# Pulmonary Pressures

4

Daniel De Backer

## Contents



There is a long-time interest for measuring pulmonary pressures. Already in the early 1900s, physicians attempted to insert catheters into the pulmonary circulation to measure pulmonary pressures [1]. Cournand first introduced in the early 1950s the use of pulmonary artery catheterization [1], but it is only with the introduction of the balloon-tipped pulmonary artery catheter (PAC), also named Swan-Ganz catheter [2], that the measurement of pulmonary artery pressures became popular in cardiovascular medicine and in the intensive care unit.

Pulmonary pressures include the pulmonary artery pressure (PAP) and the pulmonary artery occlusion pressure (PAOP). Beyond the interest of measuring these variables in specific conditions, it is important to understand the physiological role of these variables in cardiovascular and pulmonary medicine. Even if PAC is less used nowadays than at the end of the last century [3], the interest for its measured variables, including PAP and PAOP, remain [4]. Interestingly, several hemodynamic tools may serve as alternative to PAC, providing measurements of PAP and PAOP [5].

It is therefore important to understand the physiology of pulmonary pressures, their determinants, and their potential use in cardiovascular medicine and critical care.

D. De Backer (✉)
Department of Intensive Care, CHIREC Hospitals, Université Libre de Bruxelles, Brussels, Belgium
e-mail: ddebacke@ulb.ac.be


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## 4.1 Physiological Considerations

The PAP is the back pressure of the right ventricle, and is therefore an important determinant of its afterload. The right ventricle difficulty affords an acute increase in PAP, which results from right ventricular dysfunction. Accordingly, the estimation of PAP is important at bedside. PAP can be increased in various conditions, including pulmonary embolism, ARDS, chronic pulmonary, and cardiac diseases.

While systolic, diastolic, and mean PAP can be measured, the mean PAP (PAPmean) is used to define pulmonary hypertension and best reflects the afterload of the right ventricle. The normal PAPmean is around 15 mmHg, but values higher than 20 mmHg define pulmonary hypertension [6]. In acute settings, the right ventricle becomes dysfunctional for values above 35 mmHg.

PAOP reflects the left atrial pressure, which is an important physiologic variable. First, the left atrial pressure is a reflection of the left ventricular preload, and hence an important contributor of cardiac output. Second, an elevated left atrial pressure is observed in left heart diseases. Finally, it is the back pressure of pulmonary bed, and thus may contribute to pulmonary hypertension.

An elevated PAOP contributes to pulmonary edema and is a key determinant of capillary leak, together with vascular permeability. Importantly, the hydrostatic pressure at the capillary level is the true capillary pressure, not PAOP or the left atrial pressure (the measurement of true capillary pressure will be discussed below).

The use of PAOP as left ventricular preload has been criticized. Indeed, as any other "static" measurement of preload, PAOP does not reliably predict fluid responsiveness. Nevertheless, extreme values keep some value to predict fluid responsiveness [7]. More importantly, PAOP can be used as a gauge of the benefit/risk ratio: low values of PAOP are associated with minimal risk during fluid administration while high values are associated with high risk of pulmonary edema, even if fluids increase cardiac output. Accordingly, many physicians use PAOP as a safety measure during fluid challenge [8].

## 4.2 Measurements

### 4.2.1 Invasive Measurement with Pulmonary Artery Catheter

The systolic, diastolic, and mean PAP are measured from the distal end of the pulmonary artery catheter (also called Swan-Ganz catheter [2]). The principle of PAOP measurement is that flow carries the inflated balloon into a distal branch of the pulmonary artery, occluding blood flow distal to the point where the catheter with the inflated balloon wedges. The PAOP is the pressure measured from the distal end of the catheter with its balloon inflated.

Even though misnamed as PAwedge, PAOP is not identical to PAwedge pressure. PAwedge is the pressure obtained when the catheter is wedged without inflating its balloon, corresponding to a more distal branch of pulmonary artery than that obtained with the balloon inflated for PAOP measurement. PAOP also differs from the PA capillary pressure which corresponds to the hydrostatic pressure at the level of pulmonary capillaries and is obtained using a specific calculation algorithm.

PAOP reflects the pressure at the level of relatively large segments of the pulmonary artery bed; hence, this pressure mostly represents the pressure in the large pulmonary veins, and hence the left atrial pressure. PAwedge occludes smaller vessels, closer to capillaries, and hence is closer, but not equivalent, to the capillary pressure.

The true capillary pressure cannot be measured but can be calculated by several ways. The easiest is the computation by the Gaar equation [9]:

$$\text{Pulmonary capillary pressure} = \text{PAOP} + 0.4 \times (\text{PAPmean} - \text{PAOP}).$$

This formula, obtained in experimental conditions using an isolated perfused lung model, is valid only when resistances are normally distributed in the pulmonary arterial and venous tree, which is not true in disease states. Alternatively, the capillary pressure can be calculated from the

decay of the PA curve during balloon inflation [10, 11].

**Practical Advice**
PAOP is not identical to pulmonary capillary pressure, the true hydrostatic force contributing to pulmonary edema. Pulmonary capillary pressure is difficult to measure in clinical practice; accordingly PAOP is the best estimate of the force leading to pulmonary edema.

As the heart is in the chest, measurements of pulmonary pressures are influenced by pleural pressure. To minimize the influence of pleural pressure, the tip of the pulmonary artery catheter should be positioned in the West zone III, where PAP is higher than pulmonary venous pressure, itself higher than the alveolar pressure. Malposition of the catheter in West zone I or II is suggested when respiratory variations in PAOP are larger than respiratory variations in PAP [12]. Measurements should always be obtained at end-expiration.

**Practical Advice**
Pulmonary pressures should always be measured at end-expiration.

It is possible to estimate pulmonary artery transmural pressures during mechanical ventilation. The transmission index is computed as a ratio of PAOP during different phases of breathing to driving pressure: (PAOP end-inspiration – PAOP end-expiration)/(plateau pressure – PEEP).

Transmural PAOP is computed according to following formula: PAOP end-expiration – (transmission index × PEEP) [13]. For the other pressures, the same formula can be used.

**Practical Advice**
Transmural PAOP reflects the pressure contributing to pulmonary edema and left ventricular preload.

### 4.2.2 Noninvasive Measurement (Echocardiography)

Except with echocardiography, there is no other means to determine pulmonary artery pressure at bedside. In addition, the reliability of pulmonary artery pressure with echocardiography has been challenged [14, 15], but these studies were not performed in critically ill patients. In addition, the measurements were often not obtained the same day with the two techniques.

Systolic PAP is measured from the maximal velocity of tricuspid regurgitation, which estimates the systolic transvalvular gradient. Systolic PAP is computed using the simplified Bernoulli equation as 4 × (tricuspid regurgitant jet peak velocity)$^2$ + right atrial pressure (RAP).

Mean and diastolic PAP can be estimated from protodiastolic and telediastolic velocities of pulmonary valve regurgitant flow, respectively, using the same formula as above. This measurement cannot always be obtained in critically ill patients.

In addition, pulmonary hypertension is suggested by a decrease in pulmonary artery acceleration time < 90 ms or by a biphasic pulmonary flow. While a quantitative measurement cannot be obtained, this measurement is helpful to detect pulmonary hypertension when other measurements cannot be easily obtained.

PAOP can be estimated by different indices. The most accurate and easier to obtain in most conditions is the mitral inflow maximal early velocity (E) divided by mitral annulus maximal early velocity (Ea). This E/Ea ratio shows a good relation with invasive PAOP in critically ill patients [16, 17]. Nevertheless, the limits of agreement are too broad for precise numerical estimation so that this measurement is usually used as a semiquantitative assessment (PAOP low/intermediate/high) or for following the individual response to an intervention [18].

**Practical Advice**
Several echocardiographic indices can be used to estimate PAP and PAOP. Even though less precise than the invasive mea-

surements, echocardiography also allows to identify the consequences of pulmonary hypertension on the right ventricle and the source of the elevated PAOP.

**Practical Advice**
When pulmonary hypertension is identified, measuring the gradient between PAP diastolic and PAOP is helpful to discriminate precapillary and postcapillary pulmonary hypertension.

## 4.3 Pulmonary Artery Pressure in Practice

The measurement of pulmonary artery pressure is useful for several purposes: identification of the cause of right ventricular dysfunction, evaluation of the impact of therapeutic interventions, prognostic value in respiratory and cardiovascular patients, *etc*.

When PAH is identified, it is important to understand whether it is due to an increased left atrial pressure (postcapillary PAH) or caused by an increase in the resistance of pulmonary artery vessels (precapillary PAH). To differentiate both, it is useful to compute the gradient between PAPdiastolic and PAOP. A gradient below 3 mmHg indicates postcapillary PAH, while a gradient higher than 5 mmHg indicates precapillary PAH.

When right ventricular dysfunction is identified, the measurement of PAP is useful to orient into its causative mechanism: primary right ventricular cardiac dysfunction (right ventricular infarction of tricuspid disease) is associated with low or normal PAP while right ventricular failure due to an increased afterload (obstructive shock) is associated with an increase in PAP (Fig. 4.1). In addition, the severity of PAH helps to discriminate between an acute mechanism, in which PAPmean is seldom higher than 45 mmHg, and chronic ones, in which very high PAPmean levels can be observed.

**Practical Advice**
Measuring pulmonary pressures is helpful to understand the cause of right ventricular dysfunction.

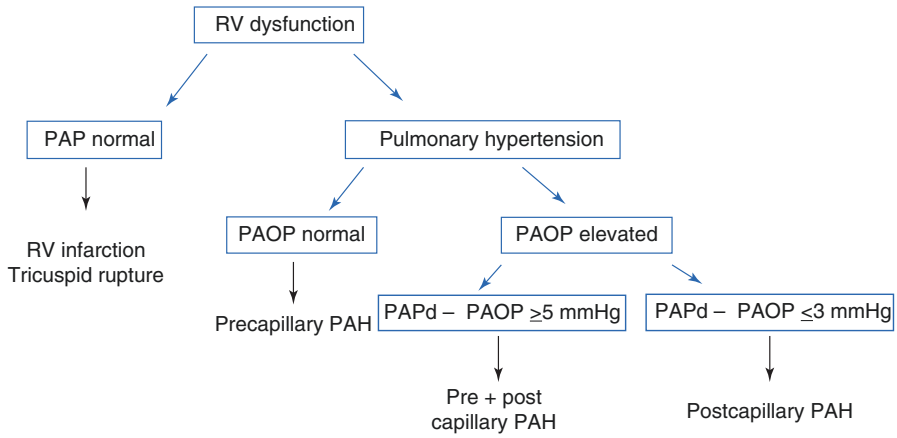


**Fig. 4.1** Usefulness of pulmonary arterial pressure measurement for the interpretation of right ventricular dysfunction. *RV* right ventricle, *PAH* pulmonary hypertension, *PAP* pulmonary artery pressure, *PAPd* diastolic pulmonary artery pressure, *PAOP* pulmonary artery occlusion pressure

The measurement of PAP is also useful to assess the impact of various interventions (*i.e.,* inhaled nitric oxide), helping to find the optimal dose of the agent.

Finally, monitoring PAP is particularly useful in the postoperative management of patients at risk to develop right heart failure during interventions such as heart transplantation, implantation of left ventricular assist device, or cure of mitral stenosis.

## 4.4 Pulmonary Artery Occlusion Pressure in Practice

PAOP can be used for several purposes: evaluation of volume status, evaluation of left ventricular cardiac function, and identification of hydrostatic pulmonary edema.

The evaluation of volume status is not straightforward. The measured PAOP is the result of volume status and systolic/diastolic properties of the left ventricle, the latter potentially influenced by right ventricular function due to ventricular interaction and pericardial constraints. And the measured value is influenced by pleural/pericardial pressure.

**Practical Advice**
PAOP reflects the balance between volume status and left heart function.

Given the complexity and multiplicity of the interactions, a low PAOP is associated with low volume status and excellent cardiac function. An elevated PAOP is associated with either severe hypervolemia, impaired cardiac function, or elevated pleural/pericardial pressure. Additional evaluation, usually with echocardiography, is useful to discriminate between these three factors. Finally, a normal PAOP can represent normovolemia in a patient with normal cardiac function, hypovolemia in a patient with impaired cardiac function, or hypervolemia in a patient with excellent cardiac function. Again, echocardiography may help to differentiate between these possibilities.

The usefulness of PAOP to guide fluid administration has been much debated [19]. Acceptably, the prediction of fluid responsiveness is relatively poor with PAOP, as with any other static measurements (filling pressures and cardiac volumes), as each patient is characterized by its own Starling relationship. Accordingly, only very low values predict fluid responsiveness, and very high values predict the absence of response to fluids.

PAOP can nevertheless be still useful for fluid management. In patients with impaired cardiac function, PAOP better predicts fluid responsiveness than cardiac volumes [20]. More importantly, PAOP can be used as a safety value. An increase in cardiac output is very unlikely in a patient with high PAOP, but also administration of fluids is very unsafe. Given the exponential relationship between PAOP and the left ventricular volume, the administration of fluids in patients with elevated PAOP will sharply raise it further, potentially leading to pulmonary edema. In these conditions, PAOP is often used as a safety value, prompting to stop fluid administration when a predefined PAOP value is reached [8].

**Practical Advice**
PAOP is a poor predictor of fluid responsiveness. However, PAOP is helpful for guiding fluid resuscitation, establishing the benefit/risk profile before fluid administration and serving as a safety variable during fluid administration.

Finally, PAOP is often used to identify hydrostatic pulmonary edema in the presence of lung infiltrates. PAOP differentiates between ARDS and hydrostatic edema, with PAOP values >18 mmHg identifying a significant contribution of hydrostatic edema [21]. It can also be used to identify weaning-associated pulmonary edema [22, 23]. Nevertheless, there are two important limitations for the interpretation of PAOP as a cause of hydrostatic pulmonary edema. First,

PAOP measurements are affected by pleural pressure, and the critical determinant of hydrostatic pulmonary edema is transmural PAOP and not intravascular PAOP. Second, lung edema occurs at a lower PAOP value in the context of increased permeability [9] and may develop at higher values in the context of chronically elevated left atrial pressure. Accordingly, the contribution of hydrostatic pressure to pulmonary edema is progressively increased rather than sharply increasing just above 18 mmHg.

**Practical Advice**
PAOP is helpful to discriminate hydrostatic from nonhydrostatic pulmonary edema.

## 4.5 Conclusions

The measurements of pulmonary artery pressures, including PAOP, are important for patient management. Even though these are less frequently measured than at the end of last century, understanding the physiology of pulmonary pressures, PAOP, and right and left ventricular functions is crucial.

**Keynotes**
- Pulmonary artery pressure is a key determinant of the right ventricular afterload.
- The evaluation of the right ventricular function should take into account PAP measurements.
- When pulmonary hypertension is diagnosed, PAOP should be measured to differentiate pre- and postcapillary hypertension.
- PAOP cannot predict fluid responsiveness. It is nevertheless useful for the guidance of fluid administration (safety limit).
- PAOP is useful to identify the hydrostatic component of pulmonary edema.

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