The loud first heart sound in left atrial myxoma

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SUMMARY The interrelation between the loudness of the first heart sound, the time interval from the Q wave to the onset of the first heart sound (QM₁), and the mitral valve closure rate was studied in nine patients presenting with left atrial myxomata. In seven patients the first heart sound was loud preoperatively and was associated with delayed mitral valve closure. After removal of the myxoma the onset of mitral valve closure returned towards normal, the mitral valve closure rate was reduced, and the first heart sound became softer. In two patients the first heart sound was normal before and after operation as were both the time of onset of mitral valve closure and the mitral valve closure rate. In neither of these patients did the myxoma completely fill the mitral orifice during diastole.

The loud first heart sound in left atrial myxoma is a useful clinical sign, and its intensity is directly related to the delay in onset of closure of mitral leaflets.

Although left atrial myxomas are rare, establishing a diagnosis is important since the risk of embolisation is high and surgical removal is usually successful with a low morbidity and mortality. Their presence may be suggested by symptoms produced by systemic emboli or by signs mimicking mitral stenosis. We were impressed by the presence of a loud first heart sound associated with left atrial myxoma and investigated the mechanism and significance of this physical sign in the last nine patients presenting to our two hospitals.

Patients and methods

Table 1 shows the relevant clinical features of the nine patients.

High frequency phonocardiograms were recorded at the mitral area, and the ratio of the mitral component of the first sound (M₁) to the aortic component of the second sound (A₂) was calculated. This ratio gives a relative indication of the intensity of M₁ and is particularly useful for comparisons of the intensity of the first heart sound within the same patient.

The interval from the onset of the QRS complex to the onset of the first high frequency vibrations of M₁ was measured (QM₁). In addition, the PR interval was measured in patients in sinus rhythm.

The rate of closure of the anterior leaflet of the mitral valve was measured from the echocardiogram. This measurement was made on the B-C slope of mitral valve motion when the cusps are closing under the influence of ventricular systole.

All the above measurements were made over a minimum of three cardiac cycles and the mean value determined.

A paired t test was used to compare the preoperative and postoperative results.

Results

In seven of the nine patients both the preoperative and postoperative echocardiograms and phonocardiograms were available for study. In two patients (cases 6 and 7) only preoperative data were available.

The patients were considered in two groups: those with a loud first heart sound (cases 1–7) and those whose first heart sound was soft (cases 8 and 9).

All the results are shown in Table 2.

CASES 1–7

Intensity of M₁—The amplitude of A₂ was used as a means of comparing the preoperative and postoperative intensity of M₁ in the five patients in whom this information was available. In these patients the mean value of M₁:A₂ fell from 2.92 preoperatively to 0.98 after removal of the myxoma (Table 2). Using a paired t test the preoperative intensity of M₁ was statistically significantly greater than the postoperative recordings (p<0.01, n=5). In the remaining

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two patients (cases 6 and 7) the first heart sound was
described as loud, and the M1:A2 ratios of 2.63 and
4.75 respectively confirm this.

**PR interval**—This has a major influence on the
intensity of the first heart sound. It was therefore
measured in the patients who were in sinus rhythm.
There was no significant variation between the
preoperative and postoperative measurements
(p=0.09, n=4).

**QM1 interval**—In the five preoperative tracings
the mean QM1 interval was 100 ms (range 90–120 ms).
After operation the mean QM1 interval fell to 64 ms
(range 60–70 ms). The preoperative and postoperative
measurements were significantly different
(p<0.01, n=5). In the patients in cases 6 and 7 the QM1
time interval was 90 and 100 ms respectively.

**Mitrval valve closure rate**—Using a paired t test, the
preoperative and postoperative mitral valve closure
slope (mean 535 mm/s) was significantly greater than
that measured postoperatively (mean 191 mm/s
(p<0.01, n=5) (Table 2).

**CASES 8 AND 9**

In these two patients the preoperative measurements
of QM1, ratio of M1:A2, and mitral valve closure rate
were similar to the postoperative measurements. The
preoperative ratios of M1:A2 were within the range
regarded as normal.7

The notable echocardiographic feature of these two
patients was the preservation of the normal diastolic
pattern of motion of the mitral leaflets (Fig. 1) rather
than the abnormal pattern typically seen with left
atrial myxoma (Fig. 2). In the two patients with a
normal M1:A2 ratio long axis parasternal views using
cross sectional echocardiography showed that the
tumour mass did not completely fill the mitral valve
orifice in diastole.

Figure 3 shows the results of the M1:A2 ratio, the
QM1 time interval, and the mitral valve closure rate in
the nine patients before and after operation.
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Fig. 1 Case 8: echocardiogram showing the tumour immediately behind the anterior leaflet of the mitral valve (AMV). Much of the space between the anterior leaflet and the posterior leaflet (PMV) is not occupied by tumour.

Fig. 2 Case 3: echocardiogram and phonocardiogram of left atrial myxoma. The tumour is seen throughout the cardiac cycle and completely fills the space between the anterior (AMV) and posterior mitral leaflets.

Fig. 3 Relation of $M_1:A_2$ ratio, $QM_1$ interval, and mitral valve closure rate in nine patients (cases 1–9) with atrial myxoma.
Discussion

The concept of “excluding a left atrial myxoma” attracts widespread attention, especially since the left heart is a not uncommon source of emboli to the cerebral circulation. Echocardiography is now established as the best technique for the diagnosis of left atrial masses, and the ease with which it can be used has led to increased demands for its application. In a busy general hospital these demands may put considerable strain on the available service. The importance of recognizing the physical signs associated with a left atrial myxoma is therefore not diminished by the availability of echocardiography.

An extremely loud first heart sound was a notable sign in seven patients with mobile prolapsing myxomas; it was absent in two other patients in whom the echocardiographic findings indicated the presence of a myxoma which did not completely fill the mitral valve during diastole.

Comments on the loudness of the first heart sound in left atrial myxomas have been made in several case reports but in others, although illustrated, the phenomenon seems to have been overlooked. Various explanations have been offered as to the cause of the loud S1 in this condition: there may be more forceful closure of the mitral valve and the loudness of S1 may be due to tumour movement produced either through the checking of the myxoma by its stalk or by it striking the atrial wall and mitral valve. Furthermore a high left atrial pressure alone may be responsible. None of these suggestions completely explain our observations.

The long QM1 interval in myxoma indicates that there is delay in mitral valve closure. Mitral valve closure therefore occurs on a later and steeper part of the left ventricular pressure curve which closes the valve more rapidly than normal. This hypothesis is supported by the observations that QM1 was shorter and the rate of closure of the mitral leaflets was slower after removal of the tumour, at which time the intensity of the first heart sound had returned towards normal. The abnormally long preoperative QM1 interval may be accounted for by the increase in left atrial pressure, which delays the left atrial—left ventricular pressure crossover, and this may be accentuated by the presence of the tumour between the mitral leaflets.

These observations on the variation of the intensity of the first heart sound are consistent with previous findings on the relation between the intensity of the first heart sound and the PR interval. As the PR interval lengths, the QM1 interval shortens and the first heart sound becomes softer. In mitral stenosis following successful mitral valvotomy, when the left atrial pressures may be presumed to be normal, the first heart sound often remains accentuated. In this situation the abnormal fibrotic structure of the mitral cusps is responsible for the loud first heart sound.

In the two patients with a normal first heart sound the M mode echocardiograms indicated that the tumour mass did not completely fill the left ventricular inflow tract. Not only does this permit physiological diastolic motion of the mitral leaflets but it allows the mitral leaflets to close at a normal rate and time.

In conclusion, a loud first heart sound is frequently present in patients with a mobile myxoma of the left atrium and is related to the delay in mitral valve closure. The mechanism of this increased intensity of the first heart sound is similar to that in normal subjects with a short PR interval, when the mitral leaflets are widely separated immediately before the onset of ventricular systole. In the absence of a short PR interval the finding of a loud first heart sound in patients in whom there is a clinical suspicion of a myxoma is an indication for echocardiography. The absence of this sign, however, does not exclude the presence of a left atrial tumour.

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The loud first heart sound in left atrial myxoma

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