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PHYSIOLOGICAL NOTE

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Pulmonary capillary pressure

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Introduction

Pulmonary capillary pressure is a primary determinant of fluid flux across the pulmonary capillary wall [1]. Increasing pulmonary capillary pressure increases fluid flux out of the capillaries into the interstitium and in the extreme induces pulmonary edema. Pulmonary capillary pressure is itself determined by the mean pulmonary artery pressure, pulmonary vascular resistance, and total blood flow. The distribution of the pulmonary vascular resistance from precapillary arterial to postcapillary venous compartments varies. Accordingly, at any given blood flow rate the hydrostatic pressure in the pulmonary capillaries depends on the magnitude of the resistance to blood flow across the pulmonary circulation and its distribution between precapillary and postcapillary vessels. Since pulmonary capillary pressure cannot be directly measured, the presence and relevance of increased pulmonary capillary hydrostatic pressures to values in excess of pulmonary artery occlusion pressure are often overlooked.

What are the components of the pressure drop across the pulmonary vasculature?

The resistance to flow across the pulmonary circulation results in the pressure drop from the large pulmonary artery to the left atrium. This resistance can be separated into arterial and venous components, with relatively little resistance seen in the compliant capacitance pulmonary capillary vessels. This physical situation can be modeled as an electrical circuit consisting of two or several resistances in series with one or several capacitors connected between the resistances (Fig. 1). The simplest model assumes one arterial and one venous resistance with one capacitance located in the capillaries [2, 3]. A three-compartment model consisting of compliant arterial, capillary and venous capacitance compartments between four resistances (resistance of large and small arterial and venous vessels, respectively) is probably more representative but does not improve the accuracy of measuring pulmonary capillary pressure [1].

Because of this series resistance interposed by a compliant pulmonary capillary network, pulmonary capillary pressure can be measured from the pressure decay profile of an acute pulmonary artery balloon occlusion maneuver. When the pulmonary artery is occluded, there is a rapid decrease in blood flow as the occluded downstream pulmonary artery discharges its blood volume sequentially into the pulmonary capillaries across the arterial resistance and then into the pulmonary veins across the venous resistance. This two-part pressure discharge is reflected in the pulmonary artery pressure decay curve. The initial rapid pressure drop approaches the pressure in the capillaries (the main capacitance component) as the blood trapped in the downstream pulmonary capillaries equilibrates with pulmonary capillary pressure. This is followed by a slower pressure decrease approaching the pulmonary artery occlusion pressure as pulmonary capillary pressure equilibrates with pulmonary venous pressure (Fig. 1a). The initial pressure drop re-

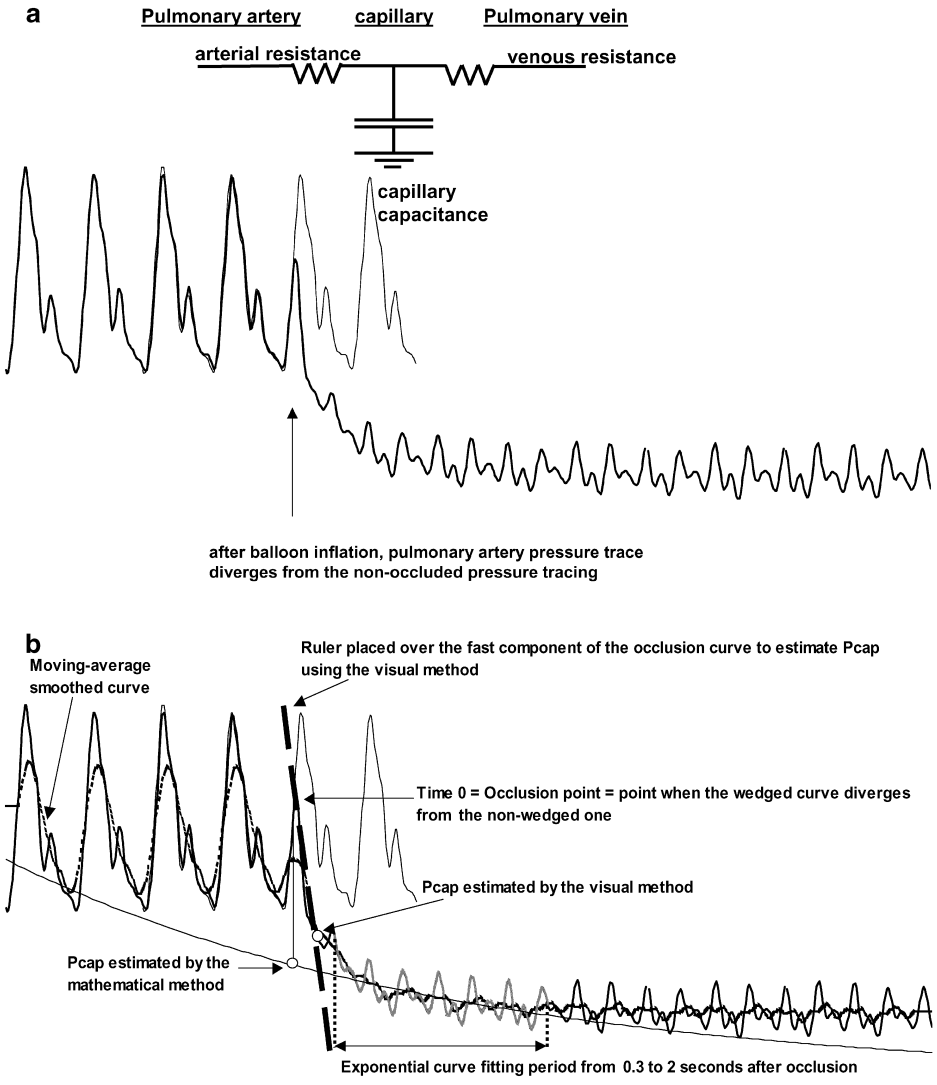

Fig. 1 A schematic representation of the electric circuit analogue of the pulmonary circulation is superimposed on the two pressure recordings. **a** Pulmonary artery pressure decay after the balloon of the Swan-Ganz catheter has been occluded. For better visualization the occlusion trace is superimposed on a nonoccluded trace, both recorded during an expiratory hold during mechanical ventilation. **b** Capillary pressure has been estimated from the trace shown in **a**. An additional trace using 20 data point moving average smoothing of the original trace (collected at 100 Hz) is superimposed on the curves. This further facilitates the visual estimation of the capillary pressure by defining more exactly the point of divergence of the occluded and nonoccluded curves. In addition, an exponential curve has been fitted on the curve 0.3–2 s after occlusion. This fitted curve has then been extrapolated to the time of occlusion to provide the capillary pressure

flects the proximal arterial resistance, and the slower pressure drop reflects the distal, venous resistance. The model shown in Fig. 1 consisting of serial resistance and capacitances does not represent the simultaneous discharge of the different capacitance components of the pulmonary circulation. Nevertheless, it provides a close approximation of the decay of pressure after a pulmonary arterial occlusion for most clinical conditions.

What is the physiological relevance of the pulmonary capillary pressure?

Pulmonary capillary pressure is a major determinant of fluid flux across the capillary wall and lung edema formation. Under normal conditions some fluid and protein is filtered through the capillary into the pulmonary interstitium and subsequently drained into the systemic circulation by the lung lymphatics. When the capacity of the lymphatics is exceeded, first interstitial and then alveolar edema ensues. The rate of fluid filtration from the capillary to the interstitium can be estimated by the Starling equation:

$$\text{Fluid efflux} = K_{fc} \times \left[\left(P_{capillary} - P_{interstitium}\right)\right] - K_d \left[\left(\pi_{capillary} - \pi_{interstitium}\right)\right]$$

where P=hydrostatic pressure, π=oncotic pressure, K_{fc}=capillary filtration coefficient (product of capillary wall hydraulic conductivity and capillary surface area), and K_d=reflection coefficient (values from 0 to 1; 0=capillary freely permeable to proteins, 1=capillary impermeable to proteins). When fluid efflux increases for any reason, lymph flow increases as well, washing out interstitial protein and decreasing $\pi_{interstitium}$, thus increasing the oncotic gradient for fluid flux back into the blood

and counteracting edema formation. When the permeability to protein increases, the influence of the term K_d ($\pi_{capillary}-\pi_{interstitium}$) in the Starling equation is reduced due to the decreased K_d as well as the decreased ($\pi_{capillary}-\pi_{interstitium}$) (loss of protein to the tissue). However, no matter what the oncotic pressure gradient, based on the Starling equation, increasing pulmonary capillary pressure always increases fluid efflux. If the capillary permeability to protein is normal, a higher capillary pressure is needed for a given rate of fluid efflux. Conversely, in the presence of increased capillary permeability, lower capillary pressure is needed for a given rate of fluid efflux.

Since the capillary pressure is the major determinant of fluid efflux from the capillaries both in normal and abnormal permeability states, division between "hydrostatic" or "cardiogenic" lung edema and "permeability" or "low-pressure" edema when pulmonary capillary pressure is unknown is artificial and arbitrary. Indeed, capillary hydrostatic pressure and capillary permeability interact in all types of lung edema. An increase in the pulmonary venous resistance increases the pulmonary capillary pressure. Under these conditions the pulmonary artery occlusion pressure or left atrial pressure underestimates the pulmonary capillary pressure [4, 5]. Furthermore, the pressure difference between pulmonary capillary pressure and left atrial pressure varies with blood flow. The higher the blood flow then for the same pulmonary venous resistance the greater the pulmonary capillary pressure and the greater the pressure drop.

How to interpret an increased transpulmonary pressure gradient?

A positive pressure gradient must exist between the pulmonary arterial diastolic pressure and the left atrium for blood to flow. Under normal circumstances this gradient is less than 6–8 mmHg, increasing slightly with increasing flow and decreasing to near zero at rest when pulmonary blood flow almost ceases during each diastole. A widening gradient between the pulmonary arterial diastolic pressure and the left atrial pressure is a signal of increased pulmonary vascular resistance, increased pulmonary blood flow, or both, and is an indicator that pulmonary capillary pressure may exceed pulmonary artery occlusion pressure. While the pulmonary artery occlusion pressure may overestimate the left atrial pressure in the presence of Starling resistor forces causing pulmonary venous collapse [6], an increased gradient between the pulmonary arterial diastolic pressure and the pulmonary artery occlusion pressure is still a valid indicator of increased capillary pressure. An isolated increase in the arterial resistance does not increase the capillary pressure by itself.

Normally two-thirds of the transpulmonary pressure drop occurs over the arterial resistance, with approximately one-third of the pressure drop occurring over the venous resistance. However, a selective increase in pulmonary venous resistance can occur and directly increases pulmonary capillary pressure in proportion to blood flow. Many normal physiological responses and disease states are associated with increased pulmonary venous resistance. Increased pulmonary vasomotor tone occurs with hypoxic pulmonary vasoconstriction. If associated with increased blood flow, as with exercise at high altitude, one can rapidly understand how high altitude pulmonary edema may occur. Disease states associated with transient massive sympathetic discharge, such as acute cerebral hemorrhage and heroin overdose, produce transient massive increases in pulmonary capillary pressure. Finally, during the reparative phase of acute lung injury, pulmonary fibrosis may occur. Fibrosis is indiscriminate of the vasculature and obstructs all vessels, thus making increased pulmonary vascular resistance a hallmark of end-stage acute lung injury. Persistent pulmonary edema in a patient with late-stage acute respiratory distress syndrome may reflect occult hydrostatic pulmonary edema.

How can the pulmonary capillary pressure be estimated at the bedside?

Bedside assessment of pulmonary capillary pressure is based on visual inspection of the pulmonary artery pressure decay during balloon occlusion using a balloon floatation pulmonary artery catheter [1, 2, 3] (Fig. 1). Ideally the occlusion should be performed during an expiratory hold to avoid the effect of dynamic changes in intrathoracic pressure and lung volume on the pressure curve. After occlusion, one sees a rapid decrease in pressure, followed by a slower pressure decrement approaching the pulmonary artery occlusion pressure (Fig. 1a). When a straight line is drawn tangent to the rapid component, pulmonary capillary pressure can be estimated as the point at which the pressure transient begins to deviate from the rapid portion of the pressure tracing (Fig. 1b). The assessment can be facilitated by the use of a strip chart recorder or a computer sampling of the signal, and by superimposing the occlusion tracing on a nonoccluded one (Fig. 1a). More sophisticated approaches include the use of moving average smoothing of the pressure signal and mathematical curve fitting of the signal (Fig. 1b). The visual inspection method has been thoroughly validated in experimental conditions and gives values very similar to those of the more complex approaches. In the clinical routine a rough estimate of capillary pressure can even be obtained directly from the monitor screen by freezing the pressure trace when measuring the pulmonary artery occlusion pressure.

To assess the risk of pulmonary edema in the presence of pulmonary hypertension and increased transpulmona-

ry pressure gradient it is necessary to estimate the capillary pressure. Importantly, once pulmonary capillary pressure is known, the arterial and venous components of the pulmonary vascular resistance can be calculated as the ratio of their respective pressure gradients (pulmonary artery diastolic to pulmonary capillary and pulmonary capillary to left atrial) to total blood flow. If pulmonary venous resistance is elevated, effective strategies to minimize pulmonary capillary pressure may include reducing total blood flow (hypothermia, sedation, paralysis) and the use of pulmonary vasodilator substances (inhaled nitric oxide, calcium channel blockers, and infusions of potent vasodilators such as prostaglandin E, prostacyclin, nitroglycerin, hydralazine) with appropriate intermittent monitoring of pulmonary capillary pressure to document its reduction.

References

1. Cope DK, Grimbert F, Downey JM, Taylor AE (1992) Pulmonary capillary pressure: a review. Crit Care Med 20:1043–1056
2. Holloway H, Perry M, Downey J, Parker J, Taylor A (1983) Estimation of effective pulmonary capillary pressure in intact lungs. J Appl Physiol 54:846–851
3. Cope DK, Allison RC, Parmentier JL, Miller JN, Taylor AE (1986) Measurement of effective pulmonary capillary pressure using the pressure profile after pulmonary artery occlusion. Crit Care Med 14:16–22
4. Pellett AA, Lord KC, Champagne MS, deBoisblanc BP, Johnson RW, Levitzky MG (2002) Pulmonary capillary pressure during acute lung injury in dogs. Crit Care Med 30:403–409
5. Benzing A, Bräutigam P, Geiger K, Loop T, Beyer U, Moser E (1995) Inhaled nitric oxide reduces pulmonary transvascular albumin flux in patients with acute lung injury. Anesthesiology 83:1153–1161
6. Fang K, Krahmer RL, Rypins EB, Law WR (1996) Starling resistor effects on pulmonary artery occlusion pressure in endotoxin shock provide inaccuracies in left ventricular compliance assessments. Crit Care Med 24:1618–1625