The British Heart Journal welcomes letters commenting on papers that it has published within the past six months.

All letters must be typed with double spacing and signed by all authors.

No letter should be more than 600 words.

In general, no letter should contain more than six references (also typed with double spacing).

Mitrail valve hypoplasia in children with isolated coarctation of the aorta

Sir,—Venugopal et al concluded that, compared with controls, patients with coarctation of the aorta have relative hypoplasia of the mitral valve which is likely to be more pronounced in patients with mitral diastolic murmurs.1 Unfortunately, they do not mention our previous study in which we found that patients with mitral diastolic rumble and coarctation of the aorta have minor abnormalities of the mitral valve or minimal mitral stenosis that significantly increase the rapid ventricular filling and pressure half times and decrease the mitral valve area.

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Peripheral vascular disease: consequence for survival and association with risk factors in the Speedwell prospective heart disease study

Sir,—We were interested in the findings of Bainton et al that a raised white cell count predicts the development of intermittent claudication.1 An epidemiological study also found a correlation between a raised white cell count and a significant risk of myocardial infarction and stroke.2 It is generally accepted that massive tissue ischaemia followed by reperfusion has an adverse effect on the systemic vascular endothelium, particularly the pulmonary microcirculation.3 Bainton suggests an important role for oxygen-derived free radicals, activated neutrophils, and endothelial mediators in this injury, resulting in a systemic increase in vascular permeability. This may be quantified by a local increase in renal permeability, which is reflected by a change in urinary protein excretion—microalbuminuria.4 We have suggested that patients with claudication undergo a series of similar less severe ischaemia-reperfusion injuries with activation of the above mechanisms. This may have an adverse effect on cardiovascular morbidity and mortality in these patients. In support of this hypothesis we found an increase in neutrophil activation, lipid peroxidation, and a rise in urinary albumin excretion after exercise in patients with claudication.5 Recently we found a decrease in neutrophil deformability, suggestive of activation, and a highly significant rise (P < 0.001) in thrombomodulin B, concentrations after exercise in patients with claudication.6 These events seem to play a part in atherogenesis, and we suggest that this may contribute to the excess cardiovascular mortality found in these patients.

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