The Gradient in Pressure Across the Pulmonary Vascular Bed During Diastole

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In the absence of narrowing of the mitral valve, of left atrial anomalous constriction, or of pulmonary vascular disease, the diastolic pressure in the pulmonary artery has been assumed by some investigators to reflect almost exactly the end-diastolic pressure in the left ventricle. This assumption has been made in studies of the circulatory dynamics at rest and with exercise, in congestive heart failure, and during the administration of digoxin.1-12

With the development of reliable techniques for catheterization of the left heart,13-24 the validity of the assumption can be tested. The objective of this study was to test this hypothesis in patients with a variety of physiological and anatomic cardiac abnormalities.

Methods

Seventy patients, ranging in age from 1 to 59 years, were subjected to right and left heart catheterization. Fifty-four patients had an atrial septal defect of the secundum type. Three of these were complicated by mild pulmonic stenosis. Four patients had a ventricular septal defect; one also had pulmonic stenosis. The remaining 12 had acquired heart disease. These included three with aortic stenosis, one with aortic insufficiency, one with mitral insufficiency, and three with so-called primary myocardial disease. Four patients with clinical mitral insufficiency and stenosis but without a diastolic gradient in pressure across the mitral valve at rest completed the group.

In the 12 patients with acquired heart disease the left ventricle was entered by the retrograde aortic or the percutaneous, direct transventricular route. Simultaneous pulmonary arterial and left ventricular pressure pulses were recorded in these. In the remainder, the left heart was catheterized through a septal defect; the recordings from the left and right sides were made within a brief interval of time of each other during a clinically stable state.

In 42 of the 58 patients with intracardiac shunts both the left atrium and the left ventricle were catheterized; and in the remainder one or the other chamber was catheterized. Right heart catheterization was accomplished by the usual venous routes. Pressure pulses were recorded by means of P23D Statham strain gauges with identically calibrated sensitivities on an Electronics for Medicine DR8 recorder. Cardiac output was measured by the direct Fick principle in 10 patients without intracardiac shunts. In each patient the relationship of the pulmonary arterial diastolic pressure to the left ventricular diastolic pressure and to the left atrial mean pressure was considered.

Results

In 56 of the 70 patients studied, there was no gradient in diastolic pressure between the pulmonary artery and the left ventricle. These included the 12 patients with acquired heart disease and 44 with intracardiac shunts.

Figure 1 shows the sphygmograms from a patient with unknown myocardial disease. There were no objective clinical manifestations of congestive heart failure, but the cardiac output was 2.52 L per minute per square meter of body surface area. At the end of diastole, pressures in the left ventricle, pulmonary artery, and pulmonary artery wedge position were at identical levels.
Figure 1

Sphygmograms from C. G., a white man, aged 57 years, with “primary” myocardiopathy. The identical pressures in the left ventricle, pulmonary artery wedge position, and pulmonary artery at the end of diastole are to be noted. The scale in millimeters of mercury is applicable to all curves. LV = left ventricle; Wedge = pulmonary artery wedge; PA = pulmonary artery; II = Lead II of conventional electrocardiograms; \( V_{PA} \) = unipolar potential within the pulmonary artery. Time lines = 0.1 second.

The response of these pressures to changing conditions was observed in the 12 patients with synchronous recordings. A simultaneous elevation in end-diastolic pressure was seen at the time of onset of a ventricular premature systole in all. Figure 2 demonstrates this event in the patient whose sphygmograms appear in figure 1. The early appearance of the premature ventricular contraction with reduction of diastolic chamber filling was reflected in the smaller premature pressure pulse in the left ventricle and in the pulmonary artery. Furthermore, at the end of the preceding foreshortened diastole, both pressures were elevated to a similar degree, 20 mm Hg, compared to the control level of 10 mm Hg.

The Valsalva maneuver performed in three patients caused rapid simultaneous elevation of these diastolic pressures (fig. 3). During prolonged straining there was a gradual decrease of both pressures in parallel. These abruptly returned to previous levels upon resumption of normal respiration.

Of the 70 patients, 14 (20%) had definite diastolic gradients of pressure between the pulmonary artery and the left ventricle. This group, all with congenital heart disease, included 13 with atrial and one with ventricular septal defect. The pulmonary artery diastolic pressure exceeded 15 mm Hg in all. These 14 appeared to be part of a different population. Since all had congenital intracardiac shunts and high pulmonary artery pressures, it may be assumed that they had some pulmonary vascular disease to account for the gradient.

In 45 of the 56 patients with normal or near normal pressures in the pulmonary artery, the left ventricular end-diastolic pressures were obtained. A close correlation between the pulmonary arterial and left ventricular diastolic pressure was found below 17 mm Hg, the highest left ventricular end-
Simultaneous left ventricular and pulmonary arterial pressure pulses before, during, and after a ventricular premature systole. Patient and symbols are same as in figure 1.

Left ventricular diastolic and pulmonary artery pressures recorded during the Valsalva maneuver in M.S., a white man, aged 47 years, with mitral insufficiency. Continuation of the Valsalva is indicated by upward shift of the electrocardiographic base lines. For more precise measurement of a possible diastolic pressure gradient, high gain amplification was used and the systolic peak of the left ventricle is not visible. Symbols and time lines are the same as in figure 1.
diastolic pressure recorded in this group. The statistical significance was demonstrated by a correlation coefficient of 0.908 (P = 0.001) (fig. 4).

A small difference between the diastolic pressure in the pulmonary artery and the left ventricle may exist when the pressure in the latter is less than 3 mm Hg (fig. 5). A similar but smaller gradient in pressure has been shown to occur in the pulmonary circulation of cats when the left atrial pressure is less than 15 cm of physiological saline. Above this level in these experiments, an increase in left atrial pressure caused an equivalent rise in the pulmonary vascular pressure.25, 26

Whether the correlation is true at higher pulmonary arterial pressure in patients who may be assumed to have no pulmonary vascular disease is not known. However, we have studied a patient with aortic insufficiency with simultaneous diastolic pressures in the pulmonary artery and left ventricle as high as 40 mm Hg, without clinical evidence of pulmonary edema (fig. 6). In another, these simultaneous

pressures were recorded at an identical level of 47 mm Hg during transient pulmonary edema; the patient had aortic insufficiency and stenosis. Data on these two patients have
The relationship between the left atrial mean pressure and pulmonary artery diastolic pressure in 46 patients. Multiple values lying in the same point are indicated by circles. The solid line represents the regression line and the dotted lines, the 95% confidence limits.

In 46 instances left atrial mean pressure was available for comparison with the pulmonary artery diastolic pressure. The correlation between these two was found to be highly significant ($r = 0.874; P < 0.001$) (fig. 7).

The relation of left heart and pulmonary artery diastolic pressures in the 14 patients with congenital heart disease and an elevated pulmonary artery pressure is shown in figure 8. For comparison the regression lines and 95% confidence limits applicable to the previous group of 45 patients with the same measurements (fig. 4) were plotted on the graph.

All the patients studied had normal sinus rhythm. Heart rates varied between 60 and 140 per minute with a mean rate of 85 (fig. 9). The 14 patients with pulmonary hypertension and a significant pulmonary artery-left ventricular diastolic pressure gradient showed a range from 70 to 130 beats with a mean of 100 beats per minute.
In 10 of the patients without intracardiac shunts, the cardiac output was 2.52 to 3.56 L per minute per square meter of body surface area. None of the patients had clinical manifestations of congestive heart failure at the time of observation.

Discussion

The effects of failure of the left ventricle on pulmonary vascular pressures were compounded years before the use of cardiac catheterization techniques. When the left atrium and mitral orifice are normal, the pressure in the pulmonary veins cannot differ much from the left ventricular diastolic pressure. Any difference between the latter and the diastolic pressure in the pulmonary artery must be ascribed to a resistance in the pulmonary arteriolar or capillary bed.

Measurement of ventricular filling pressure has been used as an indication of ventricular failure. If the ventricular end-diastolic pressure is difficult to establish, the mean atrial pressure may be used as an index of the diastolic distention of the ventricle. Although left atrial mean pressure is considered a reflection of left ventricular end-diastolic pressure in normal subjects, the validity of this correlation in disease may be in question.

In experiments on dogs large increases in left atrial pressure resulted in an equal rise of pulmonary arterial pressure. It was suggested that pulmonary arterial hypertension in left ventricular failure is due in large part to an elevation of left ventricular end-diastolic pressure. When the left ventricle fails, its end-diastolic pressure rises. As a consequence, left atrial and pulmonary venous, capillary, and arterial pressures increase. The pulmonary artery diastolic pressure thus has been assumed to be a reflection of left ventricular end-diastolic pressure.

The presence of pulmonary hypertension secondary to diseases of the lungs is well established. Furthermore this increase in pulmonary artery pressure has been recorded with normal pulmonary venous pressures, as measured by the wedged pulmonary artery technique.

The present study disclosed a close correlation between the pulmonary artery diastolic pressure with the left ventricular end-diastolic and left atrial mean pressures when these are below 17 mm Hg. Above this level a large diastolic pressure gradient existed in certain patients. All of these had congenital intracardiac shunts and probably abnormal pulmonary vascular beds. Under these circumstances the use of the pulmonary artery diastolic pressure as a measure of pressure in the left heart is invalid. Before generalizations are made however, further investigation is necessary to obtain data in patients with acquired heart disease and pulmonary hypertension, especially in those with left ventricular end-diastolic pressures in excess of 15 mm Hg, as well as in normal subjects.

In the absence of a gradient in pressure between the pulmonary artery and the pulmonary wedge position, the diastolic pressure in the former quantitatively mirrors the left ventricular end-diastolic pressure. As far as this study has gone, this has held true in patients with acquired heart disease without mitral valvular obstruction. Further investigation is planned to determine the validity and limits of these observations in patients with congestive heart failure and without obvious anatomic obstruction in the lesser part of the circulation between the pulmonic and aortic valves.

The findings presented indicate that in patients of the type studied friction across the pulmonary vascular bed is small and a state of equilibrium exists at the end of diastole.

Summary

By means of right and left heart catheterization 70 patients with congenital or acquired heart disease were examined to determine whether or not a gradient in pressure existed between the pulmonary artery and the left ventricle at the end of diastole. In the absence of mitral valvular obstruction in 56 patients there was a statistically significant correlation of pulmonary artery diastolic, left ventricular end-diastolic, and left atrial mean pressures less than 15 mm Hg which was in-
dependent of heart rate. Diastolic pressures at identical levels as high as 40 mm Hg were demonstrated in the presence of aortic insufficiency without left ventricular failure, and as high as 47 mm Hg during pulmonary edema in one patient with aortic stenosis and insufficiency. A diastolic gradient in pressure between the pulmonary artery and the left ventricle existed in 14 patients with pulmonary hypertension. These patients all had congenital intracardiac shunts and it may be assumed that they had some obstruction in the pulmonary vascular bed.

These findings suggest that within defined limits friction across the pulmonary vascular bed is so small that a state of pressure equilibrium exists at the end of diastole. When the limits are met clinically, the pressures in the pulmonary artery and in the left ventricle at the end of diastole are identical.

References


Chemical Transmitters of Nerve Impulses

When, therefore, some seven years later, Loewi described his beautiful experiments, showing that stimulation of the vagus nerve produced its inhibitor effects on the frog’s heart by the liberation of a chemical substance; and when his successive papers provided cumulative evidence of the similarity of this substance to acetylcholine, including its extreme liability to destruction by an esterase, which Loewi extracted from the heart muscle; I believe that I was more ready than most of my contemporaries for immediate acceptance of the evidence for this “Vagusstoff,” and more eager, almost than Professor Loewi himself, to assume its identity with acetylcholine.—Henry H. Dale: Some Recent Extensions of the Chemical Transmission of the Effects of Nerve Impulses. In Nobel Lectures: Physiology or Medicine. New York, Elsevier Publishing Co., 1965, p. 403.
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