MITRAL INCOMPETENCE

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We have investigated 30 adult patients in whom we believe mitral incompetence due to endocarditis was the only cardiovascular abnormality. Ten patients died and the diagnosis has been confirmed at necropsy in nine (see Appendix). Our aim has been to determine the natural history, symptoms, and signs of this uncommon lesion and to evaluate these signs in order to assess the degree of incompetence in the presence of mitral stenosis and of other valve lesions.

HISTORICAL NOTE

Corvisart (1813) and his pupil Laennec (1829) described signs that they attributed to disease of the mitral valve. Both recognized a "purring thrill" and Laennec described a rasping apical murmur, but there is no evidence that they appreciated the timing of these signs in the cardiac cycle. Hope (1849) was the first to relate the systolic murmur and thrill to mitral regurgitation—"when the valve is permanently patescent admitting of regurgitation the first sound likewise is attended with a murmur," and on another occasion, "... when blood regurgitates into the auricle, this cavity suffers in a remarkable degree." For the rest of that century apical systolic murmurs tended to be regarded as organic and caused by mitral regurgitation. A change of attitude to these murmurs must largely be attributed to Graham Steell (1906) and Sir James Mackenzie (1916), though Potain (1894) had criticized English and German authors for believing that functional mitral incompetence accounted for all apical systolic murmurs not due to mitral valve disease. Graham Steell recognized that incompetence frequently accompanied mitral stenosis but doubted if it was of any significance: he stated that it was impossible to give a description of mitral incompetence for it occurred in such a variety of conditions. He continually emphasized the importance of muscle failure rather than primary disease of the valve and said that no one ever died from mitral regurgitation. Mackenzie also held these views. This teaching was continued by Lewis (1933) who said that to regard any distinct systolic apical murmur as meaning a diseased valve was indefensible. Cabot (1926) admitted the existence of mitral incompetence but emphasized its rarity—"even in rare cases wherein mitral regurgitation without stenosis does exist, there are no physical signs by which it can be recognized or reasoned out, so that a diagnosis of mitral regurgitation without stenosis is never justified." This teaching corrected the habit of diagnosing heart disease on hearing a systolic murmur at the apex and prevented much unwarranted cardiac invalidism (Evans, 1948). In the last thirty years many writers (Sprague and White, 1926; Fishberg, 1940; Master, 1948) have considered that the apical systolic murmur has been unjustifiably relegated to an insignificant position. In the Harveian Oration of 1945 Parkinson considered that the diagnosis of mitral incompetence as a rheumatic valvular lesion should be made, though never again as a rival of mitral stenosis.

CLINICAL METHOD

These 30 patients have been seen over a period of three years. With two exceptions physical examination and X-ray screening were carried out by both of us. Electrocardiograms were taken in all cases, and logarithmic (Rappaport and Sprague, 1942) or high frequency (Leatham, 1949) phonocardiograms were recorded synchronously at the mitral and pulmonary areas in all but three. Only patients with a loud mitral systolic murmur (at least of grade four intensity using the classification of Freeman and Levine, 1933) were considered, and they were included if there was

no ischæmic heart disease, hypertension (B.P. over 160 systolic or 100 diastolic, with the exception of necropsy Case 29), or anæmia, and no evidence of other valve lesions. In fact all those where there was a possibility of functional mitral incompetence have been excluded. The patients under consideration were examined on several occasions and excluded if a presystolic or mid-diastolic murmur was heard at any time, unless necropsy showed no mitral stenosis. Patients with a history suggesting rheumatic fever within five years and those under 20 have also been excluded as we wished to confine our observations to those with relatively static disease; likewise patients with bacterial endocarditis have only been included when it was known that there was a loud apical systolic murmur, satisfying the conditions we have mentioned, before the onset of that infection. By rigorously applying these negative criteria we were left with a group that shows a constant pattern of physical signs and clinical behaviour.

CLINICAL FEATURES

The principal clinical features of the 30 patients are shown in Table II. Sixteen were between 50 and 60 years old and the oldest was 75. The natural history of this group was longer and more favourable than that of mitral stenosis where the greatest mortality lies between the ages of 30 and 45 (Baker and Musgrave, 1947). Eight of our patients were known to have a murmur for more than 25 years, the date being settled by an examination for military service in the First World War.

The well-known sex preponderance of women in cases of mitral stenosis (White, 1947) is in contrast with this series in which 25 of the 30 patients were men. Male preponderance in cases of organic mitral incompetence is also seen in Cabot's series of six autopsies where five were in men. We could find no evidence that selection explained this incidence and it appears to be a special feature of the condition. A similar male preponderance is found in isolated aortic stenosis (3 to 1 by Kumpe and Bean, 1948; up to 2.5 to 1 by Karsner and Koletsky, 1947).

A history of rheumatic fever was given by three of the five women, but by only four of the twenty-five men which is much lower than the incidence of 50 to 60 per cent given by various authors for mitral stenosis.

Many authors have drawn attention to the relatively frequent occurrence of bacterial endocarditis in patients with an isolated loud mitral systolic murmur. Thus, Sprague (1930) stated that patients with rheumatic heart disease in whom mitral incompetence persisted without the development of stenosis of marked degree were more likely to acquire bacterial endocarditis than those in whom the valve became obviously stenosed in a few years. He described the necropsy findings in 20 cases of bacterial endocarditis; the mitral valve was involved in 19 of these and in no less than 18 the valve was more than 8 cm. in circumference. Cates and Christie (1951) found stenosis in only 2 of 29 necropsies of lone mitral valve disease in a large series of patients with bacterial endocarditis. Ten of our 30 patients had bacterial endocarditis which was treated with penicillin; two of these had more than one attack and five died (necropsy in Cases 2, 15, 21, and 30). A loud apical systolic murmur had been heard in all before the bacterial endocarditis supervened. Dental extraction or other operative procedures appeared to initiate the infection in three cases. This high incidence of bacterial endocarditis is in contrast with its relative rarity in pure mitral stenosis, and cannot be explained by the longer natural history of mitral incompetence for bacterial endocarditis was not confined to patients in the older age group. The admission to hospital of patients with bacterial endocarditis has resulted in a large number showing this complication, but this does not affect its relative frequency in mitral incompetence compared with mitral stenosis. In view of the considerable risk of bacterial endocarditis in this group we agree with Master (1948) that prophylaxis with penicillin should be used invariably for such patients when undergoing any form of dental or surgical treatment.

Heart failure occurred in nine patients of whom eight died, six of them within a year of their first examination. Only two of the remaining patients complained of dyspnoea on exertion. It seems that heart failure is late in onset, but may progress rapidly in spite of treatment.

Symptoms and Signs. The only symptom of mitral incompetence, uncomplicated by bacterial endocarditis or heart failure, is palpitation and this deserves further comment. It is common experience that palpitation is a frequent symptom of neurotic ill-health. Yet palpitation was the presenting symptom in 15 of our patients and this was found to be due to frequent extrasystoles. Electrocardiograms showed that there were multiple right or left ventricular extrasystoles in all of these patients.

The pulse was usually normal; it was not small as in aortic stenosis or mitral stenosis and was never collapsing, but occasionally it appeared to be large. Five patients showed auricular fibrillation. The increased and sustained apical impulse of an enlarged left ventricle was frequently seen and more often felt, but was never as obvious as it may be in aortic valve disease or hypertension. Eighteen of 25 of our patients had clinical evidence of an enlarged left ventricle. In some a diastolic-filling wave coinciding with the third heart sound was seen and felt at the apex. Accelerated filling of the left ventricle in mitral incompetence has been demonstrated by roentgenkymography (Hirsch and Gubner, 1936). No patient had the palpable accentuated first sound of mitral stenosis or any sign of right ventricular hypertrophy. An apical systolic thrill was felt in 19; this high incidence reflects the selection of patients with loud murmurs.

Auscultation provided the most important evidence of mitral valve disease. The first sound was normal; it could usually be distinguished from the systolic murmur at the apex and was never accentuated as in mitral stenosis: these points were confirmed on the phonocardiogram (Fig. 1 and 2). Furthermore, the delay of the first sound in relation to the QRS complex that is usually

FIG. 1.—Synchronous high (HF) and low (LF) frequency phonocardiograms recorded from the apex (MA) in Case 9. A normal first sound is followed by the long systolic murmur showing a crescendo in late systole. The second sound is included in the systolic murmur at the apex in the high frequency recording but is shown in the low frequency recording. The third sound is accentuated. The time interval in this and subsequent records is 0.2 sec. (and 0.04 sec.).

present in mitral stenosis (Weiss and Joachim, 1911; Cossio and Berconsky, 1943) was not found in this series. The systolic murmur started at the first sound and it was always loud (the significance of the loudness of the systolic murmur could not be assessed in the 21 selected patients, but it was equally loud in each of the 9 necropsy cases). It was loudest at or near the apex radiating to the axilla and left scapula, a feature well known to Gee in 1893; it was sometimes heard in the region of the right scapula. Though loud at the left sternal edge the murmur was much diminished to the

FIG. 2.—Synchronous phonocardiograms, low and high frequency in the mitral area, and high frequency in the pulmonary area (PA). Case 4. There are early systolic vibrations of low intensity (inaudible) of and a late systolic murmur going up to the aortic component, the second sound, though, finishing before the pulmonary component.

right of the sternum and was either absent or faint and never more than grade 2 in the aortic area. The systolic murmur was long (pansystolic), starting immediately after the first sound and always continuing up to and including the second sound (Fig. 1). While it may be difficult to distinguish the length of the systolic murmur by auscultation alone, much reliance may be placed on the apparent absence of the second sound exactly at the apex where the systolic murmur is loudest; the second sound is easily heard in all other areas. When the second sound was split the systolic murmur extended to the aortic component but not to the later pulmonary component; this may explain the statement by Wiggers (1923), that the systolic murmur sometimes finished before the second sound. In some patients the murmur had a crescendo quality and in three it seemed to be confined to late systole; occasionally the late crescendo was preceded by a systolic click (Fig. 3). Phonocardiograms established this close relationship between the systolic murmur and sounds and showed an increase in intensity in late systole in 12 cases (Fig. 1); there were always some vibrations in early systole but these were very small in the three patients in whom only a late systolic murmur was found on auscultation (Fig. 2). One patient was thought to have a late systolic murmur in 1940 which had become pansystolic eight years later (Case 17). One patient who came to necropsy had been diagnosed as having an innocent late systolic murmur (Case 5). Bedford (1945) has emphasized, though without graphic evidence, that late systolic murmurs are not always innocent, and Wood (1950) has stated that they may be a sign of mitral incompetence; their presence in children with rheumatic carditis has been noted by Miller and Wedum (1949). It may at least be stated that a late systolic murmur is no guarantee that the patient will not develop bacterial endocarditis.

The systolic murmur of aortic stenosis may be loud at the apex and may be difficult to distinguish from that of mitral incompetence; in fact three patients who came to necropsy had been diagnosed previously as aortic stenosis. The murmur of aortic stenosis is usually loud in the aortic area and is short and confined to mid-systole, finishing before the aortic second sound (Alzamora-Castro, 1947, Fig. 4). In aortic stenosis of slight or moderate degree when the pulse is normal, the left ventricle not greatly enlarged, and aortic valve calcification absent, attention to the distribution

FIG. 3.—Phonocardiograms recorded from Cases 26 and 20 (Fig. A and B) showing a late systolic murmur preceded by a systolic click (x).

and timing and quality of the murmur can differentiate the two conditions and a phonocardiogram will give confirmatory evidence (Leatham, 1951). Ventricular septal defects may be distinguished by a more centrally placed murmur and the absence of systolic expansion of the left auricle.

Patients with a mid-diastolic or a presystolic (auricular systolic) murmur were excluded from this series (except necropsy cases). On careful auscultation there was a soft, short early diastolic murmur internal to the apex or near the pulmonary area in 13 cases (Fig. 5 and 6). It was present in five cases subsequently examined at necropsy; not one showed disease of the semilunar valves although it is appreciated that this does not exclude a slight functional incompetence, especially of the pulmonary valve. This short early diastolic murmur might sometimes be due to the continuation of mitral regurgitation into diastole after aortic valve closure and until the auricular and ventricular pressures are equalized (Wiggers and Feil, 1921). Such an explanation is supported by the electro-kymographic findings of Luisada and Fleischner (1948) who showed continuation of auricular

FIG. 4.—Phonocardiogram of a patient with aortic stenosis. The systolic murmur finishes before the second sound.

FIG. 5.—Phonocardiogram recorded from Case 6 showing the long systolic murmur and an early diastolic murmur.

FIG. 6.—Two further cases of mitral incompetence illustrating the pansystolic murmur reaching the aortic component of the second sound. In (A) the separate pulmonary component can be seen 0.07 sec. later, and after the dicrotic notch of the carotid (CAR) pulse. The short, early diastolic murmur is shown in (A) and (B) and the third sound at the mitral area in (B). (Case B by kind permission of Dr. Charles Baker.)

expansion after the second sound. Another possible cause is the elastic recoil of the over-distended auricle resulting in a flow through the mitral valve in early diastole.

In mitral valve disease the second sound, which is often split into aortic and pulmonary components, may be followed closely by the mitral snap (opening snap), and slightly later by the third sound marking the entry of blood into the ventricles and initiating the mid-diastolic murmur when this is present. Splitting of the second sound is due to delay in the pulmonary component and is maximal during inspiration; this sound is loudest at the pulmonary area and poorly conducted elsewhere. The width of the split is increased by right bundle branch block and the intensity of the pulmonary component is exaggerated by pulmonary hypertension. Under these circumstances the pulmonary component of the second sound may be mistaken for the mitral snap. This sound, specific to mitral valve disease, is usually loudest internal to the apex and at the lower end of the sternum, but when very loud may be well conducted to the mitral, pulmonary, and aortic areas. It is high pitched, often loud, and resembles the second sound in quality. The mitral snap is maximal in expiration and its timing is not affected by inspiration when splitting of the second sound is greatest. However, the only certain method of distinguishing the mitral snap from the pulmonary component of the second sound is to hear and record all three sounds. Often two "second sounds" can be heard in mitral valve disease but in the pulmonary area and lower left sternal edge during inspiration the first of these two sounds splits into aortic and pulmonary components and the later sound is the mitral snap (Fig. 7). The third heart sound is later in diastole and has an entirely different quality; it is longer, lower pitched, and usually confined to the apex (Fig. 1). The auscultatory signs of pure mitral stenosis are illustrated in Fig. 7.

The second sound in the pulmonary area was frequently split. Thirteen of 20 patients in whom phonocardiograms were recorded in the pulmonary area showed a degree of splitting that was usually greater than normal (Fig. 2 and 6A); none of these had bundle branch block. The first component of this split sound was always thought to be aortic, and it is therefore suggested that left ventricular systole was shorter than normal. The diminished resistance to left ventricular out-flow due to mitral regurgitation is probably the explanation of this shortened systole.

The pulmonary component of the second sound was not increased in intensity in any patient in this series and none showed clinical evidence of pulmonary hypertension.

The mitral snap which is frequently heard in mitral stenosis (Fig. 7) was not heard or recorded

FIG. 7.—Phonocardiogram of a patient with pure mitral stenosis showing a late first sound (nearly 0.1 sec. after R wave), split second sound (during inspiration), opening snap (S) (larger in expiration), and long mid-diastolic murmur (MDM). A=artefact; LSE=lower left sternal edge; MF=medium frequency phonocardiogram.

in a single patient. We conclude that this clear sound is related to the existence of stenosis rather than incompetence of the mitral valve. The third heart sound, which in our experience is rarely heard or recorded in pure mitral stenosis, was audible at the apex in 15 cases (Fig. 1) and shown on the phonocardiogram in all these and in four more in whom it was inaudible; it might have been normal in five who were young. Sprague and White (1926) have noted its frequency in mitral incompetence. It was not possible to relate the third sound to any other signs, but it was probably due to increased filling of the left ventricle.

A short mid-diastolic murmur was heard and confirmed on the phonocardiogram in only one of the necropsy cases (Case 12). This murmur probably arose from turbulence, like the Carey Coombs murmur in cases of acute rheumatic valvulitis, or from increased blood flow as in patent ductus arteriosus or ventricular septal defect. However, an obvious mid-diastolic murmur must exclude the diagnosis of *pure* mitral incompetence.

The principal features of pure mitral stenosis are contrasted with those of pure mitral incompetence in Table I, and the findings in the patients with mitral incompetence are shown in Table II.

TABLE I
THE PRINCIPAL FEATURES OF PURE MITRAL STENOSIS CONTRASTED WITH THOSE OF PURE MITRAL INCOMPETENCE

	Mitral Stenosis	Mitral Incompetence
Sex	Women preponderant	Men preponderant
History of rheumatic fever, etc.	In over half	Uncommon
Bacterial endocarditis	Uncommon	Common
Heart failure	Common	Late
Pulse	Small	Normal or increased
Apex beat	Palpable first sound	Suggests left ventricular hypertrophy
First sound	Loud	Normal
Second sound	Normal at apex	Inaudible at apex, being included in systolic murmur
Opening snap	Frequent	Absent
Third sound	Infrequent	Frequent, often loud
Murmurs	Mid-diastolic Presystolic With or without thrill	Systolic, loud and long : may be maximal in late systole With or without thrill
X-rays		
Enlargement of left auricle	All degrees	All degrees—usually slight or moderate
Systolic backward movement of left auricle	Frequent	Frequent
Systolic expansion of left auricle (as shown by obvious pulsation in all views)	Never	Essential for X-ray diagnosis
Electrocardiogram		
P wave	Frequently large and bifid	Normal
Ventricular preponderance	Tendency to right	Tendency to left

RADIOLOGY

Obvious systolic expansion of the left auricle is the most important radiological sign of mitral incompetence.

In the normal heart left ventricular systole is seen as an inward movement of the apex and left border of the heart; above, the pulmonary artery and aorta move outwards in systole. The right auricle forming the right border of the heart also moves inwards in systole. The right auricular border joins that of the superior vena cava in 80 per cent of normal subjects (Hirsch and Gubner, 1936), and the pulsation here is not very obvious on fluoroscopy. In some normal subjects, especially in the older age groups, the upper right border of the heart is formed by the aorta which moves outwards in systole. In the right and left oblique views the left auricle moves inwards in systole in 90 per cent of healthy subjects; in the rest there is no decisive movement though rarely the pulmonary artery region of the posterior border moves slightly

TABLE II
A SUMMARY OF OUR FINDINGS IN 30 PATIENTS WITH PURE MITRAL INCOMPETENCE

Number	Sex	Age	Length of history in years	Rheumatic fever, etc.	Bacterial endocarditis	Presenting symptom	Heart Failure	Signs at Apex				B.P.	Phono.		Radiology				Electrocardiogram			
								L.V. impulse *	Systolic murmur	Thrill	3rd sound		Systolic murmur	3rd sound	Left auricle enlarged in oblique views	Anterior view	Systolic expansion, left auricle	Left ventricle	Axis	Ventricular preponderance	Rhythm	Ventricular extrasystoles
1	M	50	40	+	0	P	0	-	Long	0	0	160/100	FS	0	+	+	+	L	LV+	N	+	
2†	M	54	5	+	0	HF	0	0	Long	+	+	145/95	FS	+	+	+	+	L	LV+	N	0	
3†	M	75	36	0	0	HF	0	+	Long	+	+	130/75	-	+	+	+	+	L	R.BBB	AF	0	
4	M	52	2	0	0	P	0	+	Late	0	0	160/80	FS ML	0	+	+	+	L	0	N	0	
5†	M	68	35	0	0	P	0	+	Late	+	+	125/80	FS ML	+	+	+	+	L	LV+	AF	+	
6	M	63	2	0	0	P	0	+	Long	+	+	140/70	FS	0	+	+	+	L	LV+	N	+	
7	M	50	36	0	0	P	0	+	Long	+	+	150/85	FS	+	+	+	+	L	LV+	AF	+	
8	M	35	10	0	0	P	0	+	Long	+	+	110/60	FS ML	+	+	+	+	L	0	N	0	
9	M	25	15	+	0	P	0	+	Long C	+	+	160/80	FS ML	+	+	+	+	L	0	N	0	
10	M	36	24	+	+	SBE	0	+	Long	+	+	125/70	FS ML	+	+	+	+	L	0	N	0	
11	M	55	2	0	0	P	0	+	Long	+	+	130/80	FS	+	+	+	+	L	0	N	0	
12†	M	55	9	0	0	P	0	+	Long	+	+	155/75	FS	+	+	+	+	L	0	N	0	
13†	M	52	8	0	0	P	0	+	Long	+	+	115/80	-	+	+	+	+	L	LV+	AF	+	
14	M	61	35	0	0	P	0	+	Long C	+	+	150/80	FS	0	+	+	+	L	LV+	N	+	
15†	M	56	2/12	0	0	HF	0	+	Long	0	0	110/75	FS	+	+	+	+	L	0	N	0	
16	F	57	41	0	0	SBE	0	+	Long C	0	0	150/90	FS ML	+	+	+	+	L	0	N	0	
17	F	52	30	0	0	P.SBE	0	+	Long	+	0	130/80	FS	0	+	+	+	L	LV+	N	+	
18	M	36	24	+	0	D	0	+	Late	+	+	150/70	FS ML	+	+	+	+	L	0	N	0	
19	M	60	4	+	0	P	0	+	Long	0	0	140/90	FS ML	0	+	+	+	L	0	N	0	
20	M	54	32	+	0	P	0	+	Long C	0	0	160/80	FS ML	0	+	+	+	L	LV+	N	0	
21†	F	23	9/12	+	+	HF	0	+	Long	+	+	110/60	-	+	+	+	+	L	0	N	0	
22	M	24	17	0	0	SBE	0	+	Long	+	+	120/65	FS	+	+	+	+	L	LV+	N	0	
23	F	49	28	+	0	SBE	0	+	Long	0	+	125/80	FS	+	+	+	+	L	LV+	N	0	
24	M	59	35	0	0	P	0	+	Long	0	+	160/80	FS ML	+	+	+	+	L	LV+	N	0	
25	M	67	1	0	0	0	0	+	Long	0	0	145/85	FS	+	+	+	+	L	LV+	N	0	
26	M	65	34	0	0	0	0	+	Late	0	0	160/100	FS ML	+	+	+	+	L	LV+	N	0	
27	M	58	7	0	0	SBE	0	+	Long C	0	0	150/90	FS	0	+	+	+	L	LV+	N	0	
28	M	65	5	0	0	HF	0	+	Long C	+	+	120/85	FS ML	+	+	+	+	L	0	N	0	
29†	M	58	35	0	0	HF	0	+	Long	+	+	150/105	FS	+	+	+	+	L	LV+	AF	0	
30†	M	63	37	0	0	SBE	0	+	Long	+	0	130/80	FS	0	+	+	+	L	0	N	0	

† = necropsy
P = palpitation
HF = heart failure
BE = bacterial endocarditis

D = dyspnoea
C = crescendo in late systole
ML = maximal vibrations in late systole

FS = fills systole
N = normal
L = left axis deviation
R = right axis deviation
AF = auricular fibrillation

-- = unrecorded

* = clinical estimation of left ventricular hypertrophy

backwards in systole (Lenègre *et al.*, 1942). Occasionally systolic uncoiling of the aorta causes a movement of the oesophagus posteriorly and upwards but this can be related to excessive movement of the aorta when viewed in the left oblique position. These movements of the normal left auricle are slight when related to the degree of activity of the whole heart.

Systolic expansion of the left auricle was appreciated as a clinical sign in a case described by Owen and Fenton (1901) where systolic pulsation of the right side of the chest was due to massive enlargement of the left auricle. Necropsy showed a grossly dilated mitral valve ring. In the same year Holzknecht, referring to the radiological appearance of the left border of the heart, said that "under pathological circumstances one sees not uncommonly an obvious middle prominence with arterial pulsation . . . one should remember mitral incompetence which produces positive pulsation of the left auricle." In an early textbook of cardiac radiology, Schwarz (1911) noted systolic filling of the left auricular appendix in mitral valve disease. Bedford (1927) observed on radioscopy that there was vigorous systolic pulsation of the left auricle from mitral reflux in two cases of mitral valve disease. Rapid serial full size X-ray films were used by Chamberlain and Dock (1927) to show sudden dilatation in early systole of the left atrium on the left border of the heart in a patient with mitral incompetence which was confirmed at necropsy. In the same year Mahaim (1927) observed paradoxical pulsation of the right and left auricles on the right border of the heart, and thought that this movement was due to thinning of the muscle wall of the left auricle and was occurring in auricular systole. With the introduction of roentgenkymography, Stumpf (1936) recorded systolic bulging of the

left auricular zone on the left border of the heart in about one-third of cases of mitral valve disease: he did not think that this indicated mitral incompetence in view of the proximity of the conus arteriosus and great vessels which normally move outwards in systole. Lemke (1939) disagreed with Stumpf; he showed by roentgenkymography systolic expansion of the left atrium on the right border of the heart in the anterior view in three cases, and attributed it to mitral incompetence. The same conclusion was reached by Gubner *et al.* (1939), Laubry *et al.* (1939), and Routier and Heim de Balsac (1942). Lenègre *et al.* (1942) examined 600 patients by fluoroscopy for pulsation in the oblique views: of 69 cases of mitral valve disease 51 had abnormal left auricular pulsation. In the right oblique position a barium swallow showed that the posterior border of the heart from the pulmonary artery (or left bronchus) impression almost to the diaphragm moved backwards in systole. In the left oblique position the same movement was seen, usually less clearly, and could be contrasted with forward movement of the left ventricle. They accepted this "systolic retropulsion" of the left auricle as evidence of mitral incompetence although it sometimes occurred in mitral stenosis with no systolic murmur. They did not consider systolic expansion of the left auricle in the anterior view because they assumed that it could only be seen on the right border of the heart when there was aneurysmal dilatation of the left auricle. By means of roentgenkymography (Heim de Balsac and Pannier, 1945; Routier *et al.*, 1950), and by electrokymography (Luisada and Fleischner, 1948; Lian *et al.*, 1948; McKinnon and Friedman, 1950; Soulié *et al.*, 1950) systolic expansion of the left auricle has been demonstrated, usually in the oblique views, in almost all cases of mitral valve disease even in the absence of a systolic murmur. The findings of auscultation and of radiology have been so much at variance that Froment *et al.* (1950) have denied that systolic expansion of the left auricle is a sign of mitral incompetence. They quoted one case; this patient showed systolic expansion although subsequent necropsy revealed a stenosed mitral valve which was competent to their water test (Froment *et al.*, 1945). They postulate that systolic expansion of the left auricle is due to displacement of blood when the fibrotic mitral valve and ring is forced like a piston deep into the left auricle. Standard textbooks of cardiac radiology tend to support the view that systolic expansion of the left auricle is one of the signs of mitral incompetence (Roesler, 1943; Schwedel, 1946; Zdansky, 1949). Systolic expansion of the left auricle following a rupture of chordæ tendineæ has been seen radiologically by Bailey and Hickham (1944), and at operation by Brock (1950). Heim de Balsac and Pannier (1945) noted that it was less likely to be seen when the left auricle was greatly dilated.

FIG. 8.—Anterior view of Case 6 showing enlargement and systolic expansion of the left auricle. The arrows indicate the direction of pulsation in ventricular systole.

FIG. 9.—Angiocardiogram of Case 6 showing enlargement of the left auricle. The enlarged left ventricle is seen below the left auricular bulge.

FIG. 10.—Kymogram of Case 6. The systolic inward movement of the left ventricle (A) may be contrasted with the simultaneous outward movement of the left auricle (B). An enlargement of the right border shows the paradoxical movement of the right (A) and left (B) auricles.

Systolic expansion of the left auricle was found in 25 of our 30 patients on fluoroscopy, including four who came to necropsy. The remaining 5 patients died without being examined for this sign. We have not been concerned with slight pulsation or with that which could only be found by kymography because such pulsation occurs in many cases of predominant stenosis. In the anterior view the left auricle expanded laterally in systole appearing as a momentary local bulge high up on the left border, and as a less obvious bulge in the upper part of the right border (Fig. 8, 9 and 10); the lower part of the right border, the right auricle, was seen to move in the opposite direction, i.e. inwards in systole. Sometimes a paradoxical movement of the left auricle could be seen through the right auricular shadow (Fig. 8 and 9). In the oblique views the barium-filled œsophagus was moved backwards by the left auricle during ventricular systole. This backward movement of the left auricle, best seen in full inspiration (Lenègre *et al.*, 1942) is not a sign of mitral incompetence for it is present in many cases of pure mitral stenosis. In our patients the left auricular movement was usually of greater degree than in mitral stenosis and was out of proportion to the degree of activity of the rest of the heart; it was always visible in the anterior view. In fact, we do not use the term systolic expansion without seeing this movement both in the anterior and oblique views, and believe that this condition must be satisfied to make a firm radiological diagnosis of mitral incompetence. Absence of systolic expansion, however, cannot be taken to mean that there is no regurgitation as has been emphasized by Heim de Balsac and Pannier (1945).

Enlargement of the left auricle was found in 26 of our 30 cases (Fig. 9 and 11); it varied from slight to moderate and it was never aneurysmal as in some cases of combined mitral stenosis and

A

B

FIG. 11.—(A) Anterior view of Case 5 showing the convex left auricular bulge on the left border of the heart, and enlargement of the left ventricle. (B) Right oblique view of Case 6 showing moderate enlargement of the left auricle posteriorly.

mitral incompetence. The remaining four patients showed no enlargement though systolic expansion was clear. Kuttner and Markowitz (1948) considered that a diagnosis of mitral incompetence was justified in children even in the absence of enlargement. In two cases the enlarged left auricle was seen posterior to the barium-filled œsophagus. Enlargement of the left ventricle in mitral incompetence has been described by many authors (Steel, 1929; Stumpf, 1936; Roesler, 1943; Zdansky, 1949), and we confirmed this in 24 of our 30 cases (Fig. 11A). In four patients there was increased pulsation of the hilar vessels, possibly the veins and, if so, comparable to the jugular pulse in tricuspid incompetence, a feature noted by Schwarz (1911) and recently by Routier and Heim de Balsac (1942).

ELECTROCARDIOGRAPHY

The electrocardiogram confirmed that there was auricular fibrillation in eight patients and sinus rhythm in the remainder. Multiple ventricular extrasystoles were recorded in twelve patients, the ectopic focus being in either ventricle. The P waves were always normal, none showing the large bifid waves characteristic of mitral stenosis. Left axis deviation was present in one-third of the cases and right axis deviation was never seen. Berliner and Master (1938) noted the shift towards left ventricular preponderance in pure mitral incompetence. Half our patients showed abnormal QRS-T changes indicating left ventricular hypertrophy (Fig. 12). These changes were not so great either in voltage increase or S-T deviation as in aortic valve disease or hypertensive heart disease.

FIG. 12.—(a) Electrocardiographic evidence of some left ventricular hypertrophy in Case 14. One ventricular extrasystole is seen. (b) Electrocardiographic evidence of moderate left ventricular hypertrophy in Case 6. One ventricular extrasystole is seen.

No patient showed cardiographic evidence of right ventricular hypertrophy, though in one case in which the diagnosis of mitral incompetence was confirmed at necropsy there was right bundle branch block. These cardiographic findings contrast with those in mitral stenosis where extrasystoles tend to be auricular, P waves abnormal, left axis deviation rare, and the QRS-T abnormal in right chest leads rather than in those from the left side.

NECROPSY FINDINGS

Confirmation of the clinical diagnosis of pure mitral incompetence was obtained in the nine patients who came to necropsy. Eight died of congestive heart failure and all of these had moderate increase in heart weight which was mainly due to hypertrophy of the left ventricle though these showed slight hypertrophy of the right ventricle also. The left auricle was moderately dilated but was never aneurysmal and the posterior wall above the mitral valve frequently (4 of 9) showed an area of pathological endocardium—the “MacCallum patch” (Fig. 13). We think that this is the effect of the regurgitant jet rather than the direct result of the rheumatic endocarditis. One patient died of coronary embolism without antecedent heart failure (Case 30) and showed moderate

left ventricular hypertrophy with no abnormality of the right ventricle. The mitral orifice was of normal size in one case and dilated in the remainder (Fig. 14). All showed thickening and deformity of the valve cusps with some calcification in two. The chordæ tendineæ were thickened in six and in two of these a chorda was ruptured (Fig. 15). Histological evidence of active rheumatism with Aschoff nodules was found in only one patient, the youngest in the series (Case 21). All the remainder showed varying degrees of scar tissue formation which might be regarded by some histologists as the legacy of past rheumatism though there was no certain evidence of this.

SUMMARY AND CONCLUSIONS

Thirty patients believed to have pure organic mitral incompetence have been investigated. The diagnosis was confirmed at necropsy in 9, in whom the clinical features were the same as in the remaining 21.

The natural history of pure organic mitral incompetence is longer and more benign than that of mitral stenosis, except that it is not infrequently interrupted by bacterial endocarditis. The ætiology may be different from that of mitral stenosis in view of its preponderance in the male sex and the rarity of a history of any form of rheumatic fever. Histological evidence of rheumatic carditis is difficult to find; yet this apparent ætiological difference may only lie in the degree of rheumatic reaction. In these general respects there are similarities with isolated aortic stenosis where males preponderate, a history of rheumatic fever is rare, and the natural course is long.

Patients with organic mitral incompetence are free of significant symptoms except in the few with heart failure, but the complaint of palpitation due to multiple ventricular extrasystoles is fairly common. The frequency of bacterial endocarditis in the course of mitral incompetence is an indication for penicillin prophylaxis in all cases undergoing surgical or dental treatment. Heart failure, though late in appearance, tends to be rapidly progressive.

There is no clinical evidence of right ventricular hypertrophy or severe pulmonary hypertension in pure mitral incompetence, and palpation frequently indicates left ventricular enlargement which may be confirmed electrocardiographically and radiologically. The loud apical murmur always fills systole and is often maximal in late systole; it extends up to, and usually embraces, the second sound; occasionally the early vibrations are small when the murmur appears to be confined to late systole on auscultation. Splitting of the second sound in the pulmonary area was wider than normal in some cases and in these the systolic murmur extended to the earlier aortic component but not to the later pulmonary component. The length of the systolic murmur, its position in systole and relationship to the sounds are as important as loudness. The systolic murmur in aortic stenosis may be loud at the mitral area but is well heard at the aortic area and is loudest in mid-systole, finishing before the second sound. The absence of a loud first sound and particularly of the opening snap in patients with mitral incompetence emphasizes the fact that these signs are related to mitral stenosis. The finding of a soft mid-diastolic murmur in one necropsy case of pure mitral incompetence means that this sign does not always indicate stenosis. The third heart sound was frequently present in these cases of mitral incompetence, which is in contrast with its rarity in pure mitral stenosis.

The most important radiological sign of mitral incompetence is obvious systolic expansion of the left auricle which should be seen in both the anterior and oblique views, especially the former. It is important that a slight degree of systolic backward movement in the right oblique view should not be taken to indicate mitral incompetence as it occurs in predominant stenosis, and conversely the absence of auricular systolic expansion does not exclude mitral incompetence if the other physical signs suggest this diagnosis.

Pure mitral incompetence has characteristic physical signs and radiological features. It is clear that this is an uncommon disease: it should never be diagnosed loosely on hearing an apical systolic murmur, but only after due consideration of the special characteristics of the murmur, the other physical signs, and radiological features. The differential diagnosis between pure mitral stenosis

and pure mitral incompetence is summarized in Table I. The presence of the signs that we have described when combined with those of mitral stenosis or other valve lesions help in the assessment of the degree of associated mitral incompetence.

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FIG. 13.—The mitral valve and MacCallum patch in Case 21.

SUMMARY OF THE NINE NECROPSY CASES

Case 2, man, aged 56. Heart weight 620 g. (body weight 60 kg.). No stenosis of mitral valve which easily admitted three fingers (Fig. 14). Both cusps puckered and thickened at contact margins. Chordæ thickened, and group of three or four from anterior cusp torn and free end covered with blood clot (Fig. 15). Other valve cusps normal; pulmonary valve ring 8.5 cm. Left ventricle dilated and hypertrophied (1.5 cm.) (Fig. 15). Right ventricle dilated and hypertrophied (0.7 cm.). Moderate dilatation of left auricle with some hypertrophy; endocardium thickened and wrinkled over posterior wall above the valve ring.

Microscopy of left auricular endocardium near valve ring showed thickening of fibrinoid tissue and infiltration by round cells and occasional plasma cells. Fibrinoid degeneration of small arteries. Left ventricular myocardium near valve ring also showed fibrinoid replacement of muscle cells and scattered small foci of round cells near arteries. No Aschoff nodes seen in any section.

Comment. No definite histological evidence of rheumatic carditis but changes suggestive of quiescent chronic rheumatism. Death due to heart failure and bronchopneumonia.

Case 3, man, aged 75. Heart weight 560 g. (body weight 52 kg.). No stenosis of mitral valve (12.5 cm. circumference). Fibrous thickening with puckering throughout free margins of mitral cusps, calcification in posterior cusp. Slight thickening and no shortening of chordæ tendineæ. Aortic, tricuspid, pulmonary valves normal. Great dilatation of left auricle. Considerable hypertrophy and dilatation of left ventricle, moderate of right ventricle and right auricle.

Fig. 14.—Dilated and thickened mitral valve ring (easily admitting three fingers) in Case 2 viewed from the left auricle.

Microscopy of anterior cusp of mitral valve showed hyaline areas, no cells except scanty spindle fibroblasts. Posterior cusp similar except scanty deposits of fibrin occupying margin of cusp and slight calcification near attachment. Both cusps entirely avascular. Aortic and tricuspid valves normal. Left and right ventricles hypertrophy of muscle-fibres and occasional foci of subendocardial fibrosis. Conspicuous hypertrophy of muscle of left auricle. Separation of fibres by diffuse fibrosis in places.

Comment. There was no definite histological evidence of rheumatic carditis in this case although the macroscopic appearances were suggestive of this condition.

Case 5, man, aged 68. Heart weight 604 g. (body weight 62.3 kg.). No stenosis of mitral valve, orifice 14 cm. in circumference. Fibrous thickening and puckering of posterior cusp and half anterior cusp. Slight thickening without shortening of some chordæ tendineæ. Aortic and other valves normal. Zone of irregular, fibrous thickening of endocardium of left auricle immediately above posterior cusp of mitral valve, and similar patch above anterior cusp. Considerable hypertrophy and dilatation of left ventricle (1.4 cm. thick), and of right ventricle. Great dilatation and very slight hypertrophy of left auricle. Considerable dilatation and hypertrophy of right auricle.

Microscopy of posterior cusp showed thickening by elastic and collagen fibres, without inflammatory infiltration. Cells restricted to a small number of spindle fibroblasts. Similar thickening affected peripheral part of anterior cusp of mitral valve.

Comment. As in Case 3, to which this bears a strong resemblance, there was no histological evidence in support of a rheumatic basis for the mitral lesion. Alternatively the fibrous thickening of the cusps may have been built up by the organization of thrombotic incrustations.

Case 12, man, aged 55. Heart very large. Dilatation of the left auricle, slight hypertrophy of the right ventricle and great hypertrophy of the left ventricle. Gross deformity and thickening of the mitral valve cusps with no stenosis. Other valves normal.

Case 13, man, aged 52. Heart weight 620 g. (body weight 56 kg.). Left ventricle moderately hypertrophied, and left auricle greatly dilated. Right ventricle dilated and slightly hypertrophied. Aortic, pulmonary, and tricuspid valves normal, Mitral valve widely patent (16.5 cm. circumference) with cusps grossly thickened

FIG. 15.—Thickened and ruptured chordæ tendineæ in Case 2. Left ventricular hypertrophy.

but not fused. Valve ring contained calcareous deposits which extended into posterior cusps. Microscopical examination of posterior mitral cusp and neighbouring tissue showed left auricular endocardium grossly thickened by irregular scar tissue and elastosis. Mitral ring vascular and enlarged by fibrosis and calcareous deposits, and among the vessels there were thick walled, muscular arterioles. Mitral cusps thickened by fibrosis.

Comment. This patient died within one year of the onset of heart failure. There were scars of past rheumatism.

Case 15, man, aged 56. Heart weight 518 g. (body weight 50 kg.). No mitral stenosis: very slight thickening of posterior cusp of mitral valve; fibrous vegetation ($3 \times 1.4 \times 1$ cm.) firmly adherent to anterior cusp; ulceration of lower 1.5 cm. of this cusp, with small friable vegetations on its surface; fusion, thickening and ulceration of attached chordæ tendineæ. Pinkish-grey granular thickening (5×3.5 cm.) over posterior wall of left auricle extending upwards from mitral valve. Other valves normal. Moderate hypertrophy without dilatation of left ventricle. Slight hypertrophy and moderate dilatation of right ventricle. Moderate dilatation and hypertrophy of left auricle, and slight of right auricle. Histological changes confined to anterior cusp of mitral valve and posterior wall of left auricle. Mitral cusp showed a thrombotic vegetation containing infected focus. Valve cusp greatly thickened by vascularized fibrous tissue and infiltrated by numerous large mononuclear cells, a few multinucleate giant cells of rheumatic type, and scantier lymphocytes. Proximal part of cusp normal. Endocardium of posterior wall of left auricle unevenly coated with a thin layer of fibrin.

Comment. The presence of preceding rheumatic endocarditis was suggested by the fusion and shortening of the chordæ tendineæ but no definite histological evidence of rheumatic carditis could be found. Bacterial endocarditis caused great injury to anterior cusp of mitral valve, resulting in extreme mitral incompetence and heart failure.

Case 21, woman, aged 23. Heart weight 434 g. (body weight 39 kg.). No mitral stenosis. Thickening (up to 0.3 cm.) of both cusps of mitral valve (10.9 cm. circumference). Succulent grey thickening of free border of anterior cusp, with granulations of similar tissue at attachments of chordæ (Fig. 13). Calcification and rupture of chordæ of this cusp except for one group at posterior commissure. A few coarse grey

rheumatic vegetations fixed to auricular side of attachments of chordæ tendineæ to posterior cusp. Calcified thrombus adherent to a few of these chordæ, three being ruptured. Area (5×2.7 cm.) of confluent grey tough vegetations in endocardium of left auricle above posterior cusp of mitral valve (Fig. 13). Remaining valves normal. Considerable hypertrophy of left ventricle; very slight of right ventricle. Hypertrophy and dilatation of left auricle. On microscopical examination abundant evidence of active rheumatic carditis affecting both cusps of mitral valve and occasional Aschoff nodes both in interventricular septum and walls of both ventricles. Fibrin vegetations on anterior cusp of the mitral valve, no organisms seen.

Comment. Histological evidence of both old and active rheumatic carditis was plentiful in this case and formed the background of a superimposed bacterial endocarditis. The latter, however, appeared to have been subdued by the use of antibiotics, as judged by the appearances of the vegetations and the spleen, though the rheumatic inflammatory process was unabated.

Case 29, man, aged 58. Heart weight 640 g. (body weight 50 kg.). Moderate left ventricular hypertrophy and great dilatation of left auricle. Right ventricle slightly hypertrophied. Mitral valve easily admitted three fingers and thickening of anterior cusp. Chordæ thickened. Other valves normal. On microscopical examination no evidence of old or recent rheumatism.

Comment. This is an example of long standing mitral valve disease without stenosis, associated with great enlargement of the left auricle, which showed systolic expansion. Mild hypertension was possibly a contributory factor but in view of the history and necropsy findings was not the primary cause of mitral incompetence.

Case 30, man, aged 63. Extensive anterior cardiac infarction was found to be due to a septic embolus (*streptococcus faecalis*), with a mycotic aneurysm in the descending branch of the left coronary artery. Heart weight 510 g. (body weight 69 kg.). Slight hypertrophy of left ventricle, none of right. Mitral valve showed no stenosis and cusps thickened, bearing numerous smooth rubbery vegetations. Microscopical examination showed organisms in vegetations, and fibrous thickening of mitral valve cusps in other areas. No definite evidence of present or past rheumatism. Other valves normal.

Comment. The mitral incompetence in this patient was slight, but it resulted in bacterial endocarditis which proved fatal from coronary embolism.

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