



Measurement of pleural pressure swings with a fluid-filled esophageal catheter vs pulmonary artery occlusion pressure^{☆,☆☆}



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ABSTRACT

Purpose: Pleural pressure measured with esophageal balloon catheters (Peso) can guide ventilator management and help with the interpretation of hemodynamic measurements, but these catheters are not readily available or easy to use. We tested the utility of an inexpensive, fluid-filled esophageal catheter (Peso) by comparing respiratory-induced changes in pulmonary artery occlusion (Ppao), central venous (CVP), and Peso pressures.

Methods: We studied 30 patients undergoing elective cardiac surgery who had pulmonary artery and esophageal catheters in place. Proper placement was confirmed by chest compression with airway occlusion. Measurements were made during pressure-regulated volume control (VC) and pressure support (PS) ventilation.

Results: The fluid-filled esophageal catheter provided a high-quality signal. During VC and PS, change in Ppao ($\Delta Ppao$) was greater than Δ Peso (bias = -2 mm Hg) indicating an inspiratory increase in cardiac filling. During VC, Δ CVP bias was 0 indicating no change in right heart filling, but during PS, CVP fell less than Peso indicating an inspiratory increase in filling. Peso measurements detected activation of expiratory muscles, development of non-west zone 3 lung conditions during inspiration, and ventilator-triggered inspiratory efforts.

Conclusions: A fluid-filled esophageal catheter provides a high-quality, easily accessible, and inexpensive measure of change in pleural pressure and provided insights into patient-ventilator interactions.

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Pleural pressure (Ppl) measurements can provide important information for the management of critically ill patients [1,2]. For example, negative swings in Ppl during spontaneous breathing give an indication of inspiratory muscle effort and work of breathing. Measurement of Ppl in mechanically ventilated patients can identify patient-ventilator dyssynchrony and unrecognized inspiratory efforts during what is thought to be controlled ventilation [3]. More recently, there has been increasing interest in the use of Ppl to obtain the best value of positive end-expiratory pressure that can maximize pulmonary recruitment while avoiding airway opening volutrauma [4,5]. Monitoring respiratory swings in Ppl also can help interpret the processes behind variations in vascular pressures during the respiratory cycle.

Pleural pressure most often has been estimated from pressure in an air-filled balloon in the esophagus (Peso) [6]. These balloons can be combined with oral or nasogastric tubes that are used for feeding or

gastric drainage. However, current commercial esophageal balloon catheters are expensive and not readily available. The appropriate balloon volume and position also need to be regularly checked, which makes long-term monitoring difficult. We have found that an inexpensive, relatively stiff tube which commonly is used for suctioning airways in children can provide an excellent measure of Ppl swings during the respiratory cycle when it is filled with fluid. Our primary objective was to confirm the reliability of this device in a series of critically ill patients. A second objective was to use measurements of Ppl to gain insight into common variations in thoracic hemodynamics that are due to heart-lung interactions in postcardiac surgery patients.

The rationale for our approach is based on a previous study in which we showed that the peak fall in pulmonary artery occlusion pressure (Ppao) during a spontaneously triggered breath closely follows the peak change in Ppl measured with an esophageal balloon [7]. In this study, we compared ventilation-induced swings in Ppl obtained with the fluid-filled catheter to changes in Ppao. We chose cardiac surgery patients because pulmonary artery catheters are routinely inserted for surgery in most of these patients at our institution. This group thus provided a readily available population for the study and, unlike the situation in most intensive care subjects, consent could be obtained when subjects were awake and competent before surgery. Subjects were studied during pressure-regulated volume ventilation (VC) just

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after surgery and then a few hours later with pressure support (PS) when they woke up.

1. Methods

1.1. Patients

We performed a single-center study in the medical-surgical intensive care unit (ICU) of the McGill University Health Center. The protocol was approved by the research ethic review board of the McGill University Health Center. All patients undergoing elective cardiac surgery (coronary artery bypass graft and/or valve replacement) were eligible. We studied 30 patients in whom preoperative consent was obtained, were sufficiently stable when they came from the operating room, and had a pulmonary artery catheter in place as part of their routine management. Exclusion criteria included gastrointestinal bleeding; anatomical esophageal abnormalities, such as esophageal varices, ulcers, or tumor; presence of hemodynamic or respiratory instability; age less than 18 years; refusal by the attending physician; or arrival too late in the ICU to make the measurements.

1.2. Protocol and measurement.

After stabilization in the ICU, subjects were placed in the supine position with the head of the bed elevated to 30°. A sterile Müllly (Unomedical, Los Angeles, CA) suction catheter (external diameter 2.7 mm, length 53 cm) was inserted with the help of a guide-wire through one nostril. The tube was filled with 0.9% saline and connected to a standard hemodynamic transducer (Transpac IV Monitoring Kit; IcuMedical, San Clemente, CA). A constant pressure of 300 mm Hg was applied to the saline bag as used for maintaining invasive arterial pressure catheters. Proper positioning of the catheter in the esophagus was confirmed during controlled ventilation by doing 3 chest compressions in the mid-chest during an end-expiratory pause [1] (Fig. 1). With this procedure, the measurement of *Peso* was considered acceptable if change in *Peso* was at least 0.85 of the change of airway pressure (*Paw*). In all subjects, the ratio was greater than 95%. Unlike air-filled catheters, pressures measured with fluid-filled catheters are dependent on a reference level. However, this was not necessary in this study because we were interested in comparing the change (Δ) in vascular and esophageal pressures. Hemodynamic pressures were based on a level 5 cm below the sternal angle [8].

The first measurement was performed just after the patient arrived in the ICU and still recovering from the anesthesia and ventilated with VC. Maximum inspiratory increase in *Ppao* and central venous pressure (CVP) were compared with the maximum increase in *Peso*.

When subjects awoke and began to trigger breaths, the mode of ventilation was switched to PS. During this mode, maximum negative deflections in *Ppao* and CVP were compared with the maximum

negative deflections in *Peso*. Measurements of *Ppao* and CVP were taken at the base of the “c” wave when visible or the base of the “a” wave when the “c” could not be identified. A second reviewer independently reviewed recordings from 20 subjects to test reproducibility of the measurements.

The measurements of *Ppl* allowed us to make some observations on heart-lung interactions and their effects on hemodynamic interpretations. These included evidence of recruitment of expiratory muscles and the appropriate time in the cycle to make hemodynamic measurements, possible development of non-west zone 3 conditions during mechanical breaths, and ventilator-triggered ventilator responses.

We have previously described 2 patterns of recruitment of expiratory muscles [9,10]. In type A, CVP falls during expiration. In type B, CVP progressively increases during expiration. We assessed the prevalence of these patterns in the current sample as indicated by the change in *Peso* with the change in hemodynamic pressure.

Second, when alveolar pressure is higher than pulmonary venous pressure, pulmonary veins collapse and produce flow-limitation or a Starling-resistor effect in what is called west zone 2 [11,12]. When alveolar pressure is higher than pulmonary arterial pressure, flow in that region is blocked in what is called west zone 1. In both west zones 1 and 2, *Ppao* reflects alveolar pressure and not left atrial pressure, as is the case in west zone 3 in which there is no obstruction to pulmonary flow [13,14–17]. During VC, evidence of non-zone 3 conditions was identified by an increase in *Ppao* that was close to the increase in *Paw*, more than 2 mm Hg greater than the increase in *Peso*, and an increase in *Ppao* that was greater than the increase in CVP. During PS, presence of non-zone 3 was suggested by a rise in *Ppao* with a fall in *Peso*. We cannot be certain that these criteria truly indicate non-zone 3 conditions, but we consider the data to be suggestive and, if frequent, worthy of further study because of the significance for right ventricular loading [17,18].

1.3. Statistical analysis

Data are expressed as mean \pm SD. Agreement between measurements was assessed by Bland-Altman plots, identity plots, and regression analysis. The bias and limits of agreement were calculated as the difference of change of esophageal pressure (Δ *Peso*) during inspiration from the difference of Δ *Ppao* or Δ CVP during inspiration (ie, Δ *Peso* – Δ *Ppao*, or Δ *Peso* – Δ CVP) because in this study, the change in *Ppao* and CVP was considered to be the independent variables (see Discussion).

2. Results

Baseline characteristics of the patients are given in the Table 1. Fig. 2A shows a typical tracing of a subject during VC and Fig. 2B an example of a subject breathing with PS. As can be seen in the figures,

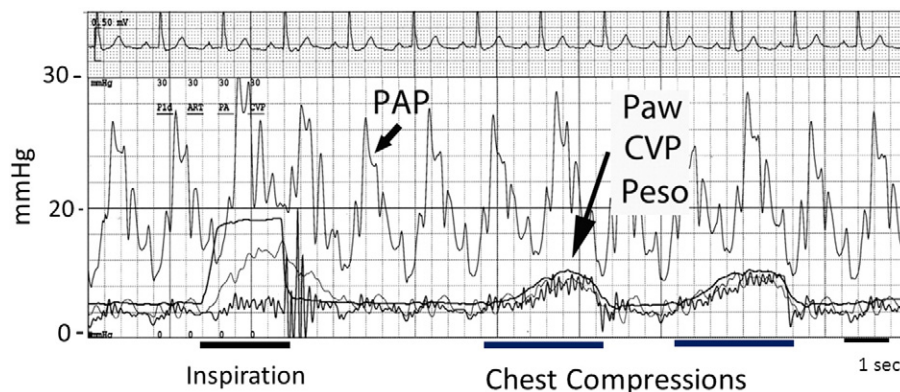


Fig. 1. Example of change in *Peso*, CVP, and *Paw* during the chest compression test. Change in CVP was identical to change in *Peso* during the compression. PAP indicates pulmonary artery pressure. All pressures are in mm Hg.

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