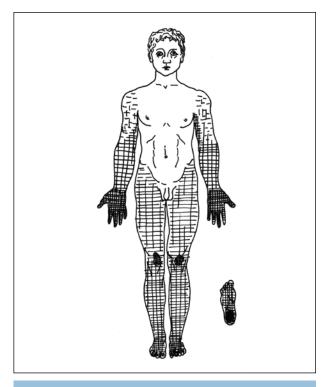
#### **NERVE DAMAGE IN LEPROSY**

Breen et al, described the features of a multiple mononeuropathy in a leprosy patient. In 1923, Monrad-Krohn, a Norwegian neurologist, described the features of a purely sensory polyneuritis, involving superficial sensory modalities in 63 Norwegian patients. (Figure 1) This sensory polyneuritis was independently verified in a group of patients in northern Nigeria, as references to Monrad-Krohn's findings were not published in the leprosy literature.



#### FIGURE I

There are, therefore, two types of nerve damage in leprosy. The sensory polyneuritis is responsible for mutilations of the extremities, trophic ulcers and Charcot's joints, which is the main reason why leprosy is a serious disorder.

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# STIGMATA OF INFECTIVE ENDOCARDITIS SHOULD BE INCLUDED IN THE COMPILATION OF THE RISK SCORE

In view of evidence suggesting that infective endocarditis-related stroke is a risk factor for post-thrombolysis intracranial haemorrhage (ICH),<sup>1,2</sup> stigmata of infective endocarditis (IE) should have been included in the variables compiled by the authors<sup>3</sup> for predicting post-thrombolysis ICH.

In one study, among 1,801 patients with acute ischaemic stroke, IE was the underlying cause of pure ischaemic stroke in 11 patients. Stigmata of IE which could be identified within the therapeutic time window for thrombolysis included clinical signs classically associated with IE as well as laboratory derangements such as anaemia, leucocytosis, and elevated C reactive protein, but it was only in a minority of cases that either clinical or laboratory stigmata could be identified. Thrombolysis was administered to four of the five patients who presented within 6 hours of symptom onset, and this was followed by ICH in all four instances. Three of the four patients died, all within 2 weeks.

Equally unfavourable results were reported in a comparison between 222 IE patients (mean age 59 years) with ischaemic stroke vs 134,048 patients (mean age 69) with ischaemic stroke in the absence of IE. The rate of post-thrombolysis ICH was significantly (p = 0.006) higher in the former category of patients.<sup>2</sup> In the former group of patients, as well, there was a significantly (p = 0.01) lower rate of favourable clinical, outcome.2 However, given the fact that thrombolysis for IE-related ischaemic stroke remains controversial,4 triage of prospective candidates for thrombolytic treatment of ischaemic stroke should include documentation of stigmata of IE identifiable within the therapeutic time window for thrombolysis, with a view to including those stigmata in the predictive score for post thrombolytic ICH.

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## JOHN FEWSTER AND SMALLPOX VACCINATION

I wish to comment on the paper regarding John Fewster by Thurston and Williams.

The authors are to be congratulated on their diligent scholarship in unearthing several facts about John Player and his letter to John Coakley Lettsome. However, I believe that they have underestimated several facts that cast grave doubt on Player's letter.

First the date: In his letter John Fewster says that it was 1768.2 Thurston and Williams' claim that this is a misprint seems lame because the Fewster letter was published in widely read journals and formed part of Pearson's evidence to Parliament while both Fewster and Jenner were alive. Neither said that it was inaccurate. John Carrick Moore, a friend of Jenner and a member of his Vaccine Society, states in his History of Vaccination that Jenner told him he learned of cowpox effects in 1768.3 Again Jenner was alive and could have corrected his friend but did not. Finally Fewster says, and Thurston and Williams accept, that Fewster was in partnership with Daniel Sutton whose revolutionary practice of variolation did not spread outside East Anglia until 1767 at the earliest.4 A catalogue of his partners published in 1768 does not list Fewster and Grove suggesting that they were not associated with him before 1768.5

Player also says that Fewster performed vaccination before Jenner's first experiment on young Phipps. It seems unlikely that this would have escaped public notice given the furore that resulted from the discovery that Jesty had vaccinated his family in 1774. Thus I believe Player's letter cannot be considered a reliable account of any of the events surrounding Fewster and the discovery that cowpox prevented subsequent smallpox.

A detailed description of this topic and the entire history of variolation can be found in my book.<sup>2</sup>

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## Authors' reply

We would like to thank Professor Boylston for his comments on our paper on John Fewster's role in the discovery of smallpox vaccination.

In response to Boylston's question over the date of John Fewster's move to Thornbury, we cannot prove that Pearson (in his abstracted version of Fewster's letter to Rolph) was mistaken in citing this as 1768. However, Fewster's autobiography in the Bristol Infirmary Biographical Memoirs states that he moved to Thornbury 'soon after' the conclusion of the Seven Years War in 1763, which would be consistent with Player's account of the dates. Pearson's Inquiry, and the evidence denigrating Jenner which he presented to Parliament, were both published while Fewster was still alive. However, this information might not have reached Fewster in rural Gloucestershire and, given his evident indifference about vaccination, might not have excited his interest.2 It should be remembered that Pearson's Inquiry was published over 30 years after Fewster's move to Thornbury, which might leave further room for error. Similarly, John Carrick Moore wrote his account 50 years after his discussion with Jenner regarding the effects of cowpox and towards the end of Jenner's life. It is possible that Jenner never read this account.

We accept Boylston's point regarding the date that variolation spread outside East Anglia, and we are interested to explore this further, as this could clarify the date when the inoculation house in Buckover was opened.

Finally, we disagree with Boylston's supposition that Fewster's putative trial with vaccination would have provoked a response from the medical establishment. Boylston bases his conjecture on the local furore that followed Benjamin Jesty's vaccination of his family in 1774, and which forced the Jesty family to move away from the area. However, Jesty's experiment was not publicised beyond the immediate area until Pearson investigated the rumour for his Inquiry some 24 years later. We do not believe that Fewster, a successful and respected practising surgeon and apothecary, would have faced the same antagonism. Even if his experiment had caused discontent among the local inhabitants, we doubt that the news would have been transmitted further afield, or its significance appreciated, as cowpox was an unknown entity in medical circles until the publication of Jenner's Inquiry in 1798.

We would like to highlight our paper's conclusion that Player's account does indeed contain inconsistencies and that his wilder claims are unsupported by independent evidence. Nonetheless, we accept that Fewster did have a role to play in the discovery of vaccination.

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## **CORRECTION**

In the Acknowledgements section of the paper entitled 'An examination of John Fewster's role in the discovery of smallpox vaccination' by L Thurston and G Williams, issue 2, pp 179–9, the name Madeleine Wright should have been Madeleine Gill.

The authors apologise for this error.