Correspondence

Hypothermic protection (26°–25°C) without perfusion cooling for surgery of congenital cardiac defects using prolonged occlusion.

SIR,—The use of periods of circulatory arrest under the protection of profound hypothermia (less than 20°C) is a well established technique to permit surgery that may be impossible on cardiopulmonary bypass. The time limits are debatable, so the statement by Professor EE Litasova and Dr VN Lomivorotov (March 1988;**43**:206–11) that "The results obtained without perfusion under deep (26–25°C) hypothermic protection suggest that 75 minutes is a safe time, in terms of brain damage, for circulatory arrest" merits critical attention. 25–26°C is quite a modest degree of hypothermia at which to seek prolonged cerebral protection from ischaemia and 75 minutes is a long period of circulatory arrest by any standards.

The anaesthetic and surgical techniques are detailed in the "Methods" section but no criteria or objective methods for establishing neurological function were employed. It appears that time to waking has to suffice. The issue of ischaemic brain damage is largely side stepped by attributing unsatisfactory outcome to air embolism or poor myocardial function. A relation was detected between duration of ischaemic time and the number of neurological complications that were recognised, and even by these rather generous criteria it does not suggest that "75 minutes is a safe time."

The authors provide nine references, of which six are to Russian publications. Their key reference, from 1963, is fully a quarter of a century old and maybe none the worse for that, but it may be! The use of circulatory arrest with surface cooling was well established in the 1950s. On the basis of oxygen consumption data—for example, those provided by Ross[1]—circulatory arrest for up to 10 minutes at 30°C was considered acceptable. Abiding by these guidelines, Sellors closed hundreds of atrial septal defects with circulatory arrest, mostly within 4–5 minutes.[2] Various combinations of surface and perfusion cooling were employed to cool to below 20°C to extend the time available and it became accepted teaching that this gave up to an hour of protection.

I reviewed the available clinical and experimental data concerning circulatory arrest at 18–20°C and found that the weight of evidence suggested that central nervous system damage was highly likely by an hour and could be demonstrated with increasing risk beyond 40 minutes.[3] Experimental work performed at the University of Alabama supported these conclusions[4] and others who have published on the subject since then have agreed with us.[5,6] It seems inherently unlikely that the anaesthetic and surgical protocols described in this paper provide protection for nearly twice as long at a temperature substantially warmer. The sparse neurological evidence provided is insufficient to convince me otherwise.

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Thyroid function and endocrine abnormalities in elderly patients with severe chronic obstructive lung disease

SIR,—We read with interest the paper by Dr S Gow and colleagues (July 1987;**42**:520–5) on endocrine abnormalities in elderly patients with chronic obstructive lung disease. Their summary concludes that "general effects of age and illness may be more important than the direct effect of hypoxia in determining hypothalamic-pituitary function in elderly patients with chronic lung disease."

As it is well known that the testis starts to fail in old age (with a rise in follicle stimulating hormone and luteinising hormone), it is not surprising that this "normal failure" will mask any other factors that may tend to lower testosterone production. We feel sure therefore that the failure of Dr Gow and her colleagues to find a correlation between serum testosterone concentrations and degree of hypoxia in their more aged hypoxic men with chronic obstructive lung disease is simply a function of age and explains the apparent discrepancy between their results and ours.

Of more interest to us is whether endocrine abnormalities occur in patients with chronic obstructive lung disease before the age that endocrine abnormalities might normally be expected. Our studies[1] suggested that they do, the most consistent being low serum testosterone concentrations that correlate with arterial oxygen tension (Pa_{O_2}) in our patients under the age of 70. Indeed, 22 patients aged 35–57 years with a mean Pa_{O_2} of 6·91 kPa had lower serum testosterone values than older patients aged 59–70 years with a mean Pa_{O_2} of 6·66 kPa (serum testosterone mean for younger 9·68 and for older 11·88 mmol/l). If aging were an important causal factor under the age of 70 we would have expected older patients to have had lower serum testosterone values for a similar reduction in Pa_{O_2}.

In a further series of 18 male patients with a mean age of 62 (range 47–72) years with chronic obstructive lung disease (Dobson *et al*, unpublished observation) we have confirmed the positive correlation between Pa_{O_2} and serum testosterone ($r = 0{\cdot}656$, $p = 0{\cdot}004$), but have been unable to show a correlation between testosterone and age ($r = 0{\cdot}006$; $p > 0{\cdot}05$).

We noted that two hypoxic patients with chronic obstructive lung disease aged 42 and 63 years studied by Dr Gow and colleagues did indeed have low testosterone concentrations with lack of obviously raised luteinising hormone. This suggests hypothalamic or pituitary suppression but gonadotrophin releasing hormone stimulation studies were not