Imaging the dissected aorta

Sm,—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 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 tool for detection and assessment of type B dissections. It is currently more reliable than angiography. 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 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 passing, 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.

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

Tomography by imaging the dissected aorta. Br Heart J 1993;70:497–8.

Sm,—De Belder et al describes how a false lumen can be detected by the cross sectional image of CT and to exclude a traumatic aortic dissection. In a critically injured patient this CT scan may be negative but the trauma of echocardiography can be used at the bedside to make a diagnosis, provided a negative test is convincing and that there is no induced hypertension and interobserver variability.

De Belder et al go on to say that it does not look impossible to know whether the cross sectional image of CT cannot prove or exclude traumatic aortic dissection. In a critically injured patient this CT scan may be negative but the trauma of echocardiography can be used at the bedside to make a diagnosis, provided a negative test is convincing and that there is no induced hypertension and interobserver variability.

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(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.1

No classification is perfect but the virtue of the current version of the classification is that it defines a category (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 valvular 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 good 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. 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. There is 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 correct, is important. We have argued elsewhere4 that the cross sectional image of CT cannot prove or exclude traumatic aortic dissection. In a critically injured patient this makes CT an unnecessary waste of time. The nature of the transoesophageal echocardiography can be used at the bedside to make an attractive, provided a negative test is convincing and that there is no induced hypertension and local interference does not make the aorta go "pop".

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Anthracyclines and the heart

Sm,—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 denervation comes from the 125I-meta-iodobenzylguanidine (MIBG) rather than from the 123I-methoxy-isobutyl iodine (MI 1).2 These radiopharmaceuticals are quite dissimilar. MIBG detects uptake mechanisms into sympathetic nerve endings as noradrenaline. It is therefore ideally suited to imaging both the distribution of sympathetic nerve endings in the heart as well as neuroendocrine neoplasms such as pheochromocytomas.4 MIBG is a myocardial perfusion agent available in cold kit form that is labelled with technetium-99m rather than with iodine-123. MIBG is a lipophilic agent taken up into myocytes in proportion to 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.3

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We thank Dr Thomas for drawing attention to this error and for apologising for the ambiguities that were caused by introducing this mistake when the technical editor misread the abbreviation MIBG for MI 1. Authors can help to avoid such mistakes by following 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

Sm,—In their otherwise excellent article Bu'Lock and Treasure5 discuss many 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 unicusp variant of aortic valvar 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 cadaver 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 RP. 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.

Familial atrioventricular septal defect: possible genetic mechanism

In the patients we studied, the aortic valve was considered to be bicuspid in six, tricuspid in five, and one may have had a unicuspid valve. Because few of these patients had visual inspection of the aortic valve (either surgically or at necropsy) we are unable to comment on the validity of the echocardiographic findings. There did not seem to be any correlation between echocardiographically determined valve morphology and the biologic determinants of survival being left ventricular (rather than aortic valve) dimensions and presence and severity of associated lesions. The patient with the apparently unicuspid valve had an excellent response to balloon valvoplasty.

It does, however, seem logical to believe that aortic valve morphology may have an important influence on the degree of relief of valve obstruction obtained, on the propensity for early re-stenosis, and on the maintenance of longer term valve competence. Studies of the relation between valve morphology and the outline of balloon dilatation might be of considerable interest. Precordial echocardiography may not be adequate for such cases, where transossephagal echocardiography or intravascular ultrasound may be of additional value.


Pedigree