Left Atrial Pressure Pulse in Mitral Valve Disease

A Correlation of Pressures Obtained by Transbrachial Puncture with the Valvular Lesion

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Left atrial pressures were determined by transbrachial puncture in 53 patients with mitral valve disease of proved type, and were analyzed by several methods. Two new methods of analysis, the rate of the y descent divided by the mean left atrial pressure, and the y descent for each 0.1-second interval divided by the mean left atrial pressure afforded the best separation of the patients with mitral insufficiency from those with mitral stenosis requiring a commissurotomy. In the presence of predominant mitral stenosis, however, the severity of accompanying mitral regurgitation could not be assessed.

It has been well established that mitral valve disease and experimentally produced mitral valvular lesions alter the contour of the left atrial pressure pulse.1-6 The purpose of this investigation was to evaluate the usefulness of several previously described1-4 and 3 new methods of analysis of the left atrial pressure in distinguishing patients with predominant mitral insufficiency from those with predominant stenosis. This study is based on the preoperative left atrial pressure curves of 53 patients with mitral valve disease of proved type.

Material

Of the patients studied, 39 were found at operation to have significant mitral stenosis. In 32 of these patients there was either no palpable regurgitation or a minimal jet considered by the surgeon to be of little functional significance. Seven of the 39 patients with mitral stenosis were found to have, in addition, "significant" mitral insufficiency. This was evidenced by the presence of a regurgitant jet of moderate or severe intensity. The valve orifice in each of the 39 patients with mitral stenosis (including those with combined mitral stenosis and insufficiency) was less than 1 cm. in diameter and a commissurotomy was indicated and performed.

Fourteen patients had essentially pure mitral insufficiency. In 7 patients this diagnosis was established at operation or autopsy. The other 7 patients presented classic clinical, roentgenologic, and electrocardiographic findings of mitral insufficiency and at left heart catheterization were shown to have no diastolic gradient across the mitral valve.

Methods

Left atrial pressures were determined preoperatively by transbrachial puncture as described by Allison and Linden7 and Facquet and co-workers.8 Local anesthesia was employed. Pressures were recorded through a no.-17 needle with a Statham P23A pressure-transducer employing either a Sanborn multi-channel recorder or a cathode-ray photographic recording system. There were no instances of hemorrhage, infection, or emphysema and the procedure was well tolerated even by severely ill patients. A detailed description of the technic of left heart pressure measurements by the transbrachial method forms the subject of a separate report.9

The components of the left atrial pressure pulse in a normal individual are illustrated in figure 1. The a wave is associated with atrial systole and follows the P wave of the electrocardiogram; the z point denotes the beginning of the second positive deflection, the c wave, associated with isometric ventricular contraction.10 Atrial pressure then falls during the early portion of ventricular systole (z descent); it again rises to a third peak, the v wave, which occurs at the time of opening of the mitral valve. The decline in atrial pressure following the v point, designated as the y descent, is associated with rapid ventricular filling, i.e., it represents the so-called diastolic inflow phase.11 The left atrial pressure then rises again in middiastole during the period of diastasis or slowed ventricular filling (designated as D in fig. 1) until the onset of the next atrial contraction.

In the nomenclature of Owen and Wood,1 P1 is the pressure at the peak of the v wave and P2 is the pressure at the termination of the y descent. T1 and T2 are the corresponding points on the time scale. The pressures at the z point and at T1 and T2 were calculated in all patients. At least 4 complexes, usually covering a complete respiratory cycle, were

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analyzed in each tracing and average values were used in the final calculations. In patients with atrial fibrillation and a ventricular rate greater than 100, the 4 longest cardiac cycles were chosen for evaluation. Mean left atrial pressures were determined by planimetric integration.

RESULTS AND DISCUSSION

There was no significant difference between the average of the mean left atrial pressures of the 39 patients with mitral stenosis and the 14 patients with mitral insufficiency. However, obvious differences were found between the left atrial pressure tracings in mitral stenosis and pure mitral insufficiency. Figure 2A illustrates a typical curve from a patient with pure mitral stenosis and sinus rhythm. The a-c complexes and v waves are of similar amplitude, the “pulse pressure”* is narrow, the y descent is slow, the mean pressure is elevated, and diastasis is absent. Figure 2B is a tracing obtained from a patient with pure mitral insufficiency and sinus rhythm. The v wave rises far above the a-c complex and the y descent is rapid. The “pulse pressure” is wide, the mean pressure is elevated, and diastasis is present.

* The term “pulse pressure” is used in this report to denote the pressure difference between the highest and lowest points of the left atrial pulse wave. It is usually represented by the difference between the pressure at the peak of the v wave and that at the termination of the y descent.

Methods for quantification of the differences in the left atrial pressure curves associated with mitral stenosis and mitral insufficiency have been described by several authors and the data from the present series were first analyzed according to their criteria. In figures 3, 4, and 6 the appropriate mathematical ratio is plotted along the abscissa. Each open circle represents a patient with sinus rhythm and each solid circle a patient with atrial fibrillation. Half circles designate those patients with mitral stenosis and significant mitral insufficiency.

Facquet and co-workers considered the v wave in terms of its “pulse pressure” and diastolic pressure. In the terminology of figure 1 this may be expressed by the ratio $\frac{P_1 - P_2}{P_2}$.

When this ratio was calculated for the patients in the present series, complete separation of mitral stenosis from mitral insufficiency was not
Fig. 3. Analysis of the left atrial pressure pulses by the previously reported methods. MS designates all patients with mitral stenosis requiring a mitral commissurotomy and M those with pure mitral insufficiency. Each open circle represents a patient with sinus rhythm and each solid circle a patient with atrial fibrillation. Half circles designate those patients with mitral stenosis and significant mitral insufficiency.

Fig. 4. Analysis of the left atrial pressure pulse in which the rate of the y descent \((R_y)\) is related to the mean left atrial pressure \((MLAP)\). The symbols correspond with those of figure 3.

apparent (fig. 3A). Allison and Linden\(^3\) related the pressure at the peak of the \(v\) wave \((P_1)\) to the pressure at the onset of ventricular contractions (\(z\) point). This may be expressed \(\frac{(P_1 - z) \times 100}{P_1}\). When this formula was applied, there was distinct overlap, particularly among patients with sinus rhythm (figure 3B). Fox and associates\(^4\) compared the height of the \(v\) wave \((P_1)\) to the mean left atrial pressure. This ratio in the present series is plotted in figure 3C. There is no clear separation of the groups regardless of the rhythm. Owen and Wood\(^5\) noted in pulmonary capillary tracings that the rate of the \(y\) descent was more rapid in mitral insufficiency than in mitral stenosis. This rate of descent \((R_y)\) is expressed by the ratio \(\frac{P_1 - P_2}{T_2 - T_1}\) (fig. 1). These investigators divided this rate of descent by the height of the \(v\) wave \((R_y/P_1)\). When this formula was applied to the left atrial pressures of the present series of patients, better separation was obtained than with the other methods (fig. 3D).

It was noted that \(P_1\) varied strikingly and the ratio \(R_y/P_1\) changed significantly according to the phase of respiration and the length of the preceding diastole. Accordingly, the rate of the \(y\) descent \((R_y)\) was related to a more stable denominator, the mean left atrial pressure \((MLAP)\) (fig. 4). This ratio provided a better separation of patients with pure mitral insufficiency from those with mitral stenosis requiring a commissurotomy. In the patients in whom a mitral commissurotomy was indicated, the ratio \(R_y/MLAP\) was always less than 4. When the ratio \(R_y/MLAP\) exceeded 4, the predominant lesion was always mitral insufficiency.

It was observed that in the presence of atrial fibrillation, marked beat-to-beat variations in the ratio \(R_y/MLAP\) occurred in a given patient. Analysis of the left atrial pulse revealed that in the presence of mitral stenosis the rate of the \(y\) descent was most rapid at its onset and diminished as diastole progressed. Therefore, in a patient with mitral stenosis, tachycardia
abbreviates the duration of the $y$ descent and thereby elevates $R_y$. Thus, the ratio $R_y/MLAP$ is a function of heart rate. Since the heart rate differs from patient to patient, and from beat to beat in a single patient with atrial fibrillation, the $y$ descent was measured at standard time intervals.

It has been noted previously\textsuperscript{12} that diastasis is absent in the left atrial pressure of patients with mitral stenosis, i.e., the $y$ descent is uninterrupted until the onset of the next atrial or ventricular contraction. It was also felt desirable to incorporate this observation into the analysis of the left atrial pressure pulse.

Accordingly, the $y$ descent divided by the $MLAP$ was calculated for each 0.1-second interval from its onset ($P_y$) to the beginning of the subsequent atrial contraction in patients with normal sinus rhythm or to the onset of the next ventricular contraction in patients with atrial fibrillation. This analysis is presented in figure 5. Four beats from each of the 53 patients were calculated. In figure 5 each symbol represents the value calculated from an individual beat. In beats in which the duration of diastole was abbreviated the ratio could only be calculated for the first 1 or 2 0.10-second periods. Hence fewer symbols are plotted as diastole progresses.

Fig. 6. Ratio of the pressure at the peak of the $v$ wave to that at the peak of the $a$ wave in patients with normal sinus rhythm. Half circles represent those patients with mitral stenosis and significant mitral insufficiency.

In the first 0.10 second there is a clear separation of virtually all beats from the patients with mitral insufficiency and those with mitral stenosis. Of the 14 patients with mitral insufficiency 7 had diastolic phases of sufficient duration to permit the calculation of the ratio for more than the first 0.10 second. Six of these 7 patients exhibited diastasis. This rise in the left atrial pressure is plotted as a “negative descent” and therefore appears below zero. Diastasis was absent in all 39 patients with mitral stenosis.
In the patients with sinus rhythm the pressure at the peak of the $v$ wave ($P_v$) was related to the pressure at the peak of the $a$ wave ($a$) and this relationship expressed as the ratio $P_v/a$. This analysis also offered a satisfactory differentiation between mitral stenosis and pure mitral insufficiency (fig. 6).

The method of analysis of Allison and Linden (fig. 3B) revealed that in patients with atrial fibrillation higher values of $P_v/a$ were noted. However, the rhythm had no discernible effect in the other methods of analysis.

**Discussion**

It is realized that the methods described for analysis of the left atrial pressure curve are to a large degree empiric. The size and distensibility of the pulmonary venous bed and left atrium and ventricle profoundly affect the left atrial pressure pulse. It is apparent that a given volume of blood regurgitated into the left atrium may result in a large or small variation in atrial pressure depending upon the volume and distensibility of this chamber and the pulmonary venous bed. The mean left atrial pressure as utilized in the formulae described takes some consideration of these important variables.

In the normal subject or the patient with mitral stenosis, the $v$ wave results only from filling of the atrium during the period in which the mitral valve is closed. In the presence of mitral insufficiency, however, blood from the left ventricle also enters the atrium at this time and further increases the pressure at the peak of the $v$ wave. When the mitral valve opens, left atrial pressure falls as blood flows from the atrium into the ventricle. In the absence of mitral stenosis rapid filling of the ventricle occurs during early diastole. In mid-diastole a rise in ventricular and hence in atrial pressure takes place as blood enters the already well filled ventricle. This rise in pressure has been termed diastasis. However, in the presence of mitral stenosis and a pressure gradient across the mitral valve, rapid ventricular filling cannot occur, the $y$ descent is slow and prolonged, and diastasis is usually absent. It should be noted, however, that diastasis might be expected to occur in those patients with mitral stenosis in whom the left atrioventricular pressure gradient falls to zero during long diastolic periods. In mitral insufficiency there is no obstruction to the flow of a volume of blood greater than normal and the fall in pressure is precipitous. Thus, in analyzing the formula presented ($R_y/MLAP$) it is apparent that in mitral insufficiency the pressure at the peak of the $v$ wave is higher and also falls more rapidly than in mitral stenosis. Both of these factors tend to increase the value of $R_y$ and hence elevate the ratio $R_y/MLAP$, and the rate of $y$ descent during the first 0.1 second.

In the clinical use of left atrial pressure measurements it must be borne in mind that elevated left atrial pressures may occur with a normal mitral valve in the presence of the elevated left ventricular end-diastolic pressure that accompanies left ventricular failure. In the absence of tachycardia such left atrial pressure pulses generally exhibit diastasis, and this criterion may be employed to rule out the presence of mitral stenosis. The absence of diastasis is a specific abnormal feature of the left atrial pulse contour in mitral stenosis. This observation is not in accord with the view that the left atrial pulse of the patient with mitral stenosis and sinus rhythm closely resembles the normal in contour but is merely elevated in height.

Extension of the technic of transbronchial puncture of the left atrium to include catheterization of the left ventricle permits measurement of pressure gradients across the mitral and aortic valves. When, however, clinical findings indicate a mitral lesion, left atrial puncture alone will aid materially in evaluation and selection of patients. When the left ventricle is not catheterized, the analysis of the atrial pressure pulse becomes even more important in the decision as to appropriate therapy. The transbronchial measurement of left atrial pressure can usually be carried out in less than 10 minutes. Its simplicity permits serial studies at frequent intervals. No untoward effects have been observed in more than 425 transbronchial punctures of the left atrium.

Combined lesions of the mitral valve present a "stenosis-insufficiency continuum" with all gradations in the severity of each. Accordingly, there is a group of patients in which determina-
tion of the predominant lesion has proved difficult. In the present series no method of analysis clearly separated those patients with pure mitral stenosis from those patients with accompanying mitral insufficiency of mild or moderate degree. However, in all these patients a commissurotomy was found indicated. On the basis of the findings presented, it is believed that the calculation of the ratio $R_y/MLAP$ (fig. 4), as well as the fractional analysis of the $y$ descent and of diastasis (fig. 5), is of distinct value in distinguishing those patients with predominant mitral stenosis from those with pure mitral insufficiency. When sinus rhythm is present the ratio $P_y/a$ is also of diagnostic value.

**SUMMARY**

Left atrial pressures were determined by transbronchial puncture in 53 patients with mitral valve disease of proved type. Thirty-nine patients had mitral stenosis and required a commissurotomy; of these, 7 had significant associated regurgitation. Fourteen patients had pure mitral insufficiency.

The left atrial pressure pulses were analyzed by several previously described methods. Of these, the ratio $R_y/v$ (Owen and Wood) proved most useful in distinguishing mitral insufficiency from predominant mitral stenosis.

The ratio $R_y/MLAP$ as well as the fractional analysis of the $y$ descent and of diastasis afforded the best separation of the 2 groups. However, in the presence of predominant mitral stenosis, the severity of accompanying mitral regurgitation could not be determined with any method of analysis.

None of the left atrial pressure curves of the patients with mitral stenosis exhibited diastasis, and the significance of this observation is emphasized.

**ACKNOWLEDGMENT**

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**SUMMARIO IN INTERLINGUA**

Le pressiones sinistro-atral eseva determinate per punction transbronchial in 53 patientes con morbo de valvula mitral de typos demonstrate. Trenta-nove patientes hadeva stenosis mitral e requireva commissurotoma. Septe de istes hadeva grados significative de regurgitation asociate. Dece-quatro patientes hadeva pur insufficientia mitral.

Le pulsus de pression sinistro-atral eseva analyzate secundo methodos previemente descripte. Le proportion $R_y/v$ (de Owen e Wood) se mostrava utilissime in le differentiation de insufficientia mitral ab predominante stenosis mitral.

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Nulle del curvas de pression sinistro-atral ab patientes con stenosis mitral exhibiva ulla diastase. Le importantia de ister observation es signalate.

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WILLIAM STOKES (1804–1878)

William Stokes was born in Dublin in 1804, the son of the Regius Professor of Medicine in the University of Dublin. The impact of Auenbrugger's Treatise on percussion, and the development of auscultation by Laennec aroused great interest at the time William Stokes attended medical school at Edinburgh.

In 1825 shortly before he graduated, he published the first systematic work in the English language on the use of the stethoscope. In other treatises, including that on Diseases of the Chest published in 1837, he described new diagnostic physical signs of pleural effusion and pneumonia and emphasized particularly the importance of correlating symptoms and physical signs in clinical diagnosis. He received international acclaim, including election to the National Institute of Philadelphia. On the death of his father, in 1845, Stokes was confirmed in the place he had held as locum tenens for twenty-seven years as Regius Professor of Medicine in the University of Dublin, a post which he occupied until his death.

His classic volume "The Diseases of the Heart and the Aorta" appeared in 1854 with its description of "Cheyne-Stokes Respiration" in connection with fatty degeneration of the heart. He refers to Cheyne's earlier description but emphasizes its diagnostic importance. Stokes in this volume also referred to his published articles on the slow pulse and its association with pseudo-apoplectic attacks with liability to sudden death now designated as "Stokes-Adams Syndrome" or disease. In this volume he advocates graduated muscular exercise for the rehabilitation of cardiac patients. Adorable descriptions of pericarditis and valvular heart disease are also presented. This work was translated into German, Italian, and French.

Practically all possible honors and degrees were accorded Stokes. Throughout his medical career he maintained close friendship with another illustrious member of the Dublin medical profession, Robert Graves. It was in large measure due to their combined efforts that the system of clinical instruction of the Dublin School of Medicine acquired world-wide fame. The statue of William Stokes, unveiled in 1876, two years before his death, now stands in the hall of the Royal College of Physicians in Dublin.—Ed.
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