Echocardiographic diagnosis of a ruptured aneurysm of the sinus of Valsalva: operation without catheterisation in seven patients

Sir,—Dr Sahasakul and colleagues (British Heart Journal 1990;64:195–8) clearly described the echocardiographic diagnosis of a ruptured aneurysm of the sinus of Valsalva, but they made no suggestion about its aetiology other than to label it a disease of "congenital origin". The ruptured sinus affected the right coronary sinus in six of their seven patients. Figure 1 in their article clearly shows the absence of the subpulmonary infundibulum and fibrous continuity between the aortic root and the pulmonary valve (fig 1B), features characteristic of a doubly committed subarterial ventricular septal defect, with patresiation of the right coronary sinus into the right ventricle (fig 1A).

The usually accepted explanation for a congenital aneurysm of the sinus of Valsalva is thinning of the wall of the aortic sinus just above the leaflet hinge, although this alone does not explain why the left coronary sinus is hardly ever involved. The association of the aneurysm of the sinus of Valsalva with a ventricular septal defect was described in Japan. Significant changes in morphology were proposed, leading to rupture of the right coronary sinus into the right ventricle in young adulthood when there was a ventricular septum in the outlet portion of the septum. It is notable that there is an increased incidence of ruptured aneurysm of the sinus of Valsalva in Oriental populations, just as there is of doubly committed subarterial ventricular septal defect. Large defects without deformity or offsetting of the aortic valve cause major haemodynamic disturbance in infancy, whereas smaller holes typically present in childhood with aortic regurgitation because of prolapse of the right coronary aortic cusp; but they may present with rupture of a sinus of Valsalva aneurysm in adult life. Indeed, during a 10 year period at our hospital 32 of 36 patients undergoing surgery for a ruptured aneurysm of the sinus of Valsalva had "subarterial infundibular" ventricular septal defects.

Thus it should be emphasised that the likely substrate for aneurysm of the sinus of Valsalva in many patients is the presence of a doubly committed subarterial ventricular septal defect with offsetting of the arterial valves. Prolapse of the right coronary aortic cusp results in elongation of the sinus and thinning of the sinus wall just above the leaflet hinge. Mechanical stress leads to dilatation of the sinus over many years, and sometimes rupture into the right heart. Patients with a doubly committed subarterial ventricular septal defect and offsetting of the arterial valves, which can be observed echocardiographically, are at risk of this latter complication.

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This letter was shown to the authors, who reply as follows:

Sir,—Dr Sullivan and Dr de Leval have correctly pointed out the association between ruptured aneurysm of the sinus of Valsalva and a doubly committed subarterial ventricular septal defect in most cases reported in the Orient. Nevertheless, there are several other causes of a ruptured aneurysm of the sinus of Valsalva, chiefly a congenital lack of fusion between the media of the aorta and the annulus of the aortic valve. Other reported causes include bacterial endocarditis, Marfan's syndrome, coarctation of the aorta, bicuspid aortic valve, trauma, and also the association with ventricular septal defect.

The reason for the high prevalence of ruptured aneurysms of the sinus of Valsalva in infantil patients is not known. We propose a genetic cause or perhaps a logistic one: patients with ventricular septal defect in the Orient may not see a doctor until symptoms develop—that is after the aneurysm has already ruptured. The latter proposal is particularly true in Thailand because many paediatric patients with ventricular septal defect went unnoticed for several years because they were born in remote provincial areas without proper medical facilities.

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Impending paradoxical embolism: a rare but important diagnosis

Sir,—I read with interest the recent case report of impending paradoxical embolism by Speckly-Dick et al. (British Heart Journal 1991;65:163–5). However, I would like to offer two differing opinions on the points they made.

First, their statement that only two of 37 reported cases of paradoxical embolism were diagnosed during life was an underestimation. All of 25 cases reviewed and discussed by Leonard et al and 10 of 27 cases reviewed by Jungbluth et al, cited by the Speckly-Dick et al in their paper as references 4 and 6, respectively, were diagnosed during life. The two cases reported by me in 1976 were diagnosed during life.

Second, the current treatment choice for patent foramen ovale or minute atrial septal defect as a cause of paradoxical embolism is non-surgical transcatheter closure. If this defect could be closed during a cardiac catheterisation, the need for open heart surgery would be obviated together with the extended hospital stay, expense, need for a procedure for the morbidity, mortality, and psychological trauma associated with surgery.

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This letter was shown to the author, who replies as follows:

Sir,—Professor Cheng is correct in saying that there are many cases of paradoxical embolism diagnosed in life and reported in the literature. However, our case report described the rare finding of impending embolism of which, to my knowledge, there are only two previously reported cases. The second point which Professor Cheng raised was that in his opinion the current treatment of choice for patent foramen ovale or minute atrial septal defect is non-surgical transcatheter closure. This is a relatively new procedure for which we have, as yet, no long term results. Surgical closure, however, is a well-established and accepted technique with very low mortality and morbidity and excellent long term results. Non-surgical transcatheter closure may well be the treatment of the future but is not yet the treatment of choice.

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