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Anticoagulants in Mitral Valve Disease

SIR,—Your leading article concerned with anticoagulants in mitral valve disease (11 March, p. 641) calls for comment.

It is stated that "most cardiac surgical centres now accept that closed mitral valvotomy is better performed under anticoagulant cover." I do not believe that this is so a routine at the Middlesex or the London Chest Hospitals and I believe that their use is the exception rather than the rule. Their introduction in this context roughly coincided with the widespread adoption of Logan's operation with the transventricular dilator, and this operation has, in itself, halved the incidence of immediate postoperative emboli in my hands (the figure stands at 2% among the last 500 patients).

Secondly, the value of the amputation of the appendage in the prevention of late emboli has been questioned. The value of this procedure was, I believe, established at a meeting of the Cardiac Society four years ago, when it was apparent that the incidence of late emboli in the Edinburgh series of over 800 patients was double that of mine in 840 patients (3%). Both these series were spread over a period of nearly 20 years. In Edinburgh the policy had been to leave the appendage; in mine it had always been amputated. It is also surely relevant that 40% of my 24 patients with late emboli had restenosis. The paper quoted in support of the view that amputation made no significant difference is based on a very much smaller number of patients.

Lastly, the incidence of late emboli is low despite an often long follow-up period, and against this background it cannot possibly

be wise to condemn almost all the patients with mitral stenosis whether operated on or not to a lifetime of anticoagulants. This is what your leader writer implies should be done, despite all the difficulties and dangers inherent in this policy.—I am, etc.,

J. R. BELCHER

London Chest and Middlesex Hospitals

SIR,—Your leading article on "Anticoagulants in Mitral Valve Disease" (11 March, p. 641) is of particular interest to us since we are engaged in analysing 20 years of experience with more than 1,000 patients. Our conclusions differ in a number of respects.

It may be that anticoagulants are of value in this condition but we remain uncertain because, to our knowledge, no controlled trial has been published. In the report from the Royal Postgraduate Medical School, the conclusion that anticoagulants protect from operative embolism is not valid.1 The anticoagulant and the control series were not contemporaneous. Without the use of anticoagulants our incidence of operative embolism fell to an even greater degree. This was clearly attributable to improvement in the surgical technique which seems to us the probable explanation of the Hammersmith experience. There is no evidence that embolism is "largely preventable". It is not uncommon in patients under good anticoagulant control.

We agree that the incidence of embolism is unaffected by removal of the atrial appendage. Like Coulshed and his colleagues from Liverpool,² we have found no close relationship between the size of the left atrial appendage and embolism except that those with small appendages are at greatest risk. This is in striking contrast to the experience from the Middlesex Hospital.³

We doubt that anticoagulant treatment is usually given to those with atrial fibrillation or with appreciable mitral stenosis. A few years ago inquiry from 20 leading centres showed that this was not the case. We also very much doubt that most cardiac surgical centres now accept that closed valvotomy is better performed under anticoagulant cover. In our experience embolism is far more frequent in mitral stenosis than in pure or dominant regurgitation. In fact, it is rare in regurgitation.

The quoted incidence of 11% in patients with sinus rhythm at the time of examination is the usual one. We agree that embolism can occur with mitral lesions which are haemodynamically insignificant. It is essential in any analysis of embolism to take account of rhythm, because embolism is most frequent in those with atrial fibrillation and particularly soon after its onset. Our series shows that the incidence of embolism in a follow-up period of 5-20 years after valvotomy is no less than before operation but is largely related to the onset of atrial fibrillation, the majority of patients having been in sinus rhythm at the time of operation. Possibly the incidence would have been greater had valvotomy not been performed. Thus we are in agreement with Szekely4 that valvotomy does not eliminate and questionably does not reduce the incidence of later embolism. It is difficult to see how systemic embolism itself can be accepted as an indication for valvotomy.