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This letter was shown to Dr Huikuri, who replies as follows:

Sir,

We are grateful to Dr Candell-Riera and his colleagues for commenting on their experience of thallium imaging in aortic valve disease. We are somewhat surprised by their reports of low specificity.

We do not know the details of how the images were assessed in their study. It is well known that the interpretation of thallium images is subjective and it is often difficult to define abnormal thallium perfusion. Misinterpretation of irregular thallium uptake in the apical region, which was commonly seen in our study (especially in severe aortic regurgitation) and has also been described by Pfisterer *et al*,¹ is a possible source of false positives. These defects are not strictly limited to the apex, but extend partly to the inferior region. These inferoapical defects should not be regarded as representing a coronary artery disease. The peak heart rate achieved during exercise was higher in patients studied by Candell-Riera *et al*. It is possible that subendocardial ischaemia occurring during maximal exercise in critical aortic valve disease can reduce the thallium uptake of the myocardial cells and result in thallium defects despite normal coronary arteries. It has been our policy not to continue exercise until

severe symptoms develop because of the possible hazards of maximal exercise testing in patients with severe aortic stenosis. This factor may explain the lower number of false positive scans in our study.

We have continued our thallium studies in patients with aortic stenosis by using dipyridamole infusion as a stress test.² In this pilot study we found thallium imaging to be 86% sensitive and 87% specific in detecting angiographically significant coronary artery disease. Because of these results we now prefer dipyridamole stress testing to dynamic exercise testing in patients with critical aortic stenosis. These patients often have a poor exercise tolerance which limits the usefulness of exercise testing in conjunction with thallium imaging. Coronary vasodilation with dipyridamole does not result in myocardial ischaemia in a hypertrophied left ventricle without coronary artery disease and may also improve the specificity. We certainly agree with Candell-Riera *et al* that none of the non-invasive methods can yet replace coronary angiography in patients with valvar disease and suspected concomitant coronary artery disease. More studies are needed.

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The cause of rheumatic heart disease

Sir,

Dr William L Proudfit's recent review entitled "John Hunter: on heart disease" is a most interesting and valuable contribution. In it he mentions the question of whether Edward Jenner or Dr David Pitcairn should rightfully be credited with having initially associated valvar heart disease with preceding rheumatic fever. He suggests that Pitcairn's claim rests upon a footnote in the second edition of

Dr Matthew Baillie's textbook published in 1797, whereas Jenner was known to have spoken on the subject to the Gloucestershire Medical Society in 1789.²

There is, however, stronger evidence than this for Pitcairn's claim. On 3 April 1810 William Charles Wells read a paper entitled "On rheumatism of the heart" to the Society for the Improvement of medical and surgical Knowledge.³ Wells was a