The oesophageal balloon for respiratory monitoring in ventilated patients: updated clinical review and practical aspects

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Abstract
There is a well-recognised importance for personalising mechanical ventilation settings to protect the lungs and the diaphragm for each individual patient. Measurement of oesophageal pressure ($P_{\text{oes}}$) as an estimate of pleural pressure allows assessment of partitioned respiratory mechanics and quantification of lung stress, which helps our understanding of the patient’s respiratory physiology and could guide individualisation of ventilator settings. Oesophageal manometry also allows breathing effort quantification, which could contribute to improving settings during assisted ventilation and mechanical ventilation weaning. In parallel with technological improvements, $P_{\text{oes}}$ monitoring is now available for daily clinical practice. This review provides a fundamental understanding of the relevant physiological concepts that can be assessed using $P_{\text{oes}}$ measurements, both during spontaneous breathing and mechanical ventilation. We also present a practical approach for implementing oesophageal manometry at the bedside. While more clinical data are awaited to confirm the benefits of $P_{\text{oes}}$-guided mechanical ventilation and to determine optimal targets under different conditions, we discuss potential practical approaches, including positive end-expiratory pressure setting in controlled ventilation and assessment of inspiratory effort during assisted modes.

Introduction
Lung-protective ventilation is associated with better outcome in patients with acute respiratory distress syndrome (ARDS) [1, 2] and the recognised standard of care [3–5]. Plateau pressure ($P_{\text{plat}}$), driving pressure ($\Delta P$) and respiratory system compliance are commonly measured bedside but do not take into account the respective contributions of the lungs and chest wall mechanics nor guarantee delivering optimal lung- and diaphragm-protective ventilation to every individual patient [6]. This is particularly relevant with significant lung inhomogeneity, when chest wall compliance is altered [7], when switching from controlled to assisted ventilation [8–11] or during difficult weaning. More advanced monitoring facilitates delivery of personalised mechanical ventilation. Pleural pressure ($P_{\text{pl}}$) estimated by oesophageal manometry enables measuring lung and chest wall distending pressures. This allows assessing the lungs...
What are the key (patho-)physiological concepts to understand when implementing oesophageal manometry?

The respiratory system consists of different structures and compartments: the airways, lung parenchyma with alveoli, pleural space, chest wall and respiratory muscles. Understanding their interaction is key to discern what drives movement of air into and outside the lungs and to understand the pressures that may aggravate lung injury. The force driving air into the alveoli must overcome opposing forces: 1) resistive pressure \( P_{\text{res}} = \text{flow} \times \text{resistance} \) due to airway resistance to airflow and 2) elastic pressure \( P_{\text{el}} = \text{volume} \times \text{elastance} \) due to the intrinsic elastic properties of the lungs and chest wall (figure 1). Elastic recoil describes the natural trend of the respiratory system to come back to its state of equilibrium, which is at end-expiration.

The equation of motion describes at any time the relationship between total respiratory system pressure \( P_{\text{tot}} \) and the elastic and resistive pressures: \( P_{\text{tot}} = P_{\text{res}} + P_{\text{el}} + \text{initial pressure at end-expiration} \left( P_0 \right) \) or \( P_{\text{tot}} = \text{flow} \times \text{resistance} + \text{volume} \times \text{elastance} + P_0 \). The exact equation also contains a pressure to overcome tissue and gas inertia, which is negligible. Using oesophageal manometry, \( P_{\text{el}} \) can be partitioned into the transmural pressure of the lungs (transpulmonary pressure \( P_L \)) and that of the chest wall (pressure across the chest wall \( P_{\text{cw}} \)) that are acting in series: \( P_{\text{el}} = P_L + P_{\text{cw}} \). Thus, the equation of motion can be written as \( P_{\text{tot}} = \text{flow} \times \text{resistance} + \text{volume} \times \text{lung elastance} + \text{volume} \times \text{chest wall elastance} + P_0 \) (figure 1). \( P_0 \) is omitted in non-ventilated subjects, since all pressures are measured relative to atmospheric pressure, or is equal to the total positive end-expiratory pressure (PEEP) during mechanical ventilation. For more precise definitions, see LORING et al. [18].

Spontaneous breathing physiology in healthy conditions

At functional residual capacity (end-expiration), with the respiratory muscles relaxed and the mouth open, the respiratory system is at equilibrium. The lung and chest wall individual resting positions are different: lung elastic recoil pushes inwards, while chest wall elastic recoil pulls outwards. This results in a slightly negative end-expiratory \( P_{\text{pl}} \) in healthy subjects (figure 2a) [19].
a) Spontaneous breathing ($P_{tot}=P_{mus}$)

End-expiration 1

- $P_{ao}=0$
- $P_{atv}=0$
- $P_{pl}=-5$
- $P_{alv}=-5$
- $P_{mus}=0$

Mid-inspiration 2

- $P_{ao}=0$
- $P_{atv}=0$
- $P_{pl}=-6.5$
- $P_{alv}=-0.5$
- $P_{mus}=2$

End-inspiration 3

- $P_{ao}=0$
- $P_{atv}=0$
- $P_{pl}=-8$
- $P_{alv}=8$
- $P_{mus}=4$

b) Controlled mechanical ventilation ($P_{tot}=P_{ven}$) and calculations

End-expiratory hold, no airway closure 1

- $PEEP_{tot}=5$
- $P_{alv}=5$
- $P_{pl}=5$
- $P_{mus}=0$
- $P_{cw}=5$

End-inspiratory hold 2

- $P_{peak}=18$
- $P_{alv}=18$
- $P_{pl}=8$
- $P_{mus}=2$
- $P_{cw}=5$

End-inspiration 3

- $P_{peak}=15$
- $P_{alv}=15$
- $P_{pl}=5$
- $P_{mus}=8$
- $P_{cw}=5$

Direct method

$\Delta P_{L, end-exp}=P_{EEP_{tot}}-P_{oes, end-exp}$
$\Delta P_{L, end-insp}=P_{plat}-P_{oes, end-insp}$

Elastance-derived method

$P_{aw}=P_{L}+P_{pl}$ and $E_{rs}=E_{cw}$

Accordingly:

$P_{L}=P_{aw} \times E_{cw}$
$P_{L, end-insp}=P_{plat} \times E_{cw}$

c) Assisted mechanical ventilation ($P_{tot}=P_{vent}+P_{mus}$)

End-expiration 1

- $PEEP=10$
- $P_{alv}=10$
- $P_{pl}=10$
- $P_{mus}=0$
- $P_{cw}=5$
- $P_{L, dyn}=10$
- $P_{aw} = 10$

End-inspiration 2

- $P_{peak}=15$
- $P_{alv}=15$
- $P_{pl}=5$
- $P_{mus}=8$
- $P_{cw}=5$
- $P_{L, dyn}=10$
- $P_{aw} = 10$

FIGURE 2 Conceptual illustrations of key respiratory physiology during a) spontaneous breathing in a non-ventilated subject, b) fully controlled mechanical ventilation with a passive patient and c) assisted mechanical ventilation. For full description, see main text. All pressures are described in cmH₂O. a) During spontaneous breathing in healthy conditions, pleural pressure ($P_{pl}$) is slightly negative at the end of expiration (situation 2). The pressure generated by the respiratory muscle pump ($P_{mus}$) creates a further drop in $P_{pl}$. $P_{pl}$ is transmitted to the alveoli resulting in a negative alveolar pressure ($P_{alv}$) and a pressure gradient between the airway opening pressure ($P_{ao}$) and $P_{alv}$ allowing tidal volume to enter (situation 2). $P_{mus}$ is the pressure needed to generate chest wall expansion as well as a drop in $P_{pl}$; therefore, $P_{mus}$ is the difference between the chest wall pressure ($P_{cw}$) and $P_{pl}$; maximum $P_{mus}$ occurs at the end of inspiration (situation 3). $P_{cw}$ is calculated by multiplying the instantaneous lung volume by the chest wall elastance ($E_{cw}$). $E_{cw}$ can be obtained during passive lung inflation or calculated as 4% of predicted vital capacity [20]. b) During passive ventilator insufflation $P_{pl}$ increases and represents the $P_{cw}$. Circuit occlusions are required to assess static transmural pressure of the lungs (transpulmonary pressure ($P_{L}$)). When there is no flow and with the airways fully open, $P_{alv}$ represents the airway pressure ($P_{aw}$) during the occlusion: total positive end-expiratory pressure (PEEP) and plateau pressure ($P_{plat}$) for end-expiratory and end-inspiratory occlusions, respectively. $P_{plat}$ is thus the sum of the $P_{L}$ and $P_{cw}$ during the end-inspiratory occlusion. Likewise, the respiratory system driving
pressure ($\Delta P = P_{\text{plat}} - P_{\text{PEEP tot}}$) includes both the lung driving pressure ($\Delta P_L$) and the driving pressure expanding the chest wall ($\Delta P_{cw}$). The direct method and elastance-derived method to calculate $P_L$ are presented. c) During assisted mechanical ventilation, both the ventilator pressure ($P_{\text{vent}}$) and $P_{\text{max}}$ contribute to lung inflation. The swing in dynamic $\Delta P = P_{\text{plat}} - P_{\text{aw}}$ will result in lower transmural pressure of the lungs ($P_{\text{aw}} - P_{\text{alv}}$) (figure 2a).

During spontaneous breathing without ventilatory assistance, inspiratory muscle contraction generates a muscle pressure ($P_{\text{mus}}$) that pulls the chest wall further outwards; $P_{\text{pl}}$ thus decreases to a more negative value. $P_{\text{pl}}$ is transmitted to the alveoli resulting in a pressure gradient between atmospheric pressure (airway opening pressure ($P_{\text{aw}}$)) and the alveolar pressure ($P_{\text{alv}}$); this drives air into the lungs, allowing tidal volume ($V_T$) to enter. Therefore, during spontaneous breathing the only source of pressure is $P_{\text{mus}}$ (i.e. $P_{\text{aw}} = P_{\text{mus}}$) and inflow occurs whenever the $P_{\text{aw}} - P_{\text{alv}}$ gradient is $>0$ (figure 2a).

Of note, during active inspiration, the pressure generated by the relaxed chest wall (i.e. $P_{\text{cw}}$) has to be overcome before airflow can start. $P_{\text{cw}}$ can only be measured during completely passive lung inflation. In this situation, $P_{\text{pl}}$ increases, representing the pressure generated by the chest wall recoil at the specific volume. $P_{\text{cw}}$ can also be computed as the instantaneous volume divided by the theoretical chest wall compliance (estimated as 4% of predicted vital capacity [20]). $P_{\text{max}}$ represents the difference between $P_{\text{cw}}$ and the change in $P_{\text{pl}}$ throughout the breathing effort, with maximal tidal $P_{\text{max}} = (V_T \times \text{chest wall elastance}) - \Delta P_{\text{pl}}$ (figure 2a). Thus, to quantify the magnitude and timing of $P_{\text{max}}$, estimation of $P_{\text{pl}}$ via oesophageal manometry is required [21].

**Physiology during mechanical ventilation**

**Controlled mechanical ventilation**

In passively ventilated patients, the only pressure source is the ventilator: $P_{\text{aw}} = P_{\text{vent}}$. Without airway flow (no resistive pressure) as during circuit occlusions, and in the absence of airway collapse, airway pressure ($P_{\text{aw}}$) measured by the ventilator equals $P_{\text{alv}}$. Estimation of $P_{\text{pl}}$ then allows for quantification of the static transmural pressure of the lungs ($P_{\text{L}} = P_{\text{aw}} - P_{\text{pl}}$; direct measurement technique, see later) and of the chest wall ($P_{\text{cw}} = P_{\text{pl}} - P_{\text{aw}} = P_{\text{pl}} - 0 = P_{\text{pl}}$) (figure 2b).

During a ventilator breath, $P_{\text{L}}$ has temporal and spatial variations. Maximal $P_{\text{L}}$ occurs at end-inspiration when total $V_T$ has entered the lungs: $P_{\text{alv}}$ then equals $P_{\text{plat}}$ measured during a short end-inspiratory occlusion. $P_{\text{plat}}$ reflects the pressure that distends both the lungs and chest wall: $P_{\text{plat}} = P_{\text{L, end-insp}} + P_{\text{cw, end-insp}}$ (measured during end-inspiratory occlusion). Likewise, the respiratory system driving pressure ($\Delta P = P_{\text{plat}} - P_{\text{PEEP tot}}$) includes both the lung driving pressure (i.e. $\Delta P_L$) and the driving pressure expanding the chest wall ($\Delta P_{cw}$) (figure 2b). Thus, for the same $P_{\text{plat}}$ and $\Delta P_{cw}$, $P_{\text{L}}$ and $P_{\text{cw}}$ differ according to the respective lung and chest wall elastances (elastance=1/compliance) [22]: higher lung elastance ("stiff" lung) will result in higher $P_{\text{L}}$ and $\Delta P_{L}$, while higher chest wall elastance ("stiff" chest wall) will result in lower $P_{\text{L}}$ and $\Delta P_{L}$ (but higher $P_{\text{cw}}$). Regarding the spatial variations of $P_{\text{L}}$, $P_{\text{L}}$ is higher in the non-dependent lung compared with the dependent regions; this gradient is exacerbated in ARDS. In this situation, for a given "global" $P_{\text{L}}$ value (estimated with $P_{\text{aw}}$, which does not include the spatial gradient), overdistension in the non-dependent lung units can occur concomitantly with collapse and atelectrauma in the dependent units.

In practice, two main methods exist to calculate $P_{\text{L}}$ from $P_{\text{aw}}$ (figure 2b). The so-called direct method [22, 23] computes $P_{\text{L}}$ as the absolute difference between $P_{\text{alv}}$ (that equals $P_{\text{aw}}$ during circuit occlusions) and $P_{\text{aw}}$. The elastance-derived method [24, 25] uses the tidal change in $P_{\text{aw}}$ (measured with circuit occlusions) to calculate the ratio between lung elastance ($E_L$) and respiratory system elastance ($E_{\text{sys}}$); $P_{\text{L}}$ is then calculated as $P_{\text{aw}} \times E_L/E_{\text{sys}}$ (see supplementary material). This approach assumes that changes in $P_{\text{pl}}$ and $P_{\text{aw}}$ are similar while their absolute values may differ. Both calculation methods are based on assumptions with possible errors. Experimental work in human cadavers and a porcine model of ARDS demonstrated that absolute $P_{\text{aw}}$ accurately reflected local $P_{\text{pl}}$ close to the measurement site (middle third of the oesophagus), corresponding to the mid-dorsal regions of the human thorax [26]. Therefore, the direct method is deemed useful to estimate $P_{\text{L}}$ in the mid-dependent lung regions. Importantly, this remains true with asymmetrical lung injury, where $P_{\text{pl}}$ equates across the two lungs [27]. In contrast, $P_{\text{L}}$ calculated with the elastance-derived method better reflected lung distending pressure of non-dependent regions [26]. Both methods therefore may have different clinical meanings in $P_{\text{aw}}$-guided mechanical ventilation (see later).
Assisted mechanical ventilation

During assisted mechanical ventilation (assist-controlled or purely assisted), the force driving lung inflation and chest wall expansion depends on the combination of the pressure provided by the ventilator and the spontaneous breathing effort: $P_{tot} = P_{vent} + P_{max}$. Therefore, during assisted ventilation it is key to understand that the pressures displayed on the ventilator monitor only reflect part of the total pressure applied to the alveoli: $P_{tot}$ results from both $P_{sw}$ and $P_{max}$ (figure 2c).

A brief history of oesophageal pressure monitoring

In 1949, Buitendijk [28] introduced the latex air-filled oesophageal balloon to study the dynamic lung elasticity in various pulmonary diseases and healthy lungs in 150 subjects. Long before that, in 1878–1880, Luciani and Rosenthal described a minimally invasive extrapleural assessment of pleural pressure by placing an open cannula in the oesophagus (cited in [28]). However, since the holes of the catheter tip were not covered with a balloon, this cannula could clog easily and the air-filled latex balloon was introduced by Buitendijk [28] as a solution to protect the catheter from oesophageal mucus while measuring pressure changes. This increased the popularity of the technique, which was further improved for assessment of both dynamic and passive respiratory mechanics in the years that followed [29–33]. With the more recent improvements of catheters, transducers and bedside monitors facilitating easy catheter insertion, use and calibration, the technique has now moved from a research tool towards a clinical modality at the bedside.

Oesophageal pressure: how do we measure it?

A practical step-by-step description of oesophageal manometry is provided in figure 3 and detailed in the following subsections.

Catheter and equipment

A variety of catheters equipped with oesophageal balloons are available for $P_{oes}$ measurement (see supplementary figure S1 for the most commonly used catheters and Modelli et al. [34] for all second-generation catheters). Some catheters are endowed with a gastric balloon for simultaneous measurement of gastric pressure. Prior to insertion (patient in semi-recumbent position), the balloon is checked for leaks by air inflation, deflated and connected to a three-way stopcock. Rigid tubing should be used to avoid underestimation of pressures due to signal dampening and phase lag (because of non-rigid tube compliance). This is especially important when fast pressure changes are of interest such as during spontaneous efforts. The catheter with extension tubing is then attached to a pressure transducer and connected to a bedside monitor or mechanical ventilator (for some examples, see supplementary figure S2); the pressure waveform should show zero pressure with an open system. Note that commonly used haemodynamic transducers are often calibrated for the positive pressure range (or including a small negative range); hence, they are especially valuable for measuring $P_{oes}$ under passive conditions but may slightly underestimate effort in the presence of excessive negative pressures. A dedicated system for measuring $P_{oes}$ or an auxiliary pressure port of a ventilator can also be used.

Inserting and filling the balloon

The catheter is gently advanced into the mid-lower third part of the oesophagus, commonly at a depth of 33–40 cm from the nostril [35]. Alternatively, it could be advanced into the stomach and then withdrawn in small steps; positive deflections in $P_{oes}$ tracing following gentle epigastric pressure verify intragastric balloon positioning. Oesophageal placement is verified by the presence of cardiac oscillations in $P_{oes}$ tracing [35] and by an occlusion test (see later). To perform the position check and measurements, the balloon is inflated with air. Balloon filling involves complete balloon deflation, equilibration at atmospheric pressure via brief disconnection, injection of maximum air volume to homogenously stretch its walls and then deflation to optimum filling volume ($V_{best}$). In theory, $V_{best}$ is the minimum volume at which tidal $P_{oes}$ swings are maximum [36], which depends on the balloon’s structural properties (length, diameter and compliance), the surrounding intrathoracic pressure and body position/gravity, but also varies depending on whether the patient is on passive mechanical ventilation or spontaneously breathing. Both too low and too high filling volume may result in emptying of the balloon in the catheter–tubing–transducer system and underestimates baseline $P_{oes}$. In contrast, too high filling volume results in an abrupt baseline increase and thus overestimation of $P_{oes}$; since walls of the relaxed oesophagus are normally apposed, distention by too much air produces a positive pressure [29], reflecting elastic recoil of an overfilled balloon. Long, wide balloons have a wider range of $V_{best}$ [12, 30, 31], and thin wall balloons have higher compliance and transmit $P_{oes}$ more precisely. In practice, we suggest using the volume proposed by the manufacturer as an initial volume and adjust accordingly, if deemed necessary depending on the occlusion test [35]. Importantly, with intrathoracic pressure increases (e.g. high PEEP, high ΔP in conditions of high lung
elastance and supine position), \( V_{\text{best}} \) may be higher than suggested [34, 36, 37]. It is thus important to periodically confirm the accuracy of the measurements by repeating the occlusion test, especially following significant changes in ventilator settings, respiratory mechanics or body position.

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**Prepare materials**
- Collect: oesophageal catheter, 2–5 mL syringe, three-way stopcock, rigid extension tubing, pressure transducer and/or dedicated module
- Check for balloon leaks by air inflation and deflation

**Insert catheter and connect to monitor**
Usually 33–40 cm from the nostril to position the balloon in the mid-lower third part of the oesophagus
Alternative: advance catheter into stomach and withdraw in small steps
- Placement is easier in semi-recumbent position
- Water-soluble lubricant aids insertion
- Pay attention to coughing or air leaking around the endotracheal tube, this may suggest tracheal insertion
- Depth markings aid in positioning
- Connect the catheter with the extension tubing and three-way stopcock to the pressure port and/or dedicated monitor

**Inflate the balloon**
- Attach the syringe and pull out any air from the balloon
- Equilibrate the system to ambient pressure: \( P_{\text{oes}} \) should be zero with the three-way stopcock open to air
- Inject maximum air volume and deflate to optimum volume; start with initial volume as described by the manufacturer: Cooper: 2 mL in, 1.2 mL out; NutriVent: 4 mL in, 1.5 mL out

**Verify cardiac oscillations in the \( P_{\text{oes}} \) tracing**
Cardiac artefacts indicate positioning in the oesophagus

**Confirm accuracy of \( P_{\text{oes}} \) monitoring with the occlusion test**

**Passive patient**
1) Perform an end-expiratory occlusion
2) Apply a gentle bilateral thoracic compression
3) Measure the increase in \( P_{\text{aw}} \) and \( P_{\text{oes}} \)

**Actively breathing patient**
1) Perform an end-expiratory occlusion
2) Wait for the next occluded inspiratory effort
3) Measure the decrease in \( P_{\text{aw}} \) and \( P_{\text{oes}} \)

4) Check if \( \Delta P_{\text{aw}}/\Delta P_{\text{oes}} \) ratio is within the 0.8–1.2 range
   The closer the ratio is to 1, the more precise the \( P_{\text{oes}} \) measurement is
   Out of range? Adjust the catheter position and/or filling volume until accuracy is confirmed

**Check the plausibility of \( P_{\text{oes}} \) values**
If baseline \( P_{\text{oes}} \) is very high, suspect balloon overinflation: deflate the balloon, reinflate and repeat the occlusion test with less air in the balloon

**Secure the catheter and start \( P_{\text{oes}} \) measurements**
**Passive patient**: perform end-expiratory and end-inspiratory occlusions to measure \( P_{\text{oes}} \) and \( P_{\text{L}} \)
**Actively breathing patient**: measure dynamic changes in \( P_{\text{oes}} \) and \( P_{\text{L}} \)

Repeat Step 5 regularly to confirm accuracy of \( P_{\text{oes}} \) measurements, especially after changes in ventilator settings, respiratory mechanics or body position

**FIGURE 3** Oesophageal manometry: a practical step-by-step approach to oesophageal pressure (\( P_{\text{oes}} \)) measurements in clinical practice. For further clinical and scientific details, see main text. \( P_{\text{aw}} \): airway pressure; \( P_{\text{L}} \): transmural pressure of the lungs (transpulmonary pressure).
Occlusion test: confirming accuracy of measurement

Adequate balloon filling volume and position should be confirmed with an occlusion test before interpreting $P_{oes}$ values. During a circuit occlusion and in the absence of airway closure, $P_{aw}=P_{adv}$. Moreover, in a closed compartment pressure changes are equally transmitted to the different anatomical components. Then, any change in $P_{aw}$, $P_{oes}$ and $P_{alt}$ should be of similar amplitude. Verification of this assumption is used to confirm the validity of $P_{oes}$ to estimate $P_{alt}$ and requires a different manoeuvre when the patient is spontaneously breathing compared with a passive patient. With breathing efforts, a dynamic occlusion test, i.e. Baydur manoeuvre, is required [13]: an end-expiratory hold is performed and the next inspiratory effort generates a decrease in $P_{aw}$ and $P_{oes}$ against the closed valve. It does not require the patient’s collaboration but can induce some discomfort in awake patients. The ratio of the $P_{aw}$ and $P_{oes}$ drop ($\Delta P_{aw}/\Delta P_{oes}$) during the occluded breath must be close to unity (between 0.8 and 1.2 [13, 38], accepting a 20% error, or ideally between 0.9 and 1.1 for higher accuracy). In passive patients, an external gentle slow manual bilateral thoracic compression is performed during an end-expiratory occlusion to confirm that the $\Delta P_{aw}/\Delta P_{oes}$ ratio is within the required range. During this positive pressure occlusion test, $P_{aw}$ and $P_{oes}$ increase similarly if $P_{oes}$ is reliable [39]. When the $P_{t}$ tracing is displayed on the screen, a flat $P_{t}$ during the occlusion test also confirms that $\Delta P_{aw}/\Delta P_{oes}$ is 1.

The occlusion test has been validated both in adults and children [38–40]. Since different factors can influence the balloon filling volume and thus $\Delta P_{aw}/\Delta P_{oes}$ ratio, it is recommended to systematically check the reliability of the $P_{oes}$ measurement before interpreting values and reposition the balloon and/or adjust the filling volume until validity is confirmed [30, 38]. Regularly checking the $\Delta P_{aw}/\Delta P_{oes}$ ratio is also important during continuous trend monitoring of $P_{oes}$ and/or $P_{t}$, since the balloon may empty over time.

Artefacts

Peristaltic oesophageal contractions or spasms generate slow and large amplitude increases in $P_{oes}$ unrelated to the respiratory cycle; reading of $P_{oes}$ values should be postponed until the signal stabilises. Cardiac contractions transmitted to the balloon can slightly distort the $P_{oes}$ signal that nevertheless usually can still be read. Optimal removal of cardiac artefacts is a topic of research [41, 42]; practically, it is recommended to take end-inspiratory and end-expiratory $P_{oes}$ values at the same time-point within the artefact.

How do we monitor and guide mechanical ventilation and in whom?

Applications during passive mechanical ventilation

Setting PEEP to avoidatelectrauma and lung collapse

Partial or complete lung tissue collapse is reflected by negative end-expiratory $P_{t}$. Negative $P_{t}$ implies atelectasis, increased lung heterogeneity and intrapulmonary shunt, and decreased end-expiratory volumes (at which lung elastance is higher). Increasing PEEP to obtain slightly positive end-expiratory $P_{t}$ (calculated with the direct method as $PEEP_{oes}$—end-expiratory $P_{oes}$ measured with occlusions) allows keeping the alveoli open at end-expiration. This approach could be of interest in ARDS or in patients with elevated $P_{alt}$ from other causes (e.g. abdominal hypertension, ascites, pleural fluids, thoracic wall abnormalities and sometimes obesity). Since the absolute $P_{oes}$ value approximates the actual $P_{alt}$ particularly well in the dependent lung regions at highest risk of collapse, $P_{oes}$-guided PEEP setting should optimise recruitment and decrease atelectrauma in these regions. Importantly, given the spatial differences in $P_{alt}$, clinicians must consider the possibility of overdistension in the non-dependent lung regions at the selected PEEP value.

Two randomised controlled trials in ARDS compared $P_{oes}$-guided PEEP setting and PEEP–inspiratory oxygen fraction ($F_{IO_{2}}$) tables. In the small single-centre EPVent-1 trial, $P_{oes}$-guided PEEP setting titrated to positive end-expiratory $P_{t}$ resulted in higher PEEP at 72 h (mean 17 versus 10 cmH$_2$O for $P_{oes}$-guided versus low PEEP–$F_{IO_{2}}$ table strategy) and improved oxygenation and compliance [23]. A trend towards better clinical outcome was also reported (but underpowered for mortality). The study was terminated early (n=61) because of improved oxygenation on interim analysis. The larger multicentre EPVent-2 trial (n=200, composite primary end-point incorporating mortality and ventilator-free days at day 28) did not find clinical benefits of targeting positive end-expiratory $P_{t}$ of 0–6 cmH$_2$O compared with empirical high PEEP (high PEEP–$F_{IO_{2}}$ table) [43]. The fact that PEEP and $P_{alt}$ between groups were similar during the first week and higher compared with other ARDS trials [44], and that $P_{oes}$-guided PEEP setting resulted in rather high end-expiratory $P_{t}$, could have contributed to the discrepancy with EPVent-1 results. Recent post-hoc reanalysis of EPVent-2 suggested better survival with $P_{oes}$-guided PEEP in patients having lower Acute Physiology and Chronic Health Evaluation (APACHE) II score (less severe multiple organ failure) and that benefits could be maximised by targeting end-expiratory $P_{t}$ tightly within 0±2 cmH$_2$O compared
with higher or more negative values [45]. Experimental work suggests that this strategy optimises the trade-off between lung collapse and overdistension also in unilateral lung injury [27].

**Limiting stress applied to the lung**

During tidal inflation the stress applied to the lung parenchyma must be as low as possible to avoid ventilator-induced lung injury (VILI) [4, 46]. Although $P_{\text{plat}}$ and $\Delta P$ limitations are cornerstones of lung-protective ventilation, these parameters do not reflect lung stress due to interpatient variability in the $E_{\text{L}}/E_{\text{es}}$ ratio [47]. Titration of end-inspiratory $P_{\text{L}}$ and $\Delta P_{\text{L}}$ could allow delivering optimised lung-protective ventilation [7, 48], and is of particular interest in patients with elevated $P_{\text{plat}}$ due to impaired chest wall mechanics [35, 43] and in severe ARDS with high lung elastance (small baby lung).

As previously mentioned, experimental work suggests that maximal $P_{\text{L}}$ of non-dependent regions is best reflected by end-inspiratory $P_{\text{L}}$ calculated with the elastance-derived method [26]. Targeting elastance-derived end-inspiratory $P_{\text{L}} <25$ cmH$_2$O to guide safe PEEP increases while preventing injurious lung stress was tested in 14 patients with ARDS due to influenza H1N1 infection referred for extracorporeal membrane oxygenation (ECMO) [7]. Seven patients (50%) showed increased chest wall elastance resulting in a wide gap between $P_{\text{plat}}$ and end-inspiratory $P_{\text{L}}$; in this subgroup, increasing PEEP using a $P_{\text{oes}}$-guided strategy up to the target $P_{\text{L}}$ improved oxygenation and prevented the use of ECMO without increasing mortality [7].

End-inspiratory $P_{\text{L}} <25$ cmH$_2$O measured with the direct method closely reflects $P_{\text{L}}$ at total lung capacity in healthy volunteers, suggesting that this threshold could be too high to prevent VILI in non-dependent regions in inhomogeneous lungs [49]. In the EPVent-2 study, end-inspiratory $P_{\text{L}} <20$ cmH$_2$O (direct method) was targeted but no effect on outcome compared with the control group was demonstrated [43]. Considering normal values at different lung volumes in healthy volunteers [49] and the risk of increased local stress in heterogeneous lungs, the end-inspiratory $P_{\text{L}}$ threshold calculated with the direct method should probably be <20 cmH$_2$O, at least in patients with inhomogeneous lungs. This, however, requires new clinical studies. Additionally, given that limiting ventilation to a target end-inspiratory $P_{\text{L}}$ aims at reducing overdistension in the non-dependent (aerated baby) lung, it is physiologically sound to use the elastance-derived method to calculate this parameter. As an alternative to using end-inspiratory $P_{\text{L}}$ and based on physiological reasoning, conservative targets for $\Delta P_{\text{L}}$ have been proposed as <15–20 cmH$_2$O in healthy lungs and <10–12 cmH$_2$O for ARDS [13, 35]. Complementary reanalysis of EPVent-1 data (n=56) reported lower $\Delta P_{\text{L}}$ in the intervention group at 24 h after enrolment, associated with improved 28-day mortality [48].

A summary of $P_{\text{oes}}$-guided ventilation targets for ARDS and their level of evidence is provided in figure 4.

**Specific population: obesity**

Morbid obesity can be associated with elevated $P_{\text{plat}}$ due to excess load imposed by the weight of the chest wall. This is frequently associated with preserved chest wall compliance [50–52] but smaller lung volumes [53]. In the situation of obesity, airway pressures can sometimes be high and $P_{\text{plat}}$ values traditionally considered unsafe in ARDS may be associated with safe $P_{\text{L}}$. End-inspiratory $P_{\text{L}}$ more accurately reflects the risk of lung stress in obesity [54]. Compared with a conventional approach, $P_{\text{L}}$-guided lung-protective ventilation in obese patients aiming for positive end-expiratory $P_{\text{L}}$ overall led to higher PEEP and restored end-expiratory volumes, improved lung elastance and oxygenation, prevented lung overdistention, and was haemodynamically tolerated [55–59]. It also decreased ARDS mortality in patients with body mass index (BMI) >40 kg·m$^{-2}$ [60]. It is important to underline that, despite significant correlations between BMI and absolute $P_{\text{oes}}$ values, there is no validated tool to estimate interindividual variability in $P_{\text{L}}$ or chest wall compliance without measuring $P_{\text{oes}}$ in obese patients [50]. Hence, an oesophageal balloon is needed to demonstrate whether high $P_{\text{plat}}$ values are safe in obesity.

**Determination of lung and chest wall elastance and compliance**

Besides titrating ventilator pressures, oesophageal manometry allows measuring and monitoring of static lung and chest wall compliance. The formulas are given in the supplementary material.

**Applications for the active patient**

**Breathing effort monitoring**

Monitoring dynamic change in $P_{\text{oes}}$ ($\Delta P_{\text{oes}}$ or $P_{\text{oes}}$ swing) is the most commonly used and readily available parameter for breathing effort estimation. However, $\Delta P_{\text{oes}}$ underestimates $P_{\text{mus}}$ (see earlier for calculation) that includes the effort needed to move both the chest wall and the lungs (figure 2a). Whereas $P_{\text{mus}}$ represents the pressure generated by all inspiratory muscles, the transdiaphragmatic pressure ($P_{\text{Di}}$) is specific...
to the diaphragm and requires a double-balloon catheter to measure gastric pressure \( P_{ga} \) and \( P_{oes} \) simultaneously: \( P_{al}=P_{ga}-P_{oes} \) (figure 5); \( P_{al} \) swing is a good estimation of effort that provided there is no significant accessory inspiratory muscle recruitment. \( \Delta P_{oes} \) 3–12 cmH2O, \( \Delta P_{max} \) 3–15 cmH2O and \( \Delta P_{al} \) 5–15 cmH2O are considered physiological ranges of effort during assisted ventilation [6, 61], but defining a “safe” range, especially upper limits, requires further study. Nevertheless, too low values may suggest ventilator over-assist and risk of diaphragmatic atrophy (strong clinical evidence [62–65]), whereas high values may cause “overuse” diaphragmatic injury (limited evidence, mostly experimental [66–69]). Recent physiological trials demonstrated the feasibility of titrating ventilator support and/or sedation to achieve lung- and respiratory muscle-protective targets [11, 70]; larger studies should evaluate effects on clinical outcomes.

Estimation of inspiratory effort with \( P_{oes} \) and/or \( P_{max} \) may be challenging in the presence of expiratory muscle activity. Significant expiratory muscle activity during expiration increases intra-abdominal and intrathoracic pressure (and thus \( P_{pl} \)), and subsequent expiratory muscle relaxation at the end of expiration results in a decrease in \( P_{pl} \); this pressure drop should be corrected for when calculating inspiratory muscle effort of the next inspiration [71, 72]. Practically, recording the increase in \( P_{ga} \) during expiration is required to quantify expiratory muscle effort, if present [73, 74], and must be subtracted from \( \Delta P_{oes} \) of the next inspiration.

The amplitude of \( P_{oes} \) swings neglects the time component of muscle contraction and does not account for intrinsic PEEP (PEEPi) if present (see later). Work of breathing (WOB) and the oesophageal pressure–time product (PTPoes) integrate these aspects [14], but are seldom used clinically due to their complexity. WOB is obtained by calculating the area of the volume–pressure \( (P_{oes} \) or \( P_{al} \)) curve, also known as the Campbell

**FIGURE 4** Summary of suggested steps for oesophageal pressure \( (P_{oes}) \)-guided titration of mechanical ventilation in acute respiratory distress syndrome during controlled mechanical ventilation. The procedure should be performed sequentially with step a), then step b) and finally step c). The level of evidence is mentioned. All pressures are described in cmH2O. \( P_{l} \): transmural pressure of the lungs (transpulmonary pressure); PEEP: positive end-expiratory pressure; \( P_{aw} \): airway pressure; \( P_{oes,end-exp} \): end-expiratory \( P_{oes} \); PEEPtot: total PEEP; \( V_{T} \): tidal volume; \( P_{l,end-exp} \): end-expiratory \( P_{l} \); \( P_{l,end-insp} \): end-inspiratory \( P_{l} \); \( P_{plat} \): plateau pressure; \( E_{rs} \): respiratory system elastance; \( E_{L} \): lung elastance.
diagram [14, 75], and only includes effort resulting in volume displacement. PTP_{oes} refers to the time integral of P_{oes}, taking into account P_{cw}, and thus incorporates isometric and miometric effort [76]. PTP_{oes} correlated to oxygen consumption of the respiratory muscles [77]. For more details, see supplementary figure S3 and an extensive review by DE VRIES et al. [78].

**Estimation of lung stress**

Inhomogeneous distribution of lung stress during spontaneous breathing may play an important role in the development or worsening of lung injury, especially with severe “solid-like” injury [79–81]. Through the
pendelluft phenomenon in inhomogeneous lungs (i.e. gas distributed between different lung regions without change in $V_t$), strong inspiratory efforts can augment regional lung stress and strain while $P_{aw}$ and $V_t$ remain the same [82]. In contrast, in mild lung injury spontaneous breathing was found beneficial for lung recruitment [83, 84].

Inspiratory changes in dynamic $P_L$ ($\Delta P_{L,dyne}$) could estimate dynamic lung stress (figure 5). Measurement requires the $P_L$ waveform displayed in real-time. $\Delta P_{L,dyne}$ is generally computed as peak $P_{L, end-expiratory}$ $P_L$ and therefore is different from the static $\Delta P_L$ (circuit occlusions), which can be difficult to obtain/read in actively breathing patients [85]. If inspiratory occlusions can be reliably obtained, the plateau phase of $P_L,dyne$ is thought to represent the stress on the non-dependent lung, whereas $\Delta P_{L,dyne}$ likely reflects approximately the maximum dependent lung stretch [86]. $\Delta P_{L,dyne}$ upper safe limits are uncertain, but values $<15–20$ cmH$_2$O were proposed [61]; however, they probably depend on lung injury severity and systemic inflammation.

**Quantification of dynamic hyperinflation and PEEP**

With dynamic hyperinflation, inspiratory effort is required to overcome PEEP, before volume displacement. Quantification of PEEP requires measuring the $P_{oes}$ drop before inspiratory flow starts (supplementary figure S3). With co-occurrence of expiratory muscle activity and PEEP, which may be common in patients with COPD [72], additional $P_{ga}$ monitoring is recommended; $P_{ga}$ drop owing to expiratory muscle relaxation at the next inspiration should be subtracted from $\Delta P_{oes}$ to avoid PEEP overestimation [72].

**Assessment of patient–ventilator interaction**

Patient–ventilator asynchronies arising from temporal or quantitative dissociation between the neural breath and ventilator-delivered pressurisation could potentially be harmful [87–89]. Careful inspection of $P_{aw}$—time and flow–time waveforms is encouraged to identify their presence to optimise ventilator settings accordingly [90]. This requires expertise and can be challenging or sometimes impossible [91]. Auto-triggered breaths, triggering delay, ineffective efforts around the cycling-off, reverse triggering and early or delayed cycling-off are very difficult to detect without monitoring patient effort, as well as triggering resulting from expiratory muscle relaxation [92]. Automated machine algorithms may increase the accuracy of waveform inspection [93–97], but continuous $P_{oes}$ monitoring remains the most precise method for identifying disynchrony (figure 5) and quantifying the magnitude and timing of disynonymous efforts and their impact on lung stress. It also enables direct assessment of adjustments in ventilator settings on patient–ventilator interaction.

**Weaning**

Both predicting weaning failure [98, 99] and enhancing mechanical ventilation weaning [100] remain important challenges to improve outcomes. Measurements of $P_{max}$, PTP$_{oes}$ and WOB are of interest as respiratory effort has been shown to increase markedly during a failed weaning trial [101, 102]. In addition, expiratory muscle activation may contribute to respiratory muscle effort in weaning failure and could be recognised with $P_{oes}$ and $P_{ga}$ monitoring [73]. $P_{oes}$ trend monitoring during weaning trials performed better than the rapid shallow breathing index to predict weaning failure [103]. $P_{oes}$ monitoring can thus be used to detect weaning failure early during the weaning trial. This could help treating potential reversible factors such as lung oedema, pain or anxiety, allows stopping earlier a spontaneous breathing trial that is going to fail and thus may potentially contribute to avoiding diaphragm injury due to diaphragm overuse. Conceptually, $P_{oes}$ monitoring during weaning seems of interest but additional studies are needed before recommending it as standard of care. $P_{max}$, PTP$_{oes}$ and WOB values associated with weaning failure also have to be determined.

**Is the oesophageal balloon gold standard?**

Despite its introduction a few decades ago and its potential interest as outlined in this review, the oesophageal balloon is only starting to be used regularly in clinical practice, in parallel with technical improvements including the recording systems. Other potentially simpler techniques have been proposed for quantification of breathing effort, and include ultrasound, electrical activity of the diaphragm (EAdi) and $P_{aw}$-derived parameters including the $P_{aw}$ deflection during a short 100 ms occlusion at the start of inspiratory effort ($P_{aw,i}$) or during a full breath occlusion ($P_{awc}$); for detailed descriptions and reference ranges, see GOLICHER et al. [61]. Ultrasound has become a well-established bedside tool for real-time visualisation of diaphragm contraction and movement, and to screen for respiratory muscle dysfunction [104]; however, diaphragm inspiratory thickening is unfortunately only weakly correlated to its pressure-generating capacity and not continuous [105, 106]. Advanced ultrasound techniques may better quantify muscle function and effort (e.g. strain imaging and shear wave elastography) and are a topic of future studies [104]. EAdi as measured via a dedicated naso-gastric catheter with electrodes reflects the
electrical activity of the crural diaphragm [107] and correlates reasonably well with breathing effort [108], but amplitudes are highly variable between subjects. EAdi could be particularly useful for identifying ventilated patients at risks for diaphragm disuse [109], to monitor within-patient changes in drive/effort or to assess patient–ventilator interaction. Notwithstanding, EAdi monitoring does not permit P0.1 estimation. Non-invasive parameters P0.1 and Pocc can be measured with almost every intensive care unit (ICU) ventilator. Although P0.1 was originally validated as a measure of respiratory drive [110], correlations with total inspiratory effort exist, but with large between-subject variations, and P0.1 was found especially sensitive for detecting low effort [111], but also for detecting high effort with some ventilators [112] or to predict relapse of respiratory failure [113]. Pocc was recently validated in a small cohort as a measure to screen for high effort (Pmax) and excessive P1 [114]. Recent work demonstrated that P0.1 and Pocc could also identify excessive P1 and the extremes of both low or high diaphragm effort specifically, with reasonable to excellent performance and with Pocc outperforming P0.1 for detecting high ΔPdi [115].

Central venous pressure swings (ΔCVP) may directly reflect P0.1 and the extremes of both low or high diaphragm effort specifically, with reasonable to excellent performance and with Pocc outperforming P0.1 for detecting high ΔPdi [115]. While P0.1 has been shown to be sensitive for detecting low effort [111], but may identify excessive effort with reasonable accuracy in experimental work [117]; this needs clinical validation. Therefore, in patients under partially assisted mechanical ventilation, the aforementioned techniques may screen for potentially low or excessive efforts (or excessive P1), but Poes remains the reference standard for quantification.

**Novel developments**

Poes integration into the ventilator monitor is now available for some machines and in vivo calibration methods [37] could potentially be automated, which improves the feasibility of Poes monitoring at the bedside. This could also enable future integration of respiratory mechanics calculations and breathing effort monitoring directly into the ventilator, as well as applications of machine learning techniques for automated detection of low/excessive efforts, identification of asynchronies or recognition and removal of artefacts, for instance.

Whereas balloon catheters were introduced because Poes monitoring with liquid or air-filled open catheters presented artefacts related to fluid menisci of surface tension effects (the balloon protected the catheter) [29], recently such catheters have been studied again but require further clinical validation [118–120]. Catheter-mounted microsensors measure Poes directly inside the oesophagus, and thus have a faster frequency response compared with balloon catheters and are not subjected to signal dampening. This allows for more accurate recording of fast pressure changes [121], but may also result in larger cardiac or peristaltic artefacts that require adequate signal filtering. Although previous solid-state sensor techniques were mainly limited by large offsets and temperature drifts [120, 122], new technological advances may overcome these limitations.

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<th>Points for clinical practice</th>
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<tr>
<td>• Poes monitoring allows partitioning of the lungs and the chest wall physiology during controlled ventilation. Tailoring ventilator settings based on the patient’s individual respiratory physiology could offer additional solutions with mechanical ventilation to improve oxygenation, including optimisation of PEEP setting.</td>
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<tr>
<td>• Poes monitoring allows measuring inspiratory effort and WOB, and assessing patient–ventilator interaction. This could facilitate providing lung- and diaphragm-protective ventilation during assisted ventilation and could optimise mechanical ventilation weaning.</td>
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<td>• Technological advances allow Poes monitoring to be implemented as part of routine respiratory monitoring in selected patients. This should stimulate the field to learn about the potential benefit of Poes monitoring in the complex critically ill.</td>
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<td>• Challenges and technical difficulties should be acknowledged before using Poes-derived values to set the ventilator.</td>
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<th>Questions for future research</th>
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<td>• Poes measurement could be a useful tool to optimise lung- and diaphragm-protective ventilation by adapting ventilator support levels to the patient’s breathing effort. Future research should focus on defining the optimal range of breathing effort, especially upper limits for safe diaphragm effort, and the impact of targeting diaphragm effort on patient outcomes.</td>
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<td>• Additional studies on the potential of Poes monitoring to individualise PEEP settings are also needed.</td>
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Summary

There is a well-recognised need for optimising mechanical ventilation to protect the lungs and the diaphragm for each individual patient [6, 61]. Measurement of partitioned respiratory mechanics and quantification of lung stress and breathing effort using $P_{\text{oes}}$ monitoring could improve our understanding of the patient’s unique respiratory physiology and allows personalisation of mechanical ventilation settings under different conditions, as extensively discussed in this review. Although clinical evidence for $P_{\text{oes}}$-guided mechanical ventilation is yet limited and challenges remain, technological improvements have made $P_{\text{oes}}$ monitoring feasible to become part of bedside respiratory monitoring in selected patients. This should encourage clinicians to develop new clinical studies aimed at identifying optimal and safe $P_{\text{oes}}$-guided targets for the management of the critically ill in order to improve ICU outcomes.

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