Editorial comment

Surgical treatment of secundum atrial septal defect in the older patient

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The natural history of secundum atrial septal defect (ASD) is not known. Many escape diagnosis in childhood but are rightly closed if recognised because surgical mortality and morbidity are extremely low and the subsequent prognosis is normal. The prognosis of ASDs closed surgically after the age of 25 years is not normal mainly because of atrial arrhythmias and thromboembolism which are not prevented[1].

ASDs are frequently discovered after investigation of a murmur or an abnormal ECG or chest X-ray found incidentally or because of symptoms from coronary or some other acquired unrelated heart disease. The development of systemic hypertension or of left ventricular dysfunction due to any cause will increase left to right shunting and lead to right rather than left sided congestive features.

Some ASDs are first diagnosed when they develop a late complication of the ASD particularly atrial flutter or fibrillation or thromboembolism which is usually associated with atrial arrhythmia. Mitral regurgitation caused by a prolapsing leaflet may develop in middle or later age. The valve will need to be repaired and the ASD closed.

ASD can also be a surprise finding in an old and sometimes very old person, and it is clear that an unknown number are never recognised at all.

ASD can be closed surgically at very low risk even in an older population but with greater morbidity than in childhood. What is less clear from the literature is the benefit of surgery[2] especially in asymptomatic individuals. Despite this the discovery of an ASD is usually taken as an indication for its closure. Published series usually describe a heterogeneous collection of people brought together for all the above reasons. In some patients there are clear indications for closure such as recurrent chest infections or marked cardiomegaly with congestive features. Late acquired pulmonary hypertension is an indication but this is sufficiently rare that prophylactic closure to prevent it is not.

In this issue 65 patients with ASD aged over 50 years and operated on over a 24 year period with a zero surgical mortality are reported[3]. This was a retrospective study and we are not told how they came to be diagnosed. Details of functional class, occurrence of arrhythmias, transient ischaemic attacks and strokes before operation were obtained from the hospital records and current status was sought by questionnaire. Reliance was therefore placed on patients' memories rather than on hospital notes for follow-up events.

Data were available in 53 of the 65 patients. Half of the patients had been asymptomatic or mildly symptomatic (functional class I or II) at the time of operation. At last follow-up 70% were in class I or II and 30% in class III or IV. Only 1 patient had deteriorated – from class III to class IV.

All the patients who had been in atrial flutter or fibrillation preoperatively were still in it and 7 out of 19 patients who had been in sinus rhythm preoperatively had developed atrial fibrillation postoperatively. Symptoms were markedly associated with these arrhythmias and lack of symptoms with sinus rhythm. Thromboembolic events were associated with atrial arrhythmias in patients who were not on warfarin. There were 8 late deaths. This was not compared with the number expected in the normal population.

Direct suture had been used in some patients and patch closure in others but no comment was made about any influence the method of closure may have had on the subsequent incidence of arrhythmias or thromboembolism, or on whether right atrial size is a determinant.

This study confirms that ASD can be closed at low risk in older patients. It offers no guidance on selection of patients but confirms that surgery fails to influence the development of atrial arrhythmias and that they are a major cause of morbidity and mortality in ASD. Time will show whether the results of device closure, by avoiding right atrial incision and suture, will be superior.

References

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