EMBOLISM IN MITRAL STENOSIS

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A man was examined for a railway company because his disability had been ascribed to his employment. He had felt quite well and had been able to perform his work as an electrician until three months before he was examined, when he was suddenly struck down in the course of his routine work by an attack of left-sided weakness. Physical examination proved that this was due to cerebral embolism secondary to mitral stenosis. It was claimed that this embolism was due to physical effort. A search throughout many textbooks and articles gave no solution to the question of the relationship between exercise and embolism in this disease, and further cases met with in the course of practice were therefore subsequently tabulated in order to attempt to find an answer.

RELATIONSHIP OF EXERTION TO EMBOLISM

The series forming the basis of this study is one of 29 patients in all, with 31 embolic incidents, two of the patients having suffered from two separate emboli separated from one another by a space of time. It is often assumed that embolism in mitral stenosis occurs only in patients in whom auricular fibrillation is present: this is not universally true. In the present series of 29 cases, 6 showed regular rhythm and 23 auricular fibrillation. The relationship of embolism to blood flow through the auricles and to exercise generally is interesting. Embolism occurred very much more frequently in patients who were ambulatory, and quite rarely in patients whose activities were restricted by the presence of congestive failure. The degree of mobility was as follows: 24 of the embolic incidents occurred in patients who had good exercise tolerance, being normally ambulatory although restricted slightly by a little shortness of breath; 4 of the incidents were in patients who were ambulatory, but had a poor exercise tolerance although signs of congestive failure were absent; and 3 of the incidents were in patients who had signs of congestive failure at the time of embolism.

These facts would at first suggest that exercise as such might be a factor in the causation of embolism when a clot is present in the left auricle; on the other hand, an analysis as to the time of onset of the embolism points to a contrary conclusion. Only twice in the whole series did an embolism occur while the patient was actively mobile. The first of these was a man with regular rhythm aged 45, who was a machine operator in active employment: he had walked on the flat to get two mugs of tea for himself and his mate; and on his return, on reaching for a spanner, he found his hand had become weak. The second was a woman, a railway porter, of 41 whose heart rhythm was also regular; whilst walking on the platform she was forced to sit down, finding that her left arm and leg had suddenly become weak.

Seven of the cases, all with auricular fibrillation, were seized by the effects of embolism while undertaking very easy physical exercise. The individual circumstances were as follows: sudden weakness of the arm occurred while engaged in brushing his hair in a man of 65; weakness of her right side overtook a woman of 45 while she was kneeling, taking cinders out of her grate; a woman of 34 suddenly became weak in her arm and leg whilst sitting, talking to a friend; a woman of 41 had sudden right-sided weakness while making tea; a woman of 45 suffered sudden numbness down her left side while she was walking about her house, having been rather more busy than usual; and a

woman of 41 had a sudden right hemiplegia while she was standing up at home. Thirteen of the whole series of patients suffered from hemiplegia or other embolic symptoms while sitting down, and nine of them while asleep in bed.

The position of the embolic lesions was as follows: 22 of them were cerebral; 3 were popliteal; and one each was axillary, femoral, retinal, splenic, and mesenteric. There was no renal embolism in this series.

DISCUSSION

Embolism in mitral stenosis has become a subject of increased importance since the introduction of anticoagulant therapy. A recent paper on resection of the left auricular appendix by Madden (1949) also raises the question as to whether patients liable to embolism are likely to be satisfactory subjects, either as regards surgical risk or as regards post-operative expectation of life. The third point of practical importance, in reference to compensation cases, has been already referred to at the commencement of this paper.

In patients with mitral stenosis in whom the narrowing is great or very considerable, there is an accumulation of blood in the left auricle, which chamber is therefore more distended than normal. When regular rhythm is present and the auricles are contracting, there is an accentuation in the onward flow of blood from left auricle to left ventricle during auricular systole, and in proportion to the degree of stenosis the chamber remains to some degree distended most of the time. When auricular fibrillation supervenes this state of auricular distention becomes permanent. The effect of permanent or nearly permanent auricular distention upon sessile clot in the chamber would be that such clot remains undisturbed or very little disturbed by the flow of blood in the chamber and by variations of pressure. If mitral disease with little narrowing of the mitral ring is considered, this mitral ring having been made rigid or somewhat rigid by chronic rheumatic endocarditis, a different physical state must occur. In proportion to the wideness of the mitral ring there will be a sudden rise of interauricular pressure at the time of left ventricular systole, due to mitral regurgitation. Here there will be both a diastolic flux and a systolic reflux of blood, similar to that of sea-water flowing in and out of a rock pool fed from above, as the tide begins to fill it. Clot in this case, unless it is closely adjacent to the wall, would stand a far greater chance of being moved to and fro, fragmented, and washed free into the blood stream. Catheterization of the left auricle being at the moment impossible of attainment, it is not feasible to check the left auricle pressures by practical experiment on the living human heart, but the facts stated seem to admit of no other explanation, and they also are compatible with the clinical observations made in the above series of cases.

The following conclusions as regards the likelihood of embolism in mitral stenosis seem to be legitimate. Embolism is not directly related to physical effort or exertion. Embolism is not usually due to clot formation in the dilated auricle of severe congestive failure. Embolism is commonest in patients whose exercise tolerance is good or very fair, and in whom the mitral orifice is therefore relatively large, allowing a considerable degree of regurgitation in addition to a slight or only moderate degree of stenosis.

REFERENCE

Madden, J. L. (1949). *J. Amer. med. Ass.*, **140**, 769.