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 task to achieve a standard approach. I have found the following guidelines for acute dissection clinically useful:

(a) Type B dissection is defined as a dissection confined to the ascending 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 method of detection and assessment of type B dissections. It is certainly more reliable than angiography. This is because the dissection in type B dissections is usually static and can be reliably imaged by CT, despite the fact that the true aortic wall may have regained 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 effort 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.

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

Sir,—Dr Partridge’s guidelines indicate his keen interest in this problem and that he has considered it carefully. I will address his points item by item. (a and b) In the original paper on which the Stanford classification is based type B is defined as dissection that does not extend proximally to the subclavian artery. In a subsequent paper there was a subtle but important change in detail.3 Type A includes any dissection that involves the ascending aorta, just as Dr Partridge writes, but type B includes all the rest, thus including some cases with arch involvement.4

No classification is perfect but the virtue of the current version of the classification is that it is clear. This is different from the group (type A) in which a challenging but achievable operation on the ascending aorta protects the patient from three lethal consequences of dissection at this site:

- Rupture into the pericardium
- Severe aortic valve regurgitation
- Occlusion of the coronary ostia

This reduces the risk from near 100% for type A to a much lower figure, made up of the risk of the operation itself and the risk associated with any residual uncorrected abnormality in the arch and descending aorta. Classification is a very interesting discipline. In this instance type A is defined by inclusion of the particular characteristic—that is, involvement of the ascending aorta—and type B is defined by exclusion of this characteristic. Both are necessary. His observations on the nuances of the interpretation of computed tomograms of the ascending and descending aorta are nicely observed and ring true.

Although I had no data or experience on which to base a comment, I was worried when de Belder et al advocated transoesophageal echocardiography to diagnose a traumatic aortic dissection. Dissection has length, so any cross sectional image will detect it. Traumatic aortic transection is a tear with an adjacent haematoma; it is not a propagating dissection. Because there are other sources of blood (rib and vertebral fractures) to cause the haematoma in trauma, it is visualisation of aortic wall discontinuity that is critical. High specificity, that is confidently excluding the diagnosis when it is present, is important. Have we argued elsewhere5 that the cross sectional image of CT cannot prove or exclude traumatic aortic dissection. In a critically injured patient this makes CT an unnecessarily expensive waste of time. The fact that transoesophageal echocardiography can be used at the bedside makes it attractive, provided a negative test is convincing and any induced hypertension and local interference do not make the aorta go "pop".

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Anthracyclines and the heart

SIR,—I thank Dr Rhoden, Dr Hasleton, and Dr Brooks for an excellent review of anthracyclines and the heart.1 I would like to point out an error. The evidence for doxorubicin-related cardiotoxicity involving myocardial adrenergic derangement comes from 121I-meta-iodo-benzylguanidine (MIBG) rather than from 123I-methoxy-isobutyl isonitrile (MIIB).2 These radiopharmaceuticals are quite dissimilar. MIBG is a uptake mimickers into sympathetic nerve endings as noradrenaline. It is therefore ideally suited to imaging both the distribution of sympathetische nerve endings in the heart as well as neuroendocrine defauna such as pheochromocytoma.3 MIIB is a myocardial perfusion agent available in cold kit form that is labelled with technetium-99m rather than with iodine-123. MIIB is a lipophilic agent taken up by myocytes independently of the sympathetic nerve endings but roughly proportionally to myocardial blood flow. MIBG is therefore used to assess the patency of coronary arteries rather than the status of the sympathetic nervous system.4

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We thank Dr Thomas for drawing attention to this error and for apologising for introducing this mistake when the technical editor missed the abbreviation MIBG for MIIB. Authors can help us to avoid such mistakes by spelling out all abbreviations and acronyms at the first mention—EDITOR


Balloon dilatation (valvoplasty) as first line treatment for severe stenosis of the aortic valve in early infancy: median term results and determinants of survival

SIR,—In their otherwise excellent article Bu-lock et al.1 have given few details of the morphology of the aortic valve itself, specifically the number of leaflets. This matter is of importance because in the so-called unicommissural and unicupid variant of aortic valvular stenosis 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 morphological 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, Jaffe HS, Jordan SC, Martin EP. Balloon dilatation (valvoplasty) as first line treatment for severe stenosis of the aortic valve in early infancy: median term results and determinants of survival. Br Heart J 1993;70:546–53.
Familial atrioventricular septal defect: possible genetic mechanism

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 Down's Syndrome. 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. Recently 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 any anatomic anomalies and all had a normal karyotype. They were examined by echocardiography and cardiac catheterization. A partial atrioventricular septal defect was diagnosed in one case and a complete defect in two cases. The daughter of a patient with atrioventricular septal defect had an isolated cleft of the mitral valve (figure 3). Anomalies differences between isolated mitral clefts and clefts associated with an atrioventricular septal defect have been described.1,2 The high frequency of isolated mitral cleft in families with atrioventricular septal defects,3-5 including our family, and its prevalence in patients with Down's syndrome (4/420 ± 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 forms1 but also in terms of the distribution of associated cardiac malformations.6 These anatomical differences and the absence of linkage in the molecular analysis of chromosome 21 in families with atrioventricular septal defect 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.

10 Melchiorri S, Digilio MC, Marino B, Giannotti A, Minguellari R, Dallapiccola B. Analysis of linkage with markers of the long arm of chromosome 1q and 8q in a family with atrioventricular canal defects. G Ital Cardiol 1993;23:7-
Balloon dilatation (valvoplasty) as first line treatment for severe stenosis of the aortic valve in early infancy: median term results and determinants of survival.

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