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.

John Sanderson,
Musgrove Park Branch,
Taunton and Somerset Hospital,
Taunton,
Somerset TA1 5DA.

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.

R N Sachs,
J Lanfranchi,
Hôpital Avicenne,
125 rue de Stalingrad,
93008 Bobigny,
France.

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.

Our study was essentially a quantitative assessment of suppressor cell function, prompted by reports of impaired function which might, in some way, allow a sequence of events terminating in congestive cardiac failure.^{1,2} We have previously reported a quantitative study of T cell populations in three groups: eight patients with congestive cardiomyopathy, eight patients with heart failure caused by coronary disease, and eight normal controls.³ There was no significant difference in the ratio of helper/suppressor cells between the groups, although there was a tendency for both heart failure groups to have higher ratios. This was due to both an increase in helper cells and a decrease in suppressor cells in both patient groups, but none of these differences was significant. These results suggested to us that any defect of suppressor cells must lie in abnormal function rather than in reduced numbers but we have failed to demonstrate a specific abnormality of suppressor cell function in the heart failure of congestive cardiomyopathy. We agree with Sanderson, however, that an immunological abnormality may be transient and detectable only in the early stage of congestive cardiomyopathy. All of our

patients in both studies had been known to have congestive cardiomyopathy for more than three months.

P J Lowry,
M D Gammage,
R A Thompson,
W A Littler,
Department of Cardiovascular Medicine,
East Birmingham Hospital,
Bordesley Green East,
Birmingham B9 5ST.

References

- 1 Fowles RE, Bieber CP, Stinson EB. Defective in vitro suppressor cell function in idiopathic congestive cardiomyopathy. *Circulation* 1979;59:483-91.
- 2 Eckstein R, Mempel W, Bolte HD. Reduced suppressor cell activity in congestive cardiomyopathy and in myocarditis. *Circulation* 1982;65:1224-9.
- 3 Lowry PJ, Thompson RA, Littler WA. Cellular immunity in congestive cardiomyopathy: the normal cellular immune response. *Br Heart J* 1985;53:394-9.

Notices

British Cardiac Society

The Annual General Meeting for 1988 will take place in Belfast on 23 and 24 March 1988, and the closing date for receipt of abstracts was 4 January 1988.

Cardiovascular intervention

A workshop on coronary angioplasty and other related procedures will be held at the London Hospital on 28 and 29 April 1988. Inquiries to Dr M T Rothman, The London Hospital, London E1 1BB.

Pacing and electrophysiology

The annual scientific meeting of the North American Society of Pacing and Electrophysiology will take place in Los Angeles on 12 to 14 May 1988. Inquiries to NASPE, 13 Eaton Court, Wellesley Hills, MA 02181, USA.

Correction

Ventricular defibrillation: the Belfast experience. *G W N Dalzell, S R Cunningham, C M Wilson, J D Allen, J Anderson, A A J Adgey*—We regret that two errors were introduced into this article published in the November issue (volume 58: pages 441-6). On page 442 the second sentence of the section entitled "Prognosis of patients in ventricular fibrillation" should have read "When ventricular fibrillation occurred *less* than four hours after the onset of symptoms (these were mainly patients with acute myocardial infarction), approximately 80% were alive three years after the initial episode." Also on page 442 the last sentence in the left hand column should have read "In 1983 we looked at 125 patients with ventricular fibrillation."