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UI 95033723
Author Anderson RH
Journal **British Heart Journal**
Title Balloon dilatation (valvoplasty) as first line treatment for severe stenosis of the aortic valve in early infancy: median term results and determinants of survival [letter; comment].
Volume 72
Issue 3
Page 300-1
ISSN 0007-0769
Year 1994 Sep
Local Msg Owned by MU HSL

Fullname Troy Norred MD
Campus Address
Local Address
Phone 882-7272
Fax
Email norred99@aol.com
Comments
Dept/Major Cardiology
Status Faculty
ID 440807683

Department Cardiology
Account Number c-3-40120-11
Delivery Mail

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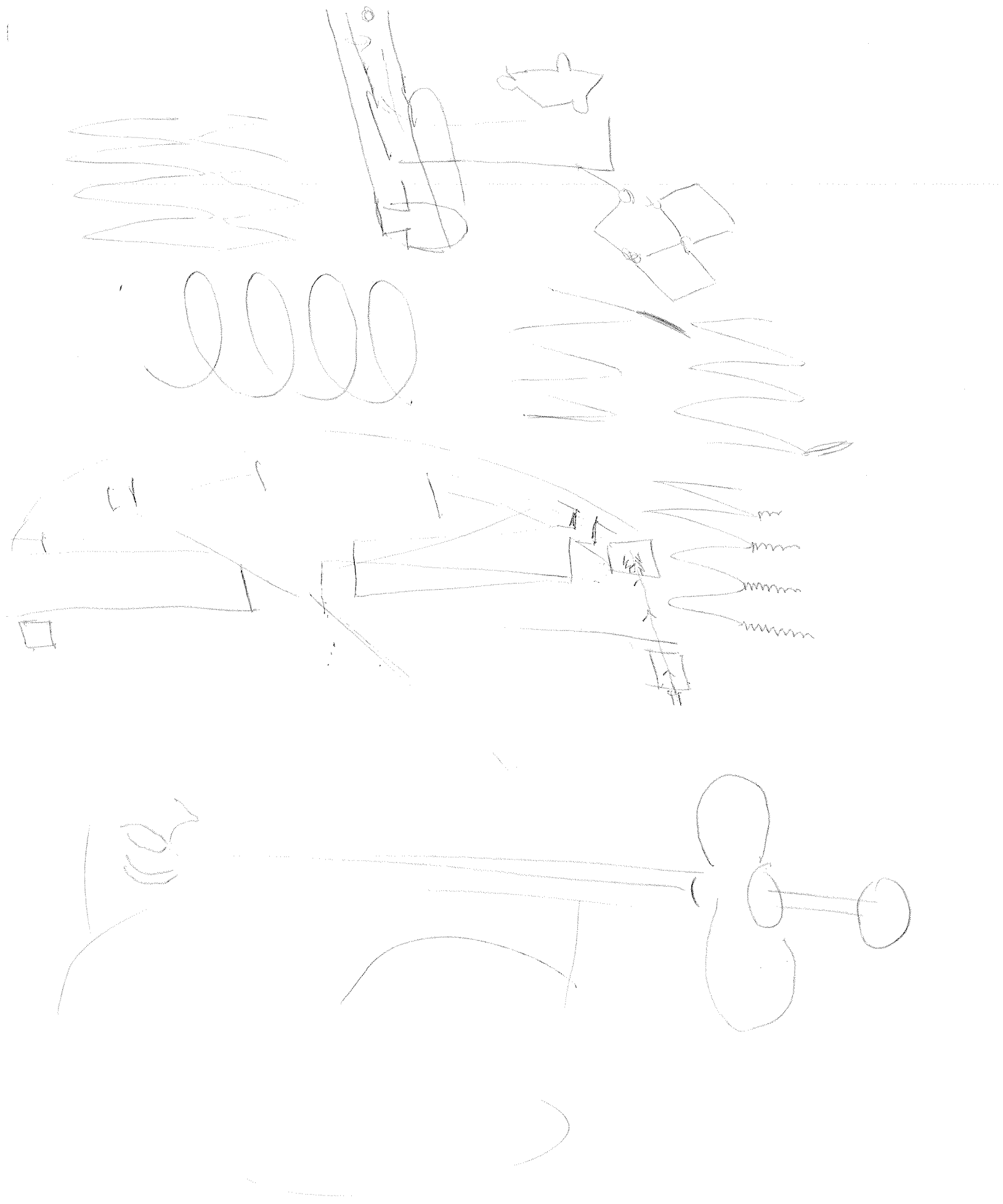
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Imaging the dissected aorta

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 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 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.

JB PARTRIDGE
Harefield Hospital,
Harefield,
Middlesex UB9 6JH

1 Treasure T. Imaging the dissected aorta. *Br Heart J* 1993;70:497-8.

2 de Belder A, Thomas M, Marrinan M. Traumatic rupture of the thoracic aorta diagnosed by transoesophageal echocardiography. *Br Heart J* 1993;70:393-4.

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 based¹ 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.² 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.^{1,2}

No classification is perfect but the virtue of the current version of the classification is that it defines a 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

associated with any residual uncorrected abnormality in the arch and descending aorta. Classification is an 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. (c, d, and e) 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 tear.³ 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 absent, is required. 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 fact that transoesophageal echocardiography can be used at the bedside makes it attractive, provided a negative test is convincing and that any induced hypertension and local interference do not make the aorta go "pop".

TOM TREASURE
Cardiac Department,
St George's Hospital,
Blackshaw Road,
London SW17 0QT

1 Daily PO, Trueblood HW, Stinson EB, Wuerflein RD, Shumway NE. Management of acute aortic dissections. *Ann Thorac Surg* 1970;10:237-47.

2 Miller DC, Stinson EB, Oyer PE, Rossiter SJ, Reitz BA, Griep RB, Shumway NE. Operative treatment of aortic dissections. *J Thorac Cardiovasc Surg* 1979;78:365-82.

3 de Belder A, Thomas M, Marrinan M. Traumatic rupture of the thoracic aorta diagnosed by transoesophageal echocardiography. *Br Heart J* 1993;70:393-4.

4 Unsworth-White MJ, Buckenham T, Treasure T. Traumatic rupture of the thoracic aorta—computed tomography may be a dangerous waste of time. *Ann R Coll Surg Engl* (in press).

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 ¹²³I-meta-iodobenzyl-guanidine (MIBG) rather than from ¹²³I-methoxy-isobutyl isonitrile (MIBI).² These radiopharmaceuticals are quite dissimilar. MIBG shares similar 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 tumours such as pheochro-

the sympathetic nerve endings but roughly proportionally to myocardial blood flow. MIBI is therefore used to assess the patency of coronary arteries rather than the status of the sympathetic nervous system.³

PAUL THOMAS
Department of Nuclear Medicine,
John Hunter Hospital,
Locked Bag 1,
Newcastle Mail Centre,
NSW 2310,
Australia

We thank Dr Thomas for drawing attention to this error and we apologise to the authors of the review for introducing this mistake when the technical editor mistook the abbreviation MIBG for MIBI. Authors can help us to avoid such mistakes by spelling out all abbreviations and acronyms at the first mention—EDITOR

1 Rhoden W, Hasleton P, Brooks N. Anthracyclines and the heart. *Br Heart J* 1993;70:499-502.

2 Valdes-Olmos RA, Ten Bokkel-Huinuk WW, Greve JC, Hoefnagel CA. ¹²³I-MIBG and serial radionuclide angiography in doxorubicin-related cardiotoxicity. *Clin Nucl Med* 1992;17:163-7.

3 Dae M. Scintigraphic assessment of cardiac innervation using iodine-123 metaiodobenzylguanidine. In: Ernst E van der Wall, Sochor H, Righetti A, Niemyer MG, eds. What's new in cardiac imaging? Dordrecht: Kluwer Academic Publishers, 1992;377-85.

4 Gross MD, Shapiro B, Thrall JH. Adrenal scintigraphy. In: Gottschalk A, Hoffer PB, Potchen EJ, eds. Diagnostic nuclear medicine, Baltimore William and Wilkins, 1988;826-7.

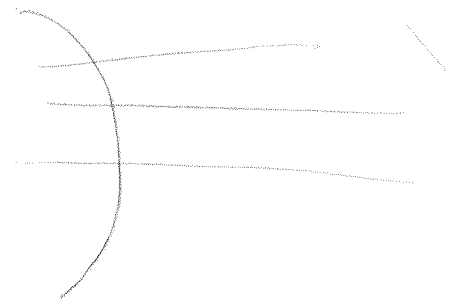
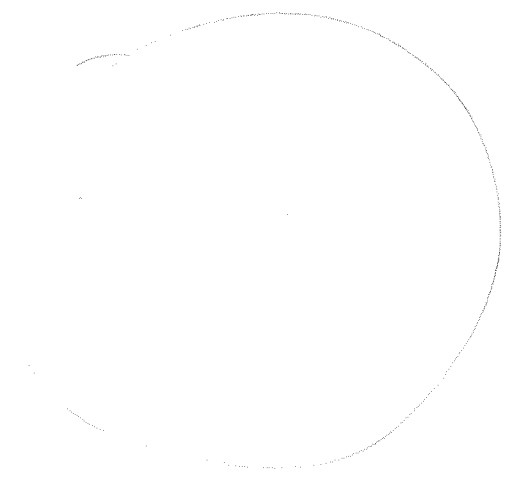
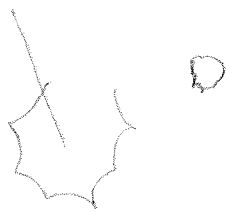
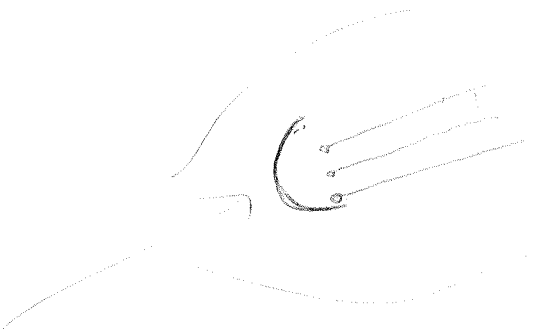
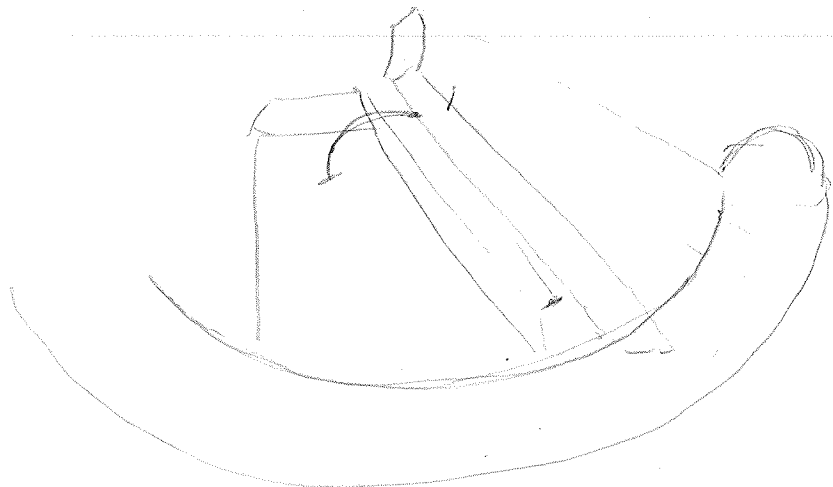
5 Rigo P, Larock M, Braat SH. Myocardial perfusion imaging with technetium-99m isonitriles: Attractive thallium substitutes? In: Ernst E van der Wall, *et al* eds. What's new in cardiac imaging? Dordrecht: Kluwer Academic Publishers, 1992;69-85.

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 and her colleagues¹ do not give any 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 unicuspid 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 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?

ROBERT H ANDERSON
Department of Paediatrics,
Royal Brompton National Heart and
Lung Institute, Dovehouse Street,
London SW3 6LY

1 Bu'Lock FA, Joffe HS, Jordan SC, Martin RP. Balloon dilatation (valvoplasty) as first line treatment for severe stenosis of the aortic valve in early infancy: median term results



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