Imaging the dissected aorta

Sir,-I read with interest and sympathy Mr Treasure’s editorial on imaging the dissected aorta.1 I agree that it is a difficult to achieve a standard approach. I have found the following guidelines for acute dissection clinically reliable.

(a) Type B dissection is defined as a dissection confined to the descending aorta—that is, any part of the aorta beyond the origin of the left subclavian artery.

(b) Type A dissection is any dissection that involves the ascending aorta, whether or not it also affects the descending aorta, and irrespective of the site of entry.

(c) Computed tomography (CT) is a reliable technique for the detection and assessment of type B dissections. It is certainly more reliable than angiocardiography. This is because the flap in type B dissections is usually static and can be reliably imaged by CT, despite the fact that the true lumen has retained a smooth, circular cross section.

(d) In type A dissection the flap in the ascending aorta is often mobile, giving a significant incidence of false negative CT examinations, but nevertheless . . .

(e) The ascending aorta is always dilated in type A dissection. If a dissection in the descending aorta is accompanied by an ascending aorta of normal size it is reasonable to exclude type A origin or extension of the false lumen.

In practice, I would like to endorse the efficacy of transoesophageal sonography in the diagnosis of traumatic aortic tear, as described by de Belder et al.2 Indeed, I have yet to hear of a falsely negative study.

* * *

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


Anthracyclines and the heart

Sir,—I thank Dr Rhoden, Dr Hasleton, and Dr Brooks for an excellent review of anthracyclines and the heart.3 I would like to point out an error. The evidence for doxorubicin-related cardiotoxicity involving myocardial adrenergic derangement comes from 125I-meta-iodobenzylguanidine (MIBG) rather than from 123I-methoxy-isobutyl isonitrile (MIBI).4 These radiopharmaceuticals are quite dissimilar. MIBI is a lipophilic agent that scintigraphic myocardial perfusion imaging with technetium-99m isonitrile: A critical evaluation of the role of technetium-99m imaging in aortic dissection. J Thorac Cardiovasc Surg 1993;105:375–8.


This study was sent to the author, who replies as follows:

Sir,—In their otherwise excellent article Bu’Lock and Hasleton,5 and in any other details of the morphology of the aortic valve itself, specifically the number of leaflets. This is of importance because in the so-called unicommissural and unicusp variant of aortic valve stenos- is recent studies have shown that the leaflet tissue is attached within the aortic root in a circular rather than a semilunar fashion.2 This arrangement would seem, on morpho- logical grounds, to militate against successful balloon dilatation: but morphologists are constantly wary of predicting outcomes in life from their observations on cadaveric hearts. For this reason it would be invaluable to know whether Bu’Lock and her colleagues had information on the number of leaflets present in the valves dilated in their patients?”

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1 Bu’Lock FA, Joffe HS, Jordan SC, Martin EP. Balloon dilatation (valvoplasty) as a first line treatment for severe stenosis of the aortic valve in early infancy: medium term results and determinants of survival. Br Heart J 1993;70:546–53.


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Familial atrioventricular septal defect: possible genetic mechanism

SIR—We read with interest the report of Kumar et al describing a family in which the mother and her two daughters by different fathers had atrioventricular septal defects not associated with trisomy 21.1 They suggest that an autosomal dominant pattern of inheritance may be involved in this pedigree, although a multifactorial or cytoplasmic mechanism cannot be ruled out.

We recently described five families in which two or more members had isolated atrioventricular septal defect. Parent-to-son transmission of concordant cardiac defect was documented in four cases. The mother was affected in three cases and the father in one.2

Later, we found an additional example of atrioventricular septal defect in four members in two generations of the same family (figure). None of the family members showed phenotypic anomalies and all had a normal karyotype. They were examined by echocardiography and electrocardiography. A partial atrioventricular septal defect was diagnosed in one case (II 5) and a complete defect in two cases (III 1 and III 2). The daughter of a patient with atrioventricular septal defect had an isolated cleft of the mitral valve (III 3).

Anatomical differences between isolated mitral clefts and clefts associated with an atrioventricular septal defect have been described.3 The latter form of isolated mitral cleft in families with atrioventricular septal defects,4 including our family, and its prevalence in patients with Down’s syndrome (4/420 vs 5/200 in our experience), however, suggests that this malformation should be included in the spectrum of atrioventricular septal defects.

Monogenic autosomal dominant inheritance with incomplete penetrance could explain the atrioventricular septal defects in the families we studied and those reported by other workers. Normal parents of affected children could be obligate carriers of the gene involved in familial atrioventricular septal defects. The father-to-daughter transmission of cardiac malformation in two cases excludes cytoplasmic inheritance in these families. Moreover, atrioventricular septal defects in patients with and without Down’s syndrome differ not only in terms of the prevalence of partial or complete forms8 but also in terms of the distribution of associated cardiac malformations.9 These anatomical differences and the absence of linkage in the molecular analysis of chromosome 21 families with atrioventricular septal defect10 suggest that the gene or genes involved in the pathogenesis of atrioventricular septal defect in “normal” children are different from those in patients with Down’s syndrome.

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