RELIABILITY OF PCWP AS AN INDEX FOR LEFT VENTRICULAR PRELOAD

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WHAT IS PRELOAD?

Preload is classically derived from the measurement of end-diastolic fibre length. Each step away from end-diastolic fibre length creates problems.

In substituting end-diastolic volume for fibre length, we assume that the ventricle is a thin-walled sphere, when in fact it is a much more complex structure [2, 7]. Thus when diastolic volume of the ventricle is increased, fibre length is increased, but not to a uniform degree in all parts of the ventricle, because the left ventricle has been shown to be both structurally and functionally non-homogenous.

Because of logistical problems in obtaining a ventricular volume, pressure measurements have been substituted. However, in substituting end-diastolic pressure for fibre length, we must additionally assume that there is a known relationship between pressure and volume. This relationship is not defined, for two reasons. First, the pressure-volume relationship of the left ventricle is curvilinear. Second, the working conditions in terms of the Frank-Starling mechanism change drastically with changes in ventricular distensibility. The same left ventricular end-diastolic pressure (LVEDP) may indicate a high volume when a ventricle has a normal compliance and a low volume when a ventricle has a decreased compliance (fig. 1). The end-diastolic volume is determined by ventricular distensibility (compliance) and the pressure acting to distend the ventricle—the transmural pressure (Ptm). The transmural pressure is the difference between LVEDP and the juxtagardiac pressure. A non-compliant ventricle or one surrounded by an increased intrathoracic pressure, requires a higher than normal intracavity pressure to achieve any specified presystolic volume. Figure 2 gives three alternative interpretations of an increased left

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Fig. 1. Influence of change of ventricular compliance on left ventricular end-diastolic pressure (LVEDP) and left ventricular end-diastolic volume (LVEDV).
VENTRICULAR END-DIASTOLIC PRESSURE: AN INCREASED DISTENSION OF A NORMALLY COMPLIANT VENTRICLE AND A NORMAL JUXTACARDIAC PRESSURE (A); A NORMAL OR DIMINISHED END-DIASTOLIC VOLUME (PRELOAD) OF A NON-COMPLIANT VENTRICLE (B); AND A NORMAL OR DIMINISHED END-DIASTOLIC VENTRICULAR VOLUME (PRELOAD) WITH A HIGH JUXTACARDIAC PRESSURE (C). CHANGES IN VOLUME OF THE LEFT VENTRICLE AT END-DIASTOLE MAY PRODUCE DIFFERENT CHANGES IN PRESSURE DEPENDING ON THE LOCATION ON THE PRESSURE-VOLUME CURVE (FIG. 3). AT A LOW VENTRICULAR PRELOAD, A LARGE INCREASE IN END-DIASTOLIC VOLUME WILL BE ACCOMPANIED BY ONLY A SMALL INCREASE IN END-DIASTOLIC PRESSURE. ON THE OTHER HAND, AT HIGH 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.

IN PRACTICE, LEFT VENTRICULAR END-DIASTOLIC PRESSURE CAN BE USED TO ESTIMATE LEFT-VENTRICULAR END-DIASTOLIC VOLUME ONLY WHEN GOOD APPROXIMATIONS OF VENTRICULAR COMPLIANCE AND JUXTACARDIAC PRESSURES ARE AVAILABLE. HOWEVER, JUXTACARDIAC PRESSURE IS OFTEN DIFFICULT TO ESTIMATE ACCURATELY AND THE VENTRICULAR PRESSURE-VOLUME RELATIONSHIP CAN VARY ABRUPTLY IN RESPONSE TO ISCHAEMIA, ONCOTIC CHANGES AND CHANGES IN TEMPERATURE [8]. OPINIONS ARE STILL DIVIDED ABOUT DRUG INDUCED CHANGES IN COMPLIANCE [4, 5]. THEREFORE, AN INCREASE IN THE LEFT VENTRICULAR END-DIASTOLIC PRESSURE MAY NOT NECESSARILY SIGNIFY FAILURE AND DILATATION OF THE VENTRICLE, AND A NORMAL PRESSURE MAY NOT INDICATE THAT ALL IS WELL. THUS THE MEASUREMENT OF END-DIASTOLIC PRESSURE MAY NOT PROVIDE AN ACCURATE METHOD OF DETERMINING THE PRELOAD OF THE VENTRICLE [9].

CHANGES IN LEFT VENTRICULAR COMPLIANCE MAY AFFECT STROKE VOLUME. FIGURE 4A (A → B) SHOWS A SUDDEN DECREASE IN STROKE VOLUME ASSOCIATED WITH NO CHANGE IN PCWP WHICH, USUALLY, WILL BE INTERPRETED AS BEING THE RESULT OF DECREASED MYOCARDIAL CONTRACTILITY. HOWEVER, IF THE VENTRICLE BECOMES LESS COMPLIANT THERE MAY BE, FOR THE SAME PCWP, A DECREASE IN LVEDV (FIG. 4B: A → B). THUS THE DECREASE IN STROKE VOLUME COULD BE THE RESULT OF A DECREASED PRELOAD (FIG. 4C: A → B) INSTEAD OF A DECREASED CONTRACTILITY.

THE TREATMENT INDICATED, AS LONG AS PCWP 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), INSTEAD OF INCREASING CONTRACTILITY WITH INOTROPES. IT IS APPARENT, THEREFORE, THAT PCWP MAY NOT BE A RELIABLE INDICATOR OF LV PRELOAD. IN CLINICAL PRACTICE IT IS ESSENTIAL TO ASSESS THE PATIENT'S RESPONSE TO THERAPY IN ORDER TO OPTIMIZE TREATMENT.

INTERPRETATION OF PULMONARY ARTERY WEDGE PRESSURE MEASUREMENTS

IT IS GENERALLY ASSUMED THAT, UNDER MOST CIRCUMSTANCES, PCWP ACCURATELY REFLECTS MEAN LEFT ATRIAL PRESSURE. HOWEVER, THIS ASSUMPTION IS CORRECT ONLY IF THE PULMONARY VASCULAR SYSTEM DISTAL TO THE CATHETER TIP (THAT IS THE PULMONARY CAPILLARIES AND
PCWP AS AN INDEX FOR LEFT VENTRICULAR PRELOAD

West and co-workers [13] demonstrated that there is a marked variation in blood flow existing within the human lung as a result of the interrelationships between the alveolar and vascular pressures (fig. 5). In the upper part of the lung (zone 1) the alveolar pressure exceeds both pulmonary arterial and venous pressure, with the
result that pulmonary capillaries are usually closed and no blood flow occurs. This effectively precludes reflection of left atrial pressures. In the central areas of the lung (zone 2) flow is primarily determined by the balance between pulmonary arterial and alveolar pressures only, because in these zones, alveolar pressure commonly exceeds pulmonary venous pressure. Balloon inflation and catheter wedging will, in fact, convert a zone 2 situation to a zone 1 situation by preventing blood flow.

Thus, if the catheter is lodged in either zone 1 or zone 2, PCWP will not reflect mean left atrial pressure, but rather alveolar pressure. In the dependent part of the lung (zone 3) the capillaries remain open, because both pulmonary artery and venous pressures exceed alveolar pressure. As a result there is free communication between the left atrium and pulmonary arteries. Wedge pressure will reflect left atrial pressure accurately only when the catheter tip is in this zone.

Lateral chest x-rays, with the catheter in the wedge position, may be needed to ascertain that the catheter is positioned correctly below the left atrium [12]. Fortunately, most of the lung is in zone 3 when a patient is supine; flow-directed catheters will usually enter zone 3 because most blood is flowing to this area [6]. The incidence of placement in other zones is greater in patients with low vascular pressure resulting from hypovolaemia, or in those with increased intra-alveolar pressures (for example as might be developed during the use of PEEP). However, as long as the catheter tip remains below the left atrium, zone 3 conditions will exist, despite high PEEP. Thus wedge pressures can still provide reasonable accurate estimates of mean left atrial pressures.

VENTILATION AND PCWP

The positive and negative swings in intrathoracic pressures associated with all forms of ventilation influence intraluminal pulmonary vascular pressure. Because intrathoracic pressure is closest to atmospheric pressure during end-expiration, regardless of whether the patient is breathing spontaneously or with the help of mechanical ventilation, all pressures should be measured at this point of the cycle to minimize the influence of intrathoracic pressure swings [10].

Errors in the measurement of wedge pressure during PEEP may be caused by the increase in the alveolar pressure at end-expiration, which increases the size of zones 1 and 2, especially in a hypovolaemic patient, and by the increase in pleural pressure associated with the increase in lung volume. This may decrease the effective transmural pressure (intraluminal pressure minus intrapleural pressure) because the increase in intraluminal pressure may be less than the increase in pleural pressure.

In any patient, the effect of PEEP on intrapleural pressure depends on that patient's lung compliance. Thus in a patient with decreased lung compliance in whom the effect on intrapleural pressure is minimal, intraluminal pressure reflects transmural pressure and actual filling pressure. However, in a patient with a normal or increased lung compliance, intrapleural pressure increases with PEEP and intraluminal pressure will no longer reflect true filling pressures.

The problem caused by PEEP cannot be overcome by measuring PCWP when PEEP is reduced or discontinued, since this would affect venous return and so create a completely different haemodynamic setting [11]. Theoretically it is better to measure transmural filling pressure, which is the difference between intravascular pressure and extravascular pressure (= pleural pressure). Pleural pressure can be measured directly [1] or by an intraoesophageal balloon [3], but this is difficult in clinical practice.

CONCLUSIONS

Many pathological conditions exist in which PCWP may not estimate left ventricular preload. Furthermore, there are many sources of error in the measurement. In view of these limitations it is vital to adopt a more critical attitude in evaluating data derived from the pulmonary artery catheters.

REFERENCES


