Review Article

Misinterpretation of pressure measurements from the pulmonary artery catheter

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Pulmonary artery catheters are now widely used for assessment of the cardiovascular system of the critically ill patient. The purpose of this review is to consider the different conditions that complicate and sometimes invalidate the interpretation of pulmonary artery pressure. We will discuss pathologic conditions of the lungs and left side of the heart that can create discrepancies between pulmonary artery and left ventricular end diastolic pressure (LVEDP) and the preload of the left ventricle. We will also examine the interaction between the lungs, the mode of ventilation and pulmonary wedge pressure measurements.

Rationale for the use of pulmonary wedge pressure

The pulmonary artery catheter (PAC) was designed to estimate left sided vascular pressures from catheterization of the right side of the heart. At the end of diastole the aortic and pulmonary valves are closed and the mitral valve is open, creating a common fluid chamber from the pulmonary valve to the aortic valve (Figure 1). At this time, there is no flow in this common chamber; there is no pressure gradient between the different parts of the chamber, assuming there is sufficient time for equilibration (i.e., a long diastolic time), and a low resistance to blood flow (i.e., normal pulmonary vascular resistance). Then the following equation applies at end diastole: PAEDP ≅ PVP ≅ LAP ≅ LVEDP (equation 1). The pulmonary wedge pressure (PWP) is the pressure read on the arteriolar side once the balloon is inflated, thus isolating the tip of the PAC from the upstream arterial pressure. This reflects pulmonary venous pressure (PVP) and by extension, pressures on the left side of the heart. We can assume: PAEDP ≅ PWP ≅ PVP ≅ LAP ≅ LVEDP (equation 2).

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Glossary

PAC – pulmonary artery catheter
PAEDP – pulmonary artery end diastolic pressure
PWP – pulmonary wedge pressure
PVP – pulmonary venous pressure
LAP – left atrial pressure
LVEDP – left ventricular end diastolic pressure
RBBB – right bundle branch block
COPD – chronic obstructive pulmonary disease
LA – left atrium
VSD – ventricular septal defect
LV – left ventricle
LVEDV – left ventricular end diastolic volume
P_A – alveolar pressure
Pa – pulmonary arterial pressure
Pv – pulmonary venous pressure
PEEP – positive end-expiratory pressure
CPAP – continuous positive airway pressure
PaO_2 – partial pressure of oxygen of arterial sample
PwO_2 – partial pressure of oxygen of wedged sample
SVC – superior vena cava
IVC – inferior vena cava
Ppl – pleural pressure
P_{AW} – airway pressure
Cl – lung compliance
Cw – chest wall compliance

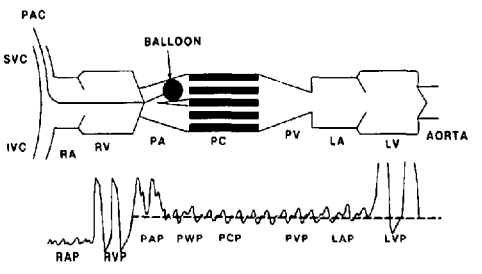

FIGURE 1 Diagrammatic representation of blood chambers between vena cava and aorta demonstrating the common chamber from the pulmonary to aortic valve at the end of diastole. Typical pressure tracings found at the sites are drawn below.

PAEDP and PWP as an approximation of left ventricular preload

How valid is equation 2? If there is disruption or obstruction of the continuous column of blood at any point between the pulmonary artery catheter and the left ventricle, the above relationship will not be accurate.

Discrepancy between PAEDP and PWP

If the time in diastole is not long enough for equilibration, as in a *tachycardia*, an end diatolic gradient will exist between the PAEDP and LVEDP. In a study by Bouchard *et al.* the left atrium was paced from 74/min to 124/min. As heart rate increased, PAEDP increased but LVEDP decreased, thus creating a significant pressure gradient (11 mmHg). With less diastolic filling time, less blood was transferred from the pulmonary vasculature to the left ventricle[2] and the left atrium was contracting against a partially closed mitral value.[3] Lappas *et al.*[4] stated that at heart rates above 115/min, PAEDP was higher than PWP.

A *right bundle branch block* (RBBB) also creates pressure gradients in the pulmonary vasculature.[5] In the presence of RBBB (Figure 2), right ventricular systole is delayed, allowing the pulmonary artery pressure to continue to fall during the *x* descent of the left atrial pressure trace. Then PAEDP is actually lower (may be up to 7 mmHg lower[5]) than the mean LAP. This will only occur when there is a normal pulmonary vascular resistance and thus rapid equilibrium between PAEDP and LAP.

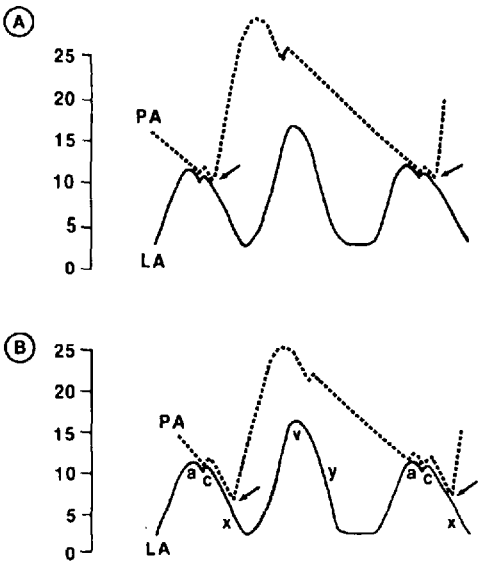

FIGURE 2 A normal left atrial (LA) and pulmonary artery (PA) pressure tracing (A) is compared to tracings with RBBB (B). The "a" wave corresponds to left atrial contraction, the "c" wave to the bulging of the mitral valve in ventricular systole, and the *v* wave to the passive filling of the atrium. Mean LAP is read between *a* and *c* waves and should correspond to LVEDP. The *x* descent represents relaxation of the left atrium, the *v* descent opening of the mitral valve. Note that the delayed right ventricular systole in RBBB is associated with a low PAEDP.

Normally, the pulmonary vasculature offers very little resistance to blood flow; at the end of diastole, flow almost ceases and the PAEDP is an adequate reflection of the LAP. When the *pulmonary vascular resistance increases*, as a result of hypercarbia, alveolar hypoxia, pulmonary embolism, COPD etc, PAEDP will be greater than the LVEDP and may not reflect LVEDP[6] because there isn't enough time for equilibration of pressures between the pulmonary artery and left ventricle during diastole. This pressure gradient will be magnified by tachycardia.[2] After open heart surgery, Mammana *et al.*[7] observed an increase in the mean pulmonary artery pressure associated with a gradient between the wedge and left atrial pressures (PWP > LAP). As a result, they[7] question the accuracy of wedge pressure measurements in assessing left sided pressures in pulmonary hypertension. But PWP remains

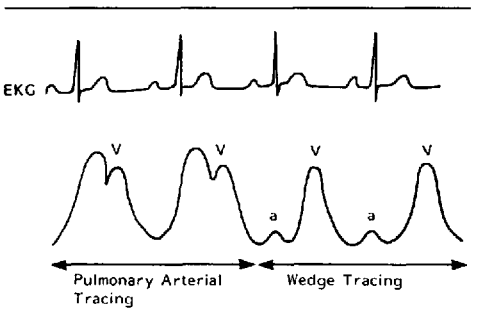

FIGURE 3 Diagrammatic representation of an ECG tracing and synchronous pulmonary artery and wedge pressure tracings indicating the appearance of a large V wave (combination of *c* and *v* waves).

closer to the LAP than the PAEDP, so PWP is preferred over PAEDP when LAP is not available.

Discrepancy between pulmonary venous pressure and left atrial pressure

The *compression* of pulmonary veins by fibrotic or neoplastic processes, although rarely encountered clinically, can cause the PWP and PVP to exceed LAP.[8] In this setting PWP may not reflect LAP.

Discrepancy between LAP and LVEDP

LAP will exceed LVEDP when there is *mitral valve obstruction* to flow in cases such as mitral stenosis,[9] an artificial mitral valve or a myxoma of the left atrium obstructing the mitral orifice.

Mitral regurgitation also creates problems. In the normal left atrium (Figure 2a and 2b) the *c* wave reflects the bulging of the mitral valve into the atrium during early ventricular systole. The *v* wave corresponds to the flow of blood into the atrium against a closed mitral valve at the end of systole.[10] In cases of mitral insufficiency the regurgitant flow of blood causes more prominent *c* and *v* waves, that are usually fused together and defined as large V waves.[11] These can be seen on both the wedge pressure trace and pulmonary artery pressure trace (Figure 3). It is important to recognize the tall V waves on the wedge trace since they can be misinterpreted as a pulmonary artery pressure tracing, stimulating further attempts to wedge the catheter which may lead to a pulmonary artery rupture.[12]

What is the end diastolic pressure of the LV in patients with large V waves? The pressure preceding the large V wave (after the "a" wave (Figure 3) should accurately represent LVEDP. Most often, LVEDP can be assessed from the diastolic PWP in the presence of large V waves. If the mean PWP is used in the presence of large V waves a falsely high assessment of LVEDP will result.

Do large V waves always signify mitral regurgitation? Fuchs *et al.*[11] studied 208 patients with suspected valvular disease. They concluded that mitral regurgitation is the most common cause of large V waves ($\geq$ 10 mmHg). However large V waves are neither highly sensitive, nor specific for severe mitral regurgitation since the predictive value of large V waves in diagnosing significant mitral regurgitation was only 64 per cent. The predictive value of trivial *v* waves (less than 5 mmHg) in excluding significant mitral regurgitation was 62 per cent. A few reports[13,14] seem to associate large V waves with acute mitral regurgitation more often than with chronic mitral regurgitation. However Fuchs postulated that the height of the *v* wave relates to the compliance of the left atrium.[15] By referring to this curve (Figure 4) there are three major factors that determine the height of the *v* wave. First, the shape and position of the left atrial pressure–volume curve; secondly the location on the curve at the beginning of atrial filling (e.g., an increase in LA volume by regurgitant flow will cause only a small increase in LA pressure when the LA is empty (flat portion of the curve) contrasted with a large increase in pressure when the atrium is

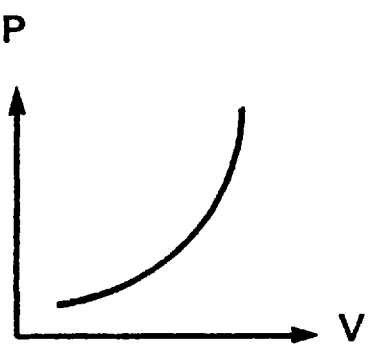

FIGURE 4 Pressure (P) volume (V) relationship in a heart chamber.

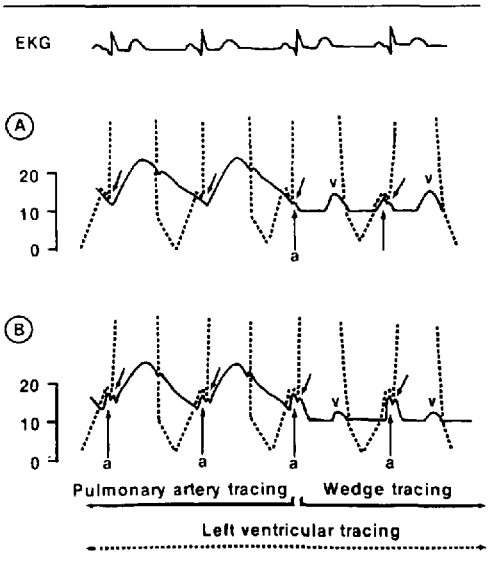

FIGURE 5 A synchronous representation of ECG, pulmonary artery and wedge pressure tracing (—) and left ventricular pressure tracings (---) in cases of a normal (A), and a stiff (B) left ventricle. The small arrow represents LVEDP, read after the peak of the "a" waves which are identified as "a" by the larger arrow.

full); and thirdly the absolute amount of blood entering the atrium during that filling. These factors explain why there is a poor relationship between the height of the *v* wave and the severity of the mitral regurgitation.[16]

Large V waves may also be observed in mitral stenosis when there is poor left atrial compliance.[11] An increase in pulmonary venous inflow as in an acute left to right shunt can also create large V waves.[17] In Fuch's study,[11] four patients had coronary artery disease and congestive heart failure, two patients had a VSD. All six patients had large V waves but there wasn't any other evidence of mitral regurgitation. These facts reemphasize the danger of making a diagnosis of mitral regurgitation based on a solitary finding of large V waves.

Another cause of discrepancy between the LAP and LVEDP is the *non-compliant left ventricle*. In this circumstance, the volume ejected by the left atrium into the relatively stiff left ventricle will cause a significant increase in left ventricular pressures at end diastole. The preload of the left ventricle can be defined as the initial stretch of the fibres of the LV just before ventricular systole, i.e., after the peak of the atrial contraction ("a" wave) (see small arrow, Figure 5a). The "a" wave is tall when left ventricular compliance is decreased (Figure 5b). Unfortunately these "a" waves may not be seen on PAP[1] or PWP[5] tracings, even with a high fidelity recording system. If we can not see the "a" wave on the PWP, we will underestimate the real LVEDP because we may be measuring LVEDP before the "a" wave. In the absence of pulmonary venous obstruction or valvular heart disease, the pressure immediately after the peak of the "a" wave of the wedge trace (although rarely seen) more truly reflects LVEDP, especially in a non-compliant left ventricle and in sinus rhythm.[5]

In the case of *aortic regurgitation*, LVEDP can be higher than LAP or PAEDP. The regurgitant flow back into the left ventricle causes premature closure of the mitral valve and prevents the left atrium and the pulmonary vascular bed from estimating the elevated LVEDP.[5,18]

Wedge pressure measurement for assessment of LV preload

What is the validity of a wedge pressure measurement in assessing the left ventricular preload? The PAC was developed to indirectly measure the preload of the left ventricle. Preload is defined as the stretch of the fibres of the LV just before the beginning of systole, or at the end of diastole. But the PAC measures pressure, not volume. The pressure–volume relationship of the LV is curvilinear (Figure 4), a high PWP or LVEDP may signify high volume for a ventricle with normal compliance or a low volume in a ventricle with decreased compliance. Changes in volume of the LV at end-diastole may produce different changes in pressure depending on the location on the P–V curve. At a low ventricular preload, a large increase in end diastolic volume will be accompanied by only a small increase in the end diastolic pressure. On the other hand, at high levels of ventricular preload (high end-diastolic volume), a small change in the end diastolic volume will be associated with a large variation in end diastolic pressure. This emphasizes the problem that the measurement of end diastolic pressure may not be accurate in measuring the preload (volume) of the ventricle.

Changes in LV compliance may affect stroke volume: e.g., a sudden decrease in stroke volume,

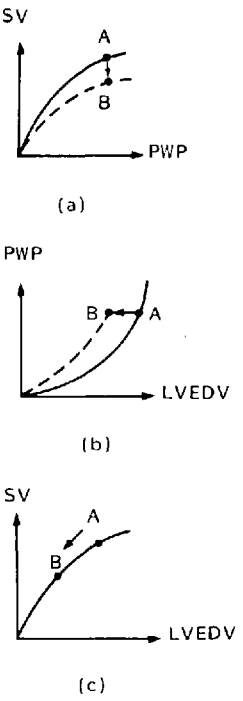

FIGURE 6 Effects of changes in left ventricular compliance on PWP, SV and LVEDV (see text for explanation).

associated with no change in PWP (A to B in Figure 6a), usually will be interpreted as decreased myocardial contractility. However, for the same PWP, a decrease in LVEDV (A to B in Figure 6b) may indicate a less compliant ventricle and decreased preload (A to B in Figure 6c) instead of a decreased contractility. The treatment indicated, as long as PWP is low and pulmonary oedema is not a problem, is to increase preload by volume infusion or reduce left ventricular afterload (and so increase compliance[19]), instead of increasing contractility with inotropes.

In clinical practice, a family of Starling curves can be constructed by plotting the PWP on the *x* axis and the stroke volume of the *y* axis (Figure 6a). This should allow us to follow and adjust treatment to see if there is displacement of the curve to the left, representing a more compliant ventricle (e.g., cardiomyopathy) or a displacement to the right, associated with a stiffer, less compliant ventricle encountered in the ischaemic or hypertrophied ventricle, pericardial disease, or in patients with increased intrathoracic pressure.[8] Practically, however, the few points we can measure may be on different curves. If we assume these few points are on one curve we may be mislead. To be certain, a large number of PWP vs. stroke volume points will need to be plotted.

Thus PWP may not be accurate as a monitor of LV preload and clinical assessment of the patient's response to therapy is vital to optimize treatment (Table).

The relationship between pulmonary pressure measurements and the lungs

Different zones of the lung

If there is collapse of the pulmonary blood column at the level of the pulmonary capillary because of alveolar pressure, the wedge pressure measured will not reflect left heart pressures but will be influenced directly by alveolar pressure.

West described three zones of the lung.[20] Only zone 3 will provide a continuous column of blood between the end of PAC and the LA since the pulmonary arterial pressure (Pa) is greater than the venous pressure (Pv) which is greater than the alveolar pressure (P_A) [Pa > Pv > P_A] (Figure 7). In zone one (P_A > Pa > Pv) or in zone two (Pa > P_A > Pv), the measured wedge pressure won't reflect LAP. The zones of the lung are physiological zones not anatomically described. Although a flow directed PAC will rarely float into zone 1, a zone 3 can be converted into zone 1 by change of position, reduced intravascular volume or application of PEEP. The relationship between the pulmonary artery, alveolar and left atrial pressure will determine whether zone 3 conditions exist.[21] The phase of the respiratory cycle will also determine zone 3 conditions. For a patient breathing spontaneously without CPAP, alveolar pressure will vary between zero (or atmospheric) and negative values during inspiration. The arterial and venous pressures are above atmospheric pressure. There will be continuity of the blood column between the end of PAC and LA and the PWP will reflect LAP. But in the same patient, the same area may convert to zone 2 during expiration as alveolar pressure becomes positive. So the different zones of the lung will

TABLE Summary of pathological conditions limiting the value of PAEDP and PWP in assessment of LVEDP and LVEDV

Level	*Conditions*	*Relations*
Pulmonary vascular bed	• tachycardia	— PAEDP > PWP > LVEDP
	• RBBB	— PAEDP < LAP ≈ LVEDP
	• ↑ PVR	— PAEDP > PWP > LAP
	• compression of pulmonary veins	— PWP ≈ PVP > LAP
LA	• mitral stenosis • artificial mitral valve • myxoma LA	LAP > LVEDP
	• mitral stenosis • mitral regurgitation • congestive heart failure • VSD	large V wave mean PWP > LVEDP diast PWP ~ LVEDP
LV	• non-compliant LV	PWP < LVEDP ΔLVEDP ≠ LVEDV
	• aortic regurgitation	— PWP ≈ LAP < LVEDP

change with the respiratory cycle. Conversely for a mechanically ventilated patient without PEEP, zone 3 during expiration may become zone 2 during positive pressure inspiration. Thus PAC pressure readings should be made at end expiration.

How can we be sure that PAC is in zone 3, the only zone in which the PWP reading is accurate? Many criteria have been used,[22,23] First the phasic wedge pressure tracing should demonstrate the *a* and *v* atrial waveforms. The waveforms should disappear promptly with balloon deflation (to a pulmonary arterial trace) and should return quickly to the wedge tracing after re-inflation. The tracing should not only reflect the ventilatory pressure.[23] Secondly, the mean PWP should be lower or equal to the pulmonary artery diastolic pressure, and lower than the mean pulmonary artery pressure (except in cases of large "V waves"). Thirdly, a continuous flush and the aspiration of blood from the distal port of the PAC should be used to exclude obstruction of the catheter tip by a clot or vessel wall. Fourthly, in the wedge position, highly oxygenated blood can be aspirated ($PwO_2 - PaO_2 \geq 10$ mmHg). Incomplete arterialization of the sample does not mean it is not in a wedged position because the tip may lie in a low ventilation–perfusion area.[24] Therefore, the first three criteria are the most important. If one of these is not verified, the catheter is probably not in zone 3 of the lung and PWP is suspect.

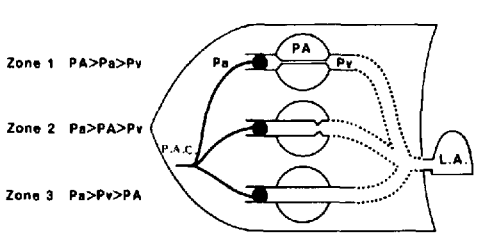

FIGURE 7 See text for a description of the effect of PAC position in the three zones of the lung.

In the supine position, most of the lung is in zone 3 and since PAC's are flow directed, they will go to the area with the greatest flow, which should be zone 3, the dependant zone.[8]

If the tip of PAC is below or at the level of the LA it might be assumed to be in zone 3, unless the patient is seriously hypovolemic or if PEEP > 10 cm H_2O is being applied.[8] An antero-posterior chest radiograph is not useful to locate the tip of PAC in relation to the LA in a supine patient. If any doubt exists that the PAC may not be in zone 3, from the above criteria, a lateral chest radiograph should be obtained.[25,26] The majority of catheter tips are usually located below the SVC or below or at the level of the left atrium.[27] However, the PAC

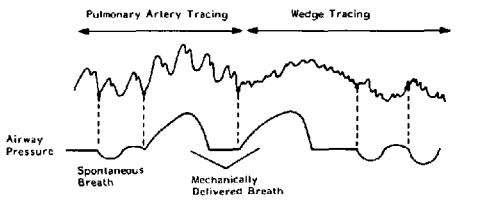

FIGURE 8 Pulmonary artery and wedge pressure tracings during inspiration and expiration of spontaneous and mechanical ventilation. PAC pressure readings should be taken at end expiration (the dotted lines).

position observed on chest radiographs is static and may not indicate the position when the wedge pressure measurement is taken. (Fluroscopy indicates the PAC is constantly moving.) Perhaps chest radiographs should be taken when the PAC is in the wedged position.

Mode of ventilation and PEEP

Whichever mode of ventilation is used, the pulmonary vascular pressures should be measured at end expiration. This assures the pulmonary vascular pressures won't be influenced by changes in pleural pressure. This is true in both spontaneously breathing or mechanically ventilated patients when PEEP is not being used.[28]

A patient's ventilatory pattern should not be changed to measure pulmonary artery pressures. Apnoea is difficult to achieve because it might be impossible for a patient in respiratory distress to hold his breath at the end of expiration with the glottis open, and critically ill patients are not always alert and cooperative. PEEP should be maintained since discontinuing PEEP may cause severe hypoxaemia, increase intrapulmonary shunt and induce a different haemodynamic situation created by a sudden decrease in mean intrathoracic pressure and a rebound hypervolemia in central vessels.[29]

In the presence of important respiratory fluctuations, the readings of pulmonary vascular pressures can be inaccurate especially when they are taken from a digital panel meter printout as there is usually a time delay in the digital printout. Also, the digital printout value is an average over time. Therefore, we may need at least a calibrated screen and at best, a paper printout of wedge and the respiratory waveform. During slow spontaneous respiration, the negative pressure of inspiration will be associated with a progressively more negative wedge pressure. At end expiration, the wedge trace returns to the baseline. So the systolic readout value in a spontaneously breathing patient, without CPAP, will reflect the correct end-expiratory wedge pressure. Conversely in the mechanically ventilated patient, without PEEP, the diastolic readout value will approximate the end-expiratory wedge pressure.

If a patient's respiratory rate exceeds the set ventilator rate many respiratory fluctuations may be seen, and it is usually impossible to approximate the end expiratory value. In these cases, measurement of airway pressures are required. By recording the airway pressure concomitantly with pulmonary pressure traces on a paper printout pulmonary wedge pressure can be determined with precision (Figure 8). End expiration can be determined by airway pressure measurement.[30]

Benumof also studied the usefulness of airway thermistors for detection of end expiration. At times, the airway pressure recordings were unsatisfactory (weak patient effort or a low resistance, high compliance circuit). In his experience, the airway thermistor (modified PAC) was very sensitive and reliable to determine end expiration.[28] The timing of end expiration will improve the accuracy of PWP, but in most patients PWP can be derived at the bedside by direct observation of the respiratory pattern without the application of respiratory timing equipment.

PEEP

The presence of positive pressure at end expiration modifies the interpretation of PWP in two ways. First, by increasing the alveolar pressure at end expiration, it can convert zone 3 of the lung to zone 2 – especially in a hypovolemic patient. Similarly PEEP can convert zone 3 or 2 to zone 1. Secondly, the positive pressure in end expiration will alter pleural pressure and so transmural pulmonary pressure.

We have already explained that we cannot measure PWP by discontinuing or decreasing PEEP because this may cause significant hypoxaemia and create a completely different haemodynamic setting.

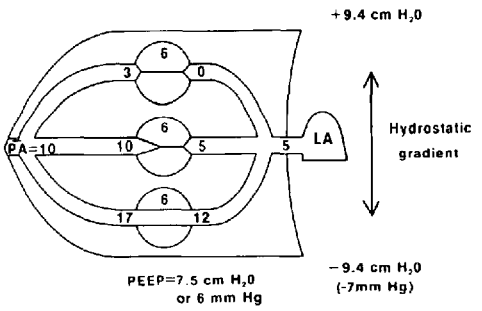

FIGURE 9 Schematic representation of the interaction between the PAC height, left atrial pressure (5 mmHg) and PEEP used (6 mmHg). At or above the LA level extravascular pressure (6 mmHg PEEP) exceeds intravascular capillary pressure and the column of blood is collapsed. In the lowest alveolus intravascular pressure (12 mmHg) exceeds the PEEP level and the vessel is held open allowing accurate measurement of PWP.

Is there a safe level of PEEP, that won't collapse the column of fluid between the end of PAC and LA? To answer this question, we have to consider four factors: the hydrostatic pressure gradient between the PAC tip and the LA, the intravascular volume, the level of PEEP and the compliance of the lung. First is the height of the PAC tip in relation to the LA. If the PAC is below the LA it will probably be in zone 3 even if the patient is on high levels of PEEP (e.g., 30 cm H_2O).[25] If the tip of PAC is above the left atrium PWP may not accurately reflect LAP.

If the intravascular volume is low, LAP will be low[26] (excluding mitral or LV disease). The continuity of blood between the tip of PAC and LA can be lost even if only small levels of PEEP are used. For example, even with levels of 7.5 cm H_2O of PEEP, if hypovolemia is present (LAP < 5 mmHg) the pressure in the alveoli may collapse the microvasculature and the wedge may not accurately reflect the LAP (Figure 9). This situation is not rare in the ICU. In the management of non-cardiogenic pulmonary oedema, increased pulmonary capillary permeability is dealt with by reducing LAP and often because of oxygenation problems high levels of PEEP (> 5 cm H_2O) are applied. This favours the conversion of zone 3 to zone 2 or 1 even if the PAC is at the level of the LA. The lower the tip of PAC in relation to the LA, the more likely the PWP will reflect LAP. Shasby[26] found that 43 per cent of pulmonary artery catheters were lodged in a position where PWP is potentially inaccurate when PEEP is being applied. They suggest a portable lateral chest *x*-ray should be taken when PEEP is being used. If the catheter is higher than the LA, it should be repositioned. This study did take radiographs with the PAC in a wedged position but, unfortunately, did not state the criteria for accuracy of wedge pressure measurements (e.g., presence of atrial *a* and *v* waves, mean PWP should be lower than, or equal to PAEDP, and lower than mean PAP etc., see above). It would be interesting to know how accurate these criteria are in the setting of Shasby's experiment.

The third factor is the level of PEEP. If PEEP levels are high, alveolar pressure is likely to collapse the pulmonary microvasculature. Referring to the following equation:

$$\Delta Ppl = \Delta P_{AW} \frac{Cl}{Cl + Cw}$$

for normal lung compliance (Cl) and chest wall compliance (Cw), the change in pleural pressure (ΔPpl) at the end of passive expiration should equal one half of the change of airway pressure (ΔP_{AW}), since in tidal volume range the compliance of the lung (Cl) equals the compliance of the chest wall (Cw). This states that in normal lungs, one half of the airway pressure change will be transmitted to zone 3 pulmonary vessels. From this equation, it was deduced[8] that if the wedge pressure increases more than half of the applied increment of PEEP, a non-zone 3 condition is likely to exist. Of course, this assumes normal lung. The fact that wedge pressure increases by less than half the increment of PEEP does not assure that zone 3 exists.[8]

The fourth factor affecting the influence of PEEP on PWP is the compliance of the lung. If the lungs are very stiff, airway pressure won't be transmitted to the microvasculature. Zapol *et al.*[31] found that PEEP up to 30 cm of water did not significantly affect vascular pressure measurements in patients with very poor lung compliance.

Many formulae[32,33] have been developed to estimate the effect of PEP on wedge pressure measurement. Since lung compliance and effects of

PEEP vary from patient to patient these formulae are not reliable.[23]

If the PWP meets the accuracy criteria what is the significance of an intravascular wedge pressure of 20 mmHg, if 10 cm H_2O of PEEP is applied to the airway?

Intravascular pressure is usually measured in relation to atmospheric pressure. The true vascular distending pressure is the difference between the intravascular pressure minus the extravascular pressure (pleural pressure). If pleural pressure equals atmospheric pressure at end expiration (no PEEP is used), the true distending pressure would equal the measured intravascular pressure.[34] The change in the intravascular pressure will accurately reflect the change in the transmural distending pressure if the pleural pressure does not change. The extent of airway pressure transmission to the pleural space will depend on airway resistance, lung compliance and thoracic compliance.[35] So the airway pressure at end exhalation should not be used as an accurate reflection of pleural pressure for measurement of transmural pressure.

Pleural pressures have been measured using two methods: inserting catheters into the pleural space and intraoesophageal balloons. Down[35] emphasizes that pleural catheters have potentially serious complications, although these are rare. The esophageal balloon is not accurate in the supine position, but only in the lateral decubitus position when PWP may not be accurate.

Since the measurement of pleural pressure is difficult in patients, transmural wedge pressure is not easily calculated. Transmural pressure is also a problem in cases of obstructive airway disease. Air trapping is well known to occur in these patients because of increased airway resistance. The passive expiratory flow is often too slow to permit evacuation of all the gas volume before the next inspiration. Pleural pressure then is still positive and lung volume is increased at the end of exhalation. This is recognized as the auto PEEP effect.[36] It can be caused by increased lung compliance or airway resistance or if the time for exhalation is shortened. This phenomenon occurs in COPD. Clinically it should be suspected if the exhalatory gas flow continues until it is interrupted by the next inspiration. Pepe *et al.*[36] found that by delaying a ventilator delivered breath and occluding the expiratory port of the ventilator circuit, the airway pressure measured when the next inflation would have begun should reflect the alveolar pressure at end expiration. This would be an accurate measurement of alveolar pressure as long as there is no spontaneous respiratory efforts or no supplemental gas flow added to the system (for nebulization or some IMV circuits). While alveolar pressure is not necessarily an accurate measurement of the change in pleural pressure it may be useful to detect the auto-PEEP effect.

To assess transmural PWP, we need pleural pressure. Clinically this is difficult. It is important to recognize that PEEP will increase intravascular pressures but effects on transmural pressures may be different. Patient management then should be guided partly by intravascular PWP and most importantly by clinical assessment when PEEP is being applied.

Technical problems

Before relying on a pressure measurement to assess left ventricular function, we should know how to calibrate and check the quality of the pressure tracing.[37]

First, the transducer should be zeroed at the level of the fourth interspace in the midaxillary line (ie., mid-left atrium).[8] Secondly, the transducer should be calibrated, usually with two other points different from the zero, to ensure a linear relationship, although the present day transducers usually are linear over a large range. These two points should also be in the range of the pulmonary artery pressure that will be measured. Many recording systems include an internal calibration system which will introduce an electrical signal directly into the amplifier, bypassing the transducer and supposing that the latter is accurate. This is not always true. To avoid that error, the calibration of the system can be verified with a known pressure created by a fluid column (1 mmHg equals 1.34 cm H_2O).

Recording systems may either underdamp or overdamp and so create inaccurate pressure readings[38] (Figure 10). When underdamped, high systolic and low diastolic pressures will be obtained. However, the diastolic pressure is less affected than the systolic.[38] The electronically determined mean pressure will be accurate, although a calculated mean pressure from the systolic and diastolic pressures may be erroneous.[39] An overdamped pressure may prevent recognition of

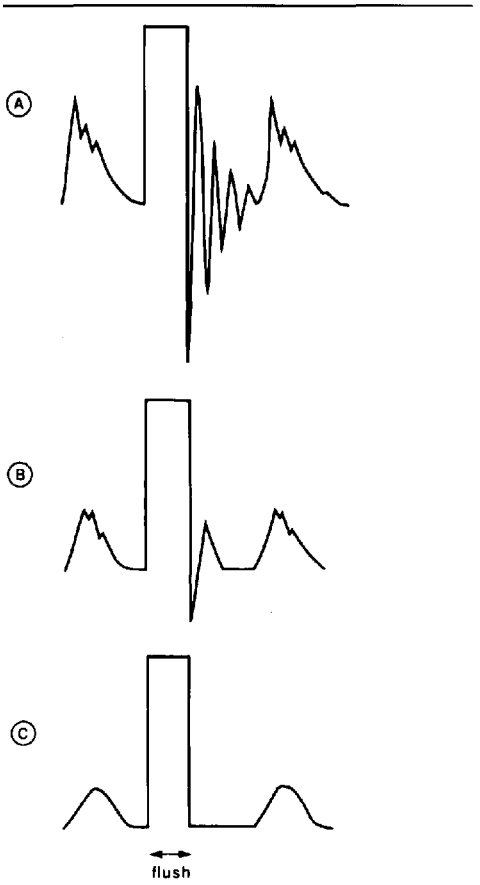

FIGURE 10 Representations of an underdamped (A), optimum damped (B) and overdamped (C) pressure tracing following a flush in early diastole. Note that systolic pulmonary artery pressure is reduced as damping increases.

large V waves on a wedge trace or may lead to misinterpretation of the damped pulmonary arterial trace for a wedge trace.[8]

How can we check the quality of the recording system at the bedside? The waveform inspection itself is inadequate for assessing the dynamic response characteristics.[38] It can easily be done by performing the square wave test by the flushing system during early diastole[38] (Figure 10). This also tests the entire system from the catheter tip to the recording system. Some resonance is warranted, but a quick return to baseline is also needed. If there is absence of oscillation, the system is overdamped, and one should correct the problem by looking for air bubbles (usually large ones) along the recording system (especially at the level of connectors), a narrow or compliant tubing, a clot over the tip of the catheter, or apposition of the catheter against the wall of the pulmonary artery. If, on the other hand, there is overshooting, the system is underdamped. It should be recognized that very small air bubbles (0.05–0.25 ml) introduced into the system can cause an underdamped trace; contrary to the large bubble of air which will cause overdamping.[39] If, after one has flushed these small air bubbles, there is still underdamping, a damping device, accudynamic (designed by Sorenson) can be included in the system. This places an air bubble separated from the circuit by a flexible diaphragm and so increases the damping coefficient without changing the natural frequency.[38]

Gardner[38] recommends testing the dynamic response by flushing at least once each shift and any time a component is changed.

This review summarizes pathologic conditions and clinical settings in which PAC pressures may not estimate left ventricular preload. But PAC pressures are only one part of the haemodynamic assessment. PAC pressures should be correlated with the cardiac output measurement and the response to fluid and other therapy. While these pressure and flows are helpful, we must be assured they are correct and that the clinical assessment retains its critical importance when treating haemodynamic problems.

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