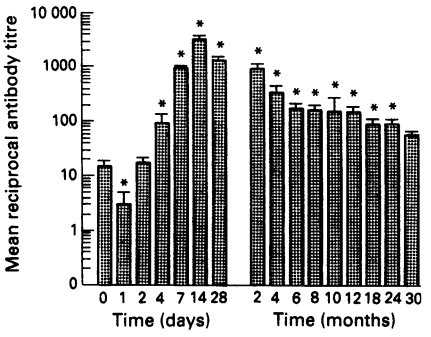

Time course of the IgG antibody response after a bolus dose of streptokinase in a mean (1SE) of 20 patients. Asterisks indicate values that are significantly different from baseline (day 0).

on the fourth day to a peak on day 14. Thereafter titres (mean (SEM) gradually declined but remained significantly raised for two years after treatment presentation, 14·63 (4); day 14; 3192 (771); 2 years, 86 (24); 2½ years, 65 (26)) (figure). No evidence of IgM antibody was found in our study indicating that the immunological response was a secondary one. We also found that specific antibody significantly impairs the action of streptokinase in vitro.[6]

We agree with Buchalter *et al* that it would be prudent to avoid repeat treatment with streptokinase for a period after the initial 72 hours of therapy, until the significance of these antibodies has been evaluated in vivo. Our findings suggest this period may be for 2½ years or more.

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Postoperative cardiac surgical care: an alternative approach

SIR,—We are grateful for Mr Treasure's appraisal of our article (*British Heart Journal* 1993;**69**:59–64. He is well qualified to assess our approach against the background of conventional clinical practice. He provides the historical background for the work and correctly defines the wide range of facilities grouped together under the title "cardiac surgical intensive care". We believe that his comment does much to reconcile the apparent conflicts between our approach and that of conventional postoperative care—showing that the only real change has been one of emphasis.

We agree that extubation is an important threshold event marking a patient's transition to a relatively straightforward recovery period. However, references to extubation practices of 20 years ago are inappropriate. Management based on a better understanding of postoperative physiology yields patients who are, as stated, "alert, haemodynamically stable, fully warm" and perfectly ready for safe extubation by any ordinary criteria. A reintubation rate of approximately 4% supports this view.

Mr Treasure asks whether we have made our case. I am sure he is aware that other units, notably the Oxford group, have already adopted our approach and confirmed the benefits, clinical and financial, that we claim. A case of voting with their feet?

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Hypoxia and the heart

SIR,—We would like to comment on the excellent editorial on hypoxia and the heart by Davies and Wedzicha (*British Heart Journal* 1993;**69**:3–5). Unfortunately they omitted a clinical situation where hypoxaemia is well documented, namely the postoperative period. The pattern of postoperative hypoxaemia is clearly defined[1] and recent studies reported in anaesthesia journals have been directed at determining the incidence of hypoxaemia and myocardial ischaemia.[2,3] A study from our department showed that 20% of hypoxaemic episodes are associated with ischaemia and that this association was related to the severity and duration of the hypoxaemia. A further study[4] in postoperative patients after aortic aneurysm repair showed a correlation between myocardial ischaemia and hypoxaemia after the withdrawal of supplemental oxygen.

We would also like to highlight the use of pulse oximetry. Davies and Wedzicha correctly suggest that hypoxia and its complications are underdiagnosed. We have shown that hypoxaemia is common in the period immediately after acute myocardial infarction and frequently missed on clinical grounds.[5] We have also shown that only 4% of coronary care units in England use pulse oximetry to guide oxygen treatment despite the fact that 80% have an oximeter available.[5] We believe from our experience in anaesthetic practice that much of this underdiagnosis of hypoxaemia is secondary to lack of monitoring and that easily correctable hypoxaemia is often not corrected with supplemental oxygen because the initial cyanosis is not noted.

We are engaged in further studies of the association between hypoxaemia and ischaemia in the perioperative period as well as the association between cardiac events or ischaemia and peri-infarct hypoxaemia.

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

SIR—We thank Wilson and Reilly for their helpful comments. Perioperative hypoxia may well be common, and interactions with anaesthetic drugs that have myocardial depressant and arrhythmogenic actions will potentially increase the risks of uncorrected hypoxia.

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Small ductus arteriosis

SIR,—May I add my support to the letters of Sturridge[1] and Glickstein *et al*[2] who recommend setting up national surveys "to discover the risk to life and health of the untreated small ductus".

In the past year I have investigated two adults in their 20s who were found on routine clinical examination to have a murmur and were subsequently, on investigation, found to have a patent ductus arteriosus with a shunt that was not haemodynamically significant and normal intracardiac pressures. In both cases the shunt was only detectable at angiography by contrast injection into the aorta. I discussed the risks of infective endocarditis and surgical closure with both patients. These risks are believed to be small. Both patients preferred to be treated medically.

I am sure it would be sensible to set up a national survey of those with a small patent ductus. Perhaps this is something that the British Cardiac Society should consider doing.

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