Mitral valvotomy and embolism

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MATERIAL

We have studied the case records of 468 patients who underwent mitral valvotomy, 363 female and 105 male, to discover the incidence and site of systemic embolism before, at, and after operation, and the relation of embolism to cardiac rhythm and to the presence of clot in the left atrium at operation.

Two hundred and seventy-two patients were operated upon in the Southampton Chest Hospital by the late E. F. Chin, K. S. Mullard, and I. K. R. McMillan during the period 1953–63, and 196 patients were operated upon by K. S. Mullard at Harefield Hospital in the period 1950–60.

Thirty-eight (8·3%) patients of this series have had a second operation for restenosis of the mitral valve, and one had a third operation. The mortality was 6·8%, 3% from embolism and 3·8% from other causes.

SYSTEMIC EMBOLISM Of 468 patients with mitral stenosis, 76 (16·3%) had a history of systemic embolism before operation. Five (1%) suffered embolism at operation and 33 (7%) after operation. Fifteen (3·2%) patients had embolism before and after operation. Ninety-nine (21·1%) patients in this series developed 114 embolic episodes before, at, and after operation (Table I).

Embolism was the prime indication for operation in 58 of the 76 patients with a pre-operative history of embolism ; thus it was the prime indication in 12·4% of the cases.

There were 15 cases of post-operative embolism among the 76 patients who suffered pre-operative embolism ; it is not known how many of these 15 occurred in the 58 patients in whom embolism was the prime or only indication for valvotomy, but it is unlikely that all the 15 post-operative cases arose in the 58 ; the proportionate figure is 8 to 9, and, if so, there would seem to be a 12 to 15% risk of post-operative embolism, if valvotomy is advised where embolism is the prime symptomatic indication.

The time of occurrence of post-operative embolism in 33 patients was as follows. In 13 patients it occurred hours after operation, in 14 after days, in two after weeks, and in four after months.

Of the 114 embolic episodes, 74 (65%) were cerebral and 40 (35%) were peripheral. We tried to find out if there was any evidence that systemic embolism occurred more commonly on the left than on the right side. There were 32 cases of right hemiplegia, 24 developed left hemiplegia, and in 18 it was impossible to discover from the records whether it was right-sided or left-sided. Of 40 peripheral episodes, 16 were in the right leg or arm and 14 were in the left leg or arm.

ATRIAL FIBRILLATION AND SYSTEMIC EMBOLISM The risk of systemic embolism in patients with atrial fibrillation, and especially at the onset of fibrillation, is high. Of 99 cases of systemic embolism, 82 occurred with atrial fibrillation, one with flutter, and 16 in sinus rhythm.

It is possible that some patients found in sinus rhythm had had an embolism during a paroxysm of atrial fibrillation which had subsequently reverted to sinus rhythm spontaneously.

CLOT IN THE HEART AT OPERATION Of our 468 cases of mitral valvotomy we found that 77 (16·4%) had clot in the left atrium and left appendage. Of these 77, only 20 (26%) had a history of systemic embolism before operation and 20 (26%) had systemic embolism at or after operation. Thirty-seven (48%) patients with clot in the left atrium did not have embolism before or after operation. It seems that clot in the left atrium does not necessarily cause systemic embolism.

TABLE I

	No. of Patients		
	Total	M	F
Mitral valvotomy	468	105	363
Restenosis of mitral valve ..	38 (8·3%)	10	28
Systemic embolism	99 (21·1%)	28	71

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HEAD VESSELS TAPED OR COMPRESSED IN RELATION TO SYSTEMIC EMBOLISM   From the patients' notes we found that head vessels were taped or compressed at operation in 44 cases; 13 had a history of embolism before operation, two had an embolism at operation, and five had an embolism after operation—three after hours, one after days, and one after months.

RESTENOSIS   Thirty-eight (8·3%) of the 468 valvotomies were for restenosis, *i.e.*, second valvotomies, and one was a third valvotomy (see Table I).

MORTALITY   There were 32 (6·8%) deaths from 468 valvotomies; 14 (3%) of these were from embolism and 18 (3·8%) from other causes.

DISCUSSION

In our series of 468 mitral valvotomies, 16·3% of patients had a history of systemic embolism before operation and 8% had systemic embolism at or after operation. From our figures it seems that the incidence of systemic embolism is higher before operation and lower after operation.

Baker, Brock, Campbell, and Wood (1952) reported 17% systemic embolism before operation and 7% after operation. Belcher and Somerville (1955) quoted 13% pre-operative embolism and 6% post-operative embolism. Szekely (1964) reported 9·6% systemic embolism from a series of 754 patients with rheumatic cardiovalvular disease. The low incidence in this latter series is because the author analysed patients with cardiovalvular disease and not patients with critical mitral stenosis. It is a known fact that tightness and calcification of the mitral valve predisposes to systemic embolism. Belcher and Gupta (1964) reported 7% cerebral embolism immediately after a second operation and 6% late emboli. The incidence of cerebral embolism (7%) was higher than after a primary operation (2·5%). Paul Wood (1954) quoted 14% systemic embolism in 150 surgical cases and 10% systemic embolism immediately complicating mitral valvotomy; he believed that the risk of embolism occurring at operation is as high as the risk of spontaneous embolism and so it is unwise to advise mitral valvotomy in order to prevent future embolism in patients who have so far been free from this complication. Bannister (1960), however, reported 21% systemic embolism from a series of 105 patients with moderate mitral stenosis and trivial symptoms in whom operation was deferred; he believed that the high incidence of embolism shows that the custom of deferring operation in patients with moderate stenosis and trivial symptoms is unsatisfactory. Our figures, and those of other authors, show that although the incidence of post-operative embolism is high, it is lower than that for pre-operative embolism, and they suggest that a history of embolism, while carrying a 10 to 15% risk of post-operative embolism, is nevertheless a good indication for operation. The condition of the myocardium plays an important part in the development of systemic embolism. Fleming and Paul Wood (1959) reported 46% systemic embolism in cases with mild mitral stenosis and myocardial dysfunction.

Cerebral embolism is more common than peripheral embolism in our series of 99 patients who had systemic embolism (114 embolic episodes); 74 (65%) were cerebral and 40 (35%) were peripheral. Paul Wood (1954) reported 75% cerebral, 33% peripheral, and 6% visceral. Belcher and Somerville (1955) reported 59% brain, with equal numbers on each side, 13% right leg, 11% left leg, 7% aorta, 4% right arm, and 7% other sites (kidney, spleen, and mesentery).

Bailey, Olsen, Keown, Nichols, and Jamison (1952) reported five right and seven left cerebral emboli. Bannister (1960) reported 22 systemic embolisms, 15 cerebral and seven peripheral.

It is generally agreed that atrial fibrillation increases the tendency to systemic embolism. It seems that the low cardiac output and the stagnation of the blood in the left atrium, especially in the left appendage, leads to the formation of clot in the left atrium. The risk of systemic embolism is high at the onset of fibrillation but it is possible for systemic embolism to develop a long time after the onset of atrial fibrillation. Bannister reported that systemic embolism occurred three times as commonly in patients who were fibrillating as in those in sinus rhythm, and nine patients developed systemic embolism between one and five years after the onset of fibrillation. Fleming and Paul Wood (1959) reported 24 cases of mild mitral stenosis and cardiac dysfunction in atrial fibrillation: 11 had systemic embolism.

Systemic embolism is not uncommon in mitral stenosis with sinus rhythm. Baker and Finnegan (1957) reported 31 cases of systemic embolism, eight in sinus rhythm.

Belcher and Somerville (1955) reported atrial fibrillation in 78% of their cases of systemic embolism; Szekely (1964) similarly reported 64%. Keen and Leveaux (1958) reported 87% in atrial fibrillation and 13% in sinus rhythm. Belcher and

Somerville noted the tendency to embolism soon after the onset of fibrillation. In our 99 cases of systemic embolism, 82 were in atrial fibrillation, 16 were in sinus rhythm, and one was in flutter. From these figures it is clear that the occurrence of systemic embolism is five times higher in patients in atrial fibrillation than in patients in sinus rhythm.

Baker and Hancock (1960) noted that atrial fibrillation may first develop soon after valvotomy. We have not investigated the incidence of this in the present series.

Turner and Fraser (1956) reported that 24% of patients who were in sinus rhythm before mitral valvotomy developed atrial fibrillation post-operatively. It is presumed that atrial fibrillation in these patients is related to traumatic pericarditis and epicarditis in the setting of rheumatic heart disease. The same workers believe that it is impossible to decrease the incidence of post-operative fibrillation by the administration of quinidine, and also that it is unprofitable to attempt to restore sinus rhythm by treatment with quinidine in the first post-operative days.

None of our patients developed systemic embolism during the administration of quinidine to restore sinus rhythm, as was the usual practice before the use of the D-C defibrillator.

Bannister (1960) found that the incidence of emboli was five times as great in patients aged 40 and over as in patients under the age of 40.

Of 468 valvotomies in our series, only 77 (16·4%) patients were found to have a clot in the left atrium. Thirty-seven of these patients (48%) had no embolism before or after operation. Paul Wood (1954) reported that only one-fifth of all the patients who had a clot in the left atrium at the time of operation developed systemic embolism. Our figures are higher, namely, one-fourth, but it seems that clot in the left atrium is not closely associated with pre-operative or post-operative embolism.

Baker and Finnegan (1957) quoted that, of 150 valvotomies, 30 (20%) patients had a clot in the left atrium at operation and only 13 gave a history of previous systemic embolism. But Belcher and Somerville (1955) found clot in the left atrium in 16% of 430 valvotomies, and observed that clot was more than twice as common (30%) in those with a history of previous embolism as in those without such a history (13%).

The risk of occurrence of a systemic embolism during operation, especially at the time of manipulation and soon after the operation, is high. The emboli can be small pieces of clot from the left atrium or pieces of calcium from a calcified mitral valve. For this reason many surgeons nowadays tape or compress the head vessels to reduce the incidence of a systemic embolism.

Of all our cases, the head vessels were taped or compressed in 44 during operation. Thirteen had had a history of systemic embolism before operation. Two systemic emboli occurred during operation and five after operation, three after hours, one after days, and one after months.

Belcher (1960) reported that in 50 valvotomies for restenosis of the valve, carotid compression was applied during the manipulation but cerebral emboli occurred in three cases (6%). This incidence was higher than that after first operations, when it was 2·5% (Belcher, 1960).

This difference may be due to the high incidence of calcification with the attendant risk of emboli due to small pieces of calcium broken off the valve.

Adams (1961) reported a case of multiple calcific cerebral embolism following mitral revalvotomy, the carotid arteries being compressed by external pressure.

Bailey *et al.* (1952) reported that cerebral embolism occurred in 5·1% of 235 cases in which methods other than carotid obstruction were employed to protect the brain at operation.

Our experience suggests that occlusion of the cerebral vessels during operation has little or no protective effect against embolism. Two of the 44 patients in whom the head vessels were taped developed emboli at operation, but only three of the remaining 424, in whom there is no record of taping the head vessels, developed embolism at operation. Of this same group of 44, four developed post-operative embolism after hours or days. Of the remaining 424, 23 developed embolism within hours or days. So far as it is permissible to reduce these figures to percentages, 9% of the group in which the vessels were taped developed embolism soon after operation, but only 5% of a much larger group in which the vessels were not taped. It seems that the vessels were taped in those cases in which the surgeon correctly anticipated a high risk of embolism, but it is not possible to say that taping the vessels had any protective effect; from our figures it did not, and it is the opinion of one of us (K. S. M.) that the additional manipulation required detracts from the prospect of satisfactory valvotomy.

The incidence of restenosis in our series was 8·3%, a figure comparable to that in other series; Fraser and Kerr (1961) report 10%; Jesus, de

Breneman, and Keyes (1962) report 6% in 672 cases.

Belcher (1960) reported 11% restenosis, and considers whether this is due to recurrent rheumatic carditis or to incomplete valvotomy. Baker and Hancock (1960) reported 1% restenosis per year, and found it always to be due to incomplete valvotomy. Our own experience is that restenosis is very largely, if not entirely, due to incomplete valvotomy, and that the incidence appears far lower following the more efficient transventricular valvotomy than after the digital valvotomies of the earlier part of the series.

The incidence of mortality in this series was 6·8%, 3% from embolism and 3·8% from other causes.

This is comparable with other series; Baker *et al.* (1952) showed 8% mortality, half due to embolism; Turner and Fraser (1956) 6·4%, of which 2·4% was due to cerebral embolism; Belcher (1960) reported 5·5% from first valvotomies and 8% from second valvotomies: Fraser and Kerr (1961) reported 8·6% mortality, 2·2% from embolism; Belcher and Gupta (1964) noted an operative mortality of 5%, of which 3% was due to embolism. All the reports, including our own, show a high incidence of operative mortality from embolism.

SUMMARY

We studied 468 patients who had undergone valvotomy to find out the incidence of systemic embolism before, at, and after operation.

The incidence of systemic embolism is twice as high before operation as at, or after, operation.

Cerebral embolism was about twice as frequent as peripheral.

Restenosis was found in 8·3% of our series.

Five-sixths of our patients with systemic embolism were in atrial fibrillation and one-sixth in sinus rhythm.

Of the patients with clot in the left atrium 26% had a history of systemic embolism and 26% developed embolism at and after operation; 48% of the patients with clot had no embolism.

Taping of the head vessels at the time of operation was not shown to have any effect in reducing the incidence of embolism.

The mortality was 6·8%, 3% from embolism and 3·8% from other causes.

REFERENCES

Adams, M. J. T. (1961). Multiple calcific embolism following mitral valvotomy. *Brit. Heart J.*, **23**, 333.
Bailey, C. P., Olsen, A. K., Keown, K. K., Nichols, H. T., and Jamison, W. L. (1952). Commissurotomy for mitral stenosis: technique for prevention of cerebral complications. *J. Amer. med. Ass.*, **149**, 1085.
Baker, C., Brock, R. C., Campbell, M., and Wood, P. (1952). Valvotomy for mitral stenosis: a further report on 100 cases. *Brit. med. J.*, **1**, 1043.
Baker, C. G., and Hancock, W. E. (1960). Deterioration after mitral valvotomy. *Brit. Heart J.*, **22**, 281.
—— and Finnegan, T. R. L. (1957). Epilepsy and mitral stenosis. *Ibid.*, **19**, 159.
Bannister, R. G. (1960). The risks of deferring valvotomy in patients with moderate mitral stenosis. *Lancet*, **2**, 329.
Belcher, J. R. (1960). Restenosis of the mitral valve. An account of 50 second operations. *Ibid.*, **1**, 181.
—— and Gupta, R. L. (1964). Restenosis of the mitral valve: a study of 100 cases treated by operation. *Brit. med. J.*, **1**, 871.
—— and Somerville, W. (1955). Systemic embolism and left auricular thrombosis in relation to mitral valvotomy. *Ibid.*, **2**, 1000.
Fleming, H. A., and Wood, P. (1959). The myocardial factor in mitral valve disease. *Brit. Heart J.*, **21**, 117.
Fraser, K., and Kerr, I. F. (1961). Mitral valvotomy: a comparison of results by two operative methods. *Brit. med. J.*, **2**, 339.
Jesus, J. R., de Breneman, G. M., and Keyes, J. W. (1962). Recurrent stenosis of the mitral valve. *Circulation*, **25**, 619.
Keen, G., and Leveaux, V. M. (1958). Prognosis of cerebral embolism in rheumatic heart disease. *Ibid.*, **1**, 1209.
Szekely, P. (1964). Systemic embolism and anticoagulant prophylaxis in rheumatic heart disease. *Ibid.*, **1**, 1209.
Turner, W. D. R., and Fraser, H. R. L. (1956). Mitral valvotomy: a progress report. *Lancet*, **2**, 525.
Wood, P. (1954). An appreciation of mitral stenosis. *Brit. med. J.*, **1**, 1051.