Suppressor T lymphocyte function in patients with idiopathic congestive cardiomyopathy

Sir,

Dr Lowry and her colleagues reported (1987;57:458–61) that they were unable to detect a defect in T lymphocyte function in their patients with dilated cardiomyopathy. At first sight this might appear to contradict our results from patients with dilated cardiomyopathy studied in Kenya where we found that about half the patients had a high helper/suppressor (OKT4/OKT8) T lymphocyte subset ratio.¹ This apparent difference may be explicable, however. All our patients with a high helper/suppressor ratio were seen within three months of the onset of their symptoms (Lowry *et al* do not state the duration of illness in their patients) and we felt that the higher ratios merely represented an active immunological reaction as part of a chronic or subacute myocarditis.

As the myocarditis “burns out”, the helper/suppressor ratio will return to normal. At this later stage there may be no immunological changes to detect. Furthermore, the high helper/suppressor ratios in our patients were caused by an increased number of helper cells rather than a significant reduction of suppressor cells, so that the changes are not likely to be non-specific and due to heart failure alone.

I would agree with Lowry *et al* that there is unlikely to be a permanent defect in T lymphocyte function that is specific to these patients but this does not rule out the possibility that an immunological reaction (which may be excessive) is part of the early pathogenesis of dilated cardiomyopathy. And this may be detected only if the patients are studied early after the onset of their illness.

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Reference

- 1 Sanderson JE, Koech D, Iha D, Ojiambo HP. T-lymphocyte subsets in idiopathic dilated cardiomyopathy. *Am J Cardiol* 1985;55:755–8.

Sir,

Lowry *et al* suggested that the defect in T lymphocyte function that they found in patients with idiopathic congestive cardiomyopathy may be a non-specific reduction that is associated with heart failure rather than with the cardiomyopathy itself. We too reached a similar conclusion in a recent paper¹: we found that the ratio of helper (T4) to suppressor (T8) lymphocytes (labelled by monoclonal antibodies) was higher because of a decrease in the percentage of T8 lymphocytes in patients with idiopathic congestive cardiomyopathy and ischaemic cardiomyopathy. The fall in T8 lymphocytes was more pronounced (and statistically significant) in patients with idiopathic cardiomyopathy than in patients with ischaemic cardiomyopathy with heart failure.

We also found that the ratio of helper T lymphocytes to responder T lymphocytes determined by *in vitro* culture was different from the ratio *in vivo*.

We believe that heart failure may impair T lymphocyte function, but this does not exclude the possibility of immunological dysfunction in idiopathic congestive cardiomyopathy.

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Reference

- 1 Sachs RN, Hatron PY, Saizy C, Lanfranchi J. Etude des sous populations lymphocytaires T par les anticorps monoclonaux au cours des cardiomyopathies dilatées primitives et alcooliques. *Ann Med Interne (Paris)* 1985;136:229–32.

These letters were shown to the authors, who reply as follows:

Sir,

We note with interest the comments of Dr Sanderson and Dr Sachs and Dr Lanfranchi.