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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

observer of irrefutable precision and independence and the secretary to that quite remarkable little society. Hunter and Baillie were also both members and regular contributors. Wells knew of Jenner because Hunter had previously read to the society an interesting letter which Hunter had received from Jenner.⁴

In the introduction to his paper in 1810 Wells detailed the development of knowledge about rheumatic heart disease. There seems little reason to challenge his account. He pointed out that Morgagni and a Dr Ferriar of Manchester had both previously reported the concurrence of rheumatism and organic disease of the heart, but had considered that the association of the two diseases was merely accidental. Then "Dr David Pitcairn, about the year 1788, began to remark that persons subject to rheumatism were attacked more frequently than others with symptoms of an organic disease of the heart. Subsequent experience having confirmed the truth of this observation he concluded that these two diseases often depend on a common cause, and in such instances therefore called the disease rheumatism of the heart. He communicated what he had observed to several of his friends and to his pupils at St Bartholomew's Hospital, to which he was then physician; but no notice, I believe, was taken of his remark in any book before it appeared in the second edition of Dr Baillie's *Morbid Anatomy*, which was published in 1797."

Wells pointed out that a Dr Odier of Geneva had subsequently mentioned the association without attribution in a book entitled *Manuel de Médecine Pratique*, published in 1803, as had Mr David Dundas, a surgeon, in a paper published in the *Transactions of the Medical and Surgical Society of London*. Since Pitcairn had indicated that he had no desire himself to write up his cases, Wells had hoped to include the details of them with those of some cases of his own, however, "I neglected to obtain this while the opportunity existed, and I now lament my indiscretion." As a result the series of 14 cases which Wells presented in 1810 and published in 1812 were all his own. The detail of his descriptions was such that there can now be no doubt that he was dealing with what has subsequently been called rheumatic heart disease. Wells must surely, therefore, continue to be regarded as the man who wrote the original definitive description, although, as he himself was most careful to point out, credit for the conceptual advance belonged entirely to Pitcairn.

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

Sir,
Credit for priority in science is usually based on the date of original publication or documented presentation at a meeting, although numerous abuses of this rule could be cited. I believe that this view is relevant to the association of rheumatic fever and valvar disease. No claim was made that Jenner was the first to make this association. Many books on "acute rheumatism" were published in the last third of the eighteenth century, especially MD theses in Edinburgh, and as I have not read these it is quite possible that the association was made before 1788.

Doctor David Pitcairn, a distinguished and beloved physician in London, was neither a medical writer nor a morbid anatomist. Credit for his relating rheumatism to heart disease rests with his friends Baillie and Wells. In the absence of knowledge of his definitions of "rheumatism" and cardiac abnormalities (before the days of auscultation), it is difficult to be certain what correlation he was making. In July 1789 Jenner described a young woman with a history of "acute rheumatism" and he found heart disease at postmortem examination.¹ Daniel Ludlow Jr reported a similar case to the same society a year later, also with necropsy findings. Jenner did not forget his aetiological hypothesis and in January 1805 he wrote to Caleb H Parry requesting return of his manuscript because a neighbour had died of heart disease "... which followed two or three severe attacks of acute rheumatism."²

This does not mean that Pitcairn had not arrived at a similar conclusion independently, but such a claim has not been documented. Fortunately, the fame of neither Jenner nor Pitcairn rests on conflicting claims. As in the case of many scientific discoveries, the observations may have been simul-