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:

- not more than 600 words and six references in length
- typed in double spacing (fax copies and paper copy only)
- 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 article by Spyrou and colleagues¹ in which *Listeria monocytogenes* was presented as an uncommon pathogen in infective endocarditis (58 cases in the literature). We have identified three further case reports²⁻⁴ and describe a patient with this condition who was recently under our care.

A 76 year old woman was admitted to our institution with a three month history of extreme lethargy, anorexia, and increasing dyspnoea. One month before admission she had been treated for a urinary tract infection and was noted to have a florid rash over both legs. A third mitral valve replacement, using a Hancock bioprosthesis, had been done 18 months before this admission. She lived in a rural community and routinely drank unpasteurised milk.

On examination she had two vasculitic lesions on the lower limbs. There were signs of mitral stenosis and tricuspid regurgitation. She did not have a fever and there were no other signs of endocarditis. Laboratory investigations revealed microscopic haematuria, polymorph leucocytosis, raised C reactive protein, low complement concentrations, and three positive blood cultures for *L. monocytogenes*. Transoesophageal echocardiography showed a very thickened mitral prosthesis with severe mitral stenosis (0.7 cm²) and moderate tricuspid regurgitation. Vegetations were seen on the bioprosthesis.

The patient was treated with ampicillin and gentamicin. Her condition slowly deteriorated with progressive cardiac and renal failure and she died 21 days after admission. Postmortem examination revealed severe prosthetic mitral stenosis with nodular excrescences filling the valvar lumen. Histological examination showed these were com-

posed of fibrin and colonies of Gram positive bacilli. A jet lesion was also noted in the left ventricle.

This case is interesting in that the source of infection was probably unpasteurised milk. Thus, it may be appropriate to advise patients with valvar prostheses to take the same dietary precautions currently given to pregnant women to avoid this infection.⁵

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- 1 Spyrou N, Anderson M, Foale R. *Listeria* endocarditis: current management and patient outcome—world literature review. *Heart* 1997;77:380-3.
- 2 Cruz JM, Fernandez R, Martinez R, et al. Endocarditis por *Listeria monocytogenes* sobre valvula protesica. *Enferm Infecc Microbiol Clin* 1994;12:221-2.
- 3 Baddour LM. *Listeria* endocarditis following coronary artery bypass surgery. *Rev Infect Dis* 1989;11:669.
- 4 Castro Cabezas M, Cramer MJ, de Jongh BM, et al. *Listeria monocytogenes* endocarditis in a patient with an aortic prosthetic valve. *Neth J Med* 1996;48:15-17.
- 5 Department of Health. While you are pregnant: safe eating and how to avoid infection from food and animals. London: HMSO, 1991.

This letter was shown to the authors, who reply as follows:

We read with interest the report of a further case of listeria endocarditis by Johnston and Trouton. 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 reasonable 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 Trouton'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 avoidance 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.¹

The original proband has now had cardiac transplantation and we are able to report the histological 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,² which is characterised histologically by increased myocyte disarray.³ 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.⁴ 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 intramyocardial eosinophils raising the possibility of Loeffler's carditis. 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.*⁵ 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 makeup⁶ remains to be seen.

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- 1 Cooke, Chambers J, Curry P. Noonan's cardiomyopathy: a non-hypertrophic variant. *Br Heart J* 1994;71:561-5.
- 2 Phornphutkul C, Rosenthal A, Nadas AS. Cardiomyopathy in Noonan's syndrome. *Br Heart J* 1973;35:99-102.
- 3 Burch M, Mann JM, Sharland M, et al. Myocardial disarray in Noonan syndrome. *Br Heart J* 1992;68:5786-8.
- 4 Wilmshurst P, Da Costa P. Probable right ventricular dysplasia and patent foramen ovale presenting with cyanosis and clubbing in a patient with characteristics of Noonan syndrome. *Br Heart J* 1995;74:471-5.
- 5 McKenna WJ, Stewart JT, Nihoyannopoulos P, et al. Hypertrophic cardiomyopathy without hypertrophy: two families with myocardial disarray in the absence of increased myocardial mass. *Br Heart J* 1990;63:287-90.
- 6 Watkins H. Multiple disease genes cause hypertrophic cardiomyopathy. *Br Heart J* 1994;72(suppl):S4-9.