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

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SUMMARY
The relationship of the left ventricular end-diastolic pressure (LVEDP) and the pulmonary arterial "wedge" mean pressure (PAWMP) to the pulmonary artery end-diastolic pressure (PAEDP) was investigated by cardiac catheterization in 71 subjects. Pressure records were obtained simultaneously or immediately consecutively. In 15 subjects with normal LV function (LVEDP, 4 to 12 mm Hg) PAEDP was within 3 mm Hg of LVEDP (r = 0.70, P < 0.01) and within 3 mm Hg of PAWMP (r = 0.69, P < 0.01) in every instance.

In 56 patients with LV dysfunction (LVEDP, 12 to 55 mm Hg), PAEDP was lower than LVEDP in 42, equal to LVEDP in six, and higher than the LVEDP in eight, who were in atrial fibrillation or had increased pulmonary vascular resistance (PVR). PAEDP correlated closely with PAWMP (r = 0.92, P < 0.001).

In 30 patients with LV dysfunction in sinus rhythm, an a wave was identified in the PA pressure tracing (PA a) prior to systole which coincided in time and magnitude to the PAWMP wave and was considered to represent retrograde transmission of the left atrial contraction wave. The PA a pressure was within 5 mm Hg of the LVEDP (r = 0.94, P < 0.001) in all except four patients, two of whom had elevated PVR.

Thus, in the presence of LV dysfunction and elevated LVEDP, PAEDP correlated well with PAWMP, but failed to represent LVEDP accurately, while PA a wave pressure closely reflected LVEDP except when PVR was markedly increased.

Additional Indexing Words:
Pulmonary arterial a wave
Retrograde pressure transmission

THE MAIN therapeutic aims in the management of patients with acute myocardial infarction are the prevention and early treatment of acute rhythm disturbances and the correction of myocardial dysfunction. Coronary care units have successfully reduced mortality from dysrhythmias, but myocardial failure still remains a therapeutic challenge. Circulatory measurements using techniques applicable to the very ill patient have been introduced to obtain more precise evaluation of myocardial function including direct measurements of the left ventricular end-diastolic pressure. Others have preferred to rely on the pulmonary arterial...
diastolic pressure as an index of left ventricular filling pressure⁶ since previous work⁷ has shown that in the absence of pulmonary vascular disease end-diastolic pulmonary arterial pressure correlates well with left ventricular end-diastolic pressure. The purpose of the present investigation was to assess the usefulness of the pulmonary artery end-diastolic pressure as an index of left ventricular end-diastolic pressure and pulmonary arterial “wedge” mean pressure in patients with and without left ventricular dysfunction studied by right and left heart catheterization.

**Methods**

Seventy-one patients, whose ages ranged from 6 to 72 years, of whom 51 were male and 20 female, were studied by right and left heart catheterization. Fifteen, investigated because of innocent systolic murmurs, had no detectable hemodynamic abnormality. Of the remaining 56 patients, 15 had predominant aortic valvar stenosis, 15 predominant aortic valvar regurgitation, 12 moderate or severe mitral valvar regurgitation, and 14 myocardial failure due to coronary heart disease or primary cardiomyopathy. Fifty-one of the 56 patients were in sinus rhythm, and five had atrial fibrillation. Excluded from this study were all patients with measurable mitral valvar obstruction or primary pulmonary vascular disease.

Following routine right heart and retrograde left heart catheterization, simultaneous pulmonary arterial wedge and left ventricular pressures were recorded. The right heart catheter was then withdrawn into the main pulmonary artery, and simultaneous or immediately consecutive left ventricular and pulmonary arterial pressures were obtained during a steady state. The right heart was entered with a 6 or 7 F Courmand catheter, and the left heart was entered with a 6, 7, or 8 F NIH or Lehman ventriculography catheter. The catheters were connected to two identically calibrated Statham P23Db strain gauges through 18-gauge polyvinyl tubes 30 cm long. The damped natural frequency of the catheter-transducer system used in our laboratory ranges from 13 Hz to 40 Hz with a damping ratio ranging from 0.38 to 0.47, depending on the size and length of the catheters used. The pressures were recorded on a multichannel oscillographic system (Electronics for Medicine) at paper speeds of 50 and 75 mm/sec. Cardiac output was determined by the method of Fick during or immediately after the pressure recordings. Pulmonary vascular resistance was calculated by standard formulae and expressed as dynes sec cm⁻⁵/m² BSA.

Left ventricular end-diastolic pressures were measured at the z point in patients in sinus rhythm, identified as a change in the velocity of upstroke of the pressure tracing occurring approximately 0.05 sec after the Q wave of the simultaneously recorded electrocardiogram.⁸ The pulmonary arterial end-diastolic pressure was measured at the lowest point of its tracing, and all measurements were averaged over a complete respiratory cycle.

In 30 patients with left ventricular dysfunction, a distinct presystolic wave was observed in the pulmonary arterial tracing (figs. 1 and 2). The a wave was recorded almost simultaneously with, and had a similar magnitude to, the a wave of the pulmonary arterial wedge tracing. The peak of this presystolic wave occurred at 0.050 to 0.153
sec after the Q wave of the electrocardiogram and was always considerably higher than the right atrial a wave recorded later in the procedure.

In each patient the relationship of the left ventricular end-diastolic pressure to the pulmonary arterial end-diastolic and a wave pressures was determined. The relationship of the mean pulmonary arterial wedge pressure to the pulmonary artery end-diastolic pressure was also analyzed.

Atrial pacing was performed in two patients, one with aortic valvar stenosis and the other with myocardiopathy, and the effects of increasing the heart rate and lengthening atrioventricular conduction were noted on these parameters.

Results
Normal Left Ventricular Function
In the 15 patients with normal hemodynamic findings, the left ventricular end-diastolic pressure (LVEDP) ranged from 4 to 12 mm Hg, the pulmonary arterial end-diastolic pressure (PAEDP) ranged from 6 to 12 mm Hg, and the pulmonary arterial wedge mean pressure (PAWMP) ranged from 4 to 12 mm Hg. A linear correlation was found between LVEDP and PAEDP ($r = 0.70$, $P < 0.01$) as well as between PAWMP and PAEDP ($r = 0.69$, $P < 0.01$). In all instances the PAEDP was within 3 mm Hg of LVEDP and within 3 mm Hg of PAWMP (figs. 3 and 4).

Left Ventricular Dysfunction
The LVEDP ranged from 12 to 55 mm Hg, the PAEDP was 8 to 38 mm Hg, and the PAWMP, 9 to 37 mm Hg in the 56 patients with left ventricular dysfunction. A linear correlation was found between LVEDP and PAEDP ($r = 0.76$, $P < 0.001$), but LVEDP exceeded PAEDP in 42 patients (range, 1 to 17 mm Hg), was equal to PAEDP in six patients, and was lower than the PAEDP in eight (range, 2 to 10 mm Hg; fig. 5). Four patients in this last group were in atrial fibrillation, and the remaining four had...
Figure 3

Relationship of the pulmonary arterial end-diastolic pressure to the left ventricular end-diastolic pressure in 15 subjects with normal left ventricular function. The regression equation and one standard error of estimate (Syx) are shown. The thin solid line represents the line of identity.

Figure 4

Relationship of the pulmonary arterial end-diastolic pressure to the pulmonary arterial wedge mean pressure in 11 patients with normal left ventricular function.

Figure 5

Relationship of the pulmonary arterial end-diastolic pressure to the left ventricular end-diastolic pressure in 56 patients with left ventricular dysfunction. The triangles represent the values of patients studied during atrial fibrillation (AF). The numbers represent the values of pulmonary vascular resistance (dynes sec cm⁻²/m² BSA) in those patients who had marked elevation of the PVR.

considerable elevation of their pulmonary vascular resistance (PVR) secondary to the effects of their left ventricular dysfunction (PVR, 530 to 1,300 dynes sec cm⁻²/m² BSA). The PAEDP was thus consistently lower than LVEDP in almost all patients in sinus rhythm who had no significant elevation of the pulmonary vascular resistance. The relationship between PAWMP and PAEDP was also linear (r = 0.92, P < 0.001; fig. 6), PAWMP being higher than PAEDP in 22 patients (range, 1 to 8 mm Hg), equal to PAEDP in five, and lower than PAEDP in 18 (range, 1 to 6 mm Hg) of the 45 patients in whom the pulmonary vascular resistance did not exceed 400 dynes sec cm⁻²/m² BSA. In three patients in whom
END-DIASTOLIC PRESSURES IN PA AND LV

Figure 6

Relationship of the pulmonary arterial end-diastolic pressure to the pulmonary arterial wedge mean pressure in 48 patients with pulmonary vascular resistance of 400 dynes sec cm⁻²/m² or less with left ventricular dysfunction. The three patients who had elevated PVR (triangles) have not been included in the statistical analysis.

Figure 7

Relationship of the height of the pulmonary arterial "a" wave to the left ventricular end-diastolic pressure in 28 patients with left ventricular dysfunction. The two patients who had elevated PVR (triangles) have not been included in the statistical analysis.

The PVR was higher (590, 700, and 800 dynes sec cm⁻²/m²), however, the PAEDP exceeded the PAWMP by 4, 10, and 5 mm Hg, respectively. PAEDP, therefore, was found to reflect PAWMP with reasonable accuracy provided severe pulmonary vascular changes were not present.

An excellent linear correlation was found between the pulmonary arterial a wave pressure and LVEDP (r = 0.94, P < 0.001; fig. 7) in the absence of pulmonary vascular obstruction. The LVEDP was higher than the PA a in 12 patients (range, 1 to 12 mm Hg), equal to PA a pressure in nine patients, and lower than PA a in the remaining seven (range, 1 to 3 mm Hg). In 26 patients the PA a wave pressure was within 5 mm Hg of the LVEDP; the two patients in whom the greatest discrepancy was found (LVEDP higher than the PA a by 6 and 12 mm Hg) had severe aortic valvar regurgitation with LVEDP values of 48 and 28 mm Hg, respectively (fig. 8). Two patients with elevated PVR had significantly higher PA a waves than LVEDP.

Atrial pacing was performed in one patient who had aortic valvar stenosis and in another who had cardiomyopathy (fig. 9). In the control state the LVEDP was significantly higher than the PAEDP, but as the rate increased and atrioventricular conduction lengthened, the LVEDP fell owing to the absence of a properly timed atrial contraction and both pressures became similar. In the patient with aortic stenosis, moreover, the PA a which could be identified only at the basal heart rate was identical to the LVEDP.

In the five patients studied during atrial fibrillation, the LVEDP was found to be equal to the PAEDP in one case and lower than the PAEDP in four, in three of which PVR was increased. The relationship between these two parameters seemed to vary with the length of diastole as illustrated in figure 10; the longer diastole was associated with equalization of pressures. This patient had severe mitral
regurgitation and elevation of the pulmonary vascular resistance but no gradient across the mitral valve.

**Discussion**

At the end of diastole, in the absence of obstructive lesions the pulmonary veins, left atrium, and left ventricle form a functionally single chamber with a minute pressure gradient in the direction of flow. During this phase of the cardiac cycle, pulmonary arterial pressure is determined predominantly by the resistance encountered by the blood as it circulates across the pulmonary vascular bed. Since this is usually a low resistance circuit and flow is least at this time, end-diastolic pulmonary arterial pressure should approximate closely the left atrial and left ventricular pressures at a point just prior to atrial and ventricular contractions. Kaltman and others\(^7\) studied the relationship of LVEDP to PAEDP in 58 patients with intracardiac shunts and in 12 patients with acquired heart disease. They found a good correlation between the two when pressures were less than 17 mm Hg and when pulmonary vascular obstruction was not present. More recently Bouchard and associates\(^8\) found a good correlation between LVEDP and PAEDP only when the values were within the normal range. In patients in whom the LVEDP was elevated, however, the

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**Figure 8**

Simultaneous pulmonary arterial wedge and left ventricular pressures (left) and pulmonary arterial and left ventricular pressures (right) of a 23-year-old man with severe aortic regurgitation. The LVEDP is 12 mm Hg higher than both the PAW and the PA “a” waves.

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**Figure 9**

Relationship between pulmonary arterial end-diastolic pressure and left ventricular end-diastolic pressure at different heart rates induced by atrial pacing. As the heart rate increases and atrioventricular conduction lengths, LVEDP falls to values close to the PAEDP. The PA “a” pressure equaled LVEDP in the first patient at the basal heart rate.

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A presystolic wave in the pulmonary arterial tracing has long been recognized; its origin has been considered obscure, or it has been tentatively attributed to the transmission of the right atrial wave of contraction to the pulmonary artery. This explanation, however, seems most unlikely, for both in normal circumstances and in the majority of disease states affecting left ventricular function, the pulmonary arterial diastolic pressure significantly exceeds the right atrial contraction pressure.

The transmission characteristics of pressure waves of the normal pulmonary arterial circulation have been studied in a pulmonary lobe in man during thoracotomy by Caro and associates who found that the forward pressure waves were transmitted better than retrograde pressure waves. When the pressures were increased, however, transmission in both directions was markedly improved, and they suggested a decrease in the normal vascular compliance due to the increased pressure as the cause. It has also been demonstrated that, allowing for certain exceptions and limitations, the pulmonary arterial wedge pressure closely resembles left atrial pressure and that the similarity between the two pressure tracings is increased when the pulmonary venous pressure is elevated as is frequently observed in mitral valvar obstruction and in left ventricular failure. The decrease in compliance caused by engorgement of the pulmonary vascular bed probably accounts in large measure for the enhanced retrograde transmission of the pressure wave. In a similar fashion, retrograde transmission of a large left atrial v wave has been recorded in the pulmonary arterial tracings of patients with severe mitral regurgitation, and Gould and Lyon recently identified large a waves in the pulmonary arterial tracings of five patients with left ventricular dysfunction.

We were able to identify pulmonary arterial a waves in the tracings of 30 patients with left ventricular dysfunction and elevated left ventricular filling pressures which by their timing and magnitude appear to represent retrograde transmission of left atrial pressure.

**Figure 10**

Simultaneous PA-LV pressure tracings of a 66-year-old man with severe mitral regurgitation and atrial fibrillation. Pulmonary vascular resistance was 700 dynes sec cm⁻²/m² BSA. The PAEDP approximates LVEDP only during longer diastolic pauses.

PAEDP was consistently lower by 2 to 18 mm Hg; our results agree with their findings.

The height of the ventricular filling pressure just before ventricular contraction is used widely as an index of ventricular function, although it is appreciated that factors other than end-diastolic volume, for example ventricular compliance, are important in its determination. In patients with left ventricular disease, the importance of a properly timed atrial contraction in elevating the LVEDP while at the same time allowing the mean filling pressure to remain low has been well recognized. Braunwald and Frahm were able to show that while the difference between the LVEDP and left atrial mean pressure averaged only 0.2 mm Hg in normal subjects, patients with left ventricular disease showed a difference of 9 mm Hg. A large left ventricular a wave is seen in most patients in sinus rhythm with chronic left ventricular disease and may well explain why the left ventricular end-diastolic pressure is usually higher than the pulmonary arterial end-diastolic pressure in these patients.
at the time of atrial contraction. In 26 of 30 instances, the magnitude of this a wave was within 5 mm Hg of the LVEDP. Discrepant values were recorded in the presence of pulmonary vascular obstruction where pulmonary arterial a pressure was usually higher than LVEDP and in two patients with severe aortic valvar regurgitation when the LVEDP exceeded the PA a pressure by 6 and 12 mm Hg. Early mitral valve closure, which most likely occurs in some patients with severe aortic valvar regurgitation, might well account for the difference in these two patients by impairing retrograde transmission of the left ventricular end-diastolic pressure.

The pulmonary arterial a waves could not be identified in the pressure tracings of the remaining 21 patients with an increased LVEDP and normal pulmonary vascular resistance. These might relate to individual compliance characteristics of the pulmonary vascular bed causing distortions and variable delays in the retrograde propagation of the pressure waves, but factors as yet unknown might also be responsible. The PAEDP correlated well with the PAWMP in this study suggesting that in the absence of pulmonary vascular obstruction the PAEDP is a useful estimate of the mean left atrial pressure.

The height of the pulmonary arterial a wave, therefore, is a reliable correlate of the LVEDP in most patients in sinus rhythm with left ventricular dysfunction and elevated filling pressures, provided that mitral stenosis and/or pulmonary vascular obstruction are excluded and early closure of the mitral valve, as seen in some patients with severe aortic regurgitation, does not occur. The height of the pulmonary arterial a wave should be a useful measurement in evaluating left ventricular function and the response to therapeutic interventions in patients with acute myocardial infarction, particularly when complicated by left ventricular failure and circulatory collapse.

References


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Self Discipline

Internal discipline is the force that duty exercises to prevent laxness in assuming responsibility. If I may be permitted the anachronism, it is the voice of conscience that tends to be scorned by the immature of all age groups. Conscience concentrates on the ultimate goal and ignores the immediate inconvenience. It makes one execute carefully the boring, the unexciting tasks of education and of life, realizing that they are stepping stones to higher spots that will be exciting. It may be more fun to talk to a sick patient from a haze of ignorance than to learn the physiology of pulmonary ventilation, but if the goal is understanding, the stimulation of the healer can be postponed while he is becoming a scientist.

A physician can be conscientious in two ways. First, scientifically; ignoring no discoverable detail of his patients’ physical and mental status. Second, humanistically, pursuing the course of kindness, compassion, tolerance, and understanding. This latter duty takes time and may easily be neglected in a busy life.—ATCHLEY, DANA W.: Acceptance of the Kober medal for 1969. Trans Ass Amer Physicians 82: 50, 1969.
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