Monitoring Pulmonary Arterial Pressure in Coronary-Artery Disease

Dennis T. Mangano, Ph.D., M.D.*

To delineate the indications for pulmonary arterial pressure monitoring, the relationship between central venous pressure (CVP) and pulmonary capillary wedge pressure (PCWP) was examined in 50 patients with coronary-artery disease and ventricular dysfunction (ejection fractions ranging from 0.26 to 0.84) prior to, during, and after coronary-artery surgery. For each patient, 30 simultaneous measurements of CVP and PCWP were made during a 36-hour period that included the awake state, the anesthetized state with and without surgery, before and after pericardiectomy, before and after cardiopulmonary bypass, and once, four, eight, and 24 hours after operation. At each point, changes in filling pressures were acutely induced by changing body position to alter venous return. The CVPs ranged from 0 to 19 torr, and the PCWPs from 0 to 31 torr.

The CVP and the PCWP correlated well ($r = 0.89$) during all measurement periods for patients who had ejection fractions greater than 0.50 without angiographically demonstrable ventricular dysynergy preoperatively. Changes in CVP ($\Delta$CVP) and PCWP ($\Delta$PCWP) over the 36-hour period also correlated well ($r = 0.94$). Normality (abnormality) of the CVP was predictive of normality (abnormality) of the PCWP for more than 96 per cent of the 450 data points. On the other hand, for patients with ejection fractions less than 0.40 or with dysynergy, the CVP did not correlate with the PCWP ($r = 0.24$), and $\Delta$CVP did not correlate with $\Delta$PCWP ($r = 0.04$). Normality (abnormality) of the CVP was predictive of normality (abnormality) of the PCWP for less than 62 per cent of the 450 data points. This study has defined subclasses of patients with coronary-artery disease for whom pulmonary arterial pressure monitoring is indicated prior to, during, and following coronary-artery surgery. (Key words: Equipment, catheters; Swan-Ganz. Heart: coronary artery disease; myocardial function; vascular pressures. Monitoring, blood pressure; central venous; pulmonary capillary wedge. Surgery, coronary artery.)

Assessment of left ventricular function in patients who are seriously ill or undergoing major surgery is important. To this end, central venous pressure (CVP) monitoring and, more recently, pulmonary capillary wedge pressure (PCWP) monitoring have been incorporated into clinical practice. Since pulmonary-artery catheterization and monitoring may be more time-consuming and costly, and may increase morbidity, evaluation of the efficacy of PCWP monitoring is of practical as well as clinical significance.

Several investigators have addressed this question and have concluded that CVP is an unreliable indicator of PCWP in patients with shock, acute myocardial infarction, hepatic failure, massive trauma, and chronic obstructive pulmonary disease with cor pulmonale. In these studies, the relationship between CVP and PCWP was not examined in detail in individual patients per se, but was examined over entire patient populations. Thus, the conclusions drawn from these studies are influenced by the biologic variation among patients, and may not reflect correlations that exist in individual patients. Furthermore, the relationship between CVP and PCWP was not examined over prolonged periods, during stress, or during various extents of hemodynamic changes. Finally, the severities of left ventricular impairment were not specifically quantified.

This study was performed to delineate the indications for PCWP monitoring by examining the relationship between CVP and PCWP during many periods of hemodynamic change in individual patients with coronary-artery disease and different degrees of ventricular impairment.

Methods

We studied 30 patients admitted for coronary-artery operations to the Veterans Administration Medical Center, San Francisco. Patients who had valvular heart disease or ventricular aneurysms were excluded. All patients had intractable angina. Sixteen had electrocardiographic evidence (Q waves) of previous myocardial infarctions. All patients had 75 per cent or greater stenosis of two or more coronary arteries. Five had histories of left ventricular failure; none had signs or symptoms of left or right ventricular failure when initially examined. Preoperatively, left ventriculography was performed in the single-plane, 30-degree right anterior oblique projection. The left ventriculograms were quantitatively characterized by longitudinal, transverse, and hemiaxial segmental

* It is recognized that pulmonary-artery catheterization can provide other important information (cardiac output, mixed venous blood values). This study has examined the efficacy in terms of its use as a pressure monitor only.
analysis according to the method of Herman et al.8 When two or more segments had less than 20 per cent of normal contraction,8 "dysynergy" was said to be present. Left ventricular dysynergy was found in 14 patients. Ejection fractions were determined using end-systolic and end-diastolic volumes obtained by the area—length method of Kennedy et al.6 Ejection fractions ranged from 0.26 to 0.84 (normal in our laboratory being 0.66 ± 0.06). All patients were cigarette smokers, but none had elevated pulmonary vascular resistance, pulmonary hypertension, or cor pulmonale. All patients were taking medications that included isosorbide and nitroglycerin; 26 were taking propranolol (40 to 480 mg, p.o.; q.d.) and two were taking digoxin. Medications were continued until one hour before the scheduled operations.

All patients were premedicated with morphine sulfate (10 mg, im) and diazepam (10 mg, p.o.). They were anesthetized with morphine sulfate (1.5–3 mg/kg, iv), and diazepam (0.25–0.50 mg/kg, iv). Pancuronium (0.05–0.15 mg/kg, iv) provided muscle relaxation, and ventilation (with 100 per cent oxygen) was controlled. Prior to induction of anesthesia, peripheral venous and radial-artery catheters were placed. A triple-lumen thermodilution pulmonary-artery catheter was inserted via the right internal jugular vein. Simultaneous measurements of CVP and PCWP were recorded on a Gilson polygraph from equisensitive Bell and Howell transducers calibrated with mercury. Before each set of measurements, zero reference point was measured as 5 cm posterior to the sternal angle, with the patient lying supine.

Prior to induction of anesthesia, with the patient in the horizontal position, CVP and PCWP were measured simultaneously at end-expiration. These pressures were then altered by changing body position from horizontal to 30-degree Trendelenburg and to 30-degree reverse-Trendelenburg with the pressure transducers continually referenced 5 cm posterior to the sternal angle in a direction perpendicular to the frontal plane of the chest. Measurements were made 2 min after each position change. These maneuvers were repeated 1) during the awake state; 2) during the anesthetized state prior to surgical incision; 3) prior to pericardiotomy; 4) following pericardiotomy; 5) 5 min and 6) one hour following cardiopulmonary bypass; and 7) one, 8) four, 9) eight, and 10) 24 hours postoperatively in the intensive care unit (ICU). For each patient, 30 simultaneous measurements of CVP and PCWP were made during the 36-hour perioperative period. Prior to bypass, no patient had ischemic changes on lead V5 (modified) of the electrocardiogram. Following bypass, a variety of abnormalities (nodal rhythms, A-V blocks, bundle-branch blocks, ST changes) were evident on the electrocardiograms of all patients. In 29 of 30 patients, these changes were transient and resolved within 12 hours. One patient had an anteroseptal myocardial infarction. The post-bypass and ICU measurements were made at the scheduled times, despite the presence of these transient abnormalities.

Every patient had electrocardiograms and myocardial enzyme evaluations on the first, second, and third postoperative days, and a pyrophosphate scan on the second postoperative day.

For each patient, correlation coefficients were computed for pre-bypass measurements (periods 1–4, above), post-bypass measurements (periods 5 and 6), ICU measurements (periods 7–10), and the entire perioperative measurement period (periods 1–10).

Results

Examination of the correlation data revealed two groups of patients with distinctly different results: Group I, 15 patients with ejection fractions greater than 0.50 who did not have areas of dysynergy assessed during preoperative angiography; and Group II, 15 patients with ejection fractions less than 0.40 or with left ventricular dysynergy. No patients in Group I had a history of left ventricular failure or needed pharmacologic inotropic support during the perioperative period. All patients with histories of left ventricular failure (5) or taking digoxin preoperatively (2) were in Group II. Two patients in Group II needed dopamine intraoperatively, and four needed it postoperatively. In one patient in Group I, an anterior wall myocardial infarction developed during the perioperative period. No patient in Group II had evidence (EKG, myocardial enzymes, pyrophosphate scan) of a perioperative myocardial infarction. No other difference (age, other medications, distribution of coronary-artery disease, aortic crossclamp time, number of vessels bypassed, length of stay in ICU) between Group I and Group II was found by use of the t test for unpaired data.

Ejection fractions for patients in Group I ranged from 0.51 to 0.84. The 30 data points for a typical Group I patient with an ejection fraction of 0.62 are shown in figure 1. For a large range of normal and elevated pressures, the CVP and PCWP correlated well over the 36-hour perioperative period (r = 0.96), and over the individual pre-bypass, post-bypass, and ICU periods. Values for CVP in the normal range (~2 to 6 torr) were accompanied by values for PCWP in the normal range (0 to 12 torr), and abnormally elevated values for CVP were accompanied by abnormally elevated values for PCWP. The regression of CVP on PCWP for the overall period was not signifi-
Fig. 1. Relationship between CVP and PCWP for one patient of Group I. Regression lines for the pre-bypass, post-bypass, and intensive care unit (ICU) periods are shown. The CVP and PCWP correlated well over the pre-bypass, post-bypass and ICU periods. Values for CVP in the normal range (−2 to 6 torr) were accompanied by values for PCWP in the normal range (0 to 12 torr), and abnormally elevated values for CVP were accompanied by abnormally elevated values for PCWP.

significantly different ($P > 0.10$, analysis of covariance) in slope or intercept from the pre-bypass, post-bypass, or ICU regression line.

In addition to the absolute values of CVP and PCWP, changes in these pressures were calculated for each pair of measurements in sequence. For example, in one patient the difference between the first and second CVP measurements was compared with the difference between the first and second PCWP measurements. The difference between the second and third CVP measurements was compared with that for PCWP. By this process a total of 29 data points relating the change in CVP ($\Delta$CVP) to the change in PCWP ($\Delta$PCWP) were obtained for the patient, and for each other patient studied. The results for this patient are shown in figure 2. Change in CVP correlated well with $\Delta$PCWP over all measurement periods.

For each of the 15 patients in Group I, CVP correlated well with PCWP over a large range of filling pressures (fig. 3). The correlation coefficients ranged from 0.72 to 0.99. For 14 of 15 patients in Group I, the slopes of the regression lines ranged from 0.52 to 0.93. For one patient (denoted by an asterisk in fig. 3), the slope was 0.18. Thus, in contrast to the situation found for the other 14 patients, for this patient, large changes in PCWP were not as well estimated by the CVP, even though the CVP and PCWP correlated well. Although the slopes and intercepts of the linear regression lines differed from patient to patient (reflecting biologic variability), little dispersion of data was found about each individual regression line. Similar results were found for $\Delta$CVP and $\Delta$PCWP; the correlation coefficients ranged from 0.86 to 0.98. No significant difference ($P > 0.10$) between the slopes (intercepts) of the pre-bypass, post-bypass, and ICU periods was found in any Group I patient. The distribution of the 450 data points for these 15 patients is shown in figure 4A. Normality or abnormality of the CVP was predictive of normality or abnormality of the PCWP for more than 96 per cent of the 450 data points. (The one patient with a perioperative myocardial infarction maintained a high correlation coefficient throughout the perioperative period. This
Fig. 3. Regression lines and correlation coefficients for the 15 patients in Group I. For each patient, the correlation between CVP and PCWP was high; little dispersion of data is found around each individual regression line. For 14 of the 15 patients, the slopes of the regression lines ranged from 0.52 to 0.93. For one patient (*), the slope was significantly lower (0.18) (see text for further discussion).

patient did not manifest signs of ventricular irritability or failure during the perioperative measurement periods.) The overall correlation coefficient was 0.85 (fig. 3).

The ejection fractions of patients in Group II ranged from 0.26 to 0.54. Seven patients had ejection fractions greater than 0.40, but all had dyssymmetry. The CVP and PCWP were uncorrelated or poorly correlated for each patient (fig. 5), and the dispersion of data about individual regression lines was large. The relationship between the ΔCVP and ΔPCWP over the 36-hour period demonstrated similar results: the correlation coefficients ranged from -0.35 to 0.44, and the slopes of the regression lines ranged from -2.11 to 0.41. Normality (abnormality) of the CVP was predictive of normality (abnormality) of the PCWP for less than 62 per cent of the data points (fig. 4B).

These relationships were examined in Group II over the individual pre-bypass, post-bypass, and ICU periods as well. For nine of 15 patients, the CVP (ΔCVP) remained uncorrelated with the PCWP (ΔPCWP) over each of these periods; no statistically significant difference was found (P > 0.10) between the correlation coefficients for these periods or between the slopes of the regression lines for these periods. However, for six of the 15 patients, significant (P < 0.05) differences were found. The pre-bypass results for these six patients were consistent with those for the other nine patients of Group II: the CVP was uncorrelated with the PCWP. However, following bypass or in the ICU, the relationship between CVP and PCWP improved markedly (fig. 6).

In these patients, examination of the right ventricular function curves (right stroke work vs. CVP) over the 36-hour period revealed that significant right ventricular dysfunction occurred along with left ventricular dysfunction during those periods in which improvement in correlation of CVP and PCWP occurred. In table I, a quantification of these ventricular function curve results is shown. For the nine-patient and the six-patient subgroups, the ratio of left ventricular stroke work index to pulmonary capillary wedge pressure (LVSWI/PCWP) decreased by similar amounts following bypass. However, the ratio of right ventricular stroke work index to central venous pressure (RVSWI/CVP) decreased more markedly in the six-patient group, indicating more significant right ventricular dysfunction. Similar changes were found for the relationship between ΔCVP and ΔPCWP.

![Graph showing regression lines and correlation coefficients](image)

**Table I**

<table>
<thead>
<tr>
<th>Group</th>
<th>N=450</th>
</tr>
</thead>
<tbody>
<tr>
<td>CVP &gt; 6 torr</td>
<td></td>
</tr>
<tr>
<td>3.5%</td>
<td>38.3%</td>
</tr>
<tr>
<td>CVP ≤ 6 torr</td>
<td></td>
</tr>
<tr>
<td>57.6%</td>
<td>0.6%</td>
</tr>
</tbody>
</table>

![Table showing distribution of data points](image)

Fig. 4A(left), distribution of the 450 data points for Group I. Normality (abnormality) of the CVP was predictive of normality (abnormality) of the PCWP for more than 96 per cent of the 450 data points obtained for patients in Group I. For Group II (B, right), normality (abnormality) of the CVP was predictive of normality (abnormality) of the PCWP for less than 62 per cent of the data points.
The relationship between the correlation coefficient and ejection fraction for all patients is shown in figure 6. The correlation coefficients for the three measurement periods are shown. Patients in Group I had correlation coefficients ranging from 0.72 to 0.98 over all measurement periods. Patients in Group II had pre-bypass correlation coefficients below 0.62. For nine patients of Group II, correlation coefficients remained below 0.75 in each measurement period. For the remaining six patients, significant improvement occurred, and the post-bypass or ICU coefficients were greater than 0.75. Similar results were found for the relationship between ΔGVP and ΔPCWP.

**Discussion**

Left ventricular end-diastolic volume (LVEDV), when combined with other cardiovascular measurements, permits an estimate of left ventricular performance. Since volume measurement is difficult in the usual clinical setting, pressure estimates, such as PCWP and CVP, have been used. However, the relationship between PCWP and LVEDV, or between CVP and LVEDV, is unknown for many disease states, and may differ considerably from patient to patient (biologic variability), even in groups without heart disease, because of differences in ventricular and pulmonary compliances.\(^\text{10-14}\) Thus, clinical use of CVP or PCWP is then limited to the relative interpretation of sequential pressure measurements in the same patient.

The efficacy of using CVP vs. PCWP monitoring for assessment of left ventricular filling pressure has been examined for various disease states.\(^\text{4,7,15}\) However, these studies did not examine the correlative relationships between CVP and PCWP in individual patients over many measurement periods during different hemodynamic states. Instead, they focused on the biologic variability between the CVP and PCWP across patient populations. Usually, one or two simultaneous measurements were made in an individual

**Table 1. Ventricular Function Results for Group II**

<table>
<thead>
<tr>
<th>PCWP or CVP</th>
<th>Nine Patients</th>
<th>Intensive Care Unit (ICU)</th>
<th>Six Patients</th>
<th>Post-bypass or ICU</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-bypass</td>
<td>Post-bypass</td>
<td>Pre-bypass</td>
<td>Post-bypass</td>
<td></td>
</tr>
<tr>
<td>LVSWI‡/PCWP</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4.8 (±0.8)</td>
<td>1.8 (±0.4)</td>
<td>2.4 (±0.5)</td>
<td>5.1 (±1.0)</td>
<td>1.9 (±0.4)</td>
</tr>
<tr>
<td>RSWI‡/CVP</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1.9 (±0.3)</td>
<td>1.1 (±0.3)</td>
<td>1.3 (±0.4)</td>
<td>2.1 (±0.6)</td>
<td>0.3 (±0.1)</td>
</tr>
</tbody>
</table>

* All values are means ± SE. The units for all values are g m/m²/ torr. LVSWI = left ventricular stroke work index; PCWP = pulmonary capillary wedge pressure; RVSWI = right ventricular stroke work index; CVP = central venous pressure. ‡ Values were averaged from data derived from the high correlation period (either the post-bypass or the ICU period). † Results were obtained by computing the stroke work-to-filling pressure ratio at each of the 50 data points for each patient. The data were averaged for each period and then averaged over the patient population.
Patient. These measurements were combined with those from other patients, and the collective set was examined. No consistent relationship was found. For example, a CVP of 6 torr might be associated with a PCWP of 8 torr in one patient vs. a PCWP of 14 torr in another patient. Such large biologic variability was found for many disease states, including shock, acute myocardial infarction, hepatic failure, peritonitis, and massive trauma.

Our results for patients in both Group I and Group II confirm this high degree of biologic variability (figs. 3 and 5). For example, CVPs of 5 torr in Group I patients were associated with PCWPs ranging from 3 to 14 torr (fig. 3). Furthermore, calculation of the correlation coefficient and linear regression line for all 450 data points of Group I demonstrates that the CVP and PCWP are uncorrelated (r = 0.21) over the entire patient population, and that the regression line has a large dispersion about it.

However, in Group I, biologic variability was not associated with variability in individual patients; individual correlation coefficients ranged from 0.72 to 0.99. These correlation coefficients are even more remarkable when one considers that hemodynamic changes induced by augmentation of venous return, anesthetic agents, mechanical ventilation, surgical incision, pericardioplexy, hypothermia (28°C on cardiopulmonary bypass), aortic cross-clamping, and even acute myocardial infarction (one patient) did not alter the relationship between CVP and PCWP. Furthermore, this relationship was maintained over a large range of normal and abnormal filling pressures. In contrast, in Group II, a correlation between CVP and PCWP could not be demonstrated for individuals or for the group as a whole (fig. 5). For nine of 15 patients in Group II, CVP and PCWP remained uncorrelated throughout all measurement periods. These patients did not evidence significant pulmonary disease or pulmonary hypertension during this study. Neither low nor high filling pressures, nor the addition of inotropic support, improved the relationship between CVP and PCWP. For the remaining six of the 15 patients, the correlation improved during the post-bypass or ICU period. Although the left ventricular function curve (stroke work vs. CVP) was depressed, the right ventricular function curve (stroke work vs. CVP) was depressed to an even greater extent and shifted to the right. These decreases in right ventricular stroke work at increased CVP indicated the occurrence of marked right ventricular dysfunction. In the face of left ventricular dysfunction, the addition of right ventricular dysfunction may cause filling pressure equalization across the interventricular septum and result in an improved correlation between CVP and PCWP. It is interesting that this occurred despite prior pericardiotomy.

Thus, the results of this study differ from the results of previous studies for two reasons. First, the relationship between CVP and PCWP was examined in individual patients; second, patients were subgrouped with respect to the severity of left ventricular impairment. Had previous studies examined the relationships in a similar manner, they too might have demonstrated a significant correlation of CVP and PCWP in patients with "reasonable" myocardial function. The presence of a strong correlating relationship between CVP and PCWP in Group II implies that CVP monitoring may be sufficient and
PCWP monitoring unnecessary in this group of patients. In contrast, PCWP monitoring would appear to be important, and CVP monitoring of little value, for assessing left ventricular filling pressures of patients in Group II.

The author thanks members of the Departments of Anesthesia, Cardiology, and Cardiac Surgery of the University of California, San Francisco, for their efforts and cooperation during the conduct of this study.

References

10. Falicov RE, Resnekov L: Relationship of the pulmonary artery end-diastolic pressure to the left ventricular end-diastolic and mean filling pressures in patients with and without left ventricular dysfunction. Circulation 42:65–73, 1970
15. Swan HJC: Central venous pressure monitoring is an outdated procedure of limited practical value. Controversy in Internal Medicine, II. Edited by FJ Ingelfinger, RV Eberi, M Finland, et al. Philadelphia, W. B. Saunders, 1974, pp 185–193