LETTERS TO
THE EDITOR

Scope
Heart welcomes letters commenting on papers published in the journal in the previous six months. Topics not related to papers published earlier in the journal may be introduced as a letter: letters reporting original data may be sent for peer review.

Presentation
Letters should be:
- no more than 600 words and six references in length
- typed in double spacing (fax copies and +
- signed by all authors
They may contain short tables or a small figure. Please send a copy of your letter on disk. Full instructions to authors appear in the January 1998 issue of Heart (page 106).

Dietary precautions and listeria endocarditis?

Sir,—We read with interest the report of a further case of listeria endocarditis by Johnston and Troughton. The consumption of unpasteurised milk and cheese made from unpasteurised milk has often been implicated in listeriosis. We assume that Johnston and Troughton’s case was the consumption of unpasteurised milk. We agreed with the advice of dietary precautions currently given to pregnant women to take the same precautions as they do (page 106).

This letter is for the authors, who reply as follows:

We read with interest the report of a further case of listeria endocarditis by Johnston and Troughton. The consumption of unpasteurised milk and cheese made from unpasteurised milk has often been implicated in listeriosis. This has led to the advice of dietary precautions for pregnant women. In view of the gravity of listeria endocarditis, it is essential to consider giving the same recommendations to patients with damaged or prosthetic valves. However, the incidence is very low and therefore it is difficult to study the final common pathway in the pathogenesis of listeria monocytogenes endocarditis.

We assume that Johnston and Troughton’s patient lived for many years in a community where she would consume unpasteurised milk and, despite having a dysfunctional mitral valve that was replaced three times, she did not develop endocarditis until she was 76 years old. The difference for pregnant women is that they need to adhere to advice of unpasteurised products for only nine months, while patients with dysfunctional valves would be condemned to a lifetime of abstinence. We believe that patients should be advised that consuming unpasteurised milk may slightly increase the risk of developing listeria endocarditis.

Histological findings in non-hypertrophic cardiomyopathy associated with Noonan’s syndrome

Sir,—We have reported the clinical and echocardiographic findings in a family with Noonan’s syndrome and a non-hypertrophic cardiomyopathy with restrictive physiology.

The original proband has now had cardiac transplantation and we are able to report the histopathological findings. There was extensive, patchy fibrosis with many areas of myocyte disarray. There was also variability in the size of the myocytes with many hypertrophied cells. There were small foci of inflammatory cells, mainly monocytes, but with eosinophils in some areas. Perl’s stain for haemosiderin and Congo red stain for amyloid were normal.

In about 25% of cases, Noonan’s syndrome is associated with hypertrophic cardiomyopathy, which is characterised histologically by increased myocyte disarray. However, there are a few cases of cardiomyopathy where the histological findings of fibre hypertrophy without disarray have been more suggestive of primary restrictive myopathy. In our case the histological findings were unusual. There was myocyte disarray, but the degree of fibrosis was greater than usually seen in hypertrophic cardiomyopathy and there were intramural eosinophils raising the possibility of Loeffler’s cardiitis. At no time was the peripheral blood eosinophil count raised, there were no echocardiographic features of endocardial disease, and there was reduced left ventricular systolic function on echocardiography that was out of keeping with Loeffler’s endocarditis, but consistent with primary restrictive cardiomyopathy.

In the absence of histological changes of primary restrictive myopathy or of infiltrative disease, the likeliest diagnosis is hypertrophic cardiomyopathy without hypertrophy as described by McKenna et al. Our case is atypical in the echocardiographic findings as well as histology. Whether it is clinically useful to continue classifying such cases under the unitary diagnosis of a condition already known to have a wide phenotypic and genetic makeup remains to be seen.

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REFERENCES