LETTERS TO THE EDITOR

To the Editor—I have noticed that respiratory stimulants such as nikethamide and doxapram are used rather frequently for the management of chronic obstructive airway disease (COAD) patients in acute respiratory failure, especially by junior doctors. The drug nikethamide is a central nervous system stimulant which may cause convulsions. It reminded me of the pharmacological experiment I did as a medical student: mice given nikethamide all died quickly after jumping violently for a short time.

There is nothing about the drug in any of the major current textbooks on medicine, respiratory medicine, or pharmacology. A literature search using Medline showed that in the past five years there has been only one study on the use of nikethamide in COAD patients with acute exacerbation. The authors concluded that "all the changes demonstrated that nothing is worthwhile with the treatment of nikethamide, but a side effect from increasing work of breathing and consumption of oxygen".[1] I believe that a drug which is ineffective and hazardous should not be used clinically. Doxapram may be a better choice in selected patients who are properly monitored and managed.[2,3] However, it should be noted that patients who are exhausted are unlikely to benefit from doxapram, and the drug may actually worsen the diaphragm dysfunction.[4] Mechanical ventilatory support should be offered instead for patients who are potentially salvageable.

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References

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To the Editor—Transfusion is the mainstay treatment for patients with thalassaemia major. It has been suggested than an annual transfusion requirement (or index) of more than 200 to 250 ml per kg mid-year body weight of packed red cells is indicative of hypersplenism.[1,2] As a result, splenectomy is indicated, to resume an annual index of 150 to 180 ml/kg as documented in splenectomised patients.

As the blood product used in Hong Kong is different from that used in other countries (Table 1), the applicability of such an approach needed verification. We retrospectively reviewed all ß-thalassaemia major patients receiving transfusion in 1993 in our unit. They were transfused at four-weekly intervals in order to maintain a mean haemoglobin level (average of pre- and post-transfusion haemoglobin levels) of 12 g/dL. The total number of units and volume of blood given, pre- and post-transfusion haemoglobin levels, and the mid-year body weight were included in the analysis. The results are shown in Table 2.

Fifty-six patients received 722 transfusions; these were divided into two groups according to whether they were splenectomised or not. The mean transfusion indices in splenectomised and non-splenectomised patients were 258 ml/kg and 315 ml/kg, respectively. Using the mean plus two standard deviations as the upper limit, three children had indices that exceeded 400 ml/kg/y. Two had progressive splenomegaly and developed thrombocytopaenia while waiting for splenectomy; no complications were detected in the other patients.

In conclusion, thalassaemic patients should be evaluated for splenectomy when their annual transfusion index exceeds 1.2 units/kg or 400 ml/kg of plasma-reduced red cells. This is in line with the upper limit recommended by the Thalassaemia International Federation of 1.5 times the annual consumption in splenectomised patients.[3] Numerical limits without reference to the type of blood product published elsewhere cannot be applied locally.

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