MITRAL REGURGITATION WITH MOBILE VALVE CUSPS

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[WITH SPECIAL PLATE]

The essential pathological difference between mitral regurgitation and stenosis as gauged by the gloved finger at operation lies in the damage done to the cusps by the inflammatory process. In pure stenosis, though the free edges of the valve have fused except for an area of about 1 cm. in the middle (Brock, 1952), the cusps are soft, pliable, and mobile, so that they can come into apposition above the aperture during ventricular systole (Fig. 1). In regurgitation, on the other hand,

FIG. 1.—Diagrammatic representation of movements of mitral valve in mitral stenosis and regurgitation. Note that the aperture remains open in each case, closure of the valve taking place above the aperture in stenosis. Very little movement of the cusps takes place in regurgitation, the cusps being converted into a shallow rigid cone with a central aperture.

the cusps are so damaged by rheumatic endocarditis that the resultant rigidity and contracture, with or without calcification, holds the orifice in a gaping position and prevents apposition of the supravulvar area (Fig. 1).

The rigidity of the cusps, together with the gaping orifice, may reasonably be taken to account for the classical heart sounds of mitral regurgitation. The soft quality of the first sound is due to the lack of movement of the cusps in ventricular systole, and the pronounced systolic murmur to the retrograde flow through the gaping orifice, which is often irregular because of calcification. Conversely, the classical heart sounds of mitral stenosis may be attributed to cusp mobility, the loud, abrupt quality of the first sound being due to vigorous propulsion of the cusp, particularly the anterior cusp,

into the atrium by the thrust of ventricular systole (Sellors *et al.*, 1953). From the experiment of palpating the movement of the anterior cusp in cases of pure stenosis while at the same time carrying out direct auscultation with a sterile stethoscope placed over the atrio-ventricular ring I find that the loud ringing first sound coincides exactly in time with the vigorous "ballooning" of the anterior cusp. However, there must clearly be another element responsible for the loud first sound of mitral stenosis, since the most mobile cusps of all are those of the normal heart, and the apical first sound of the latter is less loud than that of mitral stenosis. Wood (1954) suggests that the additional element in stenosis is the pressure gradient across the valve in diastole. This "balloons" the cusps deeply into the ventricle right up to the moment when that chamber contracts. In the normal heart, however, the left ventricle fills quickly, so that towards the end of diastole the mitral cusps tend to float into apposition, and therefore shut more quietly when the ventricle contracts. In other words, the cusps in pure stenosis move from ventricle into atrium in a more explosive fashion than do the normal cusps.

A consideration of these facts points to the conclusion that cusp mobility rather than valve closure is the cause of the characteristic first heart sound in mitral stenosis and that aperture size is only indirectly concerned.

Three cases are now presented which are exceptions to the general rule. In each instance quite gross mitral regurgitation was detected at operation although the valve cusps were soft and mobile. In each case a confident pre-operative diagnosis of pure stenosis was made because of the loud, abrupt first heart sound and the diastolic murmur.

Case 1

A married woman aged 30 years presented with increasing dyspnoea for six or seven years. She had a child at 27 years, and during the pregnancy had been very distressed. A spontaneous delivery occurred, but she had a severe attack of pulmonary oedema on the third post-natal day. She recovered a good deal for six months, but increasing dyspnoea with attacks of nocturnal dyspnoea necessitated admission to hospital. Her exercise tolerance was as follows: walking on the level, 25 yards (22.86 m.); upstairs, 4 steps; no hills; very light housework; no shopping. Pulse, 100 per minute, regular in time and force. Blood pressure, 110/75 mm. Hg. Lungs: widespread crepitations at both bases. Heart: apex beat in 5th interspace in mid-clavicular line; impulse diffuse; no thrill. Heart sounds, mitral area: first sound clear-cut; mid-diastolic murmur with presystolic accentuation; faint systolic bruit not conducted. Radiological examination (Special Plate, A) showed the heart to be enlarged in its general size, but no definite increase in vascular markings in the lung fields. The left atrium was enlarged, the aorta of normal type. A pre-operative diagnosis of pure mitral stenosis was made.

At operation the following findings were noted. A forceful regurgitant jet was palpable coming through the mitral valve. The aperture itself was quite large, measuring 2 cm. in its transverse axis. There was very little induration of either commissure and the anterior cusp was mobile. With a sterile stethoscope placed on the atrioventricular region the same findings were noted as in the ward—namely, a loud, abrupt first sound and a low, rumbling diastolic murmur with presystolic crescendo. The soft systolic murmur noted pre-operatively was not audible at operation. A note to the cardiologist concerned contained the following comment: "The anterior cusp, though shortened, was still mobile, and this was the reason we were led astray. It could be felt to 'flap' with each ventricular systole marking the typical loud first sound."

The functional result in this case has been poor. After initial improvement dyspnoea again became severe, and the patient has recently been admitted to hospital in congestive failure during a pregnancy.

Case 2

A married woman aged 32 complained of increasing breathlessness over five years. On admission her exercise tolerance was: walking on the flat, 100 yards (91.44 m.) slowly; stairs, one flight slowly; light housework. Sleeps with three pillows. An attack of pulmonary oedema with haemoptysis precipitated her admission to hospital. Pulse, 85 per minute. Blood pressure, 130/80 mm. Hg. No cyanosis or venous distension. Lungs clear. Heart: regular rhythm; apex beat in 5th interspace inside mid-clavicular line; no heave. Mitral area: presystolic-diastolic murmur; first sound abrupt and loud; soft systolic bruit not conducted. Radiography of heart (Special Plate, B) showed mitral configuration and marked enlargement of left atrium.

At operation a considerable degree of regurgitation was found to be present. The valve was also stenosed, the aperture measuring about 1 cm. and being ovoid. The anterior cusp was quite mobile, but was apparently unable to occlude the orifice. After splitting the commissures a finger was passed into the ventricle and the area of the chordae tendineae explored. The impression was gained that the free edges of the cusps were tacked down to the ventricular wall and thus prevented from closing.

The functional result in this case has been better. The patient has returned to work in a factory and has walked four miles (6.4 km.) on the flat.

Case 3

This patient, a married woman aged 37 years, complained of fatigue and dyspnoea for a period of six years. She had had one severe attack of nocturnal paroxysmal dyspnoea for which she was admitted to hospital in 1949. On the present admission her exercise tolerance was: walking on flat, one mile (1.6 km.); walking up hills, very breathless. Sleeps with one or two pillows. Pulse, 78 per minute, fibrillating. Blood pressure, 160/90 mm. Hg. Lung fields normal. Heart: apex beat in 5th interspace 4½ in. (11.5 cm.) from midline. Mitral area: diastolic murmur of low intensity; loud ringing first sound terminating in harsh systolic murmur conducted to axilla. Radiological findings are shown at C on the Special Plate.

At operation the mitral orifice was found to be gaping wide open, measuring 2 cm. by 1 cm., and there was free reflux of blood during ventricular systole. The anterior cusp was freely mobile, and it was difficult to be sure of the cause of the regurgitation. It was considered possible that the chordae tendineae attached to the anterior cusp were so shortened that, although the cusp was not itself contracted, it was restrained from coapting with the posterior cusp.

In this case the result has been indifferent. The patient's exercise tolerance has increased somewhat, but she still has precordial compression and a good deal of bronchitis.

Discussion

These three cases demonstrate exceptions to the rule that mitral regurgitation is due to rigid and contracted valve cusps, and that mobile cusps enable the valve to close adequately. They have proved to be the only exception to this rule in a series of 183 valves palpated at operation. In each case a fairly confident diagnosis of pure stenosis was made pre-operatively, because of the loud ringing first sound at the mitral area.

The cause of regurgitation in these cases is uncertain. Belcher (1956) described four similar cases, and suggested that spontaneous rupture of a chorda tendinea was responsible for the regurgitation. However, it was my own impres-

sion on palpating the subvalvular area in the ventricle in these cases that shortening rather than elongation of the chordae had occurred and that "tacking down" of the free edges of the cusps to the papillary muscles was present. Thus the cause of regurgitation may be disproportionate damage to the chordae tendineae with lesser damage to the cusps. The excessive shortening of the chordae would lead to fixation of the free margins of the valve and prevent apposition, but it would not interfere with the sudden "ballooning" of the anterior cusps into the atrium during ventricular systole (Fig. 2).

FIG. 2.—Diagrammatic representation of movements of the valve cusps in the three cases of regurgitation with mobile cusps. Note that disproportionate shortening of the chordae tendineae prevents apposition above the aperture and so allows of regurgitation.

The findings in these three cases raise the question of the cause of the typical heart sounds of mitral stenosis. There is no dispute about the apical diastolic murmurs, but the abrupt quality of the first sound has been taken to be due to violent closure of the valve at the beginning of ventricular systole. The findings here would suggest that it would be more accurate to attribute it to the cusp movement than the valve closure.

As a corollary, it may well be that the opening snap (Rouchès, 1888; Mounsey, 1953) has a similar basis and that it indicates cusp mobility rather than opening of the aperture. Of course, in the majority of cases the two are the same, and from a clinical viewpoint the distinction is academic.

Summary

Three cases are reported in which mitral regurgitation was detected at operation though both valve cusps were soft and mobile. In each case the cusps appeared to be prevented from coming into apposition by disproportionate shortening of the chordae tendineae, and in each case the loud, abrupt first mitral sound characteristic of pure stenosis was present and a pre-operative diagnosis of pure stenosis had been made.

This evidence supports the view that the loud mitral first sound in mitral stenosis is not due to valve closure, but to cusp, and particularly anterior cusp, movement. The opening snap may have the same basis.

I am grateful to Professor I. G. W. Hill, Department of Medicine, University of St. Andrews, for referring these patients for valvotomy.

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W. DRUKKER *ET AL.*: HYPERPLASIA OF ADRENAL MEDULLA

FIG. 1.—Left adrenal, removed September, 1953. Hyperplasia of medulla with elongations between cortex.

FIG. 2.—Right adrenal, removed February, 1956. Very marked hypertrophy of adrenal medulla.

D. M. DOUGLAS: MITRAL REGURGITATION

Antero-posterior and oblique views of the heart. A, Case 1; B, Case 2; C, Case 3.

J. H. KETTLE: POST-MASTECTOMY LYMPHANGIOSARCOMA

FIG. 1.—Lymph channels lined by neoplastic cells; also neoplastic cells occurring singly and in small groups. ($\times 120$.)

FIG. 2.—Neoplastic cells occurring in masses. ($\times 120$.)