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Because we receive many more letters than we have room to publish we may shorten those that we do publish to allow readers as wide a selection as possible. In particular, when we receive several letters on the same topic we reserve the right to abridge individual letters.

Letters critical of a paper may be sent to the authors of the paper so that their reply may appear in the same issue. We may also forward letters that we decide not to publish to the authors of the paper on which they comment.

Letters should not exceed 400 words and should be typed double spaced and signed by all authors, who should include their main degree.

Wasting blood

SIR,—May we suggest that recent reports of transmission of the acquired immune deficiency syndrome virus by blood transfusion should cause a reappraisal of transfusion practice in the United Kingdom. The transfusion of a single unit of blood can rarely if ever be justified and closer attention needs to be paid to the balance of risks and benefits.

In a study which we undertook of the transfusion practice of UK anaesthetists it was apparent that 10 g/dl of haemoglobin was regarded by most anaesthetists as the level below which preoperative measures to raise the haemoglobin level were indicated. We believe that by adopting 8 g/dl as a lower acceptable level for patients undergoing major surgery we could reduce the requirements for blood transfusion considerably, thus minimising the associated risks to patients.¹ For patients undergoing short operative procedures with little risk of haemorrhage even lower haemoglobin levels could be accepted provided the patient was otherwise fit.²

Several papers have recently shown that for most operations blood loss can be reasonably predicted, and the modern anaesthetist should be able to plan intraoperative transfusion regimens which protect the patient from

the dangers of haemorrhage and also avoid unnecessary transfusion.³

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- 1 Pineda AA, Braica GM, Taswell HF. Haemolytic transfusion reaction, recent experience in a large blood bank. *Mayo Clin Proc* 1978;53:378.
- 2 Graves CL, Allan RM. Anaesthesia in the presence of severe anaemia. *Rocky Mountain Medical Journal* 1970;67:35.
- 3 Stehling LC. Guidelines for ordering blood for elective surgery. *Int Anesthesiol Clin* 1982;21:45.

Noradrenaline: a circulating inhibitor of sodium transport

SIR,—Mr A Riozzi and coworkers (20 October, p 1025) describe a noradrenaline mediated, dose dependent reduction of leucocyte sodium efflux rate constant which was due to decrease in ouabain sensitive sodium pump activity. They also report that this effect was blocked by propranolol, suggesting that it was mediated by β adrenoceptors. Furthermore, they suggest a possible circulating inhibitory role for catecholamines on sodium transport.

Using mixed human leucocytes we showed a stimulatory effect of adrenaline (9 nmol/l) on both rubidium (equivalent to potassium) influx and sodium efflux and that this was mediated by β_2 adrenoceptors. We were unable to show any such effects using fresh noradrenaline (10-300 nmol/l).² Our findings are compatible with the known effects of adrenaline infusion, which causes hypokalaemia in vivo, without increased urinary potassium loss, presumably by moving potassium into cells^{3,4}; noradrenaline infusion does not affect plasma potassium concentration.⁵

Leucocytes are thought to possess β_2 adrenoceptors but not β_1 adrenoceptors,⁶ and noradrenaline is also known to act principally on α_1 , α_2 , and β_1 adrenoceptors.⁷ Consequently, it would be surprising to observe any mediated effects of noradrenaline on human leucocytes. Studies using adrenergic challenge in man have supported the contention that the relation between the sodium pump—that is, Na^+ , K^+ -ATPase—and catecholamines is mediated through β_2 adrenoceptors.⁸

The discrepancies between our results and those of Mr Riozzi and others are likely to be caused by differences in the experimental models. They exposed leucocytes, in serum, to noradrenaline for 10 minutes during loading