

Filling pressures of the heart during anaesthesia

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Summary

The validity of extrapolating central venous pressure (CVP) to left ventricular (LV) filling pressure as gauged from the pulmonary artery wedge pressure (PAWP) was investigated in 9 subjects undergoing lung resection. Correlations existed between CVP and PAWP before, during and after surgery, and between changes in CVP and PAWP during surgery. There was, however, a wide scattering of the data around the regression lines, and an inability of the CVP and changes therein to predict the actual and directional change in PAWP in specific individuals. Caution is therefore advised in assessing the LV filling pressure from the CVP in patients undergoing lung resection during any phase of the peri-operative period.

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End-diastolic filling of the ventricles is an important determinant of ventricular function (Starling's law of the heart).¹ In evaluating right ventricular (RV) function, central venous pressure (CVP) serves as an indicator of RV filling pressure, while for the left ventricle (LV), left atrial pressure (LAP) or LV end-diastolic pressure (LVEDP) may be utilized to quantify LV filling.

CVP measurement is a routine procedure, often used to judge the patient's intravascular volume and guide the administration of fluid. The CVP, combined with a clinical index of cardiac output, is used as an index of LV heart function, on the assumption that there is a correlation between CVP and LAP. However, it has been demonstrated that there is a poor correlation between CVP and LAP in patients with no heart disease² and no correlation between RV and LV filling pressures in patients with heart disease or increased pulmonary artery resistance (PAR).²⁻⁷ The balloon-tipped pulmonary artery (PA) catheter^{8,9} can be used to measure CVP, PA pressure (PAP) and PA wedge pressure (PAWP), and the use of the Swan-Ganz catheter has made bedside evaluation of LV filling pressure possible, based on the acceptance of PAWP as a more accurate index of LAP and LV filling pressure.¹⁰⁻¹⁸

Lung surgery is characterized by a rapidly changing PAR associated with one-lung anaesthesia and/or hypoxia. This problem may be further compounded by elevated resting PAP

and a reduced compliance of the pulmonary vascular bed, both of which may be accentuated by lung resection. The use of a cardiodepressant anaesthetic agent, e.g. enflurane,¹⁹ still further complicates the issue. In the light of these considerations it would appear that the conventional assumptions in the extrapolation of CVP to LVEDP are, in the context of lung surgery, completely invalid.

To examine the effect of the abovementioned combination of circumstances upon the correlation between CVP and PAWP, we collected the relevant data points (CVP, PAWP, PAR and total peripheral resistance (TPR)) from the information gained from a study in which we are at present still evaluating different cardiopulmonary risk factors in patients undergoing lung resection.

It was the aim of this study to evaluate in quantitative terms the significance of these various considerations in the assessment of CVP as an index of LV filling pressure as gauged by PAWP.

Patients and methods

Nine patients with lung disease scheduled to undergo either a lobectomy or pneumonectomy were studied. Apart from pre-operative clinical examination and routine special examinations, the patients underwent extensive lung function tests and were subjected to graded exercise with fully invasive cardiopulmonary monitoring. These studies all formed part of an extensive protocol which is still in progress.

Premedication consisted of papaveretum 0,3 mg/kg and promethazine 0,4 mg/kg, and on arrival in the induction room a 16-gauge venous cannula was introduced into a forearm vein under local anaesthesia; a 20-gauge non-tapered cannula was inserted into the radial artery after demonstration of an effective ulnar artery collateral blood flow (Allen's test), and a Cordis catheter sheath (Cordis Corp., Florida, USA) was inserted into the right internal jugular vein. A flow-directed thermol-dilution catheter (Swan-Ganz (93A131-7F); Edwards Laboratories, Puerto Rico) was introduced into the PA via the right atrium and RV utilizing the pressure trace as a guide for positioning. The PAWP was obtained by inflating the balloon with 1 ml of air, and the CVP was measured continuously thereafter via the proximal port of the catheter.

Zero levels for arterial pressure, PAP and CVP were taken at the mid-axillary line with the patient supine, and at mid-sternal height with the patient on his side during thoracotomy. The transducers used were connected to visual and digital display units, and these systems were regularly checked by means of electronic and manual manometric calibration. Values obtained from the CVP reading (in cm H₂O) were converted to mmHg by multiplying the H₂O measurement by 0,735. Apart from these measurements, cardiac output, cardiac index, stroke volume, stroke index, RV and LV stroke work, TPR and PAR were measured and calculated.

All haemodynamic measurements were made at the end-expiratory phase and no positive end-expiratory pressure was used in this study. Sufficient time was allowed for the CVP level to settle because of the slow response time of this through the Cordis catheter sheath. Cardiac output was

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determined by the thermodilution method. The mean of three values (10 ml 5% dextrose at 0°C) was used for each measurement reported here. The injections were always given by the same person at a constant speed, taking care to start the injection at the end-expiratory phase. The mean standard error of the three cardiac output values, expressed as a percentage of the mean, amounted to 5,2% ($N = 59$).

No effort was made to determine the position of the PA catheter other than ensuring that it was not in the vessel supplying the lobe or the lung to be removed. The reason for this was that the pressure tracings were used in the majority of cases to insert PA catheters and, since we tried to give this short study a clinical and practical application, the usual method of insertion was utilized without checking on the exact position of the catheter radiographically. We are aware of the possible objections in this regard.

Anaesthesia was induced with thiopentone 2 - 3 mg/kg and fentanyl 10 μ g/kg, and pancuronium 0,1 mg/kg was used to paralyse the patient before insertion of a Robertshaw endobronchial tube. All patients were mechanically ventilated with 100% oxygen to an end-expiratory carbon dioxide tension of 4,7 - 5,2%. Enflurane 1,5% was given to all the patients after induction and endobronchial intubation had been completed.

Grouping of data obtained from each patient is detailed below.

Group A

Measurements were taken before administration of enflurane, i.e. after placing of the lines (fractional inspired oxygen concentration 0,21), after a 10-minute period in which the patient was breathing 100% oxygen and after induction and intubation were completed. At the end of the study, in the recovery room, another set of values were obtained with the patient breathing spontaneously and receiving additional oxygen (40%).

Group B

Measurements in this group were taken while the patient was receiving 100% oxygen and 1,5% enflurane. During this period of the study surgery was commenced and completed, and it was also during this period that the patient was subjected to one-lung anaesthesia and that changes in PAP occurred.

Data were collected and stored off-line on magnetic tape and processed by a microcomputer. In the statistical analysis of data the paired *t*-test was used to evaluate the significance of the correlation between the paired CVP and PAWP data. Significance was assumed when $P < 0,05$.

Results and discussion

In Figs 1 and 3 the actual values of CVP and PAWP are correlated, and in Figs 2 and 4 the changes in each measurement from the control (first) reading are plotted. Details of the statistical analysis of the data are given in Table I.

Group A

In this group, i.e. using data from patients not receiving cardiodepressant drugs (that is, before or after the use of enflurane) and in whom surgery was not performed, the CVP correlated significantly with the PAWP. However, when the change in CVP (dCVP) was correlated with the change in PAWP (dPAWP), the correlation was not significant. The wide scatter of results obtained best describes the clinician's dilemma. If a dCVP of between 0 and -2 was considered (Fig. 2), this corresponded to a clinically unacceptable range of dPAWP values (-8 to +3).

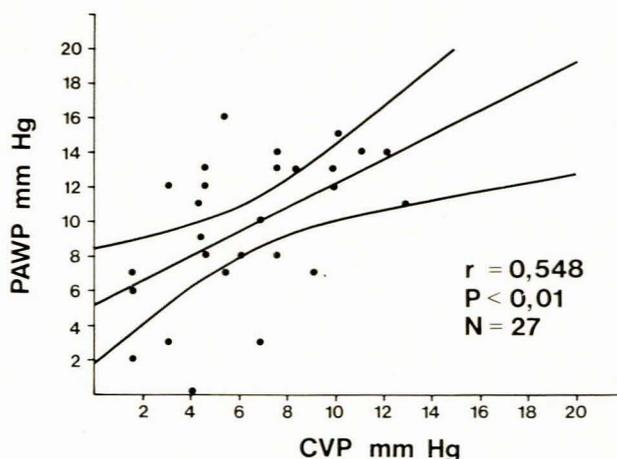


Fig. 1. Correlation between CVP and PAWP in group A (no myocardial depression and no changes in PVR). Regression analysis yielded: $y = 0,710x + 5,136$ which, when tested for linearity, was significant at the 1% level (95% confidence limits are indicated on the graph).

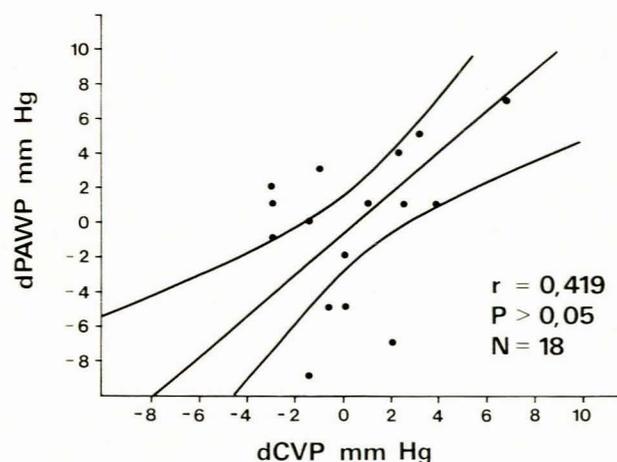


Fig. 2. Correlation between change in CVP (dCVP) and change in PAWP (dPAWP) in group A (see legend to Fig. 1). Regression analysis: $y = 1,036x - 0,848$, which was significantly linear at the 1% level (95% confidence limits are indicated on the graph).

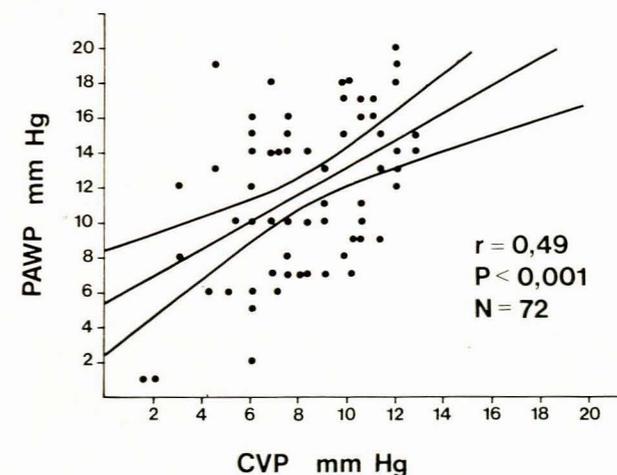


Fig. 3. Correlation between CVP and PAWP in group B (cardio-depressant drug given and subjected to changes in pulmonary vascular resistance). Regression analysis: $y = 0,819x + 4,902$ which was significantly linear at the 0,1% level (95% confidence limits are indicated on the graph).

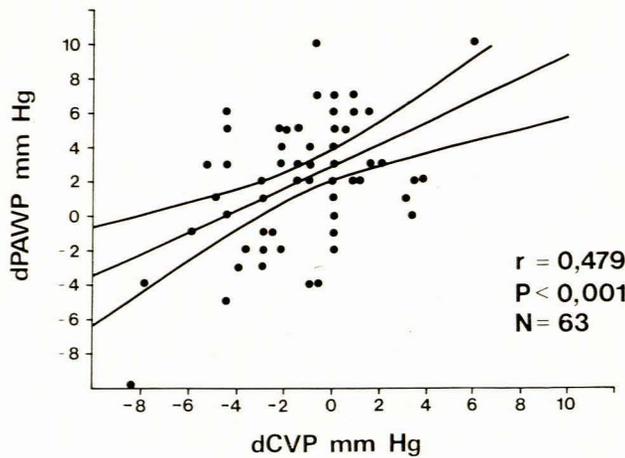


Fig. 4. Correlation between the change in CVP (dCVP) and change in PAWP (dPAWP) in group B patients (see legend to Fig. 3). Regression analysis: $y = 0,645x + 2,647$. This was linear at the 1% level (95% confidence limits are indicated on the graph).

All the CVP and PAWP results obtained were within the normal range, i.e. there was no overt cardiac dysfunction, and the question that has to be addressed is whether this discrepancy between CVP and PAWP is important to the clinician. dCVP is commonly used to give an indication of the LV filling pressure during fluid therapy, and the continuous process of decision-making during fluid replacement is determined by the direction of change of the CVP after a predetermined volume of fluid has been given. From the results depicted in Fig. 2 it would be wrong to assume a specific directional dPAWP from a directional dCVP (no correlation). However, if fluid loading is done according to preset response goals, as has been set out by Weil and Henning²⁰ (Table II), the response ceiling will warn of an approaching plateau with regard to the ratio of intravascular volume (cardiac filling volume) to myocardial function. In this sense CVP measurement may be acceptable since it implies an appropriate LV filling without

knowledge of the exact value of LVEDP. Since this method is goal-orientated, it does not have to take cognizance of LV and RV interaction and changes in the total load of the heart.

However, the correct interpretation of data will rest with the calculation of PAR, TPR, stroke volume, and LV and RV stroke work. For this a correct value for LV filling pressure is essential and, since CVP correlates poorly with PAWP, it would be wrong to use these measurements in the calculation of the appropriate data.

Group B

In this group, i.e. in those in whom a cardiodepressant drug (enflurane) was used and PAR changed due to the hypoxic vasoconstrictor response and lung surgery, the correlation between CVP and PAWP was significant (Fig. 3, Table I). The correlation between dCVP and dPAWP (Fig. 4, Table I) was also significant, but a similar argument applies to the analysis of the results obtained in both group B and group A. A good illustration of the inability of dCVP to predict dPAWP in any given patient can be obtained by considering the zero dCVP in Fig. 4. The latter corresponded to dPAWP values ranging from -4 to +7.

These findings assume specific significance if one considers the changes that occurred in PAR (range of values obtained 85 - 510 dynes-s.cm⁻⁵) and the fact that in all but one set of values a positive dCVP was associated with a positive dPAWP. However, a negative dCVP was associated with a distribution of 38% of dPAWP in a negative direction and the rest (62%) in a positive direction. The data were evaluated, and the breakdown of distribution with regard to PAR is indicated in Fig. 5. Where dPAWP increased although dCVP decreased, most patients had an increased PAR. This observation can be explained by a number of possible events, detailed below.

Under normal circumstances the interventricular septum will be deflected into the RV because of a higher intraventricular pressure in the LV and also because compliance of the LV is less than that of the RV.³ If PAR increases, the right heart has to increase its ejection pressure to overcome the new level of afterload, and this may change the degree of inter-

TABLE I. STATISTICAL ANALYSIS OF THE DATA*

	r	P	y = a (SE) x + b	F (linearity)
Group A				
CVP/PAWP	0,548	< 0,01	y = 0,710 (0,226) x + 5,136	< 0,01
dCVP/dPAWP	0,419	> 0,05	y = 1,036 (0,286) x - 0,848	< 0,01
Group B				
CVP/PAWP	0,49	< 0,001	y = 0,819 (0,174) x + 4,902	< 0,01
dCVP/dPAWP	0,48	< 0,001	y = 0,645 (0,151) x + 2,647	< 0,01

*Group A patients did not receive cardiodepressant drugs and there was no change in pulmonary vascular resistance, while the patients in group B all received enflurane and there were changes in pulmonary vascular resistance. dCVP = change in CVP; dPAWP = change in PAWP.

TABLE II. A DYNAMIC METHOD OF ASSESSING INTRAVASCULAR VOLUME IN PATIENTS WITH HAEMODYNAMIC ABNORMALITIES (ABBREVIATED; AFTER WEIL AND HENNING²⁰)

Assessment and fluid challenge		Response to fluid challenge	
CVP measurement (cm H ₂ O)	Fluid challenge (ml/10 min)	CVP increase (cm H ₂ O)	Response
< 8	200	> 5	Stop
8 - 14	100	2 - 5	Wait 10 min, reassess
≥ 14	50	< 2	Continue infusion challenge

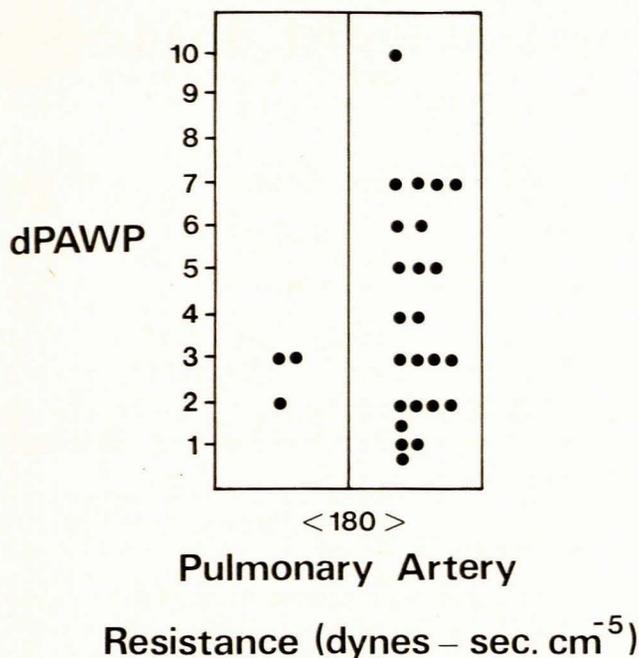


Fig. 5. The distribution of sets of data above and below a pulmonary vascular resistance of 180 dynes - s.cm⁻⁵ where dCVP was negative or zero and dPAWP was positive.

ventricular septum shift towards the RV with a resultant decrease in LV diastolic chamber volume; static diastolic compliance is expressed as dp/dv (changes in pressure per change in volume), and LV compliance has been shown to decrease under these circumstances.²¹

The second hypothesis has been put forward by Forrester *et al.*³ for the heart with a decompensated LV. Enflurane causes myocardial depression resulting in a rightward and downward shift of the filling volume-stroke volume curve. As cardiac output decreases, there is less filling of the right side of the heart (i.e. a zero or negative dCVP), but as the same stroke volume is delivered by the LV as by the RV, LV filling pressure will increase (positive dPAWP) since the RV filling volume-stroke volume curve is, under baseline conditions, to the left of the LV curve (Fig. 6). As the LV curve is displaced to the right and downwards (i.e. depressed), the change in filling pressure will be in the opposite direction.

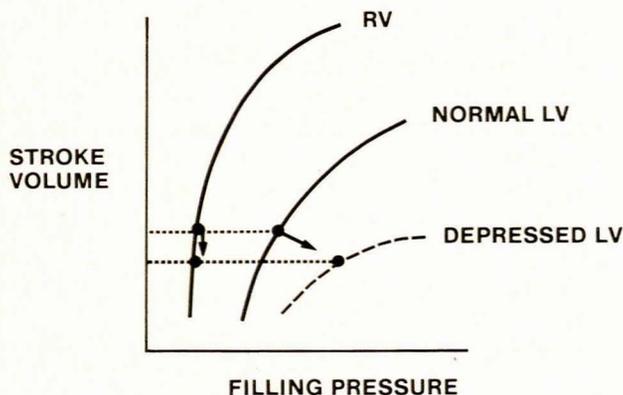


Fig. 6. Effect of a decreased LV function on the filling pressures of the RV and LV. Due to a decrease in cardiac output, the RV filling (CVP) decreases, although LV filling pressures increase. The important aspect of this figure is the demonstration of the opposite directional change which occurs when RV and LV filling are compared (from Forrester *et al.*⁵).

In summary, it may be accepted on statistical grounds that there is a correlation between both the CVP and PAWP and dCVP and dPAWP. There are, however, a number of clinically important qualifications which preclude simple extrapolation of CVP to PAWP:

1. dCVP does not necessarily reflect the dPAWP in a given individual. In the depressed myocardium (i.e. after administration of inhalational anaesthetic agents) and when there is increased PAR, there may be opposing directional changes. This will invalidate any deductions made from RV filling pressures in the assessment of LV filling pressure.

2. After consideration of the values (and the SE) of interception and slopes of the correlation lines in the regression analysis performed, it is clear that the actual value of LV filling pressure in a patient cannot be deduced from CVP measurement. For exact haemodynamic monitoring insertion of a PA catheter may be regarded as essential technology, especially in the circumstances described in this article.

Measurement of heart filling pressures is an indirect method of assessing LV end-diastolic volume. Changes in ventricular compliance constitute a major source of error in the interpretation of ventricular filling volume.²² However, until the current practical and relatively cheap methods of assessing pressures have been replaced by a bedside method for the evaluation of filling volumes, the anaesthetist, like other clinicians, will have to contend with the errors in measurements introduced by the constantly changing physiology of the patient.

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