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Non-rheumatic 'subvalvar' mitral regurgitation

Surgical pathology and results of operation in 37 patients

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Thirty-seven patients with non-rheumatic subvalvar mitral regurgitation are reported, representing 16% of all patients with mitral regurgitation submitted to open operation over a five-and-a-half-year period.

In 22 older patients with 'idiopathic' chordal lesions, the commonest finding was rupture of chordae to the posterior leaflet. The aortic leaflet chordae were most frequently involved following myocardial infarction (7 patients) or bacterial endocarditis (3 patients). Three other younger patients had ruptured chordae and two patients had rupture of the posteromedial papillary muscle following acute myocardial infarction.

The mitral valve was repaired in 16 patients with ruptured chordae, of whom only eight obtained a satisfactory late result. In the other 21 patients the valve was replaced with a mounted aortic homograft or a Starr-Edwards prosthesis. It is concluded that mitral valve repair should be reserved for patients with symmetrical rupture of the chordae controlling the centre of the posterior leaflet, as regurgitation may reappear after other forms of repair due to progressive rupture of other abnormal chordae or breakdown of the repair.

The early and late mortality in the patients with a definite antecedent myocardial infarction was much higher than in the other groups, and emergency valve replacement soon after rupture of the papillary muscle was unsuccessful in both patients.

In a previous report (Caves, Sutton, and Paneth, in press) we have defined non-rheumatic 'subvalvar' mitral regurgitation as the result of deformity, disruption or dysfunction of the chordae tendineae or papillary muscles in the absence of primary disease of the leaflets or annulus, and have discussed the aetiology and clinical findings. This paper describes the operative findings, surgical technique, and results in 37 patients with 'subvalvar' mitral regurgitation. Thirty-three of these patients were included in the previous report.

MATERIAL AND METHODS

During the period September 1965 to February 1971, 225 patients were submitted to operation for mitral regurgitation by one of us (M. P.). Functionally, all patients were class III or IV (New York Heart Association). In 37 (16%) of these patients mitral regurgitation was due to non-rheumatic rupture or stretching of the chordae tendineae (35 patients) or rupture of the posteromedial papillary muscle (2 patients). The remainder (188 patients) either gave a definite history of rheumatic fever or were found at operation to have evidence of chronic rheumatic valve disease. In some of these patients ruptured chordae were also seen, but only as part of the rheumatic disorganization of the valve structures.

Forty-four operations were performed in the 37 patients, seven patients requiring a second operation. Surgery was performed with the aid of a rotating disc pump oxygenator primed with blood/Ringer's lactate solution. The chest was opened through a median sternotomy and the mitral valve was exposed trans-septally through the right atrium.

All patients who left hospital were followed up by personal examination or by information received from the referring cardiologist. In assessing post-operative improvement, particular attention was paid to the plain chest radiograph as well as to the patient's symptoms. It has recently been shown at this hospital that changes in cardiac diameter and pulmonary vasculature are sensitive indices of 'mitral' valve function following replacement with an aortic homograft.

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SURGICAL PATHOLOGY

CHORDAE The patients have been classified as described previously, and their distribution according to aetiology, sex, and age is shown in Table I. Twenty-nine patients had ruptured chordae, and in six patients all the chordae to one leaflet (5 aortic, 1 posterior) were stretched and attenuated (Table II). Chordae controlling the posterior leaflet were involved in 19 of the 22 'idiopathic' patients. By contrast, stretching or rupture of the aortic leaflet chordae was found in six of the seven patients with a definite antecedent myocardial infarct, and in all three patients with previous bacterial endocarditis. The commonest lesion (7 patients) was rupture of all the chordae controlling the posterior leaflet. Rupture of only two or three chordae attached to the centre of the posterior leaflet was seen in another six patients. In patients with ruptured chordae it was usual to find that the immediately adjacent chordae were considerably stretched or attenuated. There were often lengths of ruptured chordae attached to the flail portion of the leaflet (Fig. 1) while it was difficult to identify the chordal attachment to the papillary muscle.

TABLE I

CLASSIFICATION, SEX AND AGE (37 PATIENTS)

Classification	No.	Sex		Age Range (Mean)
		M	F	
Chordal lesions				
1. Post-infarction[1]	7	5	2	54–70 (59)
2. Subacute bacterial endocarditis	3		3	35–54 (44)
3. Miscellaneous				
Juvenile	1	1		7
Pregnancy	1		1	20
Trauma	1	1		33
4. Idiopathic	22	16	6	45–82 (61)
Papillary muscle rupture	2	1	1	59 & 71
Total	37	24	13	

[1] Period between infarct and operation = 6 mth–2 yr

TABLE II

LEAFLET CHORDAE INVOLVED IN 35 PATIENTS

Classification	Aortic Leaflet		Posterior Leaflet		Medial Commissure	Lateral Commissure	Total
	S	R	S	R			
Post-infarction	4	2	—	1	—	—	7
Subacute bacterial endocarditis	—	3	—	—	—	—	3
Miscellaneous							
Juvenile	—	1	—	—	—	—	1
Pregnancy	—	—	—	1	—	—	1
Trauma	—	—	—	1	—	—	1
Idiopathic	1	2	1	16	1	1	22
Total	5	8	1	19	1	1	35

S = stretched chordae; R = ruptured chordae

PAPILLARY MUSCLES Two patients had infarction and rupture of the posteromedial papillary muscle. One patient, aged 71 years, was submitted to operation eight weeks after an inferior myocardial infarct and the portion of the posteromedial papillary muscle controlling the posterior leaflet was found to be ruptured. The other patient, aged 59 years, developed gross pulmonary oedema and cardiogenic shock nine days after an inferior myocardial infarct and was found to have complete rupture of the posteromedial papillary muscle at emergency operation performed the same day. One other patient with two previous episodes of postero-inferior myocardial infarction had fibrosis of the posteromedial papillary muscle and stretching and attenuation of the chordae from it to the aortic leaflet. In the remaining patients the appearance of the papillary muscles was normal.

ANNULUS In 22 patients (59%) the mitral annulus was of normal size. In eight patients it was moderately dilated, and in seven patients it was considerably dilated. There was no significant relationship between the chordal lesion and the size of the annulus. In the two patients with a ruptured papillary muscle the annulus was not dilated.

A notable feature in all patients with non-rheumatic mitral regurgitation is the lack of firm fibrous tissue in the annulus. There is much less tissue in which to place sutures for repair, annuloplasty, or valve replacement.

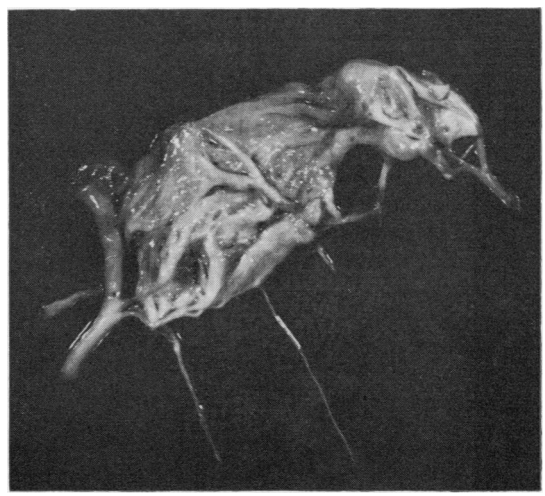


FIG. 1. *Excised posterior leaflet of the mitral valve. All the chordae tendineae were ruptured.*

LEAFLETS The uninvolved leaflets were always thin and pliable. The segment of leaflet rendered flail by chordal rupture or stretching was stretched, enlarged, and thickened, and always felt soft, quite unlike the fibrous, stiff leaflets seen following rheumatic infection. Congenital clefts or defects in the leaflets and valve calcification were not seen.

LEFT ATRIUM At operation the left atrium was of normal size or only mildly dilated in 27 patients (73%), all of whom were in sinus rhythm. The remaining 10 patients, six of whom were in atrial fibrillation, showed moderate or considerable enlargement of the left atrium. Endocardial 'jet impact' lesions were not recorded and atrial thrombus was never found.

Intracardiac pressures taken immediately before bypass revealed a 'V' wave greater than 30 mmHg in 29 patients. The 'V' wave was frequently as high as 70–80 mmHg and in one patient with partial rupture of the posteromedial papillary muscle, a 'V' wave of 115 mmHg was found, approximating to the left ventricular systolic pressure.

LEFT VENTRICLE Seven patients with chordal lesions were submitted to operation six months to two years following myocardial infarction. Two were found to have a left ventricular aneurysm, and in three others left ventricular contraction was noted to be poor. (Preoperative calculation of the ejection fraction had revealed significantly low values in all seven patients in this group (mean value $0{\cdot}44 \pm 0{\cdot}12$) when compared with the other patients with chordal lesions in whom left ventricular function was normal (mean value for ejection fraction $0{\cdot}70 \pm 0{\cdot}1$)).

In the two patients with a ruptured papillary muscle, the posterior wall of the left ventricle and left atrium felt soft and friable.

SURGICAL TECHNIQUE AND RESULTS

STRETCHED AND RUPTURED CHORDAE Forty-two operations were performed in the 35 patients, seven patients requiring a second operation (Table III). Repair of the involved mitral leaflet was the initial operation in 16 patients with ruptured chordae. In the other 19 patients the valve was replaced; in two with a Starr-Edwards prosthesis, in two with an aortic homograft (formalin-preserved) mounted on a three-pronged metal stent (FMH), and in 15 with an aortic homograft (antibiotic-sterilized and frozen at $-60°$ C) mounted in a tube of crimped Dacron (SMH) (Fig. 2).

TABLE III

SURGICAL TECHNIQUE AND RESULTS OF FIRST OPERATION IN 35 PATIENTS WITH CHORDAL LESIONS

Surgical Technique	No.	Hospital Mortality	Result		2nd Op.	Late Death
			Good	Fair		
Repair	16	1	9	1	5	3
Replacement						
SMH	15	1	13	—	1	1
FMH	2	1	—	—	1	—
S-E valve	2	—	2	—	—	1
Total	35	3	24	1	7	5

SMH = Dacron sleeve-mounted aortic homograft
FMH = frame-mounted aortic homograft
S-E valve = Starr-Edwards valve

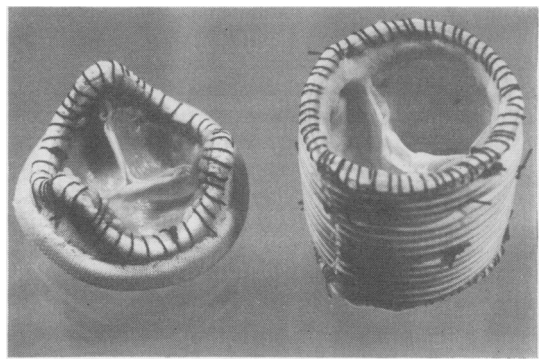


FIG. 2. (Right) *The Dacron sleeve-mounted aortic homograft (SMH) and* (left) *frame-mounted aortic homograft (FMH) used for mitral valve replacement in this series.*

TABLE IV

MITRAL VALVE REPAIR IN 16 PATIENTS

Technique	Annulo-plasty	Chordae Ruptured	Result
Reattachment	Yes	Med ½ post	Good. FU 5 yr
Reattachment	—	All post	Re-op. 1 day
Reattachment	Yes	Central aortic	Died—air embolus
Reattachment +plication	Yes	Central aortic	Re-op. 8 mth
Comm. plication	Yes	Med ½ aortic	Good. FU 5 yr
Comm. plication	—	Lat ½ post	Good. FU 4 yr (Fig. 3)
Comm. plication	Yes	Lat ½ post	Re-op. same day Died—SBE
Comm. plication	—	Med ½ post	Residual MR
Comm. plication	Yes	Med ½ aortic	Re-op. 3 mth, died (Fig. 5)
Plication both comm.	—	All post	Died 3 mth, CT
Plication both comm.	Yes	All post	Re-op. 22 mth
Plication both comm.	Yes	All aortic	Good. FU 3 yr
Wedge excision	—	Central post	Good. FU 1 yr
Wedge excision	—	Central post	Good. FU 9 mth
Wedge excision	—	Central post	Good. FU 6 mth
Wedge excision	—	Central post	Good. FU 6 mth

Med ½ = medial half; FU = follow-up; Comm. = commissure; Lat ½ = lateral half; SBE = subacute bacterial endocarditis; MR = mitral regurgitation; CT = coronary thrombosis; aortic = aortic leaflet; post = posterior leaflet.

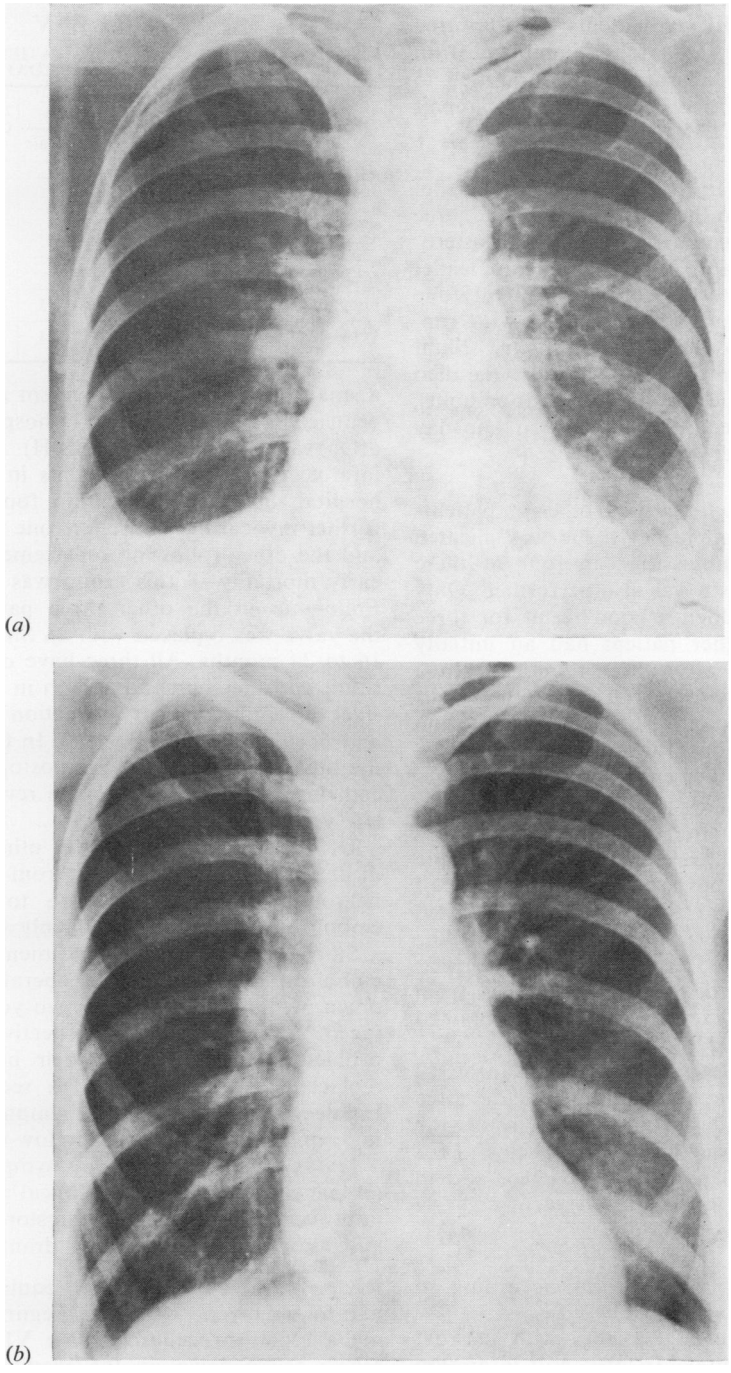


FIG. 3. *(a) Preoperative chest radiograph two months after onset of symptoms in a patient (No. 6, Table IV) with ruptured chordae controlling the posterior leaflet; (b) four years after a successful repair by the plication technique.*

Mitral valve repair Eleven patients with ruptured posterior leaflet chordae and five patients with ruptured aortic leaflet chordae were submitted to mitral valve repair. Three different types of repair were performed (Table IV).

1. *Reattachment* In four patients the leaflet margin was sutured to the papillary muscle. Three also had an annuloplasty performed at the postero-medial commissure, and in one redundant leaflet tissue was plicated into the commissure. Only one of these four patients, a man aged 34 with ruptured posterior leaflet chordae following blunt chest trauma, maintained a good result. One died from brain damage due to a presumed air embolus, and the other two required reoperation one day and eight months later, respectively.

2. *Plication into commissure* In eight patients the flail and redundant leaflet tissue was plicated into the adjacent commissure; in four of these patients an annuloplasty was also performed. Only three patients maintained a good result for three years or more. Another patient had an initially good result but died after three months from a further myocardial infarct. Of the other four patients, one has symptomatic mitral regurgitation and three required reoperation due to breakdown of the repair and further chordal rupture.

3. *Wedge excision* In four patients with rupture of only two or three chordae attached to the central region of the posterior leaflet, a wedge of leaflet was excised and the leaflet was repaired with interrupted sutures. In all four patients the annulus was not dilated and an annuloplasty was not required. The length of follow-up ranges from 6 to 12 months but all four patients have obtained an excellent result to date.

In summary, only four repairs (2 aortic, 2 posterior leaflet) have been successful for three years or more. Another four repairs using the wedge excision technique have been successful for up to one year. Two patients have died, and in the other six mitral regurgitation recurred, five of these requiring reoperation.

Aetiology and results The results according to the aetiology are shown in Table V.

Three (9%) of the 35 patients with chordal lesions died in hospital. In the post-infarction group (7 patients), one patient died from a low output on the evening of operation following resection of a left ventricular aneurysm and mitral valve replacement (FMH). A second patient with a small left ventricular aneurysm remained in left ventricular failure and died in hospital three weeks after valve replacement (SMH) from a further infarct. The other five patients in this group left hospital, but two died within four months from further myocardial infarction, one following repair and the other following replacement (SMH). The early mortality in this group was therefore 57%. Follow-up in the other three patients in whom the valve was replaced with an SMH ranges from 16 to 24 months. All three have obtained a good result and the chest radiograph in two shows considerable improvement (reduction in cardiac size and normal upper lobe veins). In the third patient the heart remains large, but postoperative cardiac catheterization at four months revealed improved left ventricular function.

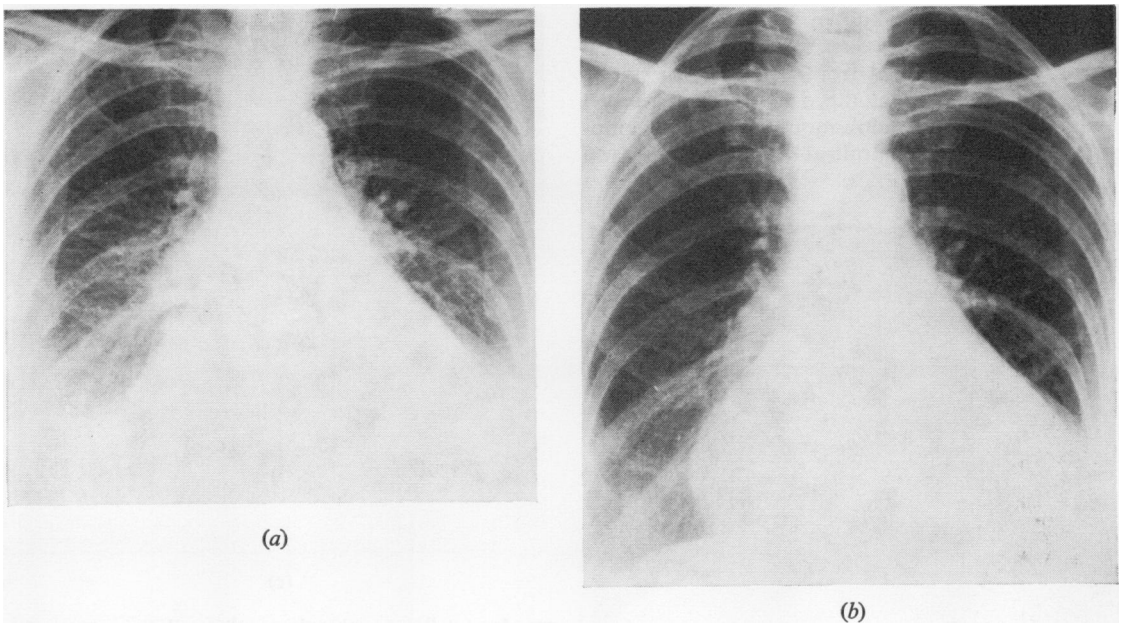
TABLE V

RESULTS OF OPERATION IN 35 PATIENTS, ACCORDING TO AETIOLOGY OF CHORDAL LESION

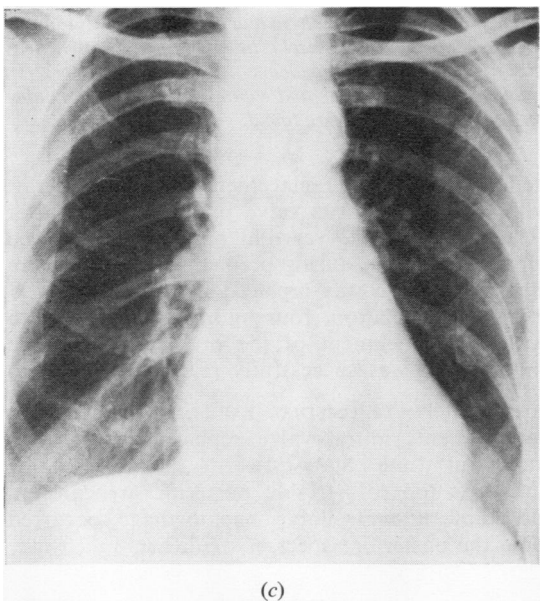
Classification	No.	Hospital Mortality	Result		2nd Op.	Late Death	Total Mortality
			Good	Fair			
Post-infarction	7	2	5	—	—	2	4 (57%)
Subacute bacterial endocarditis	3	—	2	—	1	—	—
Miscellaneous							
Juvenile	1	—	—	—	1	—	—
Pregnancy	1	—	—	—	1	—	—
Trauma	1	—	1	—	—	—	—
Idiopathic	22	1	16	1	4	3	4 (18%)
Total	35	3	24	1	7	5	8

Of the 28 patients in the other groups, one died 11 days after repair from severe central nervous system damage due to presumed air embolism. Another died suddenly 11 months after a Starr-Edwards valve replacement from cerebral embolism. Five required reoperation for breakdown of the repair, and the two youngest patients (aged 7 and 19 years respectively) developed problems associated with their homograft valve replacement which required a second operation. Eighteen patients remained symptom-free following repair or replacement (follow-up 4 months to 4 years), and one patient has symptoms following an inadequate repair. The clinical and radiological improvement following the restoration of mitral valve competence was often dramatic (Fig. 4).

Reoperation Seven patients required reoperation, five for recurrence of mitral regurgitation following a repair procedure (Table VI). In one, the repair broke down on the evening of operation, requiring emergency valve replacement with a Starr-Edwards prosthesis. Subacute bacterial endocarditis developed and the patient died 10 months later from a cerebral abscess. In two other



(*a*) (*b*)



(*c*)

FIG. 4. (*a*) *Chest radiograph only three weeks after the onset of symptoms due to rupture of the posterior leaflet chordae. The rapid cardiac and left atrial enlargement is unusual;* (*b*) *four weeks later after intensive medical treatment;* (*c*) *two months later after mitral valve replacement. The heart has rapidly returned to its normal size.*

patients the repair broke down at one day and eight months, and mitral valve replacement with a Starr-Edwards prosthesis was performed with a good result. Increasing mitral regurgitation in one patient was found at 22 months to be due to a breakdown of the repair and rupture of further chordae. Successful valve replacement (SMH) was performed. The fifth patient had an initially successful repair for rupture of a few aortic leaflet chordae and was much improved at follow-up after three months. Three weeks later he suddenly became breathless and was admitted to hospital in pulmonary oedema (Fig. 5) with signs of severe mitral regurgitation. At emergency operation rupture of all the aortic leaflet chordae

TABLE VI

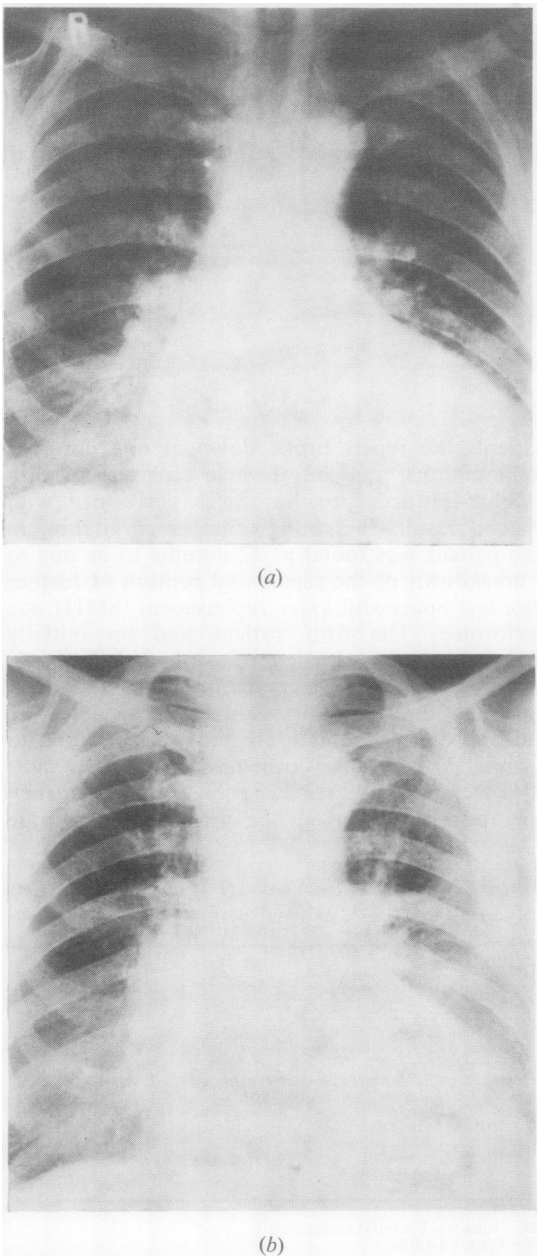
NON-RHEUMATIC SUBVALVAR MITRAL REGURGITATION RESULTS OF REOPERATION—7 CASES

Time from 1st Op.	Reason	Aetiology	2nd Op.	Result
6 hr	Failed repair	Idiopathic	S-E valve	SBE Died
1 day	Failed repair	Idiopathic	S-E valve	Good
3 mth	Rupture of more chordae	Idiopathic	S-E valve	Died[1]
8 mth	Failed repair	SBE	S-E valve	Good
22 mth	Failed repair + ruptured chordae	Idiopathic	Homograft	Good
11 mth	Calcified homograft	Juvenile	S-E valve	Good
4 mth	Detachment of homograft frame	Pregnancy	Reattach-ment	Good

[1] Died on table
SBE = subacute bacterial endocarditis
S-E = Starr-Edwards

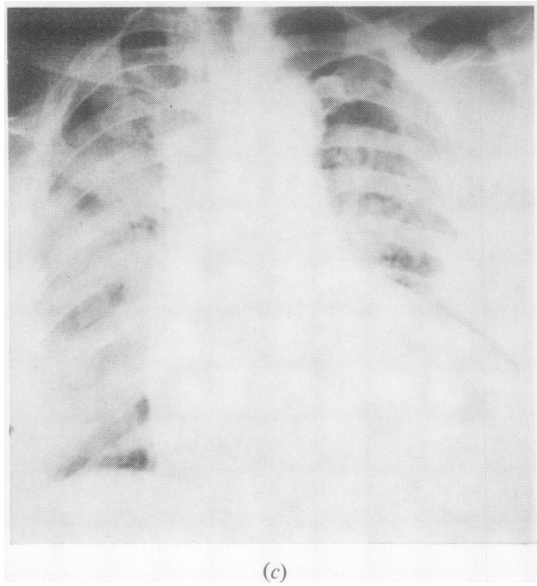
was found with breakdown of the repair, and although the mitral valve was replaced (Starr-Edwards valve), he died from a low-output state.

The youngest patient (aged 7 years) developed calcification of his sleeve-mounted aortic homograft with resultant mitral regurgitation and stenosis. Successful replacement of the homograft with a Starr-Edwards valve was performed after 11 months. The 19-year-old girl, who developed ruptured chordae during pregnancy and in whom the mitral valve was replaced (FMH), developed mitral regurgitation four months later due to posterior detachment of the prosthesis from the annulus. It was successfully reattached.



(a)

(b)



(c)

FIG. 5. (a) *Preoperative chest radiograph in a patient with ruptured aortic leaflet chordae (No. 9, Table IV); (b) three months after mitral valve repair by the plication technique. The heart is smaller and there had been great symptomatic improvement; (c) three weeks later when the patient was admitted in pulmonary oedema. At emergency operation breakdown of the repair and rupture of all the remaining aortic leaflet chordae were found.*

RUPTURED POSTEROMEDIAL PAPILLARY MUSCLE In both patients mitral valve replacement was unsuccessful (one Starr-Edwards prosthesis, one SMH) as the soft friable posterior myocardium tore, and uncontrollable haemorrhage occurred from the posterior aspect of the heart.

DISCUSSION

Rupture of the mitral chordae tendineae has been recognized in the last 12 years as an increasingly important cause of mitral regurgitation demanding surgical treatment (January, Fisher, and Ehrenhaft, 1962; Menges, Ankeney, and Hellerstein, 1964; Sanders, Scannel, Har-

thorne, and Austen, 1965; Roberts, Braunwald, and Morrow, 1966). It was soon realized that there was often no evidence of a rheumatic aetiology for the chordal lesions, and the clinical syndrome with which such patients usually present has been clearly described (Caves *et al.*, in press; Raftery, Oakley, and Goodwin, 1966; Selzer *et al.*, 1967).

The findings of a relatively normal-looking mitral valve at operation with only a few ruptured chordae, rendering a portion of one leaflet flail and incompetent, prompted many surgeons to repair the valve. A variety of surgical techniques have been described, particularly in the early reports when well proven prostheses were not available (McGoon, 1960; Morris, Penner, and Brandt, 1964; Kay, Tsuji, and Reddington, 1965; Marchand, Barlow, Du Plessis, and Webster, 1966; Hessel, Kennedy, and Merendino, 1966; Gerbode *et al.*, 1968). The number of patients in some reports was small and the long-term results were not always recorded.

Another factor complicating the assessment of the results of surgery for ruptured chordae has been the inclusion, in most series, of patients with chronic rheumatic valve disease. Apart from their different clinical presentation and haemodynamics, such patients have quite different valve leaflets and chordae. Rheumatic leaflets are thickened and fibrous and hold sutures well in contrast to the thin, soft leaflets found in the older patients with idiopathic or ischaemic chordal rupture. We believe that a distinction between rheumatic and non-rheumatic chordal rupture is as important when considering the best method of surgical treatment as it is when describing the clinical presentation, and we have therefore deliberately excluded patients with a rheumatic aetiology from this report. In this series of 35 patients with non-rheumatic ruptured or stretched chordae, two factors had an important effect on the immediate and long-term results: (1) the presence of pre-operative myocardial infarction and dysfunction, and (2) the choice of operative technique for ruptured chordae.

MYOCARDIAL INFARCTION Myocardial infarction as a cause of subvalvar mitral regurgitation was proposed by Burch, De Pasquale, and Phillips (1963) and others have described their experience with such patients (Sanders *et al.*, 1967; Spencer, Reppert, and Stertzer, 1967; Gerbode *et al.*, 1969; Klughaupt, Flamm, Hancock, and Harrison, 1969). Nine patients in this series gave a definite history and had electrocardiographic evidence of previous myocardial infarction. All seven patients in whom a chordal lesion was found at operation had impaired left ventricular function (low ejection fraction) at cardiac catheterization. In one patient a left ventricular aneurysm was diagnosed before surgery and in another patient an aneurysmal area on the left ventricle was found at operation. Six of these seven patients had lesions of the aortic leaflet chordae considered at operation to be unsuitable for repair, and mitral valve replacement (SMH) was performed. The other patient had a rupture of all the posterior leaflet chordae and the valve was successfully repaired by a plication procedure. All seven patients survived operation but four died within four months from pre-existing or further myocardial damage. Gerbode *et al.* (1969) also noted a high postoperative morbidity and mortality in this group. Although Klughaupt *et al.* (1969) did not find a history of pre-existing myocardial infarction to be an adverse factor, their results were poor in three patients in whom impairment of left ventricular function was demonstrated preoperatively.

We have found impaired left ventricular function (low ejection fraction) to be the most important preoperative factor when determining the prognosis in patients with post-infarction subvalvar mitral regurgitation. We now believe that their preoperative assessment must include coronary arteriography. If indicated, the myocardial blood supply and function can then be improved with a suitable bypass grafting or implantation procedure. Only with such a revascularization procedure is it likely that the results of mitral valve repair or replacement in this group will be significantly improved.

The first case report of successful mitral valve replacement following infarction and rupture of a papillary muscle was by Austen, Sanders, Averhill, and Friedlich (1965), who later described their experience with four further patients (Austen, Sokol, DeSanctis, and Sanders, 1968). Other reports of successful intervention in such patients have appeared (Fluck, Taubman, Cleland, and Mounsey, 1966; Horlick, Merriman, and Robinson, 1966; de la Torre, Linhart, and Bartley, 1967), while Morrow *et al.* (1967) described their experience in four patients with partial rupture of a papillary muscle coming to surgery 3 to 15 months after myocardial infarction. The usual outcome of a ruptured papillary muscle is rapid deterioration and death from congestive heart failure (Sanders, Neubuerger, and Ravin, 1957). The two patients in this series were both in pulmonary oedema at the time of surgery, eight

weeks and nine days after infarction respectively. On both occasions mitral valve replacement was performed but death occurred from uncontrollable haemorrhage.

Successful replacement has rarely been recorded in such patients earlier than 10 weeks after myocardial infarction, and it would appear that operation should be delayed as long as possible following papillary muscle rupture.

CHOICE OF OPERATIVE TECHNIQUE FOR RUPTURED CHORDAE

Mitral valve repair In these patients mitral valve repair should be considered but the result following an apparently successful repair will always depend on at least four factors: (1) the ability of the repaired tissues to hold sutures; (2) the leaflet involved; (3) the number and situation of the ruptured chordae; and (4) the strength of the remaining intact chordae.

1. Ability of repaired leaflet to hold sutures In contrast to the thickened, fibrous leaflets seen following rheumatic infection, the leaflets in patients with idiopathic chordal rupture are usually soft and thin. In our experience they hold sutures poorly, and breakdown of the repair occurred in five patients in this series due to the sutures cutting out of the leaflets. Two of these patients died following reoperation. The methods of repair used were suture of the leaflet margin to the papillary muscle (2 patients) or plication of the flail redundant leaflet into the commissural region (3 patients). As a consequence, we now believe that reattachment of the mitral leaflet to the papillary muscle (Kay *et al.*, 1965) with or without the formation of artificial chordae (Morris *et al.*, 1964; Marchand *et al.*, 1966) has no place in the treatment of ruptured chordae. These techniques have not produced good long-term results. Plication of the redundant flail leaflet into the commissural region has also not produced satisfactory results and we no longer use this technique.

If a repair is attempted, support of the sutures with small pledgets of Teflon (Gerbode *et al.*, 1969) has been recommended.

2. The leaflet involved McGoon (1960) first reported successful repair of the aortic leaflet in two patients with ruptured chordae, and Ellis, Frye, and McGoon (1966) later reported a further 19 patients in whom a similar repair was performed. It should be noted that all these patients were thought to have a rheumatic aetiology. The follow-up of these patients showed that the majority developed a significant systolic murmur later. In this series we obtained a good result with repair of the aortic leaflet in only one out of four patients, two of the other three patients requiring reoperation. Like others (Gerbode *et al.*, 1969), we now believe that mitral valve replacement is the operation of choice in patients with rupture of the aortic leaflet chordae.

In patients with ruptured posterior leaflet chordae the correct choice of operation depends on the number and situation of the ruptured chordae.

3. Number and situation of ruptured chordae In two large series (Gerbode *et al.*, 1968; Ellis *et al.*, 1966) of patients the lesion most commonly described and illustrated has been rupture of a few chordae to the central portion of the posterior leaflet. A plication procedure in these patients permits a symmetrical reduction in the size of the posterior leaflet and ensures that the whole of the remaining leaflet is supported by chordae. Our only consistent success with a repair procedure has been in this type of patient. Rather than plication we have preferred to excise the redundant wedge of leaflet, suturing the cut edges together with interrupted sutures. This procedure, first described by Hessel *et al.* (1966) and again by Manhas *et al.* (1971), has produced excellent initial results in our four patients, although the follow-up is still quite short. Recently, Kerth, Sharma, Hill, and Gerbode (1971) have stated that the late results for a plication repair in the centre of the posterior leaflet are very good.

In the light of our experience, we believe that only this type of lesion is suitable for repair, but it should be noted that it was seen in only six (17%) patients in this series.

Although one leaflet is always predominantly involved, we have never attempted repair in a patient with stretched chordae as it is usual for all the chordae to both leaflets to be attenuated and lengthened to some degree.

4. State of other chordae The 'unknown' factor in the idiopathic patients is the strength of the remaining chordae. In a previous paper (Caves *et al.*, in press) we suggested that ischaemia of the papillary muscles may play a part in chordal rupture in these patients. Whatever the aetiology, it is likely that the remaining chordae, apparently normal at operation, will also have been affected. In this series, rupture of further chordae was seen in two patients at the second operation. In one (Fig. 5), acute rupture of all the remaining chordae to the aortic leaflet con-

tributed to pulmonary oedema and death despite emergency valve replacement.

Because of this 'unknown' factor, all repair procedures must be considered to be palliative, and the morbidity and mortality associated with breakdown of the repair or rupture of further chordae must be weighed against those following mitral valve replacement.

Mitral valve replacement It is now our practice to replace the mitral valve in the majority of these patients. The frequent use of an aortic homograft reflects the trial of this type of biological prosthesis at the Brompton Hospital over the last three years. The early results with these valves have been satisfactory.

We have found it useful to roll up the leaflets with over-and-over interrupted sutures to form a stronger seat for the prosthesis. Since the annulus is not thickened and fibrosed in these patients, there is a real risk of detachment of the prosthesis if the leaflets are excised. Using this technique, we have only once seen detachment of the prosthesis postoperatively—in the 19-year-old girl with ruptured chordae during pregnancy. At the first operation it was noted that the tissues of her mitral valve were particularly soft. Successful reattachment of this prosthesis was carried out after four months (Caves and Paneth, 1972).

One aortic homograft required replacement in the 7-year-old boy due to calcification which was first noted after nine months. Successful replacement with a Starr-Edwards prosthesis was performed 11 months after the first operation. This is the only instance of calcification of an aortic homograft in the mitral area seen at this hopsital. It may be a further example of the increased tendency for children to calcify homograft valves; this has already been noted to occur in the pulmonary area (Ross, 1970).

A Starr-Edwards prosthesis was inserted at the first operation in two patients and at reoperation in five patients. Of these seven patients, one died on the table in pulmonary oedema and a second (who had both operations on the same day) died 10 months postoperatively from subacute bacterial endocarditis and a cerebral abscess. A third patient died suddenly 11 months later from a cerebral embolus.

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