

ing lipid lowering treatment. We fully agree that it is better to measure lipid concentrations before thrombolytic treatment. If such early lipid estimations are not available, values obtained within 24 hours after admission or 4 weeks later are a sufficiently valid basis for reaching a decision about early lipid intervention.

ROLAND CARLSSON
Section of Cardiology,
Department of Medicine,
Central Hospital,
S-65185 Karlstad,
Sweden

Aortic distensibility measured by magnetic resonance imaging in patients with Marfan's syndrome

SIR,—Adams and colleagues¹ reported aortic distensibility measurements based on assessments by eye of magnetic resonance (MR) and echocardiographic images in 12 patients with Marfan's syndrome. They suggest that the measurements are reproducible. Without re-scanning their patients on a separate occasion and repeating their analyses we do not see how they can conclude this. They do not specify the oscillation frequency of the ultrasound probe used for echocardiography, but at 2.5–3.5 MHz, which is typical for such work, the maximum spatial resolution would be of the same order of magnitude as the change in aortic diameter being measured. Furthermore, the errors involved in the off-line measurement of dimensions from a scan are much less than those involved in actually performing the scan.² Therefore data on the repeatability of the analysis procedure—or mean \pm SD values from repeat scans at the same visit—tell us very little, if anything, about the true reproducibility of the measurement techniques.

In any discussion of the validity of direct measurements of distensibility at a particular aortic cross-section it is important to consider the role of blood pressure. Adams *et al* measured the change in diameter (or area) at discrete cross-sections of the ascending and descending thoracic aorta but then applied blood pressure values recorded from the brachial artery to calculate aortic distensibility. Blood pressure varies along the arterial tree and amplification of the pressure pulse between central and peripheral arteries makes brachial pressure values an inaccurate measure of central aortic pressure.³ There can be absolute differences in systolic and pulse pressures of up to 20 mm Hg.³ This problem can be partly overcome by indirectly determining an average elastic property of the aortic wall based on pulse wave velocity (PWV) measurements.⁴ Such an approach does not require the blood pressure at a particular aortic cross-section to be known.⁵ This is especially pertinent because aortic distensibility indices determined from PWV measurements have good reproducibility (coefficient of variation < 10%).⁴ A transfer function can also be used to calculate central aortic systolic and pulse pressures based on non-invasively determined data on pressure waveforms in peripheral arteries.³

Papers that describe the application of MRI or echocardiography for directly measuring the change in aortic diameter (or area) between diastole and systole by eye are increasing. But not many papers report the reproducibility of such methods for non-invasively assessing aortic distensibility. Kupari *et al* reported a mean (SD) reproducibility for their MR measurements in the

ascending and descending aorta of 26.3 (23.3)% and 34.5 (32.1)%, respectively.⁶ Isnard *et al* using echocardiography reported reproducibility values of up to 23% in the ascending aorta.⁷ Dart *et al* also used echocardiography and, taking special care to obtain images exactly perpendicular to the aortic arch (to avoid measuring the change in diameter of an ellipse), obtained a mean reproducibility (SEM) in young, healthy, athletic men of 9.4 (2.9)%.⁸ Unfortunately, these latter data tell us very little, if anything, about the reproducibility of the technique in older patients with stiffer arteries in whom the change in aortic diameter during the cardiac cycle would be much reduced (as would the measurement reproducibility). In view of these data we do not find it surprising that Adams *et al* found that individual values for aortic distensibility varied by up to a factor of five depending on whether MR or echocardiographic data were used.

E D LEHMANN
Academic Department of Radiology,
St Bartholomew's Hospital,
London EC1A 7BE
K D HOPKINS
Academic Department of Medicine,
Whittington Hospital, London
R G GOSLING
School of Applied Sciences,
University of the South Bank, London

- 1 Adams JN, Brooks M, Redpath TW, Smith FW, Dean J, Gray J, *et al*. Aortic distensibility and stiffness index measured by magnetic resonance imaging in patients with Marfan's syndrome. *Br Heart J* 1995;73:265–9.
- 2 Lehmann ED, Hopkins KD, Gosling RG. Direct aortic distensibility measurements and their reproducibility. *Eur Heart J* 1994;15:1141–2.
- 3 Karamanoglu M, O'Rourke MF, Avolio AP, Kelly RP. An analysis of the relationship between central aortic and peripheral upper limb pressure waves in man. *Eur Heart J* 1993;14:160–7.
- 4 Hopkins KD, Lehmann ED, Gosling RG. Aortic compliance measurements: a non-invasive indicator of atherosclerosis? *Lancet* 1994;343:1447.
- 5 Lehmann ED. Biophysical properties of the aorta. *Lancet* 1994;344:1763.
- 6 Kupari M, Hekali P, Keto P, Poutanen V-P, Tikkanen MJ, Standertskjöld-Nordenstam C-G. Relation of aortic stiffness to factors modifying the risk of atherosclerosis in healthy people. *Arterioscler Thromb* 1994;14:386–94.
- 7 Isnard RN, Pannier BM, Laurent S, London GM, Diebold B, Safar ME. Pulsatile diameter and elastic modulus of the aortic arch in essential hypertension: a noninvasive study. *JACC* 1989;13:399–405.
- 8 Dart A, Silagy C, Dewar E, Jennings G, McNeil J, Cameron J. Direct aortic distensibility measurements and their reproducibility. *Eur Heart J* 1994;15:1142–3.

This letter was shown to the authors, two of whom reply as follows:

SIR,—Magnetic resonance imaging is an established non-invasive method of assessing anatomical structure and dimensions. We have shown in our study that aortic images obtained by this technique can be produced quickly and simply in patients with Marfan's syndrome and in healthy controls. Measurements made on these images can be reproducibly assessed by independent observers. We did not perform further scans on a second occasion and therefore cannot comment further on the reproducibility of this technique.

We agree that brachial pressure is lower than that measured directly. However, as Stefanadis *et al* have shown, the underestimation is similar in patients with coronary disease and healthy men and the pulse pressure measured indirectly by sphygmomanome-

ter correlates well with the pressure measured directly by catheterisation of the ascending aorta. Aortic distensibility determined non-invasively is closely related to that obtained using direct measurements.¹ We chose to use brachial pressure rather than pulse wave velocity because brachial pressure is more widely available and easier to use.

JACQUELINE N ADAMS
Department of Cardiology
Glasgow Royal Infirmary,
Queen Elizabeth Building,
Alexandra Parade, Glasgow G3 7ER
STEVEN WALTON
Department of Cardiology
Aberdeen Royal Infirmary,
Forresterhill, Aberdeen

- 1 Stefanadis C, Stratos C, Boudoulas H, Kourouklis C, Toutouzas P. Distensibility of the ascending aorta: comparison of invasive and non-invasive techniques in healthy men and in men with coronary artery disease. *Eur Heart J* 1990;11:990–6.

Is aspirin safe for patients with heart failure?

SIR—The prophylactic benefit of aspirin may have been overstated not only in coronary heart disease, as Cleland *et al* suggest,¹ but also in thromboembolism related to non-valvular atrial fibrillation (NVAF). In NVAF, this overstatement may be the result of failure to recognise that warfarin cannot prevent all thromboembolic events in all patients all the time,² and that aspirin may sometimes be perceived to have a prophylactic benefit because some NVAF patients have non-cardiac mechanisms of thromboembolism that are more amenable to risk modification by aspirin than by warfarin.^{3,4}

Furthermore, the risk/benefit profile of antithrombotic treatment might be more favourably disposed towards aspirin than towards warfarin in high-intensity⁵ than in low-intensity⁶ anticoagulant regimens. For thromboembolic prophylaxis, the principal disadvantage of aspirin is the unpredictability of its dose dependence with time⁷: this confounds comparisons between antithrombotic drugs and between subgroups receiving the same drug.

The seemingly intractable dilemmas of antithrombotic therapy can only be resolved by a complex trial simultaneously posing the questions "which patient?", "which drug?", "what dose?", and "what duration?"

O M P JOLOBE
Department of Medicine for the Elderly,
Tameside General Hospital,
Fountain Street,
Ashton under Lyne OL6 9RW

- 1 Cleland JGF, Bulpitt CJ, Falk RH, Findlay IN, Oakley CM, Murray G, *et al*. Is aspirin safe for patients with heart failure? *Br Heart J* 1995;74:215–9.
- 2 Archer SL, James KE, Kvernem LR, *et al*. Role of transoesophageal echocardiography in the detection of left atrial thrombus in patients with chronic nonrheumatic atrial fibrillation. *Am Heart J* 1995;130:287–95.
- 3 Kanter MC, Tegler CH, Pearce LA, *et al*. Carotid stenosis in patients with atrial fibrillation. *Arch Intern Med* 1994;154:1372–7.
- 4 Cerebral Embolism Task Force. Cardiogenic brain embolism. *Arch Neurol* 1986;43:71–84.
- 5 Stroke Prevention in Atrial Fibrillation Investigators. Warfarin versus aspirin for prevention of thromboembolism in atrial fibrillation: stroke prevention in atrial fibrillation II Study. *Lancet* 1994;343:687–91.
- 6 Ezekowitz MD, Bridgers SL, James KE, *et al*. Warfarin in the prevention of stroke associated with non-rheumatic atrial fibrillation. *N Engl J Med* 1992;327:1406–12.
- 7 Helgason CM, Bolin KM, Hoff JA, *et al*. Development of aspirin resistance in persons with previous ischemic stroke. *Stroke* 1994;25:2331–6.