Mitral Regurgitation with Rupture of Normal Chordæ Tendineæ

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Surgery for mitral incompetence still presents a challenge despite the great advance made by the introduction of prosthetic valves. In view of the high incidence of late complications of prostheses (Du Plessis *et al.*, 1965), we believe that, whenever possible, the natural valve should be preserved. Unfortunately this can be done only in cases of pure or dominant stenosis and in those of mitral regurgitation due to a localized defect or a grossly dilated annulus. Partial retention of the valve is seldom practicable because of the lack of suitable materials for leaflet substitution and the uncertainty of competency after a long intricate operation. We have, however, been able to retain the valves of six patients with severe mitral regurgitation resulting from ruptured chordæ tendineæ. This paper describes the anatomical features and surgical management of these cases and discusses the probable mechanism of the chordal disruption.

INCIDENCE OF RUPTURED CHORDÆ TENDINEÆ

Up to January 1965, ruptured chordæ were encountered in 23 of 126 consecutive mitral valve operations performed in this unit under cardio-pulmonary bypass (Table I). Of the 23 patients, 8 had undergone a previous closed valvotomy, and mitral regurgitation had either been produced or was aggravated by this procedure. This is often due to the production of chordal rupture, and a Tubb's dilator may even avulse the medial papillary muscle if the blades are opened with the instrument incorrectly placed between this muscle and the ventricular septum (Fig. 1). The degree of regurgitation following such iatrogenic chordal rupture is frequently severe, since, in rheumatic mitral

stenosis, the chordæ to an extensive length of the leaflet edge are often fused. Many authors (Hepper, Burchell, and Edwards, 1956; Osmundson, Callahan, and Edwards, 1961; Menges, Ankeney, and Hellerstein, 1964) regard bacterial endocarditis as the commonest cause of ruptured chordæ, but we were able to implicate this disease in only 4 of our 23 patients. In these 4 instances, areas of calcification or irregular nodularity were suggestive of previous bacterial endocarditis (Fig. 2). In 2 of these, the ruptured chordæ were considered to be the major cause of the mitral incompetence. In 3 cases of rheumatic mitral incompetence, due essentially to leaflet retraction, minor commissural chordæ which were irregularly thickened were ruptured, and it is probable that the rheumatic process was responsible for both the chordal disease and the disruption (Bailey and Hickam, 1944).

The 6 patients with pure severe mitral regurgitation due to major chordal rupture form a group characterized by left atrial dilatation, volu-

TABLE I
INCIDENCE OF RUPTURED CHORDÆ TENDINEÆ FOUND IN 126 CASES DURING OPEN-HEART SURGERY FOR MITRAL VALVE DISEASE

	Open-heart operations	Ruptured chordæ tendineæ	
	No.	No.	Previous closed valvotomy
Pure stenosis	8	0	0
Mixed incompetence and stenosis	94	15	8
Pure incompetence	10	6	0
Endocardial cushion defect with cleft mitral valve	14	2	0
Total	126	23	8

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FIG. 1.—The mitral valve of a patient with severe incompetence following closed valvotomy with a Tubb's dilator. The valve is viewed from the ventricular aspect and has been opened out by dividing the lateral papillary muscle and leaflet. The arrow points to the avulsed anterior head of the medial papillary muscle, which was found detached at operation and is fibrosed and shrunk. Only the tips of the lateral papillary muscles and of the posterior head of the medial muscle are attached to the specimen, because the valve was surgically excised.

minous supple leaflets, dilatation of the mitral annulus, and the absence of fibrotic or inflammatory changes in the chordæ (Fig. 3 and 4). In the 2

cases of endocardial cushion defect complicated by ruptured chordæ there was also no evidence of previous bacterial or rheumatic endocarditis.

FIG. 2.—Surgically removed mitral valve of a patient with rheumatic stenosis and incompetence. A ruptured main chorda from the lateral muscle to the centre of the anterior leaflet was found (arrow). This showed gross and microscopical features of healed bacterial endocarditis.

FIG. 3.—Heart of Case 5, a man of 68, viewed from the atrium. The mitral annulus is dilated. Both leaflets are voluminous, the posterior one shows clearly. An annuloplasty was done at the lateral commissure, and the teflon plaque over which sutures were tied is seen in position. The smaller teflon pledgets close to the free edge at the centre of the posterior leaflet mark the sites where the artificial chordae were fixed to that leaflet.

FIG. 4.—Heart of Case 5, viewed through a window cut in the posterior wall of the left ventricle. The artificial nylon chordae to the medial papillary muscle are visible, those to the lateral disappear to the left behind the cut myocardium. The intact chordae tendineae are thin and healthy in appearance. Blunt stumps of the ruptured chordae can be seen.

TABLE II
AGE, SEX, AND DETAILS OF HISTORY OF 6 PATIENTS WITH PURE MITRAL INCOMPETENCE

Case No.	Age (yr.)	Sex	Date of operation	History of		Time murmur known (yr.)	Duration of serious symptoms (mth.)
				Rheumatic fever	Subacute bacterial endocarditis or trauma		
1	46	F	1/6/61	?	0	25	8
2	41	M	17/11/61	?	0	13	3
3	36	M	4/1/63	0	0	11	6
4	49	M	21/4/64	0	0	32	2½
5	68	M	15/5/64	0	0	6	6
6	33	M	15/6/64	0	0	20	3

TABLE III
SUMMARY OF GROSS PATHOLOGICAL FEATURES

Case No.	Ruptured chordæ	Condition of remaining chordæ	Annulus	Other lesions	Anterior leaflet	Posterior leaflet
1	From lateral and medial muscles to posterior leaflet	Thick commissural chordæ	Moderately dilated	None	Voluminous	Voluminous; edge slightly thickened, rolled and flail
2	From medial muscle to posterior leaflet	Thin, elongated	Moderately dilated	None recognized	Very voluminous	Very voluminous; free edge rolled and flail
3	From medial muscle to posterior leaflet	Long and thin	Grossly dilated	Lateral perforation in posterior leaflet	Very voluminous	Voluminous; thickened, flail medial leaflet edge
4	From lateral muscle to posterior leaflet	Elongated and thin, chiefly posterior	Markedly dilated	Deep lateral cleft posterior leaflet	Voluminous	Voluminous; free edge rolled and flail
5	From lateral and medial muscles to posterior leaflet	Elongated; some nodular atheroma	Grossly dilated	Atheromatous plaques on leaflets	Voluminous; free edge thick	Voluminous; atheroma in free edge; flail
6	From lateral muscle to anterior leaflet	Adjacent to cleft thick and stiff; others long and thin	Grossly dilated	Deep lateral cleft posterior leaflet	Very voluminous and flail	Very voluminous

CLINICAL FEATURES

It is our intention in a later paper to present the detailed clinical and investigatory findings in these 6 patients. For present purposes the relevant features are summarized in Table II. Of the 6 patients, 5 were men: their ages ranged from 33 to 68 but only one was older than 50. No patient gave an unequivocal history of rheumatic fever, though 2 could recall periodic joint pains during their youth. Every patient had known of the existence of a heart murmur for years before the development of serious heart symptoms. In all instances there had been a sudden onset of cardiac symptoms within 6 months of admission to our wards, and 2 (Cases 1 and 5) had rapidly developed congestive cardiac failure necessitating continuous hospitalization from the time of deterioration. Symptoms were pronounced in all, the hearts were radiologically enlarged, and the degree of incompetence was assessed as severe. Investigations for subacute bacterial endocarditis and rheumatic activity were uniformly negative.

PATHOLOGICAL FINDINGS

The 6 patients were operated upon under direct vision with the aid of cardiopulmonary bypass. The

following descriptions are based upon the operative findings and upon the post-mortem appearances of Cases 1 and 5, the hearts of which were preserved. The ruptured chordæ of Cases 3 and 4 were excised during operation and examined histologically.

Gross Pathology. These findings are summarized in Table III. All the valves showed annular dilatation, free commissures, and mobile voluminous leaflets. The intact chordæ, particularly those adjacent to the ruptured ones, were elongated and often thinned. The appearances of the valves were remarkably similar in all cases, the only differing feature being the presence of a cause of incompetence additional to the ruptured chordæ. The posterior leaflet edge and the commissural chordæ of Case 1 were thickened, indicative of rheumatic involvement (Fig. 5). In Case 3, there was a hole 0.5 cm. in diameter lateral to the centre of the posterior leaflet. The margins of this hole were smooth and flexible though slightly thickened, and normal leaflet tissue separated the defect from the ruptured chordæ which arose from the medial papillary muscle. In the absence of any evidence of subacute bacterial endocarditis, it is probable that the defect was congenital, a belief strengthened by the fact that its situation

FIG. 5.—Heart of Case 1 with the left ventricle opened out to display the posterior leaflet. The stumps of the ruptured central chordæ tendineæ are seen to be thin and tapered. Chordæ which arose from both the medial papillary muscle (left) and the lateral papillary muscle have ruptured. The intact chordæ are thickened, suggesting a rheumatic basis.

FIG. 6.—Opened-out normal mitral valve showing deep scallop at site where congenital clefts and the punched-out hole were found in Cases 3, 4, and 6.

was precisely the same as that of the clefts encountered in 2 other patients (Cases 4 and 6). These congenital clefts in the posterior leaflet reached the annulus about a centimetre from the lateral commissure (Fig. 6). Their situation and appearance accord precisely with those of the case reported by Creech, Ledbetter, and Reemtsma (1962), and could be described as a gross exaggeration of the normal scalloping of the leaflet edge (Du Plessis and Marchand, 1964). The edges of the clefts were thickened and tethered by short

FIG. 7.—Microscopical section of chorda close to the site of rupture (Case 4), showing fragmentation and disorganization of collagen bundles but no inflammatory reaction. ($\times 34$.)

chordæ arising from the lateral papillary muscle. Jet lesions were present on the adjacent atrial endothelium. In Case 5, atherosclerotic plaques were present in the chordal insertions and the leaflet edges which were locally retracted. At the time when Case 2 was treated we were unaware of the importance of a minute search for leaflet defects, and this is the only patient in whom no obvious additional cause of incompetence was found.

In Cases 1 and 5 the affected chordæ arose from both papillary muscles; in Cases 2 and 3 the chordæ from the medial papillary muscle, and in Case 4 those from the lateral muscle were affected. In Case 6 alone were the anterior chordæ ruptured; these arose from the lateral papillary muscle (Table III). The commissural chordæ close to the annulus were never involved, it was the central ones that were affected, and these had snapped close to the free edge of the leaflet. Tapered stumps attached to both leaflet and papillary muscle were always identifiable (Fig. 4 and 5). The longer lengths were attached to the papillary muscles and their tapering suggested that they had stretched before breaking. Stretching was clearly seen in the intact chordæ immediately adjacent to the ruptured ones. In the 3 patients (Cases 3, 4, and 6) with localized posterior leaflet abnormalities, the ruptured chordæ were not situated immediately adjacent to the cleft, and indeed in Case 6 the chordæ to the anterior leaflet had broken.

Microscopical Pathology. In Cases 1, 3, and 4, the site of chordal rupture revealed fragmentation of collagen bundles but no evidence of active or degenerative disease (Fig. 7). In Cases 1 and 4, circumscribed areas of necrosis were seen in the collagenous architecture of the chordæ (Fig. 8). Collagen disease was not demonstrable by special staining of the chordæ of these 3 cases, nor was there evidence of cellular infiltration to indicate inflammatory disease.

The findings in Case 5 were different from the others in that well-marked atheroma with necrosis and calcification were shown (Fig. 9). Early thrombus formation was related to the atheromatous area, but no evidence of subacute bacterial endocarditis or active rheumatic endocarditis was detected. The findings were in keeping with the age of this man and with the severe widespread atheromatous changes of the arterial tree, which were found at necropsy.

SURGICAL TREATMENT

The 23 patients with ruptured chordæ tendineæ were treated in a variety of ways (Table IV). The procedure adopted depended upon the nature of the valve lesion, of which the chordal rupture was often only a minor feature.

In the first of the patients with "normal" chordal rupture (Case 1) an ivalon buttress was sewn beneath the flail posterior leaflet. Case 6 was treated by plication of the anterior leaflet above the insertion of the ruptured chordæ (McGoon, 1960), and by an annuloplasty which obliterated the lateral posterior leaflet cleft, raised that leaflet, and narrowed the annulus (Wooler *et al.*, 1962). Three patients (Cases 2, 4, and 5) were treated by chordal replacement and annuloplasty, and one (Case 3) by chordal replacement alone.

FIG. 8.—Microscopical section of chorda of Case 3 close to the site of rupture, showing cleft formation within the collagen bundles surrounded by a small zone of necrotic collagen. Again there is no cellular infiltration and complete absence of inflammatory reaction. ($\times 160$).

Operation for Chordal Replacement. The heart was exposed through a long left thoracotomy incision. Venous blood was drained through a $\frac{1}{4}$ in. (1.27 cm.) cannula in the right ventricular outflow tract, and oxygenated blood was returned at a flow rate of 2.2 litres/sq. m./min., through the left femoral artery. The body temperature was lowered to 30°C. without altering the perfusion rate.

The left atrium was widely opened. The papillary muscles were defined and their chordæ traced to the leaflet or to the sites of rupture. The leaflets were searched for areas of thickening, perforations, and congenital clefts. Jet lesions on the atrial walls served as useful guides for the localization of such isolated leaflet defects.

Monofilament 00 nylon was used for chordal replacement in all 4 cases. The needle was passed from the ventricular to the atrial aspect of the leaflet close to the remnant of a major chorda. It was then returned through the leaflet 0.25 in. (0.6 cm.) from its entry. The nylon was buttressed on both aspects of the leaflet with teflon pledgets and was tied firmly with 4 knots, leaving two long strands projecting from the ventricular surface of the valve. Further similar nylon sutures were then inserted until the whole flail leaflet edge was secured. In one patient (Case 4), 3 sutures (6 strands) were used, whereas in the others 2 sufficed. The free ends of the nylon were then threaded onto long slim curved skin needles and passed under vision through the appropriate papillary muscle close to its apex and about 0.25 in.

TABLE IV
TREATMENT OF ALL CASES WITH RUPTURED CHORDÆ TENDINEÆ

	No.	Ivalon bung buttress of posterior leaflet	Annuloplasty and plication leaflet	Artificial chordæ	Prosthetic valve	Survival
Rheumatic mitral stenosis and mitral incompetence	7	0	1	1	5	6
Post-closed valvotomy	8	2	0	0	6	6
Endocardial cushion defect	2	0	1	0	1	2
Pure mitral incompetence and ruptured chordæ	6	1	1	4	0	4
Total	23	3	3	5	12	18

FIG. 9.—Microscopical section through a nodule in a chorda close to the site of rupture (Case 5). This shows an atherosclerotic lesion in which there is well-marked necrosis and calcification but no inflammatory infiltration. ($\times 14$.)

(0.6 cm.) apart. The needles emerged externally through the posterior ventricular wall, care being taken to avoid transfixing or straddling major coronary vessels. Each pair of filaments was brought through a square of teflon externally.

The first time this technique was used the heart was defibrillated and the length of the artificial chordæ, necessary to prevent eversion of the leaflet during ventricular systole, was adjusted. It was found that slight traction upon the exteriorized nylon pulled the posterior leaflet against the ventricular wall and caused incompetence. Adjustment was difficult as the tendency was to tie the strands too tightly, and the knots had to be loosened several times before a satisfactory result was obtained. Subsequently, chordal length was adjusted before the heart was closed. This was achieved by grasping the leaflet edge with forceps and flapping it towards the atrium until the remaining natural chordæ were tense. When the nylon restrained the leaflet to the same extent as the natural chordæ, a mosquito clamp was applied to the threads externally and flush with the ventricular wall. Leaflet competence was later confirmed in the beating

heart by digital exploration, and the nylon was then tied over the teflon patch. A correctly adjusted loop of nylon did not dig deeply into the ventricle when the patient's own circulation was restored.

In addition to the chordal replacement, any other lesion that could cause incompetence must be corrected. The hole in the posterior leaflet of Case 3 was covered with a long strip of pericardium which was sewn to the atrial surface of the leaflet from the annulus to its free edges. The pericardial strip was left long enough to reach the apex of the papillary muscle to which it was fixed with silk sutures after the nylon threads had been adjusted for length. Despite the dilated mitral ring, annuloplasty was not done, and this proved to be a mistake since moderate regurgitation remained. In Cases 4 and 6, the deep clefts of the posterior leaflet were obliterated by including their inner edges in the lateral annuloplasties.

RESULTS

Cases 1 and 5 died in hospital; the remaining 4 survived (Table V). Minimal mitral regurgitation

TABLE V
SUMMARY OF OPERATIVE RESULTS

Case No.	Hospital Course		Post-operative	
	Murmur of mitral incompetence	Time in hospital	Symptoms	Auscultation
1	Nil until 1 week before death; then gross	Died at 6 weeks		
2	Nil	23 days	Asymptomatic 4 years later	Grade 1/6 late systolic murmur; mid-late systolic click
3	Grade 2 late systolic murmur	23 days	Marked improvement 3 years later	Grade 2-3/6 late systolic murmur; mid-late systolic click
4	Nil	20 days	Asymptomatic 2 years later	Grade 1/6 pan-systolic murmur; mid-late systolic click
5	Nil	Died at 14 days		
6	Grade 1 pan-systolic murmur	22 days	Marked improvement 1½ years later	Grade 3/6 pan-systolic murmur

remained in Cases 2 and 4 as shown by a grade 1 late systolic murmur (Barlow *et al.*, 1963; Segal and Likoff, 1964; Tavel, Campbell, and Zimmer, 1965; Barlow, 1965; Barlow and Pocock, 1965; Ronan, Perloff, and Harvey, 1965; Barlow *et al.*, 1966), and pansystolic murmur, respectively. Case 3 also had a late systolic murmur, but this was loud (grade 3) and probably denoted a more significant degree of incompetence. Case 6 had a grade 3 pansystolic murmur and moderate residual mitral regurgitation, though this was considerably less than before operation. In all 4 survivors the symptoms improved or disappeared. Systolic clicks were present in the 3 patients (Cases 2, 3, and 4) with artificial chordæ. This finding was compatible with the postulate that non-ejection clicks are usually of intracardiac origin (Barlow *et al.*, 1963; Barlow, 1965; Ronan *et al.*, 1965; Barlow *et al.*, 1966).

DISCUSSION

It is rare to find pure severe rheumatic incompetence with minimal leaflet or chordal thickening, and in most cases a degree of stenosis coexists (Saphir, 1953) (Fig. 10 and 11). Hepper *et al.* (1956) considered that bacterial endocarditis was the main cause of chordal rupture, and Osmundson *et al.* (1961) found evidence of active or healed bacterial endocarditis in 16 of 20 rheumatic valves with ruptured chordæ. These latter authors considered that rheumatic activity could produce chordal disruption but that the incompetence was then seldom severe since the commissural filaments were usually involved. Bailey and Hickam (1944) described 7 cases of ruptured chordæ which they attributed to rheumatic disease. Barber (1944) has shown that a crush injury or blow to the chest wall can rupture chordæ tendineæ.

Several workers (Frothingham and Hass, 1934; Shapiro and Weiss, 1959; January, Fisher, and Ehrenhaft, 1962) have claimed that normal chordæ can rupture, and our 6 cases fall into this category.

It is possible that the posterior leaflet perforation of Case 3 was a legacy of bacterial endocarditis, but the absence of history, the otherwise unscarred leaflet, and the fact that the defect was remote from the chordal ruptures, makes this most unlikely. Saphir (1953) maintained that, with bacterial endocarditis, the chordæ were affected by direct extension of the infective process.

Our only specific histological finding was the atherosclerosis present in the chordæ of Case 5. Features of rheumatic disease were not seen in the 3 patients (Cases 1, 3, and 4) examined, but admittedly this was a difficult histological diagnosis to make. Rokitsky (1852) and Coombs (1924) differ in their interpretation of the pathogenesis of rheumatic valve thickening but agree that the lesion is a dense nodular fibrosis associated with thickening and fusion of the chordæ. Gross and Kugel (1931) described a non-rheumatic deposition of lipid in the chordæ close to their leaflet insertion. Sokoloff, Elster, and Righthand (1950) reported degenerative chordal changes at the insertional sites in elderly people, and they described local chordal thickening characterized histologically by a loss of cellularity and increased hyalinization. They also mentioned tapering of these thickened chordæ at the insertion into the leaflet. Certainly, in our cases, the chordæ ruptured close to the leaflet edge and the stumps were tapered. However, adjacent intact chordæ were thin and stretched, and did not show the degenerative histology described by these authors.

There was, therefore, in our patients, scant evidence of rheumatic endocarditis or subacute bacterial endocarditis, and we believe that normal chordæ ruptured as a result of pre-existing and unrelated valve incompetence. This view is based upon the known presence of cardiac murmurs many years before the onset of disabling heart symptoms, the finding in 3 cases of non-rheumatic leaflet pathology, and the generally non-specific histological appearances of the ruptured chordæ. Of the 6

FIG. 10.—Series of mitral valves opened out to display the chordæ and annuli. From above downwards, normal, pure stenosis, mixed stenosis and incompetence, and dominant incompetence. Note the discrepancy in the diameters of the valves and the different chordal appearances. The pathological valves are all post-rheumatic and surgically excised.

patients with ruptured chordæ reported by Menges *et al.* (1964), 5 had also been known to have heart murmurs for many years before decompensation occurred, and though 3 of their cases had clear histories of rheumatic fever, no gross evidence of leaflet vegetation or ulceration to indicate preceding bacterial endocarditis was found at operation. In the presence of supple mitral valve components, "incompetence begets more incompetence", because of progressive annular dilatation (Edwards and Burchell, 1958; Levy and Edwards, 1962). We

believe that leaflet apposition becomes progressively less extensive as the annulus dilates, and that an increased strain is then thrown upon the chordæ, which consequently stretch and may eventually snap. When a chorda breaks the situation is suddenly aggravated, greater strain has to be borne by the adjacent intact chordæ, and serial rupture of these may then result. Our cases include rupture of the chordæ from one papillary muscle, unilateral chordal rupture with stretching of those from the opposite muscle, and central rupture of all chordæ to

a leaflet. The commissural chordæ probably survive because they are less exposed to the strain of an incompetence caused largely by annular dilatation. Sokoloff *et al.* (1950), Brock (1952), and Van der Spuy (1958) have all emphasized that the central chordæ are in line with the axes of the papillary muscle groups, and that these are the thickest of the chordæ (Fig. 10). They exercise the most selective action on the leaflets and when they rupture regurgitation becomes gross. In most rheumatic valves with ruptured chordæ, it is the commissural filaments that are directly involved by the pathological process, and incompetence from this cause is usually minor. It is, however, possible that a rheumatic valve incompetence may be aggravated by rupture of major chordæ secondary to annular dilatation in the manner postulated above. Considering the frequency of rheumatic mitral incompetence, this is a rare finding probably because such valves have thick, stronger than normal, chordæ tendineæ (Fig. 10).

The surgical technique that we have described is similar to that used by January *et al.* (1962) for their case. We have used nylon, which is probably not the most suitable material for chordal replacement because it is brittle and tends to fracture. Nevertheless, the strands have remained effective for periods of 4, 3, and 1½ years, respectively. In one patient (Case 3) an attempt was made to strengthen the repair with a strut of pericardium, and in future it is proposed to use teflon threaded through fascia lata fibres.

A cause for incompetence, additional to the chordal rupture, was found in 5 patients. The clefts of the posterior leaflet close to the lateral commissure can be easily overlooked, and a careful search for these should be made. This situation in the posterior leaflet may be a not-infrequent site of congenital defects and a perforation was also found in this position. Rheumatic fibrosis, atherosclerotic deposits, or leaflet clefts associated with endocardial cushion defects, may be the basic lesion in some instances. The additional cause of incompetence must also be corrected. Annular dilatation is a factor common to all these cases and the valve ring must be narrowed surgically.

A striking observation during operation was the absence of tug upon the exteriorized nylon strands when the valve was competent. This seems to contradict the commonly accepted view expressed by Chiechi, Lees, and Thompson (1956) that the basic function of chordæ is to take strain. With competent aortic and pulmonary valves, the pressure is borne by the support which the opposed leaflets afford each other. It is probable, therefore, that the essential function of chordæ tendineæ is to

FIG. 11.—Mitral valves mounted in closed position viewed from the atrial aspect for comparison of sizes of annuli and leaflet surface. From above downwards, normal, mixed stenosis with incompetence, pure stenosis, and dominant incompetence. The pathological valves are all post-rheumatic. The incompetent valve has a short shrunken posterior leaflet but a mobile anterior one. The chordæ are minimally thickened and the incompetence of this type of rheumatic valve could quite feasibly be aggravated by rupture of chordæ tendineæ.

restrain the leaflet edge early in ventricular systole so that the leaflet can "catch" the blood and billow in the same way as the sails of a boat catch the wind. The filled leaflets make surface contact and by their mutual support resist the rising ventricular pressure. We have used no refined techniques to measure the quantitative pull on the artificial chordæ, but Salisbury, Cross, and Rieben (1963), working with dogs, have shown by replacing a segment of chorda with a transverse displacement transducer, that the shape of its tension curve follows the contour of the intraventricular pressure curve until the aortic valve opens. Thereafter, chordal tension falls sharply, while the left ventricular pressure continues to rise. When all other chordæ were cut, however, they demonstrated that the tension on the intact one increased and continued to rise after aortic valve opening. Despite the use of nylon, our patients have survived up to 4 years without recurrence of serious incompetence, and presumably without fracture of the strands. This would indeed be remarkable if the threads had to bend and straighten many millions of times while resisting the full left ventricular pressure.

SUMMARY

A description is given of 6 patients in whom there was severe mitral incompetence associated with supple voluminous leaflets, annular dilatation, and rupture of histologically normal chordæ tendineæ. All were subjected to operation, and 4 have survived. The chordæ were replaced in 4.

It is believed that in these cases, and possibly in 2 others with endocardial cushion defects, rupture was not due to local chordal disease. The hypothesis is advanced that the chordæ ruptured because they were not adapted to resist the pressures thrown upon them by progressive mitral regurgitation caused by dilatation of the annulus of the valve. A cause of mitral regurgitation preceding the chordal rupture must, therefore, be present to account for the annular dilatation. The original, minor incompetence may be due to any congenital or acquired lesion, and such were found in five of the cases presented. All the patients were known to have had heart murmurs many years before the onset of serious cardiac symptoms. Only after the annulus has dilated sufficiently to impair wide surface apposition of the leaflets, do the chordæ come under pathological strain. Chordal rupture results in sudden aggravation of the incompetence, and serial rupture may then occur.

Our experience with the insertion of artificial chordæ indicates that they resist little strain during ventricular systole. It is suggested that the function of chordæ tendineæ is to restrain the leaflet edges

early in ventricular systole, so converting them to cup-shaped structures, the surfaces of which can oppose. As a result of the wide support which the leaflets then provide for each other, the chordæ of a competent valve are relieved of the burden of resisting the full left ventricular pressure.

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ADDENDUM

Since preparing this paper we have operated upon a further case of pure mitral incompetence in which ruptured chordæ were associated with a congenital cleft of the lateral junctional tissue of the anterior leaflet. This cleft stopped a centimeter from the annulus, and a small perforation was present in the intervening bridge of leaflet. The patient had a history very suggestive of subacute bacterial endocarditis.