TIME RELATIONSHIPS OF THE LEFT ATRIAL V WAVE IN MITRAL VALVULAR DISEASE

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Received November 24, 1960

Blood flowing into the left atrium during ventricular systole causes an increase in pressure known as the "V" wave. From the apex of the "V" wave pressure declines in the "Y" descent. Diagnostic formulae have been based upon the assumption that the summit of the "V" wave marks the opening of the mitral valve and the whole of the "Y" descent represents atrial emptying. This hypothesis is not consistent with the observation that the "V" peak may occur earlier, even before the aortic valve has closed (Luisada and Liu, 1959; Rich, 1959).

The purpose of this communication is to examine the relationship of the left atrial "V" peak to aortic valve closure and mitral opening in patients disabled from chronic rheumatic heart disease.

MATERIAL AND METHODS

Eighteen patients disabled from chronic rheumatic mitral valvulitis were selected for study on the grounds of their freedom from evidence of hypertensive, ischaemic, and aortic valvular disease. Each was examined clinically, cardiographically, and radiologically. In ten the mitral valve was examined at valvotomy or open-heart operation by Mr. G. H. Wooler.

The left heart was catheterized through the fossa ovalis (Ross, 1959) in the manner described by Nixon 1960(a). Indicator dilution curves were obtained by injecting blue dye into the left atrium. Pulmonary and mitral phonocardiograms were recorded with simultaneous brachial arterial and left atrial pressure pulses and the electrocardiogram by the N.E.P. galvanometric camera. Atrial pressure pulses were obtained with Statham P23G transducers through 90 cm. lengths of P.E. 50 catheter (Clay-Adams, Inc.). Brachial arterial pulses were obtained through Courmand needles connected to Statham P23D transducers by 30 cm. lengths of thick walled inelastic tubing of 3 mm. internal diameter. N.E.P. amplifiers were used for sound and pressure recording. By exploding a balloon inflated over the end of a catheter held close to the phonocardiograph microphone it was possible to compare transmission times from catheter tip and microphone to the camera, and to measure the time taken for the catheter-transducer-amplifier-galvanometer system to record an instantaneous fall from full scale deflection to zero pressure.

All records were obtained at a paper speed of 80 mm. per second during an expiratory pause. High or medium frequency phonocardiograph filters (Leatham, 1952) were employed. Aortic valve closure was assumed to be responsible for the first vibrations of the second heart sound in the pulmonary area. Time intervals were measured with a lens and graticule to the nearest 1/100 mm. from the onset of aortic valve closure vibrations. Measurements were averaged for at least ten beats and expressed to the nearest 1/100 second. The opening snap was identified by its high frequency vibrations occurring later than aortic and pulmonary valve closure transients. The third heart sound in mitral regurgitation occurs at the time of the annular ascent point of the left atrial pulse (Nixon, 1961). This feature prevented any possibility of confusion between a third heart sound and the earlier mitral opening snap.

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From the information obtained at clinical examination, special investigation, and operation it was possible to diagnose mitral stenosis in three cases, mitral stenosis with regurgitation in five, and mitral regurgitation in ten patients. Those with pansystolic mitral murmurs were considered disabled from mitral regurgitation when the diastolic murmur began explosively with a third sound at the annular ascent point (Nixon, 1961), when left atrial stasis waves occurred in diastasis (Nixon and Wooler, 1961), and when the long diameter of the mitral valve measured 2·0 cm. or more (Nixon and Wooler, 1960). Disability was attributed to mitral stenosis with regurgitation when the diastolic murmur began quietly immediately after the opening snap, when the pressure gradient across the mitral valve persisted throughout the longest diastolic periods, and when the long diameter of the mitral orifice measured 1·5 cm. or less.

RESULTS

Heart rhythm, left atrial “v” peak pressure, and the findings at operation are presented in the table. Time intervals from aortic valve closure to left atrial “v” peak and mitral opening snap onset, and from “v” peak to mitral opening snap onset are also tabulated.

TABLE I
HEART RHYTHM, LEFT ATRIAL “v” PEAK PRESSURE, TIME INTERVALS, AND FINDINGS AT OPERATION

<table>
<thead>
<tr>
<th>Case</th>
<th>Heart rhythm</th>
<th>Pressure at left atrial “v” peak (mm. Hg)</th>
<th>Operative findings</th>
<th>Aortic closure to “v” peak interval (seconds)</th>
<th>Aortic closure to mitral opening snap interval (seconds)</th>
<th>Left atrial “v” peak to mitral opening snap interval (seconds)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>A.F.</td>
<td>47</td>
<td>—</td>
<td>0·25</td>
<td>0·05</td>
<td>0·025</td>
</tr>
<tr>
<td>2.</td>
<td>A.F.</td>
<td>17·5</td>
<td>1·25 cm. orifice.</td>
<td>0·03</td>
<td>0·075</td>
<td>0·045</td>
</tr>
<tr>
<td>3.</td>
<td>S.R.</td>
<td>24</td>
<td>1·7 cm. orifice.</td>
<td>0·08</td>
<td>0·1</td>
<td>0·08</td>
</tr>
<tr>
<td>4.</td>
<td>S.R.</td>
<td>23</td>
<td>—</td>
<td>0·02</td>
<td>0·07</td>
<td>0·05</td>
</tr>
<tr>
<td>5.</td>
<td>A.F.</td>
<td>30</td>
<td>No regurgitation.</td>
<td>0·02</td>
<td>0·1</td>
<td>0·1</td>
</tr>
<tr>
<td>6.</td>
<td>S.R.</td>
<td>23</td>
<td>0·8 cm. orifice.</td>
<td>—</td>
<td>0·02</td>
<td>0·02</td>
</tr>
<tr>
<td>7.</td>
<td>A.F.</td>
<td>38</td>
<td>Regurgitant jet</td>
<td>0·04</td>
<td>0·04</td>
<td>0·02</td>
</tr>
<tr>
<td>8.</td>
<td>A.F.</td>
<td>27</td>
<td>1·5 cm. orifice.</td>
<td>0·04</td>
<td>0·07</td>
<td>0·03</td>
</tr>
<tr>
<td>9.</td>
<td>A.F.</td>
<td>30</td>
<td>—</td>
<td>0·03</td>
<td>0·08</td>
<td>0·05</td>
</tr>
<tr>
<td>10.</td>
<td>A.F.</td>
<td>44</td>
<td>—</td>
<td>0·01</td>
<td>0·06</td>
<td>0·05</td>
</tr>
<tr>
<td>11.</td>
<td>A.F.</td>
<td>46</td>
<td>—</td>
<td>0·04</td>
<td>0·08</td>
<td>0·04</td>
</tr>
<tr>
<td>12.</td>
<td>A.F.</td>
<td>32</td>
<td>—</td>
<td>0·04</td>
<td>0·05</td>
<td>0·01</td>
</tr>
<tr>
<td>13.</td>
<td>A.F.</td>
<td>50</td>
<td>—</td>
<td>0·03</td>
<td>0·05</td>
<td>0·02</td>
</tr>
<tr>
<td>14.</td>
<td>A.F.</td>
<td>30</td>
<td>2·5 cm. orifice.</td>
<td>0·01</td>
<td>0·09</td>
<td>0·08</td>
</tr>
<tr>
<td>15.</td>
<td>A.F.</td>
<td>33</td>
<td>Severe regurgitation</td>
<td>0·02</td>
<td>0·1</td>
<td>0·1</td>
</tr>
<tr>
<td>16.</td>
<td>A.F.</td>
<td>25</td>
<td>4·5 cm. orifice.</td>
<td>0·02</td>
<td>0·07</td>
<td>0·05</td>
</tr>
<tr>
<td>17.</td>
<td>A.F.</td>
<td>50</td>
<td>Severe regurgitation</td>
<td>0·02</td>
<td>0·07</td>
<td>0·05</td>
</tr>
<tr>
<td>18.</td>
<td>A.F.</td>
<td>62</td>
<td>Severe regurgitation</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
</tbody>
</table>
LEFT ATRIAL V WAVE IN MITRAL DISEASE

Fig. 1.—Mitral stenosis. Simultaneous pulmonary and mitral phonocardiograms, brachial arterial and left atrial pressure pulses, and electrocardiogram. 1, 2=first and second heart sounds. 0=mitral opening snap. a, c, v, and x, y=left atrial "a", "c", "v" waves and "x" and "y" descents.


Fig. 2.—Mitral stenosis with regurgitation. Simultaneous pulmonary and mitral phonocardiograms, left atrial pressure pulse and electrocardiogram.

T.M.=tricuspid followed by mitral valve closure. M.F.=medium frequency. (Other symbols as for Fig. 1.)
Fig. 3.—Mitral regurgitation. Simultaneous pulmonary and mitral phonocardiograms, left atrial pressure pulse, and electrocardiogram. $3=$third heart sound and onset of a loudly beginning diastolic murmur. (Other symbols as for Fig. 1 and 2.)

Fig. 4.—Mitral stenosis. Simultaneous left atrial, left ventricular and brachial arterial pressure pulses obtained with equisensitive transducers at cardiac catheterization. Left ventricular pressure falls below atrial at a point later than the apex of the "v" wave. This point corresponds with the mitral opening snap.
The balloon experiment proved the recording system delayed pressure events 5 milliseconds longer than sound events. Correction has been made for this lag in the table. The experiment also showed that the pressure system required 9 milliseconds to record an instantaneous fall from full scale deflection to zero pressure.

In one case of mitral stenosis with regurgitation the apex of the "v" wave occurred 0.002 sec. before aortic valve closure. In the remaining seventeen patients the "v" peak was recorded 0.01 to 0.04 sec. after aortic closure. The interval did not appear to be related to the nature of the mitral valvular lesion.

Opening snap vibrations began 0.04 to 0.1 sec. after aortic valve closure, and 0.01 to 0.1 sec. after the peak of the left atrial "v" wave. In nine patients the latter interval measured 0.05 sec. or more.

Fig. 1, 2, and 3 are examples of tracings from patients with mitral stenosis; mitral stenosis with regurgitation; and mitral regurgitation respectively.

**DISCUSSION**

Wood (1956) considered that atrioventricular valve opening was responsible for initiating the atrial "y" descent. Fox et al. (1956) and Marshall and Wood (1958) agreed with his belief, which is surprising, because their records of simultaneous left atrial and ventricular pressure pulses showed ventricular pressures falling below atrial at points appreciably later than the peak of the "v" wave. Tracings published by Braunwald et al. (1955) and Luisada and Liu (1959) showed similar features which agree with the present finding that mitral opening snaps occur 0.01 to 0.1 sec. after the peaks of the left atrial "v" waves (Fig. 4).

These observations raise a question of importance, namely, by what mechanism is left atrial pressure reduced before the mitral valve opens? If deceleration of regurgitant flow in the period of reduced ejection is responsible, it is likely that the "v" peak would occur earlier in relation to aortic closure in patients with mitral regurgitation. This does not appear to be the case. Again, if reflux of blood from atrium into pulmonary veins was responsible, some association with regurgitation rather than stenosis might be expected. Flow from atrium to ventricle through a rigid mitral orifice before the aortic cusp snapped into the "open" position is a possible explanation. The absence of any relationship between the extent of the fall and the size of the mitral orifice suggests that this is not the correct explanation.

Radner (1957 and 1958) eloquently described active left ventricular dilatation preceding the closing of the semilunar cusps and the opening of the mitral valve. Active dilatation of the atrioventricular "ring" appears to be the most reasonable explanation for the decline in left atrial pressure between the peak of the "v" wave and the mitral opening snap.

Morrow et al. (1957) and Neustadt and Shaffer (1959) attached diagnostic importance to the rate of "y" descent in the first tenth of a second from the peak of the "v" wave, believing that it was related to the rate of atrial emptying through the mitral orifice. The failure of their formulae to distinguish the various grades of mitral regurgitation may stem from the fact that the first 0.1 sec. of "y" descent can take place before the mitral valve opens.

Left atrial pressure pulses may now be obtained through the atrial septum quite simply at cardiac catheterization (Ross, 1959; Nixon, 1960(a)). Their interpretation is not so simple. In fact, if the heart rate is not slow enough for a rise in left atrial pressure to be demonstrated during the period of diastasis (Morrow et al., 1957; Nixon and Wooler, 1961), it may be impossible to distinguish cases of pure stenosis from those with severe regurgitation. Some of the reasons have been discussed previously (Nixon, 1960(b)). When Luisada and Liu (1956) and Rich (1959) claimed that the "v" peak occurred sooner in mitral regurgitation than in mitral stenosis it was hoped that the relationship in time of "v" peak to aortic closure would have diagnostic value. In the present series of cases the interval appeared to be unaffected by the presence of regurgitation. The series is small and influenced by selection, but it is unlikely that a characteristic of practical value would have failed to appear.
Rich (1959) found mitral insufficiency shortened the second sound—opening snap interval. It did not appear to do so in the present series of cases.

Julian and Davies (1957) recorded wedged pulmonary artery pressures simultaneously with phonocardiograms and observed that “v” peaks preceded the opening snaps. They considered this an impossibility and attributed their findings to artefact. It is likely that such difficulties will persist so long as wedged pulmonary artery pressures are substituted for left atrial pressure pulses.

### SUMMARY AND CONCLUSIONS

Heart sounds and left atrial pressure pulses were recorded simultaneously in eighteen patients disabled from mitral stenosis, mitral stenosis with regurgitation, or mitral regurgitation.

Diagnostic criteria and methods of investigation and measurement are described in detail sufficient for the work to be repeated.

With one exception, using the method described, left atrial “v” peaks occurred 0.01 to 0.04 sec. after aortic valve closure; and 0.01 to 0.1 sec. before mitral opening snap vibrations.

These time relationships were not apparently affected by the presence of moderate or severe mitral regurgitation.

It is suggested that active dilatation of the atrio-ventricular ring causes left atrial pressure to fall from the apex of the “v” wave to the point at which the mitral valve opens.

Diagnostic formulæ that relate the first 0.1 sec. of “y” descent to atrio-ventricular blood flow are based upon the apparently incorrect assumption that left atrial “v” wave peaks coincide with mitral valve opening.

The presence of mitral regurgitation does not appear to shorten the interval between the second heart sound and the mitral opening snap.

I am deeply indebted to Mr. G. H. Wooler, Professor R. E. Tunbridge, Dr. J. R. H. Towers, and Dr. W. Whitaker for making this work possible, and for their support and encouragement.

Mr. R. Addyman, Staff-Nurse E. M. Kirkbride and Mr. H. M. Snow are to be thanked for their enthusiastic assistance.

The Board of Governors of the United Leeds Hospitals, the Trustees of the Nuffield Foundation, the Medical Research Council and private benefactors have generously provided funds for equipment and technical assistance.

### REFERENCES


