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PHYSIOLOGICAL NOTE

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Clinical significance of pulmonary artery occlusion pressure

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Abstract *Background*: Ppao values are routinely used to assess pulmonary vascular status and LV performance. Regrettably, under many common clinically relevant conditions, even when Ppao values are measured accurately, Ppao values at baseline and in response to therapy often reflect an inaccurate measure of cardiovascular status.
Results and conclusions: Thus, caution should be used when applying measures of Ppao in determining therapy if changes in RV volume, hyperinflation, or LV diastolic compliance are simultaneously occurring.

Keywords Bedside measurements · Pulmonary hemodynamics · Left ventricular performance · Critically ill patients · Pulmonary artery occlusion pressure · Pulmonary vascular status

Introduction

Balloon floatation pulmonary arterial catheterization has permitted the bedside estimation of pulmonary hemodynamics and left ventricular (LV) performance of critically ill patients. Present-day pulmonary artery catheters can routinely estimate cardiac output and right ventricular (RV) ejection fraction, by the thermodilution technique, mixed venous oxygen saturation, by reflective oximetry, and three intrapulmonary vascular pressures: right atrial, pulmonary arterial and pulmonary artery occlusion pressure. Of all of these, pulmonary artery occlusion pressure (Ppao) is perhaps subject to the most error in its measurement and interpretation. The previous "Physiological Note" discussed problems in the accurate measure of Ppao at the bedside. In this one we discuss the physiological significance of Ppao measures.

Pulmonary artery occlusion pressure is used most often in the bedside assessment of: (a) pulmonary edema, (b) pulmonary vasomotor tone, (c) intravascular volume status and LV preload, and (d) LV performance. We address each separately.

Pulmonary edema

Acute pulmonary edema can be life threatening because of the systemic hypoxemia that it creates. Pulmonary edema can be caused by either elevations in pulmonary capillary pressure (hydrostatic or secondary pulmonary edema), increased capillary and/or alveolar epithelial permeability (primary pulmonary edema), or a combination of the two. If pulmonary capillary pressure increases above 18–20 mmHg, increased fluid flux across the capillary membrane occurs, promoting alveolar flooding. However, if capillary or alveolar cell injury is present, alveolar flooding can occur at much lower pulmonary capillary pressures. Measures of Ppao are commonly used to determine the cause of pulmonary edema. Thus Ppao values lower than 18–20 mmHg suggest a nonhydrostatic cause,

whereas values higher than 18–20 mmHg suggest a hydrostatic cause of pulmonary edema [1]. However, these are not hard values.

Ppao may be lower than 18–20 mmHg in a patient with secondary pulmonary edema if either the Ppao increase had been transient and is now gone, or if pulmonary capillary pressure significantly exceeds Ppao. Transient severe LV dysfunction can transiently increase Ppao during upper airway obstruction with vigorous inspiratory efforts (inspiratory stridor, obstructive sleep apnea), unstable angina (reversible ischemia), and arrhythmias. Increased pulmonary capillary pressure can occur due to massive sympathetic discharge (e.g., intracerebral hemorrhage and heroin overdose), which rapidly reverses, but the pulmonary edema lingers. Furthermore, persistently elevated pulmonary capillary pressures may coexist with normal Ppao values if pulmonary venous resistance is increased and cardiac output not decreased (e.g., high altitude pulmonary edema, pulmonary venoocclusive disease, and end-stage acute respiratory distress syndrome).

Ppao may be higher than 18–20 mmHg in a patient without hydrostatic pulmonary edema. Since Ppao is measured relative to atmospheric pressure, elevations in pleural pressure artificially elevate Ppao values. Any increase in pleural pressure increases measured Ppao. Hyperinflation, either intrinsic or extrinsic, increases pleural pressure. Furthermore, when active expiratory muscle effort persists, pleural pressure increases.

Pulmonary vasomotor tone

Increased pulmonary arterial pressure (Ppa) impedes RV ejection, causing RV dilation and a decreased cardiac output. If pulmonary hypertension occurs rapidly, as with massive pulmonary embolism or marked hyperinflation, acute cor pulmonale and cardiovascular collapse also occurs. Pulmonary hypertension can be due to either an increase in pulmonary vasomotor tone or passive increases in Ppao due to LV failure. The pulmonary circulation normally has a low resistance, with pulmonary arterial diastolic pressure only slightly higher than Ppao and mean pulmonary arterial pressure thus a few mmHg higher than Ppao Global pulmonary vascular resistance, by Ohm's law, equals the ratio of the driving pressure (mean Ppa–Ppao) and flow (cardiac output). Normal pulmonary vascular resistance is between 1.8 and 3.1 mmHg l^{-1} min^{-1}. Usually these values are multiplied by 80 to give normal pulmonary vascular resistance range of 150–250 dynes s^{-1} /cm^{-5} of. Thus by measuring Ppa, Ppao, and cardiac output in patients with pulmonary hypertension, one may determine whether the increase in Ppa is due to increased pulmonary vascular resistance (PVR) or a passive pressure build-up. If pulmonary hypertension is associated with an increased PVR then the causes are primarily within the lung, whereas if PVR is normal then LV dysfunction is the more likely cause [2].

Regrettably, PVR is not a good measure of pulmonary vasomotor tone. Pulmonary vascular pressure does not decrease linearly from input to output and may vary from region to region due to lung distention, structural damage and acute processes, such as hyperinflation, pneumonia, emphysema, pulmonary fibrosis, and acute lung injury. Thus PVR as a lumped parameter may not identify local injury or define why PVR is elevated. As alveolar pressure increases above Ppao (West zone 2 conditions) alveolar pressure becomes the backpressure to pulmonary blood flow. Thus measures aimed at decreasing pulmonary vasomotor tone (e.g., inhaled nitric oxide) have little effect on Ppa [3]. Furthermore, with nonhomogeneous lung disease blood flow is preferentially shifted to those circuits with the lowest resistance, thus making the lung vascular pathology appear less than it actually is. By examining the change in Ppa in response to interventions that alter cardiac output, one may obtain a better understanding of the determinants of pulmonary hypertension. If the extrapolated zero-flow pulmonary artery pressure created from such a maneuver is much higher than Ppao, Ppao is probably not the downstream pressure to flow thus measures aimed at reducing zone 2 conditions (reverse hyperinflation) should be more effective at decreasing Ppa.

Intravascular volume status and LV preload

Hemodynamically unstable patients often benefit for fluid resuscitation, as manifested by increases in organ perfusion and function, resolution of lactic acidosis, and increased survival. A fundamental tenant of such therapy is that fluid resuscitation increases LV end-diastolic volume (EDV), and that such increases in EDV translates into increased cardiac output. For a given level of contractile function, increasing LV EDV increases both LV stroke volume (and thus cardiac output) and LV stroke work. It is difficult to make repeated measures of LV EDV at the bedside to titrate fluid resuscitation and vasoactive therapy. Ppao values are often taken to reflect LV filling pressure, and by inference LV EDV. Operationally, subjects with cardiovascular insufficiency and a low Ppao are presumed to be hypovolemic and initially treated with fluid resuscitation, whereas patients with similar presentations but and elevated Ppao are not. Although there is no accepted high and low Ppao values for which LV under filling is presumed to occur, Ppao values lower than 10 mmHg are usually used as presumed evidence of a low LV EDV, whereas values higher than 18 mmHg suggest a distended LV [4].

Regrettably, of all the uses of Ppao in the management of the critically ill, this one use is the least accurate. The reasons for this are multiple and relate to the

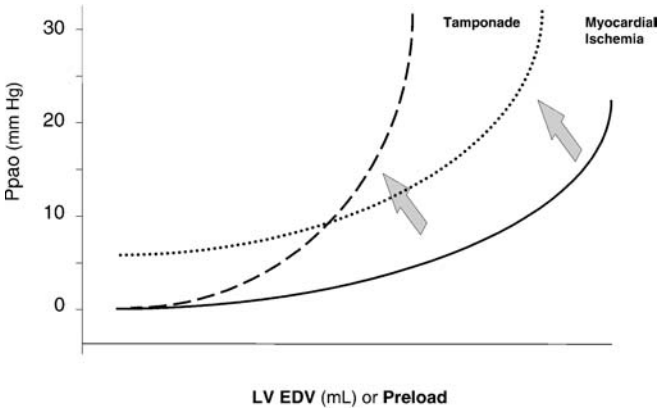

Fig. 1 Schematic representation of the relationship between left ventricular end-diastolic volume (LV EDV) and pulmonary artery occlusion pressure (Ppao) under a variety of circumstances. *Solid line* Idealized LV diastolic compliance; *other two curves* increased external pressure and volume constraint (tamponade) and diastolic stiffening (myocardial ischemia), respectively

determinants of LV diastolic compliance and contractile function (Fig. 1) [5]. First, the relationship between Ppao and LV EDV is curvilinear and may be very different between subjects. Thus neither absolute values of Ppao or changes in Ppao define a specific LV EDV or its change. Second, Ppao is not the distending pressure for LV filling. Assuming that Ppao approximates left atrial pressure, it would poorly reflect LV end-diastolic pressure because it poorly follows the late diastolic pressure rise induced by atrial contraction and does not measure pericardial pressure, which is the outside pressure for LV distention. Thus changes in pericardial pressure alter LV EDV independently of Ppao. Hyperinflation, tamponade, and active inspiratory and expiratory muscle activity can rapidly alter pericardial pressure. Finally, even if one knew that pericardial pressure and Ppao do accurately reflect LV end-diastolic pressure, LV diastolic compliance can vary rapidly, changing the relationship between LV filling pressure and LV EDV. Myocardial ischemia, arrhythmias, and acute RV dilation can all occur over a few heartbeats. Thus it is not surprising that Ppao is a very poor predictor of preload responsiveness. The use of Ppao as a measure of LV EDV and preload responsiveness has not been validated by clinical trials. Accordingly, using Ppao to predict response to fluid resuscitation is not recommended, except at the extremes of Ppao values, and during those conditions one rarely needs to measure Ppao to make the correct diagnosis.

LV performance

As stated above, LV EDV is a fundamental determinant of stroke volume and LV stroke work. The bedside assessment of LV performance is important in determining the causes of cardiovascular insufficiency and the potential of the patient to response to fluid challenge, increasing arterial pressure and afterload reduction. Although many factors converge on the resultant LV stroke volume, including valvular function, synchrony of contraction, and diastolic filling time the four primary determinants of LV performance are preload (LV EDV), afterload (LV wall stress, which is itself the product of LV EDV and diastolic arterial pressure), heart rate, and contractility. To the extent that Ppao mirrors LV EDV, Ppao can be used to construct Starling curves that plot Ppao vs. LV stroke work (LV stroke volume × developed pressure). Patients with heart failure can be divided into four groups depending on their Ppao (>or <than 18 mmHg) and cardiac index values (>or <2.2 l min^{-1} m^{-2}) [4]. Those patients with low cardiac indices and high Ppao are presumed to have primary heart failure, and low Ppao hypovolemia. Those with high cardiac indices and high Ppao are presumed to be volume overloaded, and low Ppao increased sympathetic tone.

Again, as above, if LV diastolic compliance is reduced or pericardial pressure increased, Ppao underestimates LV EDV. This interaction is the primary reason why both acute pulmonary embolism-induced cor pulmonale and PEEP-induced hyperinflation were erroneously thought to cause myocardial depression. In both clinical scenarios baseline Ppao values markedly increase without a proportional increase in stroke volume. Accordingly, the same limitations on the use of Ppao in assessing LV preload must be considered when using it to assess LV performance. Thus in subjects without lung or pericardial disease, tamponade, or pulmonary embolism the relationship between Ppao and LV stroke work can be used to assess LV performance.

Summary

Ppao values are routinely used to assess pulmonary vascular status and LV performance. Regrettably, under many common clinically relevant conditions, even when Ppao values are measured accurately, Ppao values at baseline and in response to therapy often reflect an inaccurate measure of cardiovascular status. Thus caution should be used when applying measures of Ppao in determining therapy if changes in RV volume, hyperinflation, or LV diastolic compliance are simultaneously occurring.

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