

MISUSE OF BLOOD

To the Editor: In recent months an increasing number of patients have been referred to us by doctors from outside with a standard note: '... breathlessness ... tiredness ... anaemia (4-8 g/100 ml) ... blood transfusion given ... Hb now 12 g/100 ml ... please see and treat'. Many of these patients have required urgent blood-letting and dialysis for iatrogenic pulmonary oedema and renal failure.

Few doctors realize the many disadvantages of, and the limited indications for, blood transfusion. The disadvantages are: (i) infection — thrombophlebitis, hepatitis, etc. (ii) allergy, including renal shutdown; (iii) incompatibility reactions, including renal shutdown; (iv) sensitization, making future transfusions or transplants difficult; (v) volume overload and cardiac failure; (vi) worsening of uraemia; (vii) limited availability and enormous cost (R16-R20 per unit); and (most important) (viii) delay and confusion in diagnosis and treatment of the cause of the anaemia.

Management of Anaemia

Acute haemorrhage (e.g. obstetric, acute haemolysis, trauma, gut): Packed cells *only* should be given, to keep the Hb at 8-10 g/100 ml (haematocrit $\pm 30\%$), or above 12 g/100 ml in pregnancy. In massive, ongoing haemorrhage, fresh whole blood should be given after 6 units of packed cells.

Chronic bleeding (e.g. slow gut losses, menorrhagia) or anaemia of any other cause: Blood transfusion is contraindicated, except (a) for urgent anaesthetic — transfuse to Hb 8 g/100 ml; (b) if Hb is below ± 4 g/100 ml or if there is frank high-output cardiac failure or cardiac or brain ischaemia — 1-2 units only; (c) in severe trauma or sepsis — transfuse to Hb 8-10 g/100 ml with packed cells.

For example, the majority of anaemic patients require only iron replacement (oral, rarely intravenous) for chronic bleeding or frank iron deficiency; folic acid or vitamin B₁₂ for macrocytic anaemia; rarely, steroids for ongoing haemolysis; and diagnosis and treatment of other causative disease, e.g. infection, renal failure, cancer, leukaemia.

In patients without obvious bleeding, a blood smear will show hypochromic microcytic anaemia in most — in which case only iron replacement is needed. Other anaemic patients or those without an obvious cause should have a bone marrow examination and blood urea estimation done.

No patient should have blood transfusion without regular checking of the urine, blood electrolytes and urea (Azostix is better than nothing). Diuretics should *not* be given routinely.

Although the Hb may rise by only 1 g/100 ml per week, replacement of deficient iron, folate or vitamin B₁₂, etc., produces dramatic symptomatic improvement within a few hours.

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ASSOCIATION OF LACTIC ACIDOSIS WITH BIGUANIDE THERAPY

To the Editor: I write to endorse the views expressed by Drs Botha, Vinik and Jackson¹ that it is not possible to predict which patients with uncomplicated diabetes are likely to develop lactic acidosis on biguanide therapy. I should like to reiterate the point they made, that all biguanides impair lactate clearance and are contraindicated in patients with renal, cardiac and hepatic insufficiency. I should, however, also like to emphasize (in the light of their preamble, 'it would be useful if one or another of the biguanides proved to be free of this ... lactic acidosis ... complication'), that all 3 of the biguanides in current therapeutic use have been shown to be associated with lactic acidosis.^{2,3} While in no way implying that the authors meant anything but this, it is imperative that the prescribing doctor is left in no doubt of this risk with all 3 agents. Until such time

as the relative risk from each of the 3 drugs is known, it is preferable not to refer to any particular agent as the 'drug of choice', since this connotation is bound to instil a false sense of security in the prescriber.

The key to the safe use of biguanides today is meticulous attention to detail, with exclusion of patients in whom contraindications are known to exist, and close surveillance of those in whom complications and contraindications might arise. Anything less might well lead to a spate of cases of lactic acidosis from the indiscriminate use of the currently favoured biguanides (and there are those who favour metformin⁴), and precipitate a ban on the use of all these agents.

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HYCANTHONE — A DANGER ASSOCIATED WITH ITS USE

To the Editor: I should like to draw the attention of your readers, especially those in peripheral hospitals, to a recent fatality in a patient treated with hycanthone. Full details of this report will be published at a later date.

Suffice it to say at the present that this patient received an appropriate dose of hycanthone, and became jaundiced and restless. Promethazine hydrochloride was administered, but coma ensued and the child died 2 days later. Autopsy revealed features of acute massive hepatic necrosis.

As stated by Gane,¹ hycanthone is contraindicated in patients who have concurrent infections, or who have been ill in the past month, or have known or suspected liver damage, or are receiving hepatotoxic drugs. The use of chlorpromazine in conjunction with hycanthone is specifically contraindicated.

We should be grateful for further details of any cases in which hycanthone may have been a contributing factor to a patient's death, such as for example the report by Kallmeyer *et al.*²

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ERRATUM

In the article entitled 'Autopsy findings in mental patients', by Gillian Cole, on page 534 of the *SAMJ* of 17 September 1977, the second sentence of the second paragraph under the subheading 'Subdural Haematoma' on page 535 ought to have been omitted. It read as follows: 'In the remaining 3 cases the diagnosis was not made, nor was there sufficient time to make it.'