that doubt the effectiveness of non-invasive techniques forget that it is their infalibility that limits the value of myocardial perfusion imaging and not the technique itself. Instead of ignoring it they should follow the practice of their colleagues who are able to manage patients without subjective measurements of myocardial perfusion rather than subjective impressions of the severity of disease made from the coronary arteriogram. They might then find that they can make many more therapeu- tic decisions without the need to refer for their cathers, and we shall be spared the debate about the role of cardiac catheterisation in the district general hospital.

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Acromegalic heart disease

Sir,—We disagree with several of the statements in the article by Rodrigues et al entitled “Subclinical cardiac dysfunction in acromegaly: evidence for a specific disease of heart muscle” (1989;62:185-94).

In the summary Rodrigues et al say that “this is the first study to find evidence of subclinical cardiac dysfunction in acromegaly”, and in the discussion they say that “Left ventricular diastolic function has not previously been studied in acromegaly”. I would like to draw attention to the paper of Bertoni and Morandi entitled “Impaired left ventricular diastolic function in acromegaly: an echocardiographic study”, which relaxa- tion times of the left ventricle were shown in acromegalic patients in 1987.1

On the basis of the paper by Lie and Grossman, Rodrigues et al suggest that the fibrosis which is partly responsible for the diastolic filling disturbance is a consequence of an inflammatory process. Van den Heuvel et al took biopsy specimens of the right ventricle of an acromegalic patient and later examined abnormalities of the left ventricle were shown in acromegalic patients in 1987.2 On the basis of the paper by Lie and Grossman, Rodrigues et al suggest that the fibrosis which is partly responsible for the diastolic filling disturbance is a consequence of an inflammatory process. Van den Heuvel et al took biopsy specimens of the right ventricle of an acromegalic patient and later examined abnormalities of the left ventricle were shown in acromegalic patients in 1987.3

These results prove that the main patho- logical background for the acromegalic heart disease is left ventricular hypertrophy, with some contribution from myocardial fibrosis and the increased collagen content of the myocardium. We found the dilated form of the disease in a few of our patients not to be associated with inflammatory process; these patients were burned-out cases with lower serum concentra- tions of growth hormone than those with hyperthropy.4

Rodrigues et al cited the second edition of Feigenbaum’s Echocardiography (not pub- lished in 1979 but in 1976) as saying that echocardiography is an insensitive method of assessing left ventricular function. However, this is not the opinion given in the fourth edition published in 1986.5 Radionuclide ventriculography is probably a better method of assessing the ejection fraction than echocardiography. But because there are several other indices of left ventricular func- tion (for example, enlargement of the left ventricle and segmental wall movement abnormalities) echocardiography cannot be deemed to be a good method of evaluating left ventricular function.1

In Rodrigues et al’s paper apart from 13 self-citations, there are only three references from 1982-84 and one of these was an abstract, dated 1986. The references 1 cite were published between 1983 and 1987.

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This letter was shown to the authors, who reply as follows:

Sir,—In reply to Csanady’s letter we would like to make the following comments. Bertoni and Morandi published a paper on left ven- tricular diastolic function in 1987, but our study was largely completed in 1986 and was presented in part to the British Cardiac Society in December 1986.1

Csanady seems to regard the seminal work on pathological findings of Lie and Grossman in favour of the necropsy findings in a patient described by van den Heuvel et al. Necropsy findings in only one patient could not dispel the finding of a left ventricular hypertrophy. Furthermore, in our study we tried to assess the influence of myocardial hypertrophy by studying left ventricular mass derived echocardiographically and planned that the echocardiograms were obtained from a group of patients who had no correlation between peak filling rate abnormalities and left ventricular wall thick- ness or left ventricular mass. Therefore, it is unlikely that the abnormal peak filling rate seen in our patients was merely a reflection of hypertrophy.

Though we agree that echocardiography can be used not only to measure ejection fraction but also to assess the anatomical consequences of left ventricular dysfunction, such as left ventricular enlargement and wall-motion abnormalities, the point we made in our discussion was that subtle changes in diastolic relaxation were shown better by radionuclide ventriculography. Indeed, Feigenbaum pointed out the drawbacks and pitfalls of calculating ejection fraction by cross sectional echocardiography and did not go into any detail about the assessment of such subtle diastolic abnormalities.2

We referred to our previous validation studies in the text to establish the quantitative success and reproducible nature of the radionuclide technique for assessing subdi stolic left ventricular dysfunction. Many methods have been used to study a large group of patients with a rare condition. Previous studies did not deal with the same group of patients (Feigenbaum’s echocardiography used to exclude obstructive coronary artery disease.5 Thus our suggestion that radionuclide ventriculography is better than echocardiography for picking up subtle left ventricular diastolic dysfunction is justified.

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Severe haemolytic anaemia after replacement of the mitral valve by a St Jude medical prosthesis

Sir,—The interesting case report by Feld and Roth (1989;62:473-6) of severe haemolytic anaemia after mitral valve replacement with a St Jude medical prosthesis highlights the difficulty in detecting mitral parapros- thetic leaks. Despite clinical evidence of mitral regurgitation in their patient, careful echocardiography with Doppler studies failed to detect any prosthetic abnormality and left heart catheterisation was required to show severe paraprosthetic regurgitation.

We have also seen a patient with echocardiography with a 5 MHz phased array trans- oesophageal transducer (HP 21362A) was helpful in nine patients (mean age 59 years) with mitral prosthetic regurgitation. One of them also had severe haemolytic anaemia after the insertion of a 27 mm St Jude mitral prosthesis. Mitral regurgitation was detected by transoesophageal echocardiography, includ-
RITA trial protocol.

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