Esophageal Pressure Measurements in Patients With Acute Respiratory Distress Syndrome

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Esophageal balloons are used in the respiratory monitoring of critical care patients. After the esophageal pressure is measured, the corresponding pleural pressure in the thorax can be projected, enabling lung-thorax compliance to be partitioned into chest-wall compliance and lung compliance. The esophageal balloon allows determination of transpulmonary pressures and a correspondingly individually tailored approach to respiratory care, such as patient-specific titration of positive end-expiratory pressure for patients with extrapulmonary acute respiratory distress syndrome. Esophageal balloon monitoring provides critical information for selecting ventilation strategies to use in patients with acute respiratory distress syndrome. (Critical Care Nurse. 2016;36[5]:27-36)

When esophageal balloons are placed in critically ill patients receiving mechanical ventilation, the balloons are typically placed by nurses. The balloons allow respiratory care staff and physicians to monitor otherwise inaccessible respiratory values. This article describes the use of esophageal balloons and provides an in-depth analysis of the associated respiratory mechanics to elucidate the use of these devices in critical care practice. This analysis includes the placement of these balloons, the “dynamic occlusion” technique that is commonly used to verify the correct positioning of the balloons, and the associated physiology that describes the critical advantage that such balloons provide to patients with extrapulmonary origins of acute respiratory distress syndrome (ARDS).

Rationale for Increased Use of Esophageal Balloons: Pleural Pressure Measurements

Appropriate and correct estimation of patients’ pleural pressures is a subtle and crucial component of patient care that, according to Laurent Brochard,1 “is underused in everyday practice.” Measurement of esophageal pressure (P_{Es}) is used to estimate transpulmonary pressure (P_{TP});2-4 which is of utmost importance,
as it permits calculation of the distending pressures of the lung, chest wall, and respiratory system. Measurements of esophageal pressure have also been used to calculate work of breathing and the pressure developed by the inspiratory muscles, as well as to guide weaning from mechanical ventilation. A detailed analysis of patient-specific respiratory mechanics is required to avoid mechanical stress, such as overdistention produced by inappropriate ventilating volumes (volutrauma) or positive end-expiratory pressure (PEEP) that is insufficient to optimize alveolar recruitment and avoid cyclic opening and closing of alveoli (atelectrauma). The esophageal balloon allows such analysis to take place in real time.

The clinical use of esophageal pressure requires a fundamental understanding of the clinical implications of intrapulmonary and extrapulmonary impairments. Measurement of esophageal pressure may be used to determine when it is appropriate to exceed the recommended plateau pressures (Pplat) of 30 cm H2O as dictated by the ARDS clinical network (ARDSnet). The ARDSnet was established in 1994 in order to determine safe ventilating volumes and pressures for patients with ARDS; the resulting standards have since been applied to patients receiving mechanical ventilation in general. The ARDSnet provides an excellent set of patient-independent guidelines. An improved approach that uses appropriate adjustments for a patient’s pathophysiology, including use of in situ estimates of pleural pressures via the esophageal balloon, has the potential to lead to improved outcomes in specific subsets of patients with ARDS.

**Physiological Background**

The respiratory system can be partitioned into various components: the airways, alveoli, and chest wall.

These 3-dimensional structures are conjoined by the pleural space. The pressure at the airway opening (P AO) is called the airway pressure. In unintubated persons during quiet breathing, P AO is the mouth pressure and is equal to atmospheric pressure unless pressure is applied to the nose and mouth. The pleural pressure is the pressure in the pleural space; in a healthy upright human, the pleural pressure (Ppl) remains negative throughout the entire resting respiratory cycle (Figure 1). The continuously negative pleural pressure can be attributed to the elastic tendency of the lung to recoil to a smaller volume versus the tendency of the chest wall to expand. These 2 opposing forces generate a negative pressure in the pleural space at rest, thus preventing the lungs from collapsing. The pressure difference across the lung is termed the transpulmonary pressure (PTP); transpulmonary pressure is the pressure required to inflate the lung, also known as the distending pressure: \( P_{TP} = P_{AO} - P_{pl} \) and thus is equal to the airway opening pressure (P AO) minus the pleural pressure (Ppl). During spontaneous inspiration, pleural pressure becomes more negative, resulting in an increase in transpulmonary pressure, increased lung volume, and ventilation. At the end of resting expiration (when all airflow has ceased), the remaining gas volume in the lungs is known as functional residual capacity, and the pressures at the airway opening (P AO) and alveoli (P A) have equilibrated (are equal). This equilibration is the reason why there is no airflow at the resting expiratory level.

Altering the pressure applied by a mechanical ventilator to the airway opening during inspiration results in changes to the transpulmonary pressure, lung volume, and ventilation. Measurements of pleural and airway opening pressure allow clinicians to calculate transpulmonary pressure, as well as compliance of the lung and of the chest wall. Without measurement or estimates of pleural pressure, the compliance of the entire respiratory system as a whole can be calculated. Compliance is defined as the change in volume divided by the change in pressure and is a measure of the distensibility of the lung-thorax system. This compliance value is influenced by the compliances of the lung and of the chest wall, which includes the abdomen.

During an end-inspiratory pause, when airflow is zero, the alveolar pressure is equal to the pressure at the airway opening, as pressures equilibrate when flow is zero. The end-inspiratory plateau pressure (Ppl) is a
measurement of the alveolar distending pressure, the pressure applied to the airways and alveoli in communication with the ventilator. The PEEP is the baseline pressure in the lungs and is critical in preventing alveolar collapse upon exhalation; PEEP also helps restore functional residual capacity to normal when compliance is low. Applying PEEP to the lungs will increase the plateau pressure during an end-inspiratory pause. Esophageal balloons are used to estimate the pleural pressure, separate it from the lung-thorax plateau pressure, and recover the transpulmonary plateau pressure to ensure safe ventilation. Esophageal balloons permit the estimation of the true transpulmonary plateau pressure of the lungs to evaluate ventilating volumes and pressures, which may be higher than the levels recommended in the ARDSnet guidelines, and to select PEEP levels for effective recruitment of functional residual capacity and improve oxygenation (which go hand in hand) on a patient by patient basis. Table 1 summarizes the important calculations of respiratory mechanics that will be used throughout, including those values only recoverable from esophageal balloon measurements.

Figure 1 Measuring intrapleural pressure with an esophageal balloon. Intrapleural pressure is transmitted through the esophagus to the balloon-tipped catheter.

Table 1 Important calculations related to interpretation of data from esophageal balloons

<table>
<thead>
<tr>
<th>Name</th>
<th>Equation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tidal volume</td>
<td>$\Delta V = V_T$</td>
</tr>
<tr>
<td>Transpulmonary plateau</td>
<td>$\Delta P_{TP} = \Delta P_{AO} - \Delta P_{pl}$</td>
</tr>
<tr>
<td>pressure</td>
<td></td>
</tr>
<tr>
<td>Respiratory system</td>
<td>$C_{RS} = \frac{\Delta V}{\Delta P_{AO}}$</td>
</tr>
<tr>
<td>compliance</td>
<td></td>
</tr>
<tr>
<td>Compliance partitioning</td>
<td>$\frac{1}{C_{RS}} = \frac{1}{C_L} + \frac{1}{C_W}$</td>
</tr>
<tr>
<td>Lung compliance</td>
<td>$C_L = \frac{\Delta V}{\Delta P_{ AO} - \Delta P_{ pl}}$</td>
</tr>
<tr>
<td>Chest wall compliance</td>
<td>$C_W = \frac{\Delta V}{\Delta P_{ pl}}$</td>
</tr>
<tr>
<td>Esophageal pressure</td>
<td>$\Delta P_{ES} = \Delta P_{pl}$</td>
</tr>
<tr>
<td>Transpulmonary plateau</td>
<td>$\Delta P_{TP} = \Delta P_{AO} - \Delta P_{ES}$</td>
</tr>
<tr>
<td>pressure</td>
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The following nomenclature is used: $\Delta X = X_{\text{exp}} - X_{\text{ins}}$. Here, $X_{\text{exp}}$ is the value of $X$ at the end of normal inspiration; $X_{\text{ins}}$ is the value of $X$ at the end of expiration. Using this nomenclature, $\Delta V = V_{\text{ins}} - V_{\text{exp}} = V_T$, where $V_T$ is the tidal volume.
Of note, respiratory system compliance alone does not enable poor lung compliance to be distinguished from poor chest wall compliance; the inability to partition lung and chest wall compliance may lead to inaccuracies when assessing the severity of a patient’s pulmonary impairment and ventilator management strategies. Determination of the chest wall compliance allows the lung compliance to be projected from the net compliance of the respiratory system without interference; edema in the chest wall, abdominal distention, paralytic agents, or simple changes in the patient’s position will not affect the calculation of the lung compliance as a result.\textsuperscript{13} The figure of merit is $C_L$, calculated as $\Delta V / (\Delta P_{A0} - \Delta P_{pl})$. As described in the next section, it is clinically reasonable to substitute the esophageal pressure for the pleural pressure directly, thus recovering the transpulmonary pressure as $\Delta P_{TP} = \Delta P_{A0} - \Delta P_{pl} = \Delta P_{A0} - \Delta P_{Es}$. One may monitor the compliance of the lung to optimize patients’ outcomes and reduce ventilator-associated injury risks directly.\textsuperscript{5-7,9,12}

**Pleural Pressure Measurement Techniques**

Pleural pressure can be directly measured through invasive methods but is accompanied by a high risk of pneumothorax.\textsuperscript{8} These methods typically involve puncturing the chest wall and invading the pleural cavity in several locations and should be avoided.\textsuperscript{14} Alternatively, the pressure in the lower third of the esophagus closely parallels the pressure in the adjoining pleura, and that pressure can be measured by using esophageal balloon technology.\textsuperscript{2,4,13} In fact, esophageal pressure may “represent a better measure of the over-all elastic behavior of the lung than any local pleural pressure.”\textsuperscript{14} This measurement is accurate when taken in an upright human lung without the weight of the mediastinum compressing the esophagus and generating large regional variances in pleural pressure.\textsuperscript{8}

The relationship between esophageal pressure and pleural pressure, and its measurement with an esophageal balloon, was first outlined in 1949 in a thesis by Buytendijk,\textsuperscript{2} and further measurements in the following years confirmed the effectiveness of the technique in the early 1950s.\textsuperscript{3,4} Starting in the late 1950s, results of several studies\textsuperscript{15,16} have strongly supported the use of esophageal pressure as a surrogate for pleural pressure. More recent publications have supported the notion that transpulmonary pressure at the end of inspiration (PTPplat), as a direct measure of lung stress, “might be a better indicator of injury than plateau pressure alone.”\textsuperscript{7} One may infer the transpulmonary pressure from the open airway pressure at the end of inspiration and the pleural pressure, in which esophageal pressure is used in lieu of pleural pressure. Additionally, as most measurements are performed using differences between various pressures and volumes, the reported offset between esophageal pressure and pleural pressure does not affect the calculation of most relevant respiratory factors.\textsuperscript{15} Calculation of the transpulmonary plateau pressure ($P_{TP\,Plat}$) is of utmost importance (Table 1).

**Esophageal Balloon Placement Techniques and the Dynamic Occlusion Method**

Evaluation of correct placement of the esophageal balloon uses the process described by Baydur et al,\textsuperscript{17} the “dynamic occlusion technique.” The dynamic occlusion technique ensures that the changes in esophageal pressure are sufficiently similar to changes in pleural pressure to allow clinically significant calculations of lung compliance, which is assumed in the preceding equations.\textsuperscript{15} Appropriate balloon placement is described in the following paragraphs.

Most esophageal balloons are now available as a single system, typically comprising a thin polyethylene catheter with several holes, possibly in a spiral pattern, on the distal end. This catheter is placed inside the “balloon,” which serves 2 purposes: first, prevention of occlusion of the catheter holes, and second, maintenance of a thin column of air to measure pressure in the surrounding area. Ventilators, such as the Carefusion Avea (BD Worldwide) and Hamilton Galileo (Hamilton Medical), provide integrated esophageal pressure measurements. It is also possible to use available pressure transducers to make these measurements.

For a nasal balloon, the deflated balloon (and catheter) is typically inserted through the nares into the posterior part of the pharynx; if the patient is conscious, he or she is instructed to swallow. The balloon is then passed through the esophagus and into the stomach and is inflated with 2.0 mL of air. Then 1.5 mL of air is withdrawn, to leave...
0.5 mL of air in the balloon. The balloon is then attached to the ventilator or other transduction device.\textsuperscript{13}

If the balloon is properly placed in the stomach, a positive pressure deflection will be observed during inspiration. The balloon is then slowly withdrawn into the esophagus, where inspiration will induce a negative pressure deflection. From the point of initial negative deflection, the balloon is withdrawn another full length of the balloon, which is typically 10 cm, to ensure that the entirety of the balloon is within the esophagus. Cardiac oscillations should then appear on the esophageal pressure ($P_{ES}$) waveform. The tip of the catheter is typically within 40 cm (SD, 5 cm) from the nares.

After insertion of the balloon, the dynamic occlusion test\textsuperscript{17} is performed in patients who are spontaneously breathing: at the end of expiration, the airway is occluded, and the ratio of the esophageal pressure ($P_{ES}$) to the airway opening pressure ($P_{AO}$) is measured during several (3-5) inspiratory attempts against the closed airway. The 2 pressure differences should be equal ($AP_{P_{pl}} = AP_{P_{ES}}$); by measuring this ratio, the validity of the measurement can be determined and appropriate balloon placement can be confirmed (Figure 2).

The dynamic occlusion test is used to ensure the linearity of the relationship between pleural pressure and esophageal pressure. Thus one can treat the changes in these pressures as clinically equivalent, as outlined in the physiological background section. Occluding the airway prevents airflow, and the lung-thorax system will equilibrate. If the patient is not spontaneously breathing, an alternative method as described by Lanteri et al\textsuperscript{18} can be used, in which correct placement of the esophageal balloon can be verified via observation of cardiac oscillations of the pressure waveform.

**Treatments That Use Esophageal Balloon Measurement**

The use of esophageal balloon measurements to manage several pulmonary disease states and guide the determination of the corresponding ventilator parameters is a suitable demonstration of the effectiveness of partitioning the lung-thorax compliance. Obesity, ascites, ARDS, and other disease states can lead to increased pleural pressure and require a corresponding increase in PEEP.\textsuperscript{19} It is possible that esophageal pressure can be elevated independently of intrapulmonary and extrapulmonary ARDS; for example, obesity and ascites can cause increased esophageal pressure. Under these conditions, the pleural pressure, and correspondingly the esophageal pressure, is increased because of the increased body mass index in obesity or the increased intra-abdominal pressure in ascites.\textsuperscript{20,21} Esophageal balloon monitoring of pleural pressure analogues has many uses besides ARDS treatment, although the latter is the main focus of this article.

One of the primary clinical uses of esophageal balloons is in patients with ARDS.Gattinoni et al\textsuperscript{22} studied patients using both computed tomography and esophageal balloon measurements to identify differences in respiratory system mechanics due to different causes of ARDS. They reported that ARDS patients could be partitioned into 2 groups: 1 with direct pulmonary causes and 1 with extrapulmonary causes.\textsuperscript{22} Esophageal pressure measurement to estimate pleural pressure may be of greatest benefit in monitoring patients with extrapulmonary ARDS.

Discrimination between causes of ARDS is a critical measure; the net respiratory system compliance can be influenced by the compliance of the lung (a “pulmonary” cause) or by an “extrapulmonary” cause; either type will reduce overall respiratory system compliance. Patients with a direct pulmonary source of ARDS, such as aspiration or pneumonia, had low lung compliance but relatively normal chest wall compliance. Gattinoni et al\textsuperscript{22} attributed these findings to alveolar filling and consolidation, which is not very responsive to increases in PEEP. The low lung compliance in these patients suggests that an increase in

![Figure 2](image-url)
PEEP will not increase alveolar recruitment. Extrapulmonary ARDS often manifests as atelectasis, associated with a stiff chest wall or an intra-abdominal process; therefore, the potential for alveolar recruitment is greater than in a primary pulmonary abnormality (Figure 3).

Patients with an extrapulmonary source of ARDS (eg, sepsis, pancreatitis, peritonitis) have relatively normal lung compliance, albeit with a stiff chest wall/abdomen and increased pleural pressure. The underlying lung in these patients is affected primarily by compressive atelectasis rather than alveolar consolidation and has a significant potential for alveolar recruitment with PEEP. In addition, patients with ascites, pleural effusion, and possibly obesity also have elevated airway pressures and a greater tendency to externally compress the lung, causing hypoxemia. This category of patients with elevated airway pressure and hypoxemia has the greatest potential to benefit from determination of transpulmonary pressure for adjustment of tidal volume and PEEP. An increase in PEEP above ARDSnet guidelines can still be safe for the patient and effective to counter the compressive pressure caused by these types of abnormalities.

As an extension of the ARDSnet recommendation to keep the airway opening plateau pressure at less than 30 cm H₂O, it is common to maintain the transpulmonary plateau pressure at less than 25 cm H₂O. Transpulmonary plateau pressures approaching 30 cm H₂O result in overdistension of remaining normal alveoli, which are still found to exist in nondependent portions of the lung in patients with ARDS. Overdistension can damage the alveolar wall as well as their adjacent capillaries; this situation has been called “volutrauma” contributing to ventilator-induced lung injury (VILI). However, in patients with extrapulmonary sources of ARDS (ascites, pleural effusion, and possibly obesity), it is quite likely that airway opening plateau pressures greater than 30 cm H₂O do not induce VILI because these conditions make pleural pressure positive rather than negative. One may better evaluate the cause of increased airway pressure by measuring the transpulmonary plateau pressure.

Measurement of airway opening pressure and esophageal pressure during an expiratory pause allow the determination of the end-expiratory transpulmonary pressure: 

\[ P_{\text{TP PEEP}} = P_{\text{ALVEOLUS}} - P_{\text{ES}} \]

This end-expiratory transpulmonary pressure is also commonly called \( P_{\text{TP PEEP}} \). If pleural (esophageal) pressure is higher than the pressure inside the alveoli, the alveoli will collapse. A negative \( P_{\text{TP PEEP}} \) indicates that pleural pressure is greater than alveolar pressure and that PEEP is insufficient to prevent alveolar collapse. Increasing the PEEP to be at least equal to the esophageal pressure during expiration has the potential to prevent collapse and recruit alveoli. It is common to maintain a PEEP level that generates a \( P_{\text{TP PEEP}} \) of 0 to 2 cm H₂O to optimize alveolar recruitment. Part of the mechanism of VILI is the repeated cyclic closing and reopening of alveoli; using a PEEP level that is adequate to prevent this form of shear injury may also decrease VILI.

One significant limitation of esophageal balloons and a source of inaccuracy is the influence of the weight of the heart and mediastinum on esophageal pressures. Supine positioning places the heart superior to the balloon and falsely elevates the measured pressure. Talmor et al subtracted 5 cm H₂O from esophageal pressure values to compensate for this effect, agreement has not yet been reached on an optimal method of correcting \( P_{\text{ES}} \). Reverse Trendelenburg positioning reduces the magnitude of this effect and is generally recommended for measurement.
A 5-foot 10-inch (1 m 78), 380-pound (171 kg) man was transferred to a regional medical center from an outlying hospital with ARDS associated with pancreatitis and sepsis. He was stabilized on ARDSnet ventilator settings with a tidal volume of 5 mL/kg ideal body weight, 90% oxygen, and PEEP of 14 cm H₂O. Despite these settings, his oxygenation remained marginal and his end-inspiratory plateau pressure exceeded 30 cm H₂O (range, 32-35 cm H₂O). The patient was frequently asynchronous with the ventilator, which precluded further reductions in tidal volume. The patient’s abdomen was very firm on palpation; the decision was made to institute esophageal pressure monitoring. After sedating the patient further, the nurse and respiratory therapist achieved satisfactory placement of the esophageal balloon. The esophageal and airway opening pressures were measured; Figure 4 shows esophageal and airway opening pressures during an end-expiratory pause, as well as the effect of both those pressures on transpulmonary pressure. The same measurements (esophageal pressure and airway opening pressure) were made during an end-inspiratory pause (Figure 5). Table 2 outlines the mathematical calculation of transpulmonary pressure from those measurements.

These values were interpreted as follows: the $P_{TP} - PEEP = -4$ cm H₂O (without adjusting the $P_{ES}$ for the weight of the mediastinum) indicates that the set PEEP of 14 cm H₂O is less than the esophageal pressure of 18 cm H₂O, resulting in alveolar collapse at end-exhalation as well as impaired oxygenation. The set PEEP was increased to 18 cm H₂O to improve oxygenation; this allowed the inspired oxygen concentration to be reduced to 50%. The improvement in lung recruitment also resulted in an increase in lung compliance. Measurements were repeated after the PEEP was increased, with results shown in Table 3.

Although the increase in PEEP increased both the alveolar and esophageal end-inspiratory plateau pressures, the $P_{TP\ Plat} = 11$ cm H₂O (less than the guideline value of 25 cm H₂O). The medical team thought that allowing an increase in tidal volume to 8 mL/kg ideal body weight in order to eliminate patient-ventilator
Measurement of esophageal pressure with esophageal balloons allows projection of pleural pressure followed by partitioning of the compliance of the lung-thorax system into its individual components. The pulmonary and extrapulmonary causes of ARDS result in elevated airway pressure, alveolar collapse, and hypoxemia, although the mechanisms of the diseases differ. Use of esophageal pressure measurements helps to determine adequate PEEP levels to recruit alveoli and to evaluate for true overdistention of the lungs. When esophageal pressure measurements are used, it is reasonable to exceed the PEEP and end-inspiratory airway opening plateau pressure recommended in the ARDSnet guidelines. “The time is now right to apply the knowledge obtained with esophageal pressure to improve the management of critically ill and ventilator-dependent patients.”

Financial Disclosures
None reported.

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References
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- Measurements of esophageal pressure have been used to calculate work of breathing and the pressure developed by the inspiratory muscles, as well as to guide weaning from mechanical ventilation.
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- The PEEP is the baseline pressure in the lungs and is critical in preventing alveolar collapse upon exhalation; PEEP also helps restore functional residual capacity to normal when compliance is low.
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