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SIR,—The editorial by Salmon, Keeton, and Sethia (*British Heart Journal* 1993;69: 479-80) on achieving a balance between surgery and interventional catheterisation was, to say the least, uncharitable. It contained not a single positive comment on either of the two new interventional cardiac catheterisation techniques of laser pulmonary valvotomy¹ and ductal stenting with pulmonary artery banding.² Reports of first experiences with new techniques are all too easy to douse with negativity. There must be some positive points to be made about both the new techniques.

The fact that pulmonary atresia with intact septum and favourable associated anatomy may be treated with low risk by surgery does not mean we should not look to improve further upon the results of traditional treatment. Rosenthal and his colleagues showed that it is possible to perform an adequate valvotomy in the presence of an atretic pulmonary valve without opening the chest.¹ Surely there is something positive, exciting and progressive in that—even though Rosenthal *et al* clearly accept that their selection criteria need reviewing. I and my colleagues at Killingbeck have also used laser pulmonary valvotomy to treat patients with pulmonary atresia due to an imperforate pulmonary valve who would otherwise

have been candidates for conventional surgical valvotomy. We attempted the technique in five infants. In two laser valvotomy followed by balloon dilatation produced excellent results with residual outflow gradients of less than 20 mm Hg, some pulmonary regurgitation, and no significant atrial shunt in either direction. In one there was residual infundibular stenosis and a residual right to left atrial shunt; the child is now two years old, mildly cyanosed, and is awaiting elective surgery. Two of the infants were discharged from Killingbeck Hospital within 48 hours of the procedure. The technique was unsuccessful in the remaining two cases (in one the right ventricular outflow tract was perforated without ill effect and in the other the procedure was abandoned because of inability to position the laser guiding catheter satisfactorily): both babies were successfully treated surgically. We have had no deaths and no major complications. Is it not positive progress to treat 60% of candidates for open pulmonary valvotomy by low risk interventional catheterisation? Our early experience of this technique leads us to recommend laser valvotomy as first line treatment in this group of patients.

It may be more difficult to justify positive comments concerning any form of treatment for the hypoplastic left heart syndrome. It is, however, inappropriate to damn our attempts² at short term palliation of these patients by comparing our results with those of a single centre in the United States.³ No centre in the United Kingdom has been able to attain Norwood's surgical results. Indeed, my overall impression from colleagues around the United Kingdom is that the results of the stage 1 Norwood operation have been so disappointing here that the operation has all but been abandoned in favour of either no treatment or cardiac transplantation. Organs for small babies are in such short supply that almost all such patients will die awaiting a donor organ. Ductal stenting combined with pulmonary artery banding was introduced as a joint attempt between physicians and surgeons at Leeds and Newcastle to assess the possibility of providing short-term palliation for babies whose parents wished for active treatment. To date we have attempted the combined procedure in seven neonates with technical failure in one, death due to persisting heart failure in four, and survival (at home, gaining weight) until transplantation, at the ages of four and six months respectively, in two cases.

Only 28% survival till transplantation may not sound much to be proud of, but it does represent a substantial improvement upon the mortality of virtually 100% that we offered before the trial of our new protocol. Short-term banding of the branch pulmonary arteries had not caused major distortion of the vessels in our patients who underwent transplantation: the 8 mm diameter ducts appeared adequate for systemic blood flow despite substantial weight gain and there was no evidence of coronary or cerebral ischaemia to support the statement in the editorial that coronary or cerebral blood flow "must ultimately become severely compromised" (this is not so). A more balanced view of combined pulmonary artery banding and ductal stenting would be that, at the very least, it increases the chance of an infant with hypoplastic left heart syndrome surviving until a donor heart is available.

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Streptokinase resistance: when might streptokinase administration be ineffective?

SIR,—We read with interest the recent report by Buchalter *et al* concerning streptokinase readministration.¹ However, we have some reservations about the interpretation of their immunological findings.

Buchalter *et al* adapted an ELISA to assess anti-streptokinase IgG and used normal sera for the reference standard curve. In the patients studied none had high antibody titres on admission but by days 7-10 titres were significantly different from "normal". The finding of specific antibodies at presentation before the administration of the antigen and a time lag of 7-10 days resulting in a significant rise in titre is typical of a secondary immunoglobulin response² and not a primary response as stated by Buchalter *et al*. If the patients had been studied every day Buchalter *et al* would almost certainly have found the titres were gradually increasing (as shown in our paper³ and did not suddenly rise by days 7-10).

They found that neutralisation titres to streptokinase had risen above the normal range by days 3-4 and because the neutralisation titre is probably caused by antibody, this result suggests an early rise in specific immunoglobulin. The difference in findings between the neutralisation titre and the ELISA assays may reflect the sensitivity of the assays rather than another class of antibody, but unfortunately, Buchalter *et al* do not state the sensitivity and reproducibility of their methods. If they seriously considered the possibility of a primary response, it would have been important to look for evidence of antigen specific IgM.

The widespread presence of anti-streptokinase resistance (attributed to antibodies) in the population has been well documented and is presumably caused by previous streptococcal exposure.⁴ It has also been shown that resistance to streptokinase develops after treatment with streptokinase and the time course of this neutralisation has been described.⁵ We have studied the immunoglobulin response to streptokinase in patients with acute myocardial infarction and found the time course of the response was similar to the neutralising titre as described elsewhere⁵ which suggests that the response related to antibody. We found that patients presenting with acute myocardial infarction had low titres of anti-IgG (subclass IgG1) and after treatment with streptokinase these initially disappeared (1-3 days). Subsequently there was a rise

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 post-operative 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 post-operative period. The pattern of post-operative hypoxaemia is clearly defined¹ 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 study⁴ 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 arteriosus

SIR,—May I add my support to the letters of Sturridge¹ 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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