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Original Article

Mitral valvar anomalies and discrete subaortic stenosis

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Abstract On the basis of our clinical experience, we hypothesized that the role of mitral valvar anomalies in the development and recurrence of discrete subaortic stenosis might be underestimated. From January 1994 to October 2000, the anatomy of the mitral valve and its relationship to the other components of the left ventricular outflow tract were studied by echocardiography in a series of 73 consecutive patients referred to our institution for surgical correction of discrete subaortic stenosis. In all patients for whom it was considered advisable, surgical correction of the mitral anomaly was performed, together with resection of the fibro-muscular subaortic stenosis. One or more mitral valvar anomalies were found in 35 patients (48%). They could be grouped into five categories: insertion of a papillary muscle into the aortic leaflet, insertion of a papillary muscle into the ventricular wall, "muscularization" of the subaortic portion of the aortic leaflet, anomalous insertion of the valvar tissue into the ventricular wall, and accessory valvar tissue. In all cases with anomalous mitral valvar anatomy, surgical correction was feasible. It consisted of transection of the anomalous papillary muscle or its attachment, resection of accessory valvar tissue, and/or patch enlargement of the aortic leaflet. The incidence of mitral valvar anomalies associated with subaortic stenosis is probably underestimated. Our data suggest that they should be systematically searched for during the evaluation of all cases of subaortic stenosis. Their surgical correction is generally feasible, and might improve the mid and long term results.

Keywords: Subaortic stenosis; mitral valvar abnormality; obstructive cardiomyopathy

Discrete subaortic stenosis is a perplexing disorder. Although its anatomic expression, evolution and pathogenesis have been extensively studied, its surgical treatment is still controversial and disappointing, particularly because of the high incidence of recurrence, even in recently published surgical series.[1,2] There is good reason to believe that many, if not all cases of discrete subaortic stenosis may be the acquired consequence of primary congenital anomalies of the left ventricular outflow tract.[2–6] It is thus logical to think that surgical treatment should be aimed not only at relieving stenosis but also at correcting, insofar as is possible, the primary factors which are potentially responsible for the recurrence. Some years ago, we discovered severe mitral valvar anomalies within a short period of time in four patients referred for correction of discrete subaortic stenosis. The mitral valvar anomaly had not been detected before in any of these four patients, including two of them who had been previously treated in our institution. We hypothesized that the role of the mitral valve in the development of subaortic stenosis might be underestimated, even though it has been recognized for many years.[7,8] We subsequently studied the anatomic features of the mitral valve more systematically in all patients referred for discrete subaortic stenosis, as well as the relationship of this valve to the other components of the left ventricular outflow tract. The aim of this study is to present our echocardiographic and surgical findings, together with the consequences of these findings for the surgical treatment of patients.

Materials and methods

From January 1994 to October 2000, 73 patients were referred to our institution for surgical treatment of so-called discrete subaortic stenosis. The indication for surgery was a significant ventriculo-aortic

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pressure gradient, defined as a peak pressure of greater than 40 mm of mercury, and/or aortic regurgitation. Of the patients, five had previously undergone surgical resection of subaortic stenosis, including four in our own institution. Another one had been treated by percutaneous dilation. In addition, three patients had been previously treated for ventricular septal defect, and five for aortic coarctation. In this study, we excluded patients referred for closure of a significant ventricular septal defect associated with subaortic stenosis, and those who had to be treated for aortic coarctation associated with intracardiac defects during the same procedure. All these latter patients were neonates in whom subaortic stenosis was part of a Shone's syndrome. We also excluded nine patients operated on for the association of subaortic stenosis with atrioventricular septal defect and common atrioventricular junction. In this subset, nonetheless, we also found anomalies involving the left component of the common atrioventricular valve comparable to those involving the mitral valve in the patients herein described. The patients with common atrioventricular junction were excluded only because their left valve is not strictly comparable in morphology to the mitral valve. In those included in the series, the age at operation ranged from 44 days to 61 years, with a mean of 9.5 years. Only 11 patients were over 15 years of age. In all these patients, we performed a thorough analysis of the mitral valve by means of echocardiography and Doppler examination (Acuson 128 XP from 1994 to 1999, Hewlett Packard Sonos 5500 since January 1999), in addition to the usual study of discrete subaortic stenosis. All the patients were studied by the same operator (L.C.), with the adults being additionally studied by another operator (M.C.M.). Our analysis aimed at describing morphologically and using different view all the components of the valvar and subvalvar apparatus. We also sought to define the relationship of each component with the parietal and septal ventricular structures and the aortic valve. The movements of the aortic (anterior) leaflet relatively to the ventricular septum, the area of subaortic stenosis, and the aortic valve were also described. No effort was made to obtain quantitative measurements, because of the extreme variability of the anatomic features. All the operations were performed by the same surgeon (Y.L.). During surgical repair, the mitral valve was inspected through the aortotomy as completely as possible, generally after resection of the fibro-muscular obstruction. Specific repair of any identified mitral valvar anomaly was performed when indicated. No patient died, and none had any residual significant pressure gradient after the operation.

Results

Echocardiographic and operative findings

In the overall group of patients, even in those who had none of the other mitral valvar anomalies which we will described below, it was noted that the position of the papillary muscles was generally abnormal. When viewed transversely from the apex, there was a counterclockwise rotation of the axis of both papillary muscles (Fig. 1). During repair, it was not

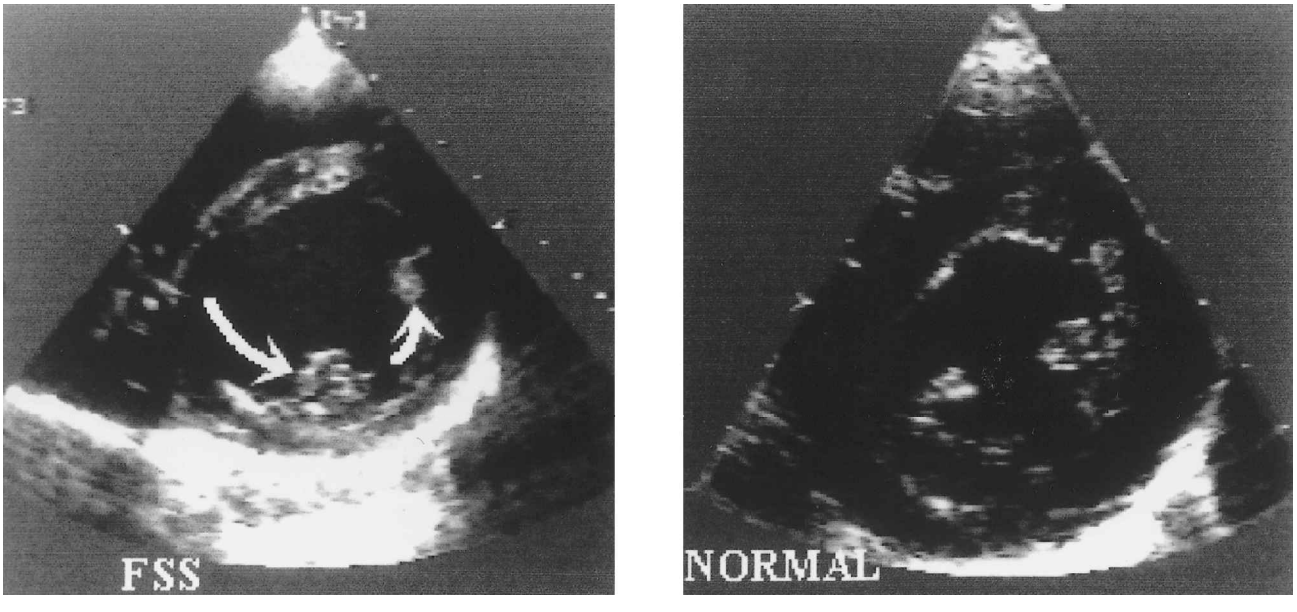


Figure 1.
Axial rotation of the mitral valve as seen in most cases of discrete subaortic stenosis, even without other morphologic anomalies. This rotation is seen in the transverse echocardiographic view. Note the anomalous position of the papillary muscles (left) compared to normal (right).

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Table 1. Distribution of mitral valvar anomalies in 35 patients.

Patient no.	PM to A. leaf.	PM to V. wall	Muscullariz.	Anom. inser.	Acces. tissue
1	−	−	+	−	−
2	−	−	−	+	−
3	−	−	−	+	−
4	+	−	−	−	−
5	+	−	−	−	−
6	+	−	−	−	−
7	−	−	−	+	−
8	−	−	−	+	−
9	−	−	−	+	−
10	−	+	−	−	−
11	−	−	−	+	−
12	+	−	−	+	−
13	−	−	+	−	−
14	+	−	+	−	−
15	−	−	−	+	+
16	−	−	+	−	−
17	−	−	+	−	−
18	+	−	−	+	−
19	−	−	+	−	−
20	−	−	−	+	−
21	−	+	+	+	−
22	−	−	−	+	−
23	−	−	−	+	+
24	−	−	−	+	−
25	−	−	+	−	−
26	−	+	−	−	−
27	−	−	−	+	−
28	−	−	+	+	−
29	−	+	+	+	−
30	+	−	−	−	−
31	−	−	−	+	−
32	−	−	−	+	−
33	−	−	−	+	−
34	−	+	−	−	−
35	+	−	+	−	−
Total	**8**	**5**	**11**	**20**	**2**

Abbreviations: PM to A. leaf: anomalous insertion of a papillary muscle into the aortic leaflet of the mitral valve; PM to V. wall: anomalous insertion of a papillary muscle into the ventricular wall; Muscullariz: muscularization; Anom. inser.: anomalous insertion of the mitral valve into the ventricular septum or parietal wall; Acces. tissue: accessory tissue

possible to analyze the situation of the subvalvar apparatus in the ventricular cavity, but it was noted that the left part of the aortic leaflet tended to be situated more anteriorly and closer to the ventricular wall than normal, even after resection of the fibromuscular stenosis. This rotation of the mitral valve on its longitudinal axis tended to narrow the lateral part of the outflow tract.

We found morphologic anomalies of the mitral valve in 35 out of the 73 patients who underwent surgical repair. Five types of anomaly were identified. In several patients, more than one type was detected (Table 1).

Anomalous insertion of a papillary muscle directly into the aortic leaflet of the mitral valve (found in 8 patients)

Echocardiography, in longitudinal views, revealed one head of a papillary muscle, generally the posteromedial one, to be in continuity with the aortic leaflet of the mitral valve. The image was much denser than that produced by second order tendinous cords. The location of the anomalous insertion was variable, but was distant from the free edge of the leaflet in all cases. At the level of the insertion, the leaflet was deviated anteriorly within the left ventricular outflow tract, particularly during systole. This angulation was always in front of the fibrous shelf (Fig. 2). In transverse view, the mass of the anomalous muscle could be detected anterior to the mitral leaflet and contributed to the obstruction of the outflow tract.

The surgical findings confirmed that, in all these 8 patients, an abnormal muscular structure was arising from the "head" of an otherwise normal papillary muscle. The thickness of this anomalous muscle was variable. After checking that the free edge of the aortic leaflet of the mitral valve was normally supported by first order cords, the abnormal attachment was simply transected or, when it was long, resected.

Anomalous insertion of a papillary muscle directly into the ventricular wall (found in 5 patients)

Echocardiographically, this anomaly was similar to the previous one, but the anomalous muscle was inserted directly into the ventricular wall instead of the mitral leaflet (Fig. 3). It created, nevertheless, anterior systolic deviation of the leaflet into the left ventricular outflow tract.

The existence of the anomalous muscle was confirmed at surgery in all cases. After transection, the aortic mitral valvar leaflet moved posteriorly, contributing to the enlargement of the lower part of the ventricular outflow tract.

In six other patients, anomalous cords attaching one of the papillary muscles to the ventricular wall were visualized. Their role in the genesis of the outflow tract obstruction was not evident, but they were nevertheless transected.

Muscularization of the subaortic portion of the aortic leaflet (found in 11 patients)

When seen at echocardiography, this anomaly was characterized by a marked angulation of the aortic leaflet of the mitral valve. The portion situated between the hingepoint of the aortic valvar leaflets and the summit of the angle was much thicker than the lower portion, and nearly motionless. The hinge

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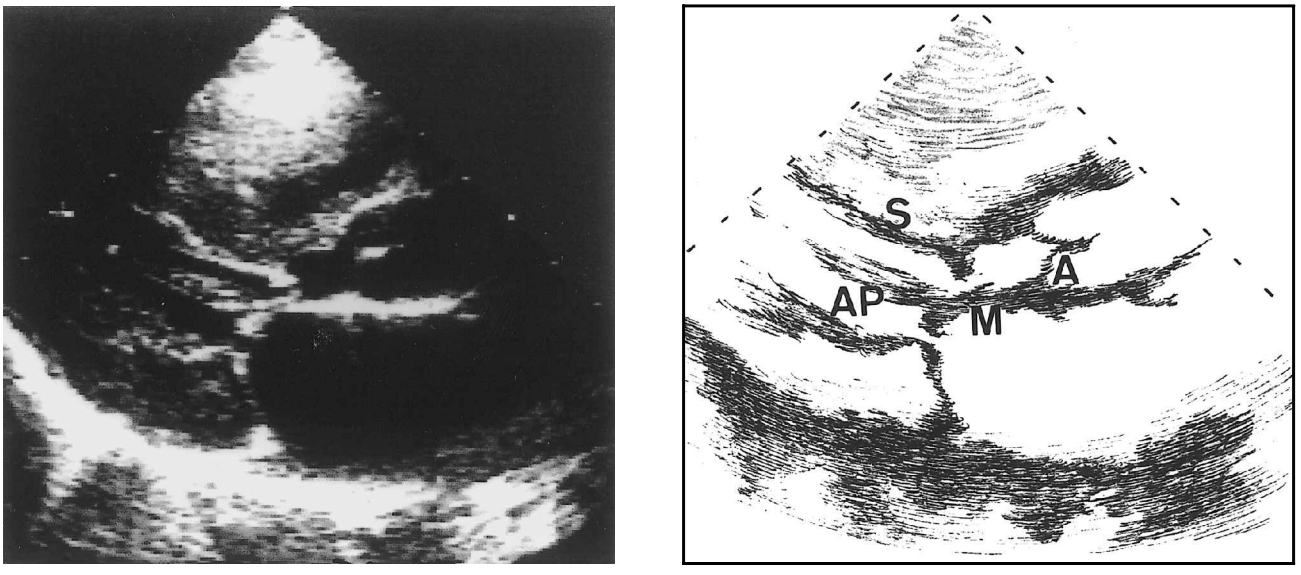


Figure 2.
Anomalous insertion of papillary muscle (AP) into the aortic leaflet of the mitral valve (M). S = ventricular septum; A = aortic valve.

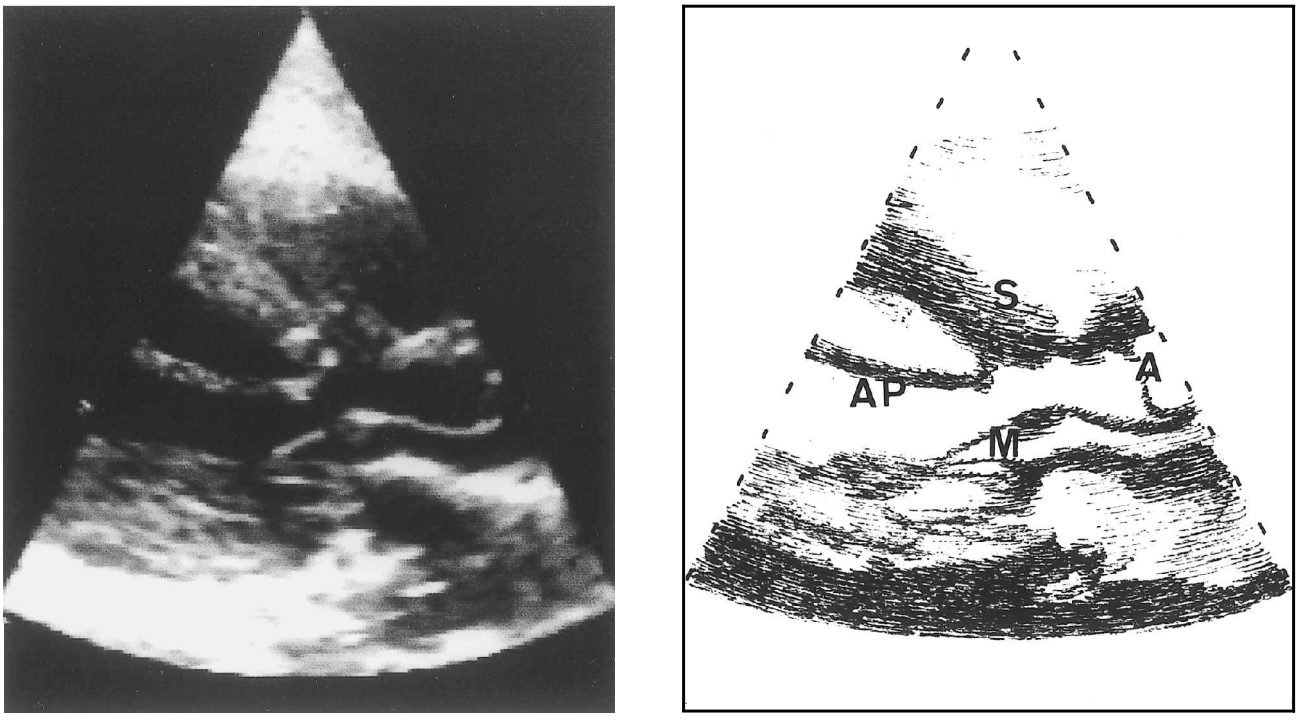


Figure 3.
Anomalous insertion of papillary muscle (AP) into the ventricular septum (S). M = Mitral valve; A = aortic valve.

point of the aortic leaflet of the mitral valve itself was distant from the aortic valve (Fig. 4). In the first case, this had been interpreted as an increased mitro-aortic distance, but this was not confirmed by the operative findings.

At surgery, in all cases there was a normal continuity found between the leaflets of the aortic and the mitral valves. The anomaly was not the position, but the nature of the subaortic portion of the aortic leaflet of the mitral valve. Instead of the usual white fibrous tissue, it was made of muscle. This was not obvious in most patients, because a thin layer of white tissue covered the muscle. Even though this part of the leaflet was thicker than the rest of the valve, it was much thinner than the wall of a complete muscular subaortic infundibulum as seen in

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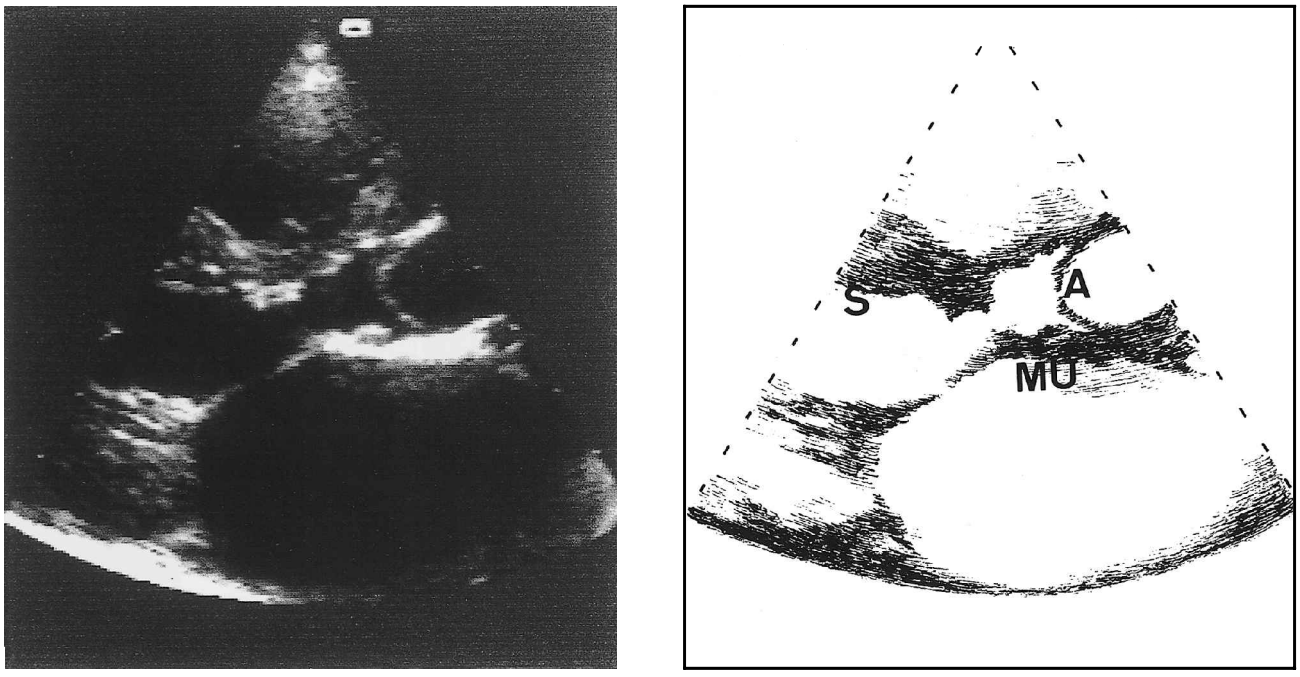


Figure 4.
"Muscularization" of the aortic leaflet of the mitral valve. The subaortic portion of the leaflet, made of muscular tissue (MU), is thicker than the other one. The limit between the anomalous portion and the normal one is the summit of the angle. A = aortic valve; S = ventricular septum.

anomalies of ventriculo-arterial connection. Unlike a complete muscular infundibulum, it could be mobilized using an instrument. This was confirmed by the incision of the leaflet, from the hingepoint of the leaflets of the aortic valve to the normal fibrous zone of the mitral valvar leaflet. The muscular nature of the subaortic zone then became evident. This anomaly was treated by patch enlargement of the leaflet, using autologous pericardium, as described by Carpentier[9] and Chauvaud et al.[10] for other types of subaortic stenosis. One of the patients needed reoperation because of patch dehiscence. Postoperatively, angulation of the aortic leaflet of the mitral valve was no longer observed, and its hinge point was contiguous to the aortic valve.

Anomalous attachment of the aortic leaflet of the mitral valve to the ventricular wall (found in 20 patients)

At echocardiography, using a transverse view, the direct attachment of the mitral valvar leaflet was clearly seen (Fig. 5). In many cases, interpretation of the images proved difficult, in particular in distinguishing between short tendinous cords and direct attachment of the valvar tissue itself. The point of anomalous attachment was also difficult to find using conventional echocardiographic views. It could be situated on either the septal or parietal sides of the left ventricular outflow tract. From the long axis view, the anteriorly displaced part of the leaflet was visible inside the left ventricular outflow tract. The anomalous attachment was, in all cases, in continuity with the fibrous component of the subaortic stenosis.

The anomalous attachment was confirmed at surgery in all cases. It differed from the frequent extension of the fibrous subaortic ring into the aortic mitral valvar leaflet. The anterior distortion of this leaflet persisted after blunt dissection of the fibrous ring, and the anomalous attachment needed to be transected in order to free the left ventricular outflow tract. In two cases, the anomalous attachment was so short that this transection created a perforation of the leaflet, which was repaired by direct suturing. In four subsequent cases with very short or direct attachment, the technique was modified. The leaflet of the mitral valve was deliberately incised close to the attachment, which was left intact. The defect was then repaired using a patch of autologous pericardium.

Accessory tissue of the leaflet of the mitral valve (found in 2 patients)

At echocardiography, an abnormal image was seen moving in the left ventricular outflow tract. It was pushed towards the aortic orifice during systole, and returned to a lower position during diastole (Fig. 6).

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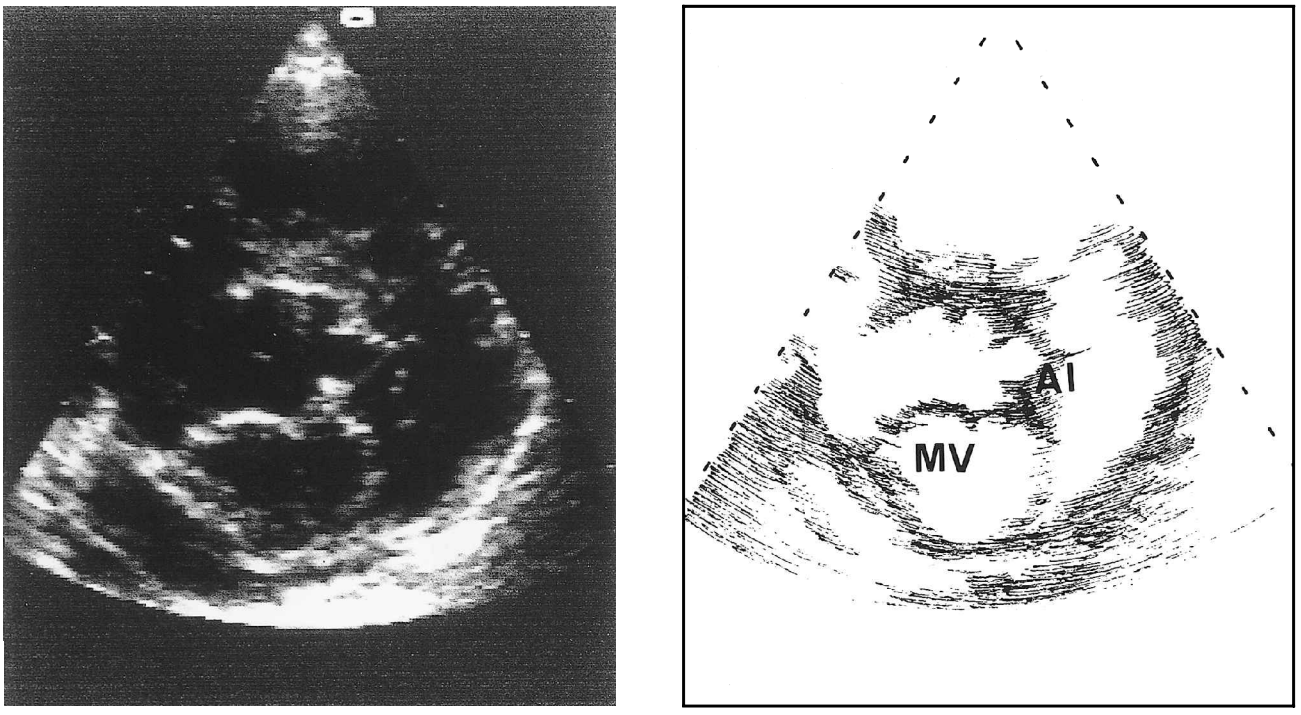


Figure 5.
Anomalous insertion (AI) of the mitral valve (MV) to the left ventricular wall.

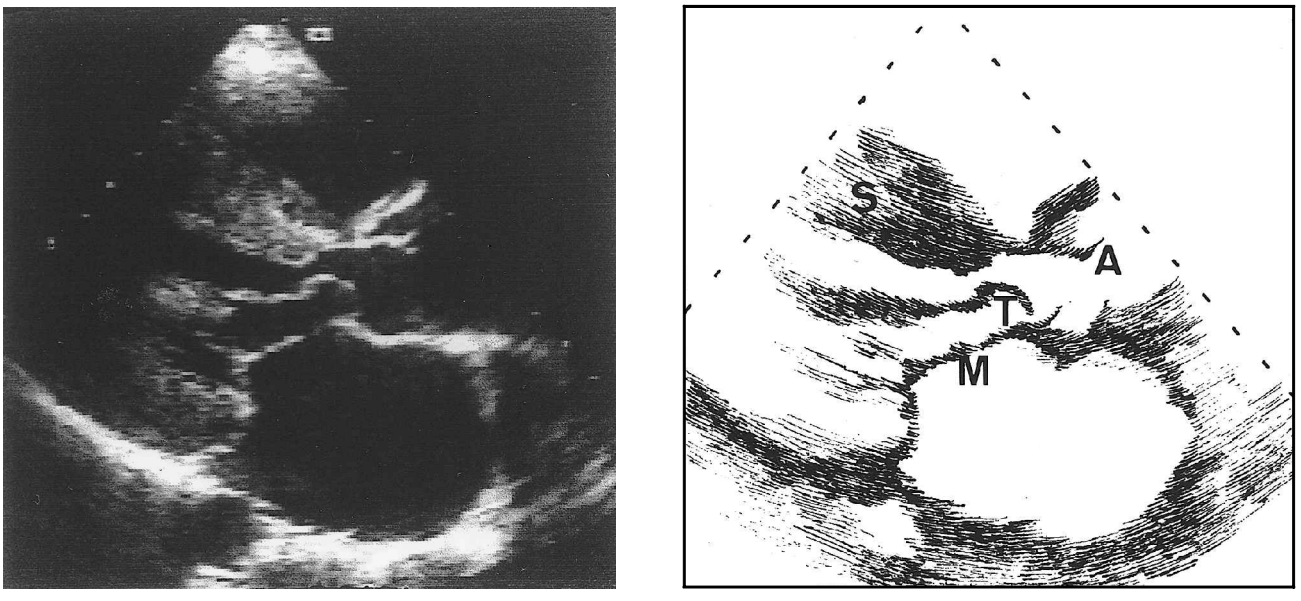


Figure 6.
Accessory tissue derived from the leaflet of the mitral valve (T) bulging into the outflow tract, beneath the aortic valve (A). In this case, the accessory tissue was attached to an anomalous papillary muscle which can be seen on this echocardiographic view. M = aortic leaflet of the mitral valve; A = aortic valve.

Its attachment to the mitral valvar apparatus was difficult to analyze preoperatively, but it was associated with other anomalies. In one case, a papillary muscle was anomalously attached to the aortic leaflet of the mitral valve. In the other, the aortic leaflet was attached directly to the ventricular wall.

At surgery, the anomalous mass was easily seen in both cases. It did not differ, macroscopically, from

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normal valvar tissue. In each case, it was attached either to a papillary muscle, or to the aortic leaflet, in such a way that the blood stream ballooned it in the left ventricular outflow tract. It was resected without difficulty.

Mid-term results and reoperations

At a mean follow-up of 3.3 years, with a range from 2 months to 6.8 years, reoperation proved necessary in 8 patients. Mitral insufficiency was the cause of reoperation in 4 children. In two patients, this was due to detachment of a pericardial patch placed on the aortic leaflet of the mitral valve. In the fourth one, it reflected incomplete analysis of the mitral valvar anatomy during the first operation. A cleft in the aortic leaflet had been overlooked during the repair of discrete subaortic stenosis, which included detachment of anomalous insertions of the mitral valve on the ventricular septum. On reoperating, it was found that only the leftward and superior part of the leaflet had been detached. The left inferior half of the cleft leaflet was detached from the septum and sutured to the superior one, with a good result. Reoperation in another 4 patients was needed because of recurrent subaortic stenosis. All 4 patients had anomalies of the mitral valve which had either gone unrecognized or been insufficiently treated at the first operation. In 3 patients, anomalous insertions of the mitral valve had been left intact. One of these patients also had muscularization of the aortic leaflet. In the last patient, recurrence of discrete subaortic stenosis was almost expected. He was 3 months old at the first procedure, during which an anomalous attachment of the mitral valve was noted but not resected. This was because of the small size of the aortic valvar orifice, making hazardous any manoeuver through the orifice. Upon reoperation, at four years of age, the anomalous insertions could be safely resected and a pericardial patch was inserted on the aortic leaflet. All the other patients are alive and well, with no significant subaortic stenosis and absent or trivial mitral insufficiency.

Discussion

Our findings suggest that the incidence of mitral valvar anomalies associated with discrete subaortic stenosis is much higher than generally expected. In this series, more than half of the patients had at least one abnormality of the mitral valve, not taking into account the axial rotation which could also be observed in patients who had an otherwise normal mitral valve. This striking observation does not seem to be due to a bias of selection. All but two patients were referred to our institution for the surgical resection of apparently banal subaortic stenosis. Four of them had been previously treated by us and, during the first hospitalization, the mitral valvar anomaly had been overlooked. It is thus extremely likely that the incidence of such anomalies is greatly underestimated just because they are not systematically searched for. It is of interest to note that we were unable to unearth in the literature any study which reports the results of such a systematic study of the mitral valvar anatomy in a cohort of apparently banal discrete subaortic stenosis. This is in contrast with the number of papers[3,7,8,11–19] which describe either isolated cases of mitral valvar anomaly, suggesting that these cases are exceptional, or short series of one specific type of mitral anomaly, ignoring other possible types. In our opinion, it is important to analyze systematically the mitral valvar anatomy in all cases of discrete subaortic stenosis, whatever surgical conclusions are drawn from this systematic study. Not to do so would be illogical, since the aortic leaflet of the mitral valve is an integral part of the left ventricular outflow tract.

Even if our study has demonstrated an incidence of mitral valvar anomalies higher than previously reported, all the lesions found have been previously described. Lampros and Cobanoglu,[2] for example, found an axial rotation of the mitral valvar apparatus in 83% of their patients, although they included patients with atrioventricular septal defect in their series. In those with a common atrioventricular junction, of course, it is the rule to find axial rotation of the left ventricular papillary muscles. Anomalous insertion of a papillary muscle has been described by Klues et al.[20] in adults with hypertrophic myocardiopathy. The incidence of 13%, in their series of 78 patients, was relatively important. Tasdemir et al.[21] reported the same anomaly in a single patient. The same type of anomalous insertion has also been described, in a few cases of discrete subaortic stenosis.[13,19] The lesion that we call muscularization of the anterior leaflet could be the same entity that Rosenquist et al.,[14] and more recently Di Donato et al.[15] and Kleinert and Geva,[6] reported as "increased mitro-aortic separation". In particular, the echocardiographic image presented in these last two articles is exactly the same as the one we found in the patients who, following incision of the aortic leaflet, showed muscularization of the subaortic portion of the mitral valvar leaflet. Anomalous attachments of the mitral valve to the ventricular wall have also been described by Vairo et al.[17] Accessory mitral valvar tissue has frequently been recognized as a cause of subaortic obstruction.[8,12,16,18] Our findings suggest that the anomalous tissue may be the result of an abnormal portion of the valvar tissue attached in such a way that the flow of blood progressively

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distends this part of the valve, as suggested by Ow et al.[18] The supernumerary mitral valve described by Hatem et al.[11] and the diverticulums of the aortic leaflet described by Agathos et al.[22] could have been produced by a similar mechanism. The concept of mobilization of the fibrous trigones, suggested by Yacoub et al.[23] as a means of relieving subaortic stenosis, is not in contradiction with our findings, since it also aims at treating the anomalous position of the mitral valvar apparatus in the left ventricular outflow tract.

The role of the mitral valvar anomalies that we found in our study is still conjectural. Among the anomalies that we discovered, very few were, in themselves, hemodynamically obstructive. As recently stated by Freedom,[3] "for a number of years, it has been speculated that the occurrence of subaortic left ventricular outflow tract obstruction results from a specific anatomic substrate promoting abnormal and likely turbulent flow dynamics". It may be hypothesized that any anatomical or positional variation of the mitral valve can induce this type of flow, and thus promote the development and/or the recurrence of discrete subaortic stenosis. The fact that the obstructive fibrous shelf, or the so-called diaphragm, was situated exactly at the level of the mitral valvar anomaly in all our patients favours this hypothesis. In our view, this justifies our efforts in repairing this anomaly insofar as this is possible.

Regarding the surgical strategy, except for the axial rotation, all the types of mitral valvar anomalies that we have discovered thus far are generally amenable to at least partial repair. It is obvious that, preoperatively, the feasibility of repair must be analyzed for each specific case. Anomalous attachments or insertions should not be transected if they are essential to the valvar continence. This must be thoroughly analysed during the operation. In some patients, we chose not to treat some anomalous insertions when we felt that such a maneuver could compromise valvar function.

The results of our study are, in many ways, comparable to those of Klues et al.,[24] who found an unexpected incidence of anomalies through systematic study of the mitral valvar anatomy in a cohort of 94 patients with hypertrophic cardiomyopathy. We were particularly impressed by the fact that they frequently discovered, in their adult patients, the same type of anomalous insertion of a papillary muscle as the one we found in several of our children. The results of Hagège and coworkers[25] also tend to confirm the unexpected incidence of mitral valvar anomalies in obstructive cardiomyopathy. Kofflard et al. [26] proposed to treat this disease by constructing an extension of the aortic leaflet, as we did in several of our children. Levine et al.[27] demonstrated that experimental displacement of a papillary muscle causes systolic anterior motion of the mitral valve and subaortic obstruction. This may suggest that, at least in some patients, hypertrophic cardiomyopathy and discrete subaortic stenosis may have a common physiopathology, related to anatomic variations of the mitral valve. Bruce and coworkers[28] found, in four cases, various types of discrete subaortic stenosis in a cohort of patients referred to their institution for presumed hypertrophic cardiomyopathy. The recent description, by Conte et al.,[29] of the familial occurence of discrete subaortic stenosis is another argument in favour of this hypothesis. Should this be confirmed, the nosologic frontiers between these two types of cardiopathy would have to be revisited, as well as the therapeutic approach.

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