Evaluation of Pulmonary Arterial End-Diastolic Pressure as an Indirect Estimate of Left Atrial Mean Pressure


SUMMARY
Simultaneously recorded values for mean left atrial pressure (LAMP) and end-diastolic pulmonary arterial pressure (PAEDP) were compared in 28 patients with acute or chronic cardiac disease. Atrial pressure was varied by alterations in blood volume or the administration of inotropic agents. One hundred twenty paired values were divided into three groups according to the pulmonary vascular resistance which was calculated on each occasion. Although LAMP and PAEDP were shown to correlate significantly at all levels of pulmonary vascular resistance, worthwhile estimates of LAMP could only be obtained from PAEDP when the pulmonary vascular resistance was normal.

Additional Indexing Words:
Heart rate Pulmonary vascular resistance Stroke output

It is widely accepted that the Frank-Starling mechanism applies to the intact human heart and indices of performance are frequently related to end-diastolic pressure in the ventricle. The technical and ethical problems of obtaining direct measurements of left ventricular pressure in the acutely sick have led to the use of end-diastolic pulmonary arterial pressure (PAEDP) as an indirect index of left ventricular end-diastolic pressure, although Bouchard and associates have shown that in patients with chronic left ventricular disease left ventricular end-diastolic pressure and PAEDP can differ considerably and vary independently. If the heart is normal, mean left atrial pressure (LAMP) is little different from left ventricular end-diastolic pressure, but it can be appreciably different in the presence of left ventricular disease. However, the relationship between mean atrial pressure and stroke work, which then reflects the characteristics of both atrial and ventricular performance may be of greater therapeutic relevance in severe heart failure than the assessment of ventricular performance alone.

The advantages of obtaining information about the behavior of the left heart from the relatively simple bedside procedure of right heart catheterization using flow-guided catheters (which do not wedge) prompted examination of the relationship between PAEDP and LAMP in an attempt to determine under what circumstances PAEDP can be safely assumed to reflect LAMP.

Methods
Twenty-eight patients with acute or chronic heart disease were studied. Those with chronic disease, congenital (four patients) or valvular (18) were studied following cardiac surgery; the remainder were studied after acute myocardial infarction (two patients) or pulmonary embolism (four patients).

Left atrial pressure was measured through a fine Teflon catheter (ID, 0.4 mm) which was placed in situ during open-heart surgery or
introduced transseptally via the right internal jugular vein. This latter technic, which will be reported separately, has been developed to allow transseptal puncture to be performed at the bedside in orthopneic, hypotensive patients. During evaluation of the method, it has only been employed in patients in whom it was judged that direct measurement of left atrial pressure was essential to their subsequent management.

Pulmonary arterial pressure was measured through a flow-guided nylon catheter (ID, 0.5 mm). This was also used to record cardiac output by the thermal dilution technic, each value being the mean of at least three estimations.

All pressure measurements were related to the sternal angle and were made using Statham P23Db transducers and an Elema-Schonander recorder. The frequency response for the pulmonary arterial catheter system is 100% to 10 Hz and diminishes to 50% at 20 Hz without resonance. For the left atrial catheter system, the frequency response is 100% to 20 Hz. Mean pressures were obtained electronically.

Pulmonary vascular resistance was calculated from the formula:

\[
PVR = \frac{PAMP - LAMP}{Q}
\]

where PVR = pulmonary vascular resistance (units)

PAMP = mean pulmonary arterial pressure (mm Hg)

LAMP = mean left atrial pressure (mm Hg)

Q = cardiac output (L/min).

Simultaneously recorded values for end-expiratory LAMP and PAEDP were selected, covering changes in atrial pressure produced by acute alterations in blood volume (eight subjects), the administration of isoproterenol (isoprenaline) or epinephrine (adrenalin) (five subjects) or both change in volume and inotropic drugs (13 subjects). On each occasion, cardiac output was determined immediately after these pressure records had been obtained, and PAMP, LAMP and heart rate were recorded while the output measurements were made.

Results

One hundred twenty paired values of LAMP and PAEDP were selected to provide 40 pairs in each of three groups, distinguished
PULMONARY ARTERIAL END-DIASTOLIC PRESSURE

77

by the level of pulmonary vascular resistance as follows:

Group A, nine subjects: PVR normal, \( \leq 2 \) units (range, 0.9 to 2.0; mean, 1.6; \( \text{sd}, 0.27 \))

Group B, 11 subjects: PVR 2.1 to 3.9 units (range, 2.1 to 3.9; mean, 2.8; \( \text{sd}, 0.50 \))

Group C, 15 subjects: PVR \( \geq 4 \) units (range, 4.0 to 12.5; mean, 6.2; \( \text{sd}, 2.16 \)).

Of the 11 subjects in group B, four appear solely in this group; four are also represented in group A and three in group C.

Correlation between the paired values is shown graphically for the three groups in figures 1 to 3.

Correlation coefficient, regression equation, and standard error for the three groups are as follows:

Group A: \( r = 0.9471 \)

\[ y = 0.9109x - 0.708 \]

where \( y = \) LAMP, and \( x = \) PAEDP

\( \text{se} = 1.213 \)

Group B: \( r = 0.8718 \)

\[ y = 1.132x - 5.45 \]

\( \text{se} = 2.805 \)

Group C: \( r = 0.761 \)

\[ y = 0.6572x - 3.68 \]

\( \text{se} = 4.028 \)

Values for heart rate in the three groups are:

Group A: mean heart rate, 91 beats/min (range, 68 to 121; \( \text{sd}, 13.4 \))

Group B: mean heart rate, 87 beats/min (range, 62 to 130; \( \text{sd}, 19.7 \))

Group C: mean heart rate, 96 beats/min (range, 57 to 120; \( \text{sd}, 18.5 \))

Discussion

Calculations of pulmonary vascular resistance require the simultaneous and accurate measurement of both cardiac output and the drop in mean pressure across the pulmonary circulation.

Although the frequency responses of the catheter systems used in this study are less than that of conventional cardiac catheters, a linear response to 10 Hz and no resonant frequency is sufficient for intravascular pressure recording.\(^{10, 11}\)

The thermal-dilution technic which was used to measure cardiac output enables multiple determinations to be performed rapidly because there is no detectable recirculation of the thermal indicator, and repeated injections can be given with safety. On each occasion, at least three thermal-dilution curves were recorded, and the mean values were used for the calculation of pulmonary vascular resistance. In some subjects, particularly those with atrial fibrillation and therefore with varying stroke outputs, the difference between successive values for cardiac output exceeded 0.5 L/min. When this occurred, six or more thermal-dilution curves were recorded in rapid succession, and the mean value was calculated. Even when the circulation time is prolonged, six thermal-dilution curves can be recorded in 5 min.

In acutely sick patients, steady-state conditions are difficult to achieve. In this study, observations were made at least 15 min after any change which had been induced, and mean pressures were recorded during the inscription of the thermal-dilution curves.

The circulatory changes which were applied (alteration of left atrial pressure, infusion of acid solutions, action of isoprenaline) are known to alter pulmonary vascular resistance in man,\(^{12}\) and the calculated value for pulmonary vascular resistance in any individual varied with the experimental conditions. The levels of pulmonary vascular resistance which were used to define the three groups were chosen arbitrarily, and seven subjects appear in two adjacent groups as a result of the changes which were induced.

A highly significant correlation between LAMP and PAEDP \((P < 0.001)\) could be demonstrated at all levels of pulmonary vascular resistance, but there were marked differences between the individual paired values in groups B and C. These findings are reflected in the regression equations, and the figures for correlation coefficient and standard error in the three groups show that the estimation of LAMP from PAEDP is unlikely to be accurate when the pulmonary vascular resistance is abnormally elevated.

The heart rate might be expected to influence the relationship between LAMP and
PAEDP because a tachycardia would decrease the time available for the establishment of equilibrium between these two pressures. In the three groups reported here, the highest mean heart rate occurred in group C in which there was least correlation between LAMP and PAEDP. However, there was no significant difference ($P > 0.05$) from the mean heart rate in group A in which correlation was excellent. The range of heart rates in the two groups was also similar (68 to 121 in group A and 57 to 120 in group C).

These results suggest that worthwhile estimates of LAMP from PAEDP can be obtained only when the pulmonary vascular resistance is normal. When the pulmonary vascular resistance is abnormal, PAEDP does not provide an accurate estimate of LAMP, although changes in PAEDP are associated with changes in LAMP in the same direction but of differing magnitude.

The relationship between stroke output or stroke work and changes in PAEDP may give some guide to the performance of the left heart, particularly in clinical situations where the pulmonary vascular resistance is unlikely to be raised (namely, no history of chronic cardiorespiratory disease, normal arterial gas tensions, and no evidence of pulmonary embolism). However, the findings presented here and the uncertainty of all methods of assessing pulmonary vascular resistance, emphasize the desirability of direct measurement of pressure in the left atrium or ventricle, even in the acutely sick.

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Circulation. 1970;42:75-78
doi: 10.1161/01.CIR.42.1.75
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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