Reply to Eisenhut

TO THE EDITOR—We thank Dr Eisenhut for his comments regarding possible myocarditis associated with human parechovirus-3 (HPeV-3) infection [1]. With respect to the cohort of 118 children we reported on from New South Wales, Australia [2], and further review of the medical records, 10 children had trans-thoracic echocardiograms that did not demonstrate evidence of myocarditis. All evaluations were performed in young infants aged 5–95 days at presentation. Eight children had serum troponin levels measured and 6 of these were >50 ng/L on at least 1 occasion (highest 189 ng/L). Four of the children with raised troponin levels also had echocardiograms. Two other children had normal serum creatinine kinase levels. At least 19 children had electrocardiograms (ECGs), the majority of which showed sinus tachycardia only. One ECG demonstrated supraventricular tachycardia in a child who also had an echocardiogram (only abnormality was a patent foramen ovale), both of which were normal on follow-up. Another ECG demonstrated occasional ventricular ectopics and sinus tachycardia. One child diagnosed with severe left ventricular dysfunction due to a congenital cardiac defect had an abnormal ECG, and serum troponin peaked at 7125 ng/L. She required
extracorporeal membrane oxygenation and a left ventricular assist device followed by surgery. The majority of children with abnormal liver function tests had an aspartate aminotransferase to alanine aminotransferase ratio of ≥2, which could be consistent with myocyte rather than hepatocyte origin of these enzymes to some extent [3].

Although 63% of children required fluid boluses, anecdotally there was no response to fluid administration. Only 2 children required inotropic support; 1 was for bradycardia associated with encephalopathy and a complete set of normal cardiac investigations. The level of tachycardia noted in the children was at times disproportionate to the level of fever, but the pattern appeared to be in keeping with the pattern of fever spikes and resolved with resolution of fever. Furthermore, it is possible that “pain” may have contributed to tachycardia in some children; 93% of children in this series required analgesia for what was perceived to be pain or distress by parents and pediatricians.

Some of the children with HPeV-3 infection in our series may have had some degree of transient myocarditis that contributed to tachycardia and elevated troponin levels. Although formal cardiac investigations were only performed on a small minority, these were the children of greatest clinical concern, and the results were reassuring. In addition, most of the children in our series have been followed up locally, and we are aware of none who have had persistent tachycardia or developed cardiomyopathy as a result of parechovirus infection. We feel that it is unlikely that any of the children in our series had myocarditis of long-term significance but agree with Dr Eisenhut that evidence for myocarditis should be sought in children presenting with tachycardia and signs of severe HPeV-3 infection.

**Notes**

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All authors have submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest.

Conflicts that the editors consider relevant to the content of the manuscript have been disclosed.

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**References**


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