Pitfalls in the interpretation of pulmonary capillary wedge pressure

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Summary

The measurement and interpretation of pulmonary capillary wedge pressure (PCWP) is often vital to the correct management of patients. In this article some pitfalls associated with PCWP measurement are discussed. The physiology of PCWP, preload and the Starling curve are described. PCWP indirectly measures left ventricular end-diastolic volumes; the interpretation of this correlation is altered under certain circumstances, and these are described.

Physiology

Systemic blood pressure is the product of cardiac output and total peripheral resistance (TPR). Therefore an increase in either TPR or cardiac output will cause an increase in blood pressure. The cardiac output is influenced by heart rate and stroke volume, and the latter variable is influenced by preload, ventricular contractility and afterload. Afterload is the tension developed in the wall of the ventricle during systole. An increase in the stroke volume may therefore result from an increase in ventricular preload, from an increase in contractility or from a decrease in the afterload or impedance to ventricular ejection. The sarcomere is the fundamental unit of contraction in cardiac muscle. Starling's law of the heart states that 'the energy of ventricular contraction, however measured, is a function of the length of the muscle fibres prior to contraction'. Therefore, at any given functional state of the ventricle, the force of its contraction is dependent upon the end-diastolic muscle tension or end-diastolic volume, i.e. preload. It follows from the above that the stroke volume or cardiac output produced by any ventricle will depend on its initial fibre length or end-diastolic volume. This has given rise to determination of the so-called Starling curve. On such curves we can predict that an optimum end-diastolic fibre length or preload will give rise to optimum cardiac output. By increasing the end-diastolic ventricular volumes to this point, cardiac output will increase. Any further increase in the end-diastolic ventricular volume will not also further increase cardiac output. A patient is therefore given fluids to increase cardiac output until his preload is optimized. If the cardiac output needs to be increased further, either vasodilators or inotropic agents are then added. Clinically, end-diastolic ventricular volume is represented by left ventricular end-diastolic volume (LVEDV). Until recently this could not be accurately measured directly and therefore had to be indirectly determined by measuring a pressure. The concept behind this is: as LVEDV increases, so left ventricular end-diastolic pressure (LVEDP) increases; as LVEDP increases, so there will be an increased pressure in the left atrium — this will be reflected by an increased pulmonary venous pressure, and since there are no valves in the pulmonary vasculature the increased pulmonary venous pressure will be reflected by an increase in pulmonary capillary wedge pressure (PCWP). This has been documented in animal studies and in normal humans. To summarize, we measure a PCWP via a right-sided pulmonary artery catheter with its balloon blown up. This will give an index of LVEDV. In clinical practice we try to manipulate PCWP to optimize cardiac output, and at the same time to minimize fluid extravasation across the pulmonary vasculature.

It is pertinent to point out that the above statements assume various relationships. They assume that there is a direct relationship between LVEDV and LVEDP. They also assume that there is no obstruction at the mitral valve. The third assumption is that PCWP measurement records the pressure within the pulmonary arteries without the influence of intrathoracic pressures, i.e. transmural pressures. The above three assumptions hold true in normal human subjects.

Pitfalls

Mitral valve obstruction

From the above we can see that an obstruction to flow between the left atrium and the left ventricle (a mitral stenosis or an atrial myxoma) will falsely raise pressures measured proximal to that obstruction. These conditions will result in a higher wedge pressure than would normally occur for the
same end-diastolic ventricular volume if there is no obstruction to flow.

**Mitrval valve incompetence**

Similar to the giant ‘v’ wave that occurs in the neck when tricuspid incompetence is present, a giant ‘v’ wave occurs on the left side of the heart when mitral incompetence is present. This can be reflected on a pulmonary artery wedge pressure tracing as a large wave just after the beginning of systole. To measure wedge pressure accurately in a patient with such a condition this giant ‘v’ wave must be totally ignored and the resultant pressure taken as being indicative of the preload.

**Left ventricular compliance**

Compliance is defined as volume change per unit of pressure change. We assumed that left ventricular volume change would represent a specific pressure change, therefore as LVEDV rises so there would be similar rise in pressure. The left ventricular compliance curve is not a straight line relationship, i.e. a given change in volume will cause a specific change in pressure. On top of this there are various compliance curves for diseased states of the ventricle. A still ventricle will have a higher pressure change for a given volume change which would not be the case with a more compliant ventricle. This is important in interpreting a specific wedge pressure measurement in relation to LVEDVs. The significance of this is related to the Starling curve; as LVEDV increases, cardiac output will increase, and LVEDVs are indirectly measured from a wedge pressure. The following two examples illustrate this: in a patient with a flabby left ventricle, increasing the ‘low’ wedge pressure above the plateau of LVEDVs on the Starling curve will not improve the patient’s cardiac output. The opposite occurs with a stiff ventricle, where a high measured wedge pressure is not indicative of optimum left ventricular volumes; therefore, raising this further may improve cardiac output.

I would like to point out here that although under these circumstances an increased pressure may increase cardiac output, it is the pressure in the pulmonary vasculature per se that determines extravasation of fluid across that area of vasculature, i.e. the higher the pressure, no matter what the volumes are, the more extravasation of fluid across that membrane.

Therefore, for a specific end-diastolic volume, the stiffer the ventricle and the higher the measured wedge pressures, the more chance there is of extravasation of fluid into the lungs, whereas with a more compliant ventricle no matter what the volumes, the lower the pressure will be for any specific volume and therefore the less the chance of fluid flux into the lungs from the left side of the heart. This concept is illustrated by the following equation (a mathematical expression of the Starling equation):

\[ J_v = K_{fc} \left( (P_c - P_f) - \delta (\pi_c - \pi_t) \right) \]

where \( J_v \) = net volume flow, \( K_{fc} \) = filtration coefficient, \( P_c \) = pulmonary microvascular pressure, \( P_f \) = intrastitial pressure, \( \delta \) = the Staverman reflection coefficient, \( \pi_c \) = capillary onotic pressure, and \( \pi_t \) = tissue onotic pressure.

**Intrapleural pressure swings — spontaneous respiration**

Measurement of the pulmonary artery pressures obviously involves catheterization of the pulmonary artery and measurement of pressures within it. This necessitates measurement of a combination of intravascular and intrathoracic pressures (Fig. 1).

**Intrapleural pressure swings — controlled mechanical ventilation**

The pressure changes during the phases of respiration when the patient breathes spontaneously have been described. The concepts are similar when a patient is placed on a respirator. Here the changes in pressure during specific phases differ, i.e.
during inspiration the intrathoracic pressure becomes positive and elastic recoil allows the pressure to go towards normal during the expiratory phase. Again the end of expiration is the phase of no flow between the mouth and the alveoli, and therefore intrathoracic pressures would be nearest to ambient pressure at the end of expiration, when wedge pressures are best measured.

There is even more confusion as regards vascular pressure changes in the patient who is on a ventilator but is breathing spontaneously between cycled ventilator breaths (intermittent mandatory ventilation). The same logic is used with regard to no-flow states and the end of expiration. In other words, no matter what respiratory assistance is being used (controlled mechanical ventilation, positive end-expiratory pressure (PEEP), intermittent mandatory ventilation, etc.), PCWP and PAWP pressure measurements are assessed at end-expiration.

**Digital read-outs**

The measurement of systemic arterial pressures via an intraarterial line and a digital read-out is a generally accepted procedure. The changes in arterial pressures with changes in respiratory phases are minimal, except when pulsat paradoxicus is present. Most monitoring systems used for these digital read-outs are specifically made for arterial pressures. They update every 5 - 10 seconds, and within that time period as an integral part of their mechanism will automatically read the highest pressure as the systolic pressure and lowest pressure as the diastolic pressure. With wave forms such as those obtained in the pulmonary artery (except under normal respiratory excursions) these digital read-outs are totally misleading because they can never interpret end-expiratory pressures. Therefore, except when there are minimal intrathoracic swings in pressure during respiratory cycles, a graphic trace must be used and the digital read-outs ignored. This is the only means of timing the pressure measurements accurately and thus of obtaining end-expiratory pressures. Emphasis must again be placed on the timing of the pressure measurement even with graphic traces because end-expiration is important. To be even more precise, end-expiratory readings should be averaged over three respiratory cycles, ensuring that measurements are made with an open glottis.

A suggested way around the problems of interpreting combined vascular and intrapleural pressures is the use of an oesophageal manometer. Theoretically such a manometer reads intrapleural pressures. These pressures can be subtracted from PCWP measurements and a transmural pressure can therefore be obtained. There are some problems with such procedures. Firstly, in the supine position the weight of the mediastinum and the degree of lung inflation may affect oesophageal pressure measurement, and the exact position of the catheter tip is important if correct oesophageal pressures are to be measured.

**Zones of the lung**

The effects of gravity will influence vascular pressures within the lung. West *et al.* have described various zones in the lung according to the hydrostatic gradients involved (Fig. 3). Zone I is the upper part of the lung where the alveolar pressure is greater than pulmonary arterial pressure, which in turn is greater than pulmonary venous pressure. Lower down in the lung is zone II; here the pulmonary arterial pressure is greater than alveolar pressure, which is greater than pulmonary venous pressure. The lowest part of the lung is termed zone III where the pulmonary arterial pressure is greater than pulmonary venous pressure, which is greater than alveolar pressure. Theoretically, if a catheter is placed in zone I, the pressure measured would be alveolar pressure. There is no correlation between such a pressure measurement and LVEDP. Therefore, to be sure that pressure measured by a pulmonary artery catheter is representative of LVEDP there must be a continuous gradient of fluid between the left ventricle and the tip of such a pulmonary catheter. This most definitely does not occur within zone I, but does occur within zone III. Any intermediary zone will influence pressures to an unknown extent. For this reason some authors suggest taking lateral radiographs to show the tip of the catheter in relation to the left ventricle.

**Fig. 2.** Pulmonary arterial pressures (systolic, diastolic and wedge) during various respiratory manoeuvres: (a) — normal respiratory cycle; (b) — respiratory distress; (c) — controlled mechanical ventilation; (d) — intermittent mandatory ventilation (see text for further details) (PAP = pulmonary artery pressure, PAS = pulmonary arterial systolic pressure, PAD = pulmonary arterial diastolic pressure, PAW and PAWP = pulmonary arterial wedge pressure or PCWP, inh = inspiration, exh = expiration, resp = respirator breath, spont = spontaneous breath).

**Fig. 3.** Zones of the lung. From the above diagram it is apparent that a catheter measuring pressures from zone I would measure alveolar pressure, and a catheter in zone III would measure pulmonary venous pressure (P_a = alveolar pressure; P_a = pulmonary artery pressure; P_v = pulmonary venous pressure).
The higher the alveolar pressure in any specific patient, the greater the discrepancy in the above interpretation of pressure. Also, the lower the venous pressures in the pulmonary artery (hypovolaemia), the less the validity of such measurements will be, because the catheter tip may be measuring alveolar pressures.

High alveolar pressures will occur with PEEP. The effect of PEEP on West et al.’s zones of the lung is as follows: zone I falls into zone II and more zone II areas fall into zone I. In such a patient the lateral chest radiograph becomes more important as regards the position of the pulmonary artery catheter tip in relation to the heart position.21

PEEP

PEEP has other effects on the lung, the pressures within the lung and the interpretation of such pressures. First of all, PEEP increases functional residual capacity. It does this by maintaining a pressure at the end of expiration and therefore a higher mean intrathoracic pressure. This positive pressure will decrease venous return but increase the measured pressures both in the vasculature and in the thorax. Although the measured pressure increases, the transmural pressure (i.e. pressure difference between inside vasculature and outside vasculature) in fact decreases due to diminished venous return. PEEP also increases the pressure against which the right ventricle has to pump, i.e. the right ventricular afterload. This will decrease the right ventricular stroke volume just as an increased systemic afterload will decrease left ventricular stroke volume. Thus, PEEP decreases venous return (right ventricular preload) and increases right ventricular afterload, both of which will decrease right ventricular stroke volume. Depending on how much the right ventricular afterload is increased, the right ventricle may dilate accordingly. This dilatation within a fixed pericardial cavity must impinge on left ventricular filling, which will decrease left ventricular compliance. The decreased left ventricular compliance will be manifested by an increased left ventricular filling pressure for any LVEDV.

The problem of pressure interpretation within the thorax and PEEP is compounded by the fact that for any level of PEEP administered to the mouth there is no measurement of just how much reaches the intrapleural space. This will depend on thoracic wall and lung compliance.

To summarize the vascular effects of PEEP — right ventricular afterload is increased, which may impinge on left ventricular filling pressures, therefore giving a higher wedge pressure for any ventricular volume. Also, venous return is decreased and transmural pressures within the pulmonary vasculature are decreased. The latter problem may be overcome by oesophageal pressure measurement.

Increased right ventricular afterload

The penultimate pitfall of pulmonary artery pressure measurement to be discussed involves increased right ventricular afterload due to any cause. Much of the previously mentioned logic and references apply here too. Most respiratory diseases will increase pulmonary vascular resistance and therefore by definition increase right ventricular afterload. The effects of increased right ventricular afterload have just been discussed, i.e. increased right ventricular afterload will decrease right ventricular stroke volume but increase right ventricular end-diastolic volumes. These increased right ventricular end-diastolic volumes will impinge on the intraventricular septum and therefore decrease left ventricular compliance. This will cause a higher wedge pressure for any LVEDV.

Pericardial restriction—tamponade, effusion and constriction

Either inflation of the pericardial sac or fluid within the sac can result in decreased left ventricular compliance (Fig. 1). This leads to a falsely high wedge pressure reading for any transmural LVEDV.

The preceding discussion covers pitfalls of pulmonary artery pressure measurements that are not always apparent. Other problems common to the measurement of any pressures will still apply to pulmonary artery pressure measurement — in other words accurate transducing, frequency responses, damping, zeroing, etc. should all be taken into account when measuring any pressure within the vascular system.

I would like to thank Dr A. Kark for reviewing the manuscript, Mrs V. L. Rex for typing it, and the Photo-illustration Unit of the University of the Witwatersrand for help in the preparation of the figures.

REFERENCES