Evaluation of Pulmonary Arterial End-Diastolic Pressure as an Estimate of Left Ventricular End-Diastolic Pressure in Patients with Normal and Abnormal Left Ventricular Performance

By Richard J. Bouchard, M.D., James H. Gault, M.D., and John Ross, Jr., M.D.

SUMMARY

It has been suggested that the pulmonary arterial end-diastolic pressure (EDP) may accurately reflect the level of left ventricular EDP, and therefore be useful in the continuous monitoring of left ventricular EDP in acutely ill patients. Accordingly, pulmonary arterial pressure was recorded simultaneously with left ventricular pressure in 24 patients with normal left ventricular function and in 26 patients with left ventricular myocardial disease and elevated EDP (range 13 to 38 mm Hg; average 22 mm Hg). In patients with normal left ventricular function, the EDPs in the left ventricle and pulmonary artery were equal (range 5 to 12 mm Hg; average 8 mm Hg; maximum difference ± 4 mm Hg). In contrast, in 20 of the patients with impaired left ventricular function, left ventricular EDP was consistently higher than pulmonary arterial EDP, exceeding the pulmonary arterial EDP by 2 to 21 mm Hg (average 8 mm Hg); in 12 of these 20 patients, the pulmonary arterial EDP was 12 mm Hg or less, the upper limit of normal for left ventricular EDP. The left ventricular diastolic pressure prior to atrial contraction correlated more closely with pulmonary arterial EDP. In six patients in whom increases in systemic arterial pressure were induced by methoxamine, and in two patients in whom spontaneous increases in systemic arterial pressure occurred, left ventricular EDP increased by 2 to 11 mm Hg (average 6 mm Hg); pulmonary arterial EDP remained unchanged or increased only slightly (less than 3 mm Hg) in six of the patients, and increased by 4 and 5 mm Hg in the two remaining patients. During increases in heart rate induced by atrial pacing, left ventricular EDP declined in 12 of 14 patients, while pulmonary arterial EDP increased, resulting in a consistent disparity in these pressures (average 11 mm Hg) at heart rates in excess of 124 beats/min.

These data indicate that pulmonary arterial EDP does not provide an accurate estimate of left ventricular EDP in patients with chronic left ventricular disease, and in addition it often fails to reflect acute alterations in left ventricular EDP.

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Cardiac pacing    Methoxamine

IT WOULD BE of considerable value to have a safe and accurate means for monitoring left ventricular end-diastolic pressure, since this measurement provides an

From the Department of Medicine, University of California, San Diego, School of Medicine, La Jolla, California 92037.
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Address for reprints: James H. Gault, M.D., Milton S. Hershey Medical Center, Pennsylvania State University School of Medicine, Hershey, Pennsylvania 17033.

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important indicator of changes in left ventricular performance. Several investigators have proposed that the left ventricular end-diastolic pressure may be estimated indirectly from the pulmonary arterial end-diastolic pressure, a measurement susceptible to continuous monitoring in acutely ill patients. Although the functional continuity between the pulmonary artery and left ventricle could permit equilibration of these pressures during the normal diastolic period, the possibility was considered that acceleration of heart rate might prohibit such equilibration. In addition, the transient pressure wave produced by an abnormally forceful atrial contraction might result in a directionally opposite disparity between these pressures. Accordingly, the present study was undertaken to examine the relation between pulmonary arterial and left ventricular end-diastolic pressures in patients with normal left ventricular function and in patients with myocardial disease at rest and during acute alterations in left ventricular end-diastolic pressure.

Methods

Fifty patients were studied at diagnostic cardiac catheterization. In 24 patients the left ventricular end-diastolic pressure (EDP) and cardiac output as well as other indices of left ventricular function including end-diastolic volume and ejection fraction were normal. In 26 patients with left ventricular disease, the left ventricular EDP was elevated. Five of these patients had left ventricular hypertrophy associated with aortic valve disease, four had idiopathic hypertrophic subaortic stenosis, and 17 had myocardial disease without a mechanical cardiac lesion. Patients with mitral stenosis, and patients with elevated pulmonary vascular resistance, in whom pulmonary arterial EDP might be expected to exceed left ventricular EDP, were specifically excluded from this study.

All but three patients were in normal sinus rhythm; two had atrial fibrillation, and one had complete A-V dissociation. Studies were performed in the postabsorptive state in the supine position following premedication with sodium pentobarbital, 100 mg intramuscularly. Left ventricular pressure recordings were obtained with 7 or 8 Fr. thin-walled saline-filled catheters introduced by the retrograde arterial technique. Pulmonary arterial pressure pulses were measured in 14 patients by means of a catheter-tip micromanometer.* The pulmonary arterial pulse amplitude was calibrated against the simultaneous tracing obtained by a conventional manometer system. In 36 patients, conventional manometer recordings of pulmonary arterial pressure pulses were made by means of saline-filled 7 or 8 Fr. cardiac catheters. All recordings were made at a rapid paper speed (100 mm/sec) using a multichannel oscillographic recorder.

In all patients the pulmonary arterial and left ventricular EDPs were recorded simultaneously in the resting state during quiet respiration. Left ventricular EDP was identified 0.04 to 0.06 second after the onset of the QRS complex at a point often represented by a trough on the pressure pulse following the atrial contraction wave, and immediately preceding the rapid upstroke of left ventricular pressure (fig. 1). P-wave pressure was defined as the left ventricular pressure just prior to the ventricular manifestation of the atrial contraction wave at a point occurring approximately 0.04 second after the onset of the P wave in the ECG.

In six patients with normal left ventricular EDP and systemic arterial pressure, pulmonary arterial EDP and left ventricular EDP were compared in the resting state and during the acute elevation of the left ventricular EDP accompanying increases in systolic arterial pressure of from 20 to 44 mm Hg induced by methoxamine, 0.3 to 0.6 mg/min intravenously.

In 14 patients, the influence of heart rate on the relation between pulmonary arterial EDP and left ventricular EDP was examined. A 6 Fr. bipolar electrode was placed in the coronary sinus. Left atrial pacing via a coronary sinus electrode was employed because of the empirical observation that it was possible to maintain a normal or near-normal temporal relation between the P wave and QRS by this technique. Heart rate in the resting state prior to left atrial pacing averaged 74 beats/min (range 58 to 94). Heart rate was increased by pacing in graded increments of 10 beats/min at 2- to 4-min intervals. Simultaneously recorded pulmonary arterial EDP and left ventricular EDP were examined at an average paced heart rate of 124 beats/min (range 82 to 136)

Results

Measurements of Left Ventricular and Pulmonary Arterial Pressures in the Resting State

The relation between left ventricular and pulmonary arterial EDP is shown in all patients in figure 2.

*Statham Laboratories SF-1.
In 24 patients with normal left ventricular EDP, the pulmonary arterial EDP and left ventricular EDP were generally similar at rest, differing by 4 mm Hg or less ($P < 0.2$). In 26 patients with chronic elevation of left ventricular EDP, left ventricular EDP exceeded pulmonary arterial EDP in 20, the disparity in pressures ranging up to 21 mm Hg (average 8 mm Hg). In addition, pulmonary arterial EDP was normal (12 mm Hg or less) in 12 patients in whom left ventricular EDP was elevated (range 13 to 27 mm Hg; average 18 mm Hg).

In patients with chronic left ventricular dysfunction, the disparity in left ventricular EDP and pulmonary arterial EDP appeared to result from the augmentation of left ventricular pressure by the atrial contraction wave. Accordingly, the influence of the atrial contraction wave on this relationship was examined by comparing pulmonary arterial EDP and left ventricular diastolic pressure immediately preceding the atrial contraction wave (pre-a-wave left ventricular pressure) in the resting state in all patients with normal sinus rhythm (fig. 3). Regression analysis of these data indicates that pulmonary arterial EDP does provide a relatively accurate estimate of pre-a-wave left ventricular pressure ($\sigma_p = \pm 2.2$ mm Hg).

*Figure 1*

High-fidelity catheter-tip micromanometer recordings of pulmonary arterial pressure are superimposed on the simultaneously recorded high-gain left ventricular pressure pulse obtained by means of a conventional catheter-manometer system in a patient with normal left ventricular function (M.R., upper panel) and in a patient with left ventricular hypertrophy (J.L., lower panel). Pressure, in mm Hg, is shown on the vertical axis. The arrow in each panel indicates the point of measurement of left ventricular end-diastolic pressure.
Effects of Acute Alterations in Left Ventricular End-Diastolic Pressure

Figure 4 illustrates the effect of acute elevation of left ventricular EDP produced by methoxamine infusion on the pulmonary arterial EDP-left ventricular EDP relation in a patient with normal hemodynamic findings. The data for pharmacologically induced increases in left ventricular EDP are summa-

**Figure 2**

Measurements of pulmonary arterial end-diastolic pressure (EDP) on the vertical axis are compared with simultaneous measurements of left ventricular end-diastolic pressure on the horizontal axis in all patients; those with normal left ventricular EDP are indicated by open circles and those with elevated left ventricular EDP by closed circles. The dashed line represents the line of identity between these measurements.

**Figure 3**

Measurements of pulmonary arterial (PA) end-diastolic pressure (EDP), on the vertical axis, are plotted together with corresponding measurements of left ventricular pressure immediately prior to the atrial contraction wave (pre-a) on the horizontal axis in all patients. The dashed line indicates identity of these measurements. The solid line represents the regression analysis relating these two variables. The regression equation and correlation coefficient (r) are shown in the lower right corner.

**Figure 4**

Simultaneous high-gain recordings of pulmonary arterial and left ventricular pressure, obtained by means of a conventional catheter-manometer system, are shown before (upper panel) and during (lower panel) the intravenous administration of methoxamine, in a patient with normal left ventricular function. The level of brachial arterial pressure (B.A. PR.) corresponding with these measurements is shown above each panel. Pressure, in mm Hg, is shown on the vertical axis. The arrow indicates the point of measurement of left ventricular end-diastolic pressure in each panel.
Simultaneous measurements of pulmonary arterial (vertical axis) and left ventricular (horizontal axis) end-diastolic pressure (EDP) are shown in six patients before (open triangles) and during (closed triangles) methoxamine-induced elevation of systemic arterial and left ventricular end-diastolic pressures. Measurements obtained in two additional patients during spontaneously occurring elevations of systemic arterial pressure are indicated by cross-hatched triangles. The dashed line denotes identity of these measurements.

Effects of Acute Alterations of Heart Rate on Left Ventricular and Pulmonary Arterial End-Diastolic Pressure

The data in 14 patients studied by left atrial pacing are shown in figure 6. When heart rate was increased from an average control value of 74 beats/min (range 58 to 102) by pacing to an average level of 124 beats/min (range 82 to 136), the left ventricular EDP declined in 12 of 14 patients by an average of 6 mm Hg (range -1 to -15 mm Hg), while pulmonary arterial EDP increased by an average of 5 mm Hg (range +1 to +13 mm Hg); pulmonary arterial EDP reached levels greater than 12 mm Hg in eight of the nine patients in whom left ventricular EDP was normal. The disparity between pulmonary arterial EDP and left ventricular EDP was shown to increase further as more rapid rates were achieved, both with atrial pacing and in two patients with atrial fibrillation (figs. 7 and 8).

Discussion

Since the pulmonary artery and the left ventricle are in continuity during diastole, it might be expected that the pulmonary arterial and left ventricular pressures would tend to equilibrate during a normal diastolic filling
period, assuming pulmonary vascular resistance to be normal and the mitral valve to be unobstructed. Indeed, it has been shown that when left ventricular EDP is normal, and the diastolic filling period is of sufficient duration, pulmonary arterial and left ventricular EDPs are approximately equal. If, in fact, the correlation between these two measurements were valid under all circumstances, the measurement of pulmonary arterial pressure might then provide a simple means of estimating left ventricular performance, both in patients with chronic left ventricular disease and by continuous monitoring in patients with acute alterations in left ventricular function. However, it has not been shown previously whether this relation is maintained when the diastolic filling period is attenuated by tachycardia, or when the left ventricular EDP is elevated in the presence of acute or chronic left ventricular dysfunction.

In the present study, a substantial divergence of pulmonary arterial and left ventricular EDPs was noted with surprisingly modest increases in heart rate (greater than 115 beats/min), well within the range observed with congestive heart failure, shock, or acute myocardial infarction. The disparity in EDPs under these conditions resulted from both a decrease in left ventricular EDP and an increase in the pulmonary arterial EDP. It may indeed be true that, as in two patients in the present study, the pulmonary arterial EDP may more closely approximate left ventricular
EDP when the latter is elevated at more rapid heart rates, as might occur in the presence of acute decompensation in left ventricular performance. Comparative measurements are not available in such patients during sinus tachycardia. Nonetheless, it is apparent that the directional change in pulmonary arterial EDP was similar in patients in whom left ventricular EDP rose and in those in whom it decreased during pacing, so that the predictive value of pulmonary arterial EDP must be seriously questioned in the presence of tachycardia.

In addition, in patients with both acutely and chronically elevated left ventricular EDP, the atrial contraction wave resulted in an augmentation of left ventricular pressure at end-diastole that was not reflected in the pulmonary arterial pressure pulse, resulting in a wide disparity between these two measurements at normal heart rates. Although Falicov and Resnekov have recently noted a close correlation between left ventricular EDP and a point on the upstroke of the pulmonary arterial pulse, which they designated as pulmonary arterial "a" wave pressure, we were able to define such a point in only one of 14 patients with high-fidelity pulmonary arterial micromanometer tracings. In the resting state and at heart rates less than 120 beats/min there is a general correlation between pulmonary arterial EDP and left ventricular diastolic pressure measured prior to the "a" wave, "pre-a-pressure." However, with very modest increases in heart rate, this point occurs before completion of the previous ventricular systole and hence cannot be identified. In addition, the pertinence of this measurement as an index of left ventricular function remains to be clarified. In a small number of additional patients in whom we have compared left atrial and left ventricular pressures, the pre-a-wave left ventricular pressure appears to closely approximate left atrial mean pressure. Similar findings have been reported by other investigators. However, with acute elevations of left ventricular EDP averaging 8 mm Hg in the present study during spontaneous or drug-induced increases in arterial pressure, pre-a-wave left ventricular pressure exhibited little or no increase (average +3 mm Hg), suggesting that this measurement may be considerably less sensitive to changes in left ventricular function than left ventricular EDP. Braunwald and Frahm have previously demonstrated that when left ventricular EDP is increased by acutely induced hypervolemia there is a concordant increase in left atrial mean pressure in normal subjects but a proportionately smaller increment in left atrial mean pressure in patients with chronic left ventricular dysfunction. The authors concluded that by this mechanism the left ventricle is able to avail itself of the Starling effect at a lower mean filling pressure. The disparity between left ventricular EDP and left atrial mean pressure at rapid rates has been found to result from both the attenuation of the diastolic filling period and from contraction of the atrium against a completely or partially closed A-V valve. Direct measurements of left ventricular pressure in patients with acute myocardial infarction have shown a generally parallel increase in mean left ventricular diastolic pressure and left ventricular EDP. Conversely, in view of the well-known ability of acute hypoxemia to induce increases in pulmonary vascular resistance, and in view of the documented frequency of hypoxemia with acute myocardial infarction, it is possible that in the latter pathological circumstance the pulmonary arterial pressure may be elevated, independent of change in pulmonary venous pressure.

On the basis of these observations, the accuracy of estimation of left ventricular EDP from measurements of pulmonary arterial EDP must be seriously questioned in patients with more rapid heart rates. Although no measurements were made in patients with acute myocardial infarction where altered hemodynamics may differ from drug-induced changes in EDP in the normal left ventricle, the findings of the present study suggest that the validity of the estimation of left ventricular EDP from the pulmonary arterial pressure must be more clearly demonstrated before
pulmonary arterial EDP can be employed as an index of left ventricular performance.

References

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