A myeloproliferative disease and pseudohyperkalaemia

Sir,

A 76-year-old woman was admitted after dislocating her left shoulder in a fall. Her past medical history included osteo-porosis and cerebrovascular disease. Routine investigations revealed a sodium of 135 mmol/litre, potassium of 6.4 mmol/litre, urea 7.0 mmol/litre, creatinine 85 µmol/litre, haemoglobin of 17.2 g/dl, white cell count 21.1x10^9/litre and platelet count of 679x10^9/litre. There was no evidence of haemolysis. An electrocardiogram was normal. Despite potassium-lowering treatment with intravenous insulin and dextrose, serum potassium did not fall below 6.0 mmol/litre. Blood samples collected in a heparinised test tube and standard gel tube, allowing for analysis of plasma and serum specimens respectively, resulted in corresponding potassium values of 4.9 mmol/litre and 6.1 mmol/litre. No harm was incurred in terms of potential iatrogenically-induced hypokalaemia. Cytogenetic studies and bone marrow biopsy established an underlying myeloproliferative disease as responsible for the leucocytosis and thrombocytosis.

Pseudohyperkalaemia was recognized as a clinical entity by Hartmann and Mellinkoff in 1955, and has been the presenting feature of acute and chronic myeloproliferative disorders and leukaemoid reactions from malignancies (Bronson et al, 1966; Bellevue et al, 1975; Kerr et al, 1985; Ho et al, 1991).This façade of false hyperkalaemia, apart from precipitating unnecessary potassium-lowering interventions, is harmless. Hyperkalaemia is caused by the lysis of platelets and leucocytes occuring after venesection. The use of anticoagulation prevents any cellular degradation and artifactual release of potassium. Remember that platelets which contain 70 mEq/litre of potassium can 'spill' their contents in view of their fragility in certain in vitro circumstances (Bronson et al, 1966; Ho et al, 1991).

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SORTED

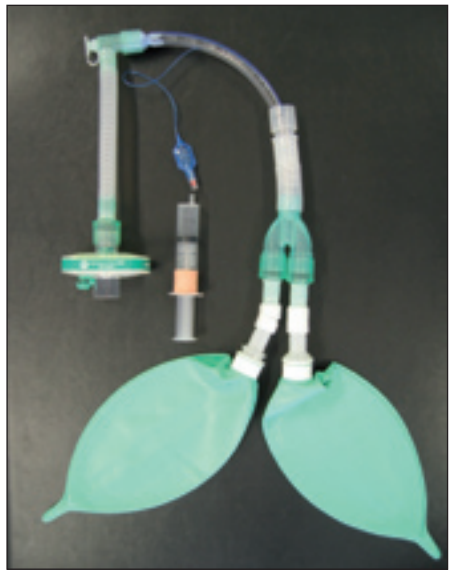
Sir,

We would like to share a teaching aid we have devised to illustrate the anatomy, and to an extent physiology, of the trachea, bronchus and lungs; the Selly Oak Respiratory Tree Educational Device (SORTED). This has proved useful for educating school children, medical students, theatre practitioners and anaesthetists of all grades.

Figure 1. SORTED system.

The design is simple (*Figure 1*) and can be made from recycled consumables from theatre to save money (for infection control reasons do not recycle used catheter mounts or filters), or using new components at a cost of about £7. The trachea, bifurcation and main bronchi are constructed from breathing circuit tubing and connector, and each lung from a 2000 ml re-breath bag.

Figure 2. The SORTED system intubated with a cuffed, pre-cut size 9 endotracheal tube connected to angle piece and filter.



The 'trachea' can be attached to a self-inflating bag (Ambu-bag) or circuit of an anaesthetic machine (*Figure 2*). Once the 'lungs' are inflated hand or mechanical ventilation can be demonstrated, as well as how peak end expiratory pressure acts.

As well as ventilating the 'lungs', this can be intubated with most sizes of endotracheal tube and shows the position of the inflatable cuff in relation to the 'carina' (*Figure 2*). Using smaller endotracheal tubes, the left and right main 'bronchi' can be intubated, illustrating how this can easily occur and the subsequent outcome. The use of the bougie and bronchial blockers can also be demonstrated, and we believe a similar system could be created to allow practice with a flexible bronchoscope. Smaller re-breath bags could be used to show paediatric volume lung capacities.

The SORTED system is easy and inexpensive to construct, and has a wealth of uses for interested individuals and groups.

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