the cardiac chambers and the great vessels

Right atrial transmural pressure (RAP_{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). RAP_{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 RAP_{TM} cannot be superimposed, as frequently proposed.

At the interstitial level, the impact of P_{pl} 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_{pl} 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).

Figure 2 Effect of PEEP on transvascular pressure [adapted from [19]]. The left panel represents the example of a mechanically ventilated ARDS patient with 10 cmH$_2$O PEEP. Assuming that the transmission of PEEP to the pleural/interstitial pressure is about one-third, the pleural pressure measured by P$_{es}$ is 4 cmH$_2$O and the transvascular pressure 11 cmH$_2$O for an intravascular pressure of 15 mmHg. In contrast, ventilated in ZEEP conditions, this patient would have a P$_{es}$ of –3 cmH$_2$O and then an increase in the transvascular pressure to 18 cmH$_2$O for the same intravascular pressure, exposing him to the risk of pulmonary edema by fluid leakage. PEEP, positive end-expiratory pressure; P$_{es}$, esophageal pressure; P$_{tv}$, transvascular pressure.

How to measure P$_{es}$ 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 P$_{pl}$ (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 P$_{es}$ is more altered by cardiac artifacts (30) and also tends to overestimate the value of P$_{es}$ (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 P$_{es}$ 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 P$_{aw}$ and the P$_{es}$ (28).

What are the limits?

Due to the esophagus wall elastance, the filling volume can be responsible for an overestimation of P$_{es}$, even if the balloon is partially inflated (31). In contrast, it has been suggested that P$_{es}$ could be underestimated if the balloon is unfilled (25,27,32). This might suggest that only respiratory-related changes of P$_{es}$ (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 P$_{es}$ (i.e., maximizing ΔP$_{es}$) (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 P$_{esCAL}$ = P$_{es}$ – P$_{ew}$ (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 P$_{es}$ (34).

Each commercially available balloon catheter system has its own properties (material, compliance, recommended filling volume…), which exposes P$_{es}$ 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 P$_{es}$ 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 P$_{es}$ measurement is rarely performed.
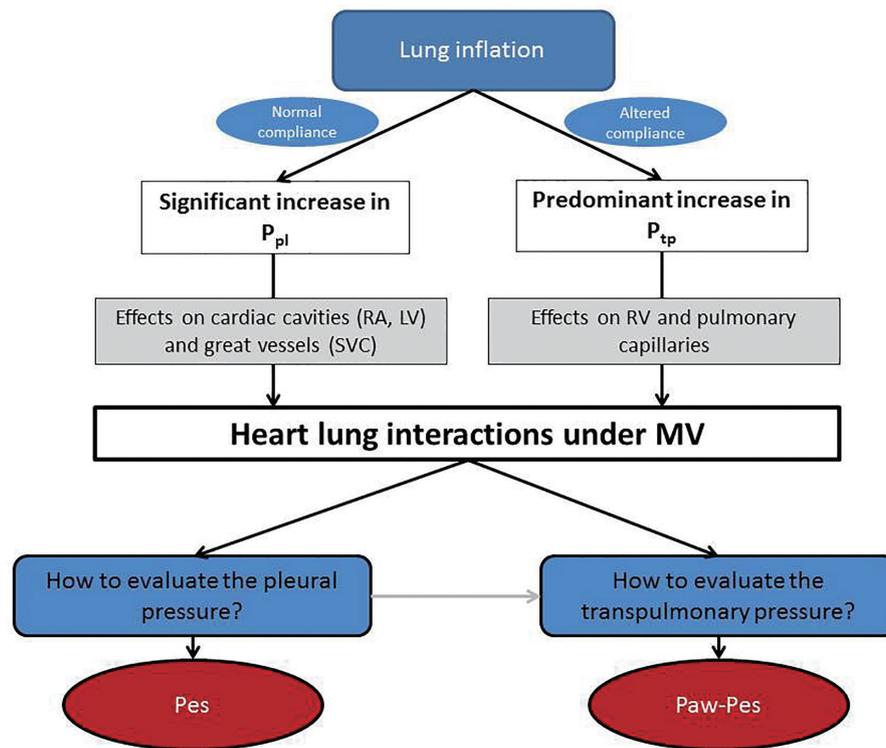
Figure 3 Mechanisms of the involvement of P_{es} in heart-lung interactions under positive pressure mechanical ventilation. Under mechanical ventilation, the mechanisms of heart-lung interactions can differ greatly in the case of preserved or altered lung compliance. Heart-lung interactions rely on the increase in P_{pl} and on P_{TP} in patients with preserved or altered compliance, respectively. P_{aw}, airway pressure; LV, left ventricle; MV, mechanical ventilation; P_{es}, esophageal pressure; P_{pl}, pleural pressure; P_{TP}, transpulmonary pressure; RA, right atrium; RV, right ventricle; SVC, superior vena cava.

In the last part of this review, we will describe how the use of P_{es} 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_{es} 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_{es} 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_{pl} (preload effect) from those for whom the main effect of lung inflation will be related to increased P_{tp} (afterload effect). Indeed, management will be dramatically different. In both mechanisms, intravascular RAP is increased due to the transmission of P_{pl} to the right atrium. But in the case of a decrease in systemic venous return driven by P_{pl}, RAP_{TM} is decreased (*Figure 4*) (37,38), while it is increased in the case of increased RV afterload driven by P_{tp} (*Figure 4*). Increased resistance to venous return is a key factor in the decrease in systemic venous pressure driven by P_{pl} (39). It was suggested to occur at least in the superior vena cava (SVC), using transesophageal

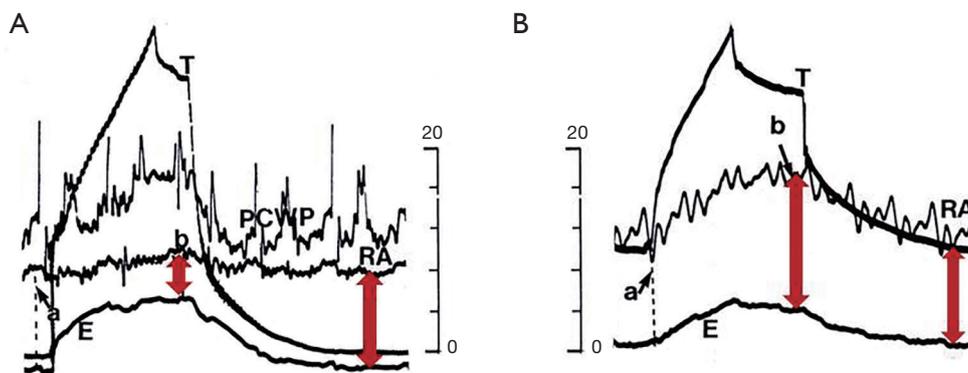　　　atm.amegroups.com　　　*Ann Transl Med* 2018;6(18):351

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.

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 increased RV afterload driven by P_{tp}, measuring P_{es} is mandatory when physicians want to invasively determine the effect of a PEEP trial on RV function in ARDS (*Figure 5*) (43).

The use of P_{es} to understand the effect of positive pressure ventilation on the LV is also very important, in particular in patients with severe dilated cardiomyopathy. Indeed, LV afterload may be approached by the LV constraint (σ) expressed as $σ = LVP_{tm} × R/t$, where LVP_{tm} represents the LV transmural pressure, R the LV radius and t its thickness. In the case of dilated cardiomyopathy,

R increases and t decreases, leading to a huge rise in σ. 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_2O, 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 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).
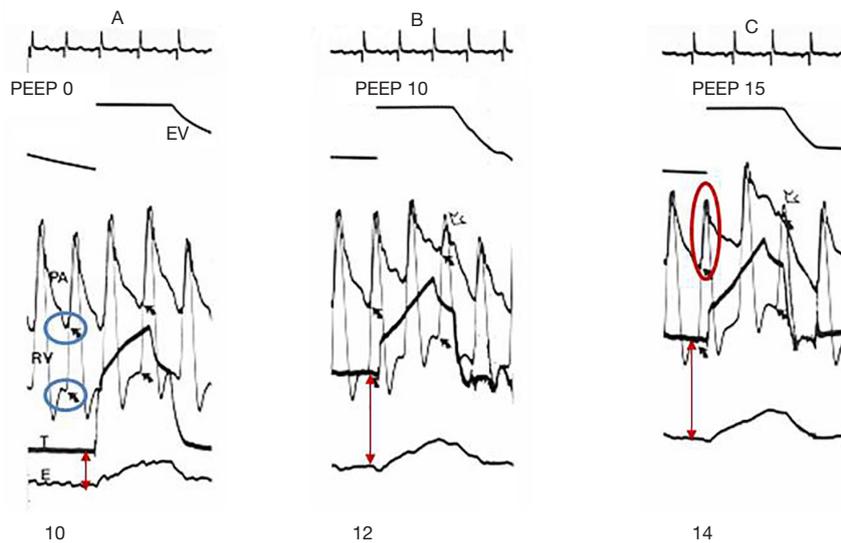
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Figure 5 Evaluation of a PEEP trial effect in ARDS using P_{es}. The left panel (A) represents the PA, RV and esophageal pressures recorded in ZEEP conditions. P_{TP} (red arrow) is low and the RV isovolumetric pressure (between the 2 black arrows), which reflects RV afterload, is 10 cmH$_2$O. In 10 (B) and 15 (C) cmH$_2$O PEEP, P_{TP} increases significantly at end-expiration and is responsible for an increase in RV afterload, attested by an increase in RV isovolumetric pressure to 12 and 14 cmH$_2$O, respectively. Note that the PA pulse pressure (red circle), which reflects the RV stroke volume, decreases with the increase in RV afterload. Blue circles represent closing of the tricuspid valve and opening of the pulmonary valve with the beginning of RV ejection. PEEP, positive end-expiratory pressure; ARDS, acute respiratory distress syndrome; PA, pulmonary artery; P_{TP}, transpulmonary pressure; RV, right ventricle.

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, RAP_{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_{tp} 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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