INFLUENCE OF ELEVATED PULMONARY VASCULAR RESISTANCE ON THE RELATIONSHIP BETWEEN CENTRAL VENOUS PRESSURE AND PULMONARY ARTERY OCCLUDED PRESSURE FOLLOWING CARDIOPULMONARY BYPASS

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FULL EVALUATION of the haemodynamic status of many critically ill surgical patients requires the use of a flow-directed balloon tipped pulmonary artery catheter.¹⁻³ The pulmonary artery occluded pressure (PA_o) is used as a measure of left atrial pressure which is close to the left ventricular end-diastolic pressure (LVEDP)⁴ in patients with no valvular disease. Central venous pressure (CVP), on the other hand, reflects the functional right ventricular filling pressure.

Previous studies have demonstrated a poor correlation between CVP and PA_o in patients with myocardial infarction,⁵ hepatic failure,² peritonitis,² multiple trauma² and chronic cardiac¹ or obstructive lung disease with cor pulmonale.¹ Many of these disease states are associated with marked pulmonary vascular hypertension. We have previously noted an elevation of pulmonary vascular resistance (PVR) in some patients following cardiopulmonary bypass (CBP) for saphenous vein aortocoronary^{6,7} bypass surgery (A-C Bypass).

In the critically ill patient it is important to know when CVP may not be relied on as an index of LVEDP. This information would allow us to select patients that require a Swan-Ganz catheter for measuring PA_o thus avoiding unnecessary exposure of patients to the complications of this technique.

The potential problems in attempting to relate CVP with PA_o are the pulmonary vasculature, biventricular function and ventricular compliance. When the pulmonary vascular resistance (PVR) is high, CVP might be expected not to relate to PA_o . It is also possible that differences in left and right ventricular compliance or function will remove any CVP to PA_o relationship.

This study assessed the clinical validity of CVP monitoring as a reflection of PA_o in unselected patients following CPB. We also investigated the

importance of the elevation of PVR in dissociating the right and left ventricular filling pressures in these patients following surgery.

METHODS

We studied 30 patients undergoing saphenous vein aortocoronary bypass surgery. These patients were randomly selected cases with no pre-operative evidence of congestive heart failure or valvular heart disease. Twenty-eight of the 30 patients were smokers. Following induction of anaesthesia but prior to surgery the following invasive monitoring devices were inserted: a 7 Fr. Swan-Ganz catheter into the pulmonary artery through the basilic vein under pressure trace monitoring and a polyethylene catheter through the internal jugular vein for simultaneous measurement of CVP. Cardiac output (Q) was measured using a thermal indicator. Pulmonary vascular resistance was calculated from the formula $(\bar{PA} - PA_o)/\bar{Q} \times 1332^8$ where $\bar{PA}$ is the mean pulmonary artery pressure (mm Hg), PA_o is the mean pulmonary artery occluded pressure (mm Hg) and $\bar{Q}$ is the mean of three or four cardiac output determinations (ml/sec) made at each study.

These measurements were made in each of 30 patients. The control study was made following the induction of anaesthesia but before operation. The post-operative study was made in the intensive care unit within one hour of the end of the operation and the AM_1 measurement was made the following morning (16–18 hours following operation). All patients were mechanically ventilated at constant tidal volumes (10–15 ml/kg) at all study times.

Placement of the pulmonary artery and CVP catheters was checked on the post-operative chest radiograph. Patients were excluded from the study if either catheter was not appropriately placed in the thoracic cavity.

All patients were treated on the basis of routine clinical assessment as well as CVP and PA_o data. Management included digitalization, fluids and diuretics as indicated clinically. No intentional

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TABLE I
MEAN $\pm$ SEM FOR ALL 24 PATIENTS AT EACH STUDY TIME

	Control	Post-op	AM ₁
CVP (cm H ₂ O)	9.3 $\pm$ 0.8	12.3 $\pm$ 0.9	12.3 $\pm$ 0.6*
PA _O (mm Hg)	11.0 $\pm$ 0.8	11.1 $\pm$ 0.7	11.8 $\pm$ 0.7
Q (l/min)	5.1 $\pm$ 0.3	6.5 $\pm$ 0.6*	7.1 $\pm$ 0.5*
PVR (dyne. sec. cm ⁻⁵)	67.7 $\pm$ 6.9	107.9 $\pm$ 14.5*	72.6 $\pm$ 0.6

*Denotes significantly different from control value.

volume loading or aggressive diuretic therapy was undertaken to exaggerate intravascular pressure changes.

Data was evaluated using an unpaired t-test for groups of unequal numbers and linear regression analysis. If $p < 0.05$ the changes were considered significant.⁹

RESULTS

In six patients CVP catheter position was not ideal for reliable measurements. Thus only 24 patients were included in evaluation of the data. In all patients the pulmonary artery catheter was in the correct position. In three additional patients technical difficulties before completion of the study prevented PVR measurement on at least one occasion.

The mean CVP, $\dot{Q}$, and PVR data for all 24 patients are listed in Table I. There is a significant increase in PVR in this group (Table I) at the post-operative study time.

When the relationship between CVP and PA_O in all 24 cases is examined (Figure 1) there is no significant correlation. Accurate prediction of the PA_O from CVP measurement is impossible. But many of these patients have an increased PVR at the post-operative time (Table I).

In order to examine the importance of the increased PVR in causing the dissociation of right from left ventricular filling pressures, we arbitrarily separated the patients into two groups depending on their PVR. In order to apply a stringent test to this comparison, a HIGH PVR group was chosen in which the change in PVR was greater than 20 dyne sec cm⁻⁵ between measurements. The LOW PVR group is composed of patients in whom the change in PVR between measurements was less than 20 dyne sec cm⁻⁵ (Figure 2). This value (20 dyne sec cm⁻⁵) is less than one standard deviation from the control mean PVR in all patients. Thus, if a change in PVR is responsible for the poor correlation between CVP and PA_O, this relationship (CVP to PA_O) should be improved by

FIGURE 1 Pulmonary artery occluded pressure (PA_O) related to central venous pressure (CVP) measurements in 24 patients at each study time.

considering only patients with a very small change in PVR.

There is a statistically significant although not a good relationship between CVP and PA_O at each study time ($n = 38$, $r = 0.3$, $p < 0.05$) in the HIGH PVR group. In the LOW PVR group there is no positive correlation between CVP and PA_O. Accurate prediction of PA_O from CVP measurement was not possible in either group.

In 15 of 22 measurements made in the HIGH PVR group the direction of change of PA_O (Δ PA_O) was the same as the direction of change of CVP (Δ CVP) (Figure 3). However, in the LOW PVR group, there was no correlation between Δ PA_O and Δ CVP (Figure 4) and in 10 of 15 measurements the direction of change in CVP was opposite to that of PA_O as indicated in quadrants 1 and 3 of Figure 4.

FIGURE 2 Pulmonary vascular resistance (PVR) values in HIGH PVR group and LOW PVR group at each study time.

FIGURE 3 Change in CVP vs change in PA_0 between study times in HIGH PVR group.

DISCUSSION

The clinical usefulness of CVP monitoring assumes a relationship between right and left ventricular function and filling pressures. Civetta and Gabel² have demonstrated that in patients with no cardiopulmonary disease, CVP and PA_0

change in a similar direction in response to volume loading. In patients with coronary artery disease, impaired left ventricular reserve may be present, and Civetta and Gabel showed a very poor correlation between CVP and PA_0 in these patients following surgery.² The potential problems in relating CVP to PA_0 are the pulmonary vasculature, altered right or left ventricular function and altered ventricular compliance.

Ventricular dysfunction associated with cardiopulmonary bypass per se has been reported.¹⁰ The disturbance of normal biventricular function that results may cause the dissociation of right and left side filling pressures noted in our study. However, if the cause of the poor correlation between CVP and PA_0 is an increase in PVR, with an elevation of right ventricular end-diastolic pressure and CVP, then, by eliminating the PVR change in a similar group of patients, one may recover the normal physiological relationship between right and left side filling pressures.

In our study the correlation coefficient between CVP and PA_0 was poor when all patients were considered (Figure 1). However, the predictability of PA_0 from CVP measurement was not improved by considering only those patients with a small change in PVR (LOW PVR group) (Figure 4). Thus it seems that the inconsistent relationship between these two pressures is a result of altered ventricular function or compliance and not altered PVR.

Can we then explain the dissociation between right and left ventricular filling pressures if in-

FIGURE 4 Change in CVP vs change in PA_0 between study times in LOW PVR group.

creased PVR is not a critical factor? In the normal heart, the right ventricular filling pressure (F_1) at any given stroke volume (for example 100 ml) is lower than left ventricular filling pressure (F_2) (Figure 5). A shift to a depressed left ventricular function curve may result in an elevated left ventricular filling pressure (F_3) at a lower stroke volume (for example, 75 ml). If right ventricular function is unchanged, the decreased venous return as a result of reduced left ventricular output causes a slight reduction in right ventricular filling pressure (F_4). Therefore left ventricular dysfunction can result in the shift of CVP and PA₀ in opposite directions. CVP would have fallen from F_1 to F_4 but PA₀ has increased from F_2 to F_3 (Figure 5).

We can also consider a ventricular compliance curve with volume as the independent variable (Figure 6). If volume increases, pressure must rise. Because the thick-walled left ventricle is normally less compliant than the thin-walled right ventricle, any change in left ventricular end-diastolic volume results in a greater change in left ventricular end-diastolic pressure than would occur on the right side for a similar increase in volume. However, if the pressure-volume relationship of the left ventricle is shifted to the left (as it may be with reduced ventricular compliance after cardiopulmonary bypass), the same change in volume (ΔV) would result in a marked rise in pressure. This could also account for part of the discrepancy between CVP and PA₀ found in our patients. In this situation CVP would rise minimally while PA₀ would show a marked increase (Figure 6).

Although the disparity between CVP and PA₀ may be accounted for both by altered left ventricular function and compliance, there may in these patients be an element of impaired right ventricular function as well.

Myocardial infarction may complicate A-C

FIGURE 5 Effect of depressed left ventricular function curve on the relationship between right and left ventricular filling pressures (CVP-PA₀) (see text).

FIGURE 6 Effect of altered left ventricular compliance on relationship between right and left ventricular filling pressures (CVP-PA₀) (see text).

bypass procedures¹¹ and altered left ventricular function and compliance can result.⁵ These direct effects of depressed left ventricular function upon both right and left ventricular filling pressures have been reviewed by Forrester,⁵ to account for the disparity between CVP and PA₀ found after acute myocardial infarction. There is evidence of left ventricular dysfunction following A-C bypass surgery with no evidence of myocardial infarction.¹⁰ Whether this dysfunction is secondary to altered contractility, preload or afterload, it may explain the poor correlation between CVP and PA₀. Right ventricular infarction may also occur following a revascularization procedure and would result in an altered relationship of CVP to PA₀.

Although there is no correlation between the absolute values of CVP and PA₀, CVP would still provide a practical guide to fluid management if it qualitatively reflected changes in PA₀ in each patient. Directional changes in serial CVP measurements often (20/37) reflected similar directional changes in PA₀ (quadrant 2 and 4 of Figures 3 and 4). However, on many occasions (17/37) changes in CVP did not reflect changes in PA₀ (quadrants 1 and 3 of Figures 3 and 4). This was most marked in the LOW PVR group.

In 10/15 LOW PVR group measurements, directional changes in CVP did not reflect PA₀ changes (quadrants 1 and 3 of Figure 4). In the HIGH PVR group, 7/22 measurements of CVP and PA₀, changes were in opposite directions (quadrants 1 and 3 of Figure 3). Thus, the presence of a low PVR in patients did not improve the reliability of CVP as a monitor of left ventricular filling pressure.

In states associated with HIGH PVR, the pul-

monary artery end-diastolic pressure (PAEDP) may exceed the PA_0 . One must consider the possibility that under these conditions the PA_0 measured may not reflect true left atrial pressure. Lappas, *et al.*¹² have demonstrated the reliability of PA_0 as a reflection of left atrial pressure in cardiac surgical patients, some of whom demonstrated HIGH PVR. Rahimtoola, *et al.*¹³ reported an average gradient of 6.7 torr between PAEDP and PA_0 in patients with elevated PVR following acute myocardial infarction. Thus PA_0 would seem to be a reliable guide to left atrial filling pressure even in the presence of increased PVR while PAEDP becomes unreliable under these circumstances.

The passage of a flow-directed pulmonary artery catheter and measurement of PA_0 is a safe bedside procedure.^{14,15} However, several complications of the technique have been recognized. These include arrhythmias,^{16,17} thromboembolism,^{18,19} pulmonary ischaemia,¹⁹ haemoptysis,²¹ pulmonary haemorrhage,²² perforation of the pulmonary artery,¹⁷ intracardiac knotting of the catheter,^{23,24} local infection,⁴ and sepsis.⁴ We disagree with Toussaint *et al.*¹ who stated that balloon tipped pulmonary artery catheters carry no greater risk than CVP catheters. We agree that complications are rare and may be reduced to a minimum by meticulous attention to sterility on insertion and maintenance with the precautions outlined by Foote *et al.*²⁰ In our patients, the only complication resulting from the insertion or maintenance of the Swan-Ganz catheter was frequent transient arrhythmias on insertion. The decision to use this monitoring device must be influenced by the training and experience of the nursing and medical staff involved.

Routine use of these catheters in a critically ill patient is often invaluable in separating the contribution of heart disease and pulmonary disease to the clinical state of the patient.²⁵ Our study demonstrates that unselected patients undergoing routine aortocoronary bypass surgery may be safely and reliably monitored with a Swan-Ganz catheter.

We have previously measured an increase in extravascular lung water in a similar group of patients following bubble oxygenation.⁶ A major factor in the post-perfusion lung syndrome is increased hydrostatic pressure due to impaired myocardial performance following surgery.

Mundth and Austen¹¹ have reviewed the relative influence of various operative risk factors on surgical mortality following A-C Bypass. Patients

with pre-operative congestive heart failure or severe left ventricular dysfunction as demonstrated by haemodynamic parameters have a high operative mortality.¹¹ Our patients had no evidence of congestive heart failure or severe left ventricular dysfunction. This group has a peri-operative mortality of less than three per cent in this centre. Patients in the high risk category (poor left ventricular performance pre-operatively) should be monitored in the post-operative period with left ventricular filling pressure measurements to detect early left ventricular failure and to optimize management.

In our hospital monitoring with Swan-Ganz catheters is not used at present for all patients undergoing A-C bypass. In the low risk group of patients we do not insert pulmonary artery catheters routinely at operation. However, if these patients are not responding to therapy as indicated by clinical examination in the early post-operative period, insertion of a flow-directed balloon tipped catheter is essential for accurate fluid management and precise monitoring.

The question arises, if there is little relationship between CVP and PA_0 , how have clinicians managed patients successfully with CVP monitors? Clearly, repeated clinical examination of peripheral perfusion, urine output and specific gravity, and pulmonary radiographs have contributed to that management. In patients with low cardiorespiratory reserves, CVP will not be adequate to adjust fluid and drug therapy and PA_0 measurement will assist in fine tuning management of high-risk patients.

The one instance where CVP monitoring is a more sensitive index of the progression of the physiological process is in "restrictive heart disease" (cardiac tamponade).²⁶ In this condition, which can occur following cardiac surgery, the "pressure plateau phenomenon"²⁷ results in elevated and near-equal right atrial, PAEDP, and PA_0 pressures. This emphasizes the importance of recording both CVP and PA_0 pressures when these are available. It also emphasizes the fact that right atrial pressure measurement provides valuable information to the clinician who understands the factors that can influence CVP, and also the limitations in assuming a relationship between right and left ventricular filling pressures. Right and left ventricular filling pressures must always be interpreted after clinical assessment of the patient and do not replace clinical evaluation of haemodynamic status.

SUMMARY

This study has demonstrated that CVP measurement is an unreliable index of left ventricular filling pressure in unselected patients undergoing A-C bypass procedures. The influence of altered PVR on the disparity between right and left ventricular filling pressures is not of prime importance. The inconsistent relationship between CVP and PA₀ is primarily a result of ventricular dysfunction and not altered PVR. This suggests that following A-C bypass surgery patients cannot be grouped with respect to altered PVR as the variable which determines whether CVP will be a reliable guide to changes in left atrial pressure.

Routine use of Swan-Ganz catheters would improve the precision of monitoring and fluid administration in all patients following CPB. However, it would also increase the cost and complexity of routine care and add a very low incidence of complications to all patients having cardiac surgery. At present, we monitor high risk patients from the time of surgery and advocate early insertion of Swan-Ganz catheters in low risk patients if they are not responding to appropriate management.

RÉSUMÉ

Cette étude, effectuée chez des malades soumis à un pontage aorto-coronarien, démontre que la mesure de la pression veineuse centrale ne constitue pas un indice fiable de la pression de remplissage ventriculaire gauche. On ne peut imputer à des modifications des résistances vasculaires pulmonaires les discordances entre les pressions droites et gauches; de telles discordances reflètent plutôt une dysfonction ventriculaire. Ceci suggère que les malades ayant subi un pontage aorto-coronarien ne peuvent être classifiés selon les modifications observées des résistances vasculaires pulmonaires afin de déterminer si la pression veineuse centrale peut être utilisée comme guide du remplissage ventriculaire gauche.

L'usage routinier d'un cathéter de Swan Ganz permettrait, après chirurgie coronarienne, un monitoring plus précis et une administration plus rationnelle des liquides. D'autre part, il contribuerait à élever le coût et à augmenter la complexité des soins routiniers tout en ajoutant la possibilité de complications inhérentes à la technique. Il faut dire cependant que l'incidence

de complications liées à un tel monitoring est très basse.

Pour le moment, nous installons des Swan Ganz aux malades à risque élevé, et nous conseillons d'installer un tel cathéter de façon précoce en période post-opératoire, lorsque les malades qui n'en ont pas ne répondent pas bien au traitement habituel.

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