The College of Anaesthetists' scientific meeting in May 1990 brought an area of everyday practice relating to fluid and electrolyte balance, which many believe to be mundane, into high profile and exploded some of our cosy myths with well validated new facts. New agents for the management of metabolic acidosis will shortly become available for general use and their rational application will be a matter of great importance.

This issue of the Journal is based on that scientific meeting, which aimed to challenge old concepts in acid-base, sodium, potassium and calcium balance and to debate the choice of fluids for resuscitation and for prevention of perioperative renal failure.

Cohen presents a radical revision of the traditional explanations for acid excretion, elaborating the key role of the liver in this respect [4] and drawing on the work of Häussinger, Steeb and Gerok [7] showing that in liver disease, particularly cirrhosis, plasma bicarbonate concentration is related inversely to the ability of the liver to synthesize urea. In renal failure, by contrast, the uraemic acidosis is most probably related to failure of nitrogen excretion as $\text{NH}_4^+$ by the kidney and its conversion to urea by the liver, with production of protons producing acidosis [2]. Whatever the mechanisms of production of acidosis, we must revise our views of its automatic, unthinking treatment with sodium bicarbonate, which in many experimental and clinical studies has been shown to worsen intracellular acidosis related to hypoxic ischaemic conditions [1, 10].

It is hard to believe that so much controversy has raged in the past decade over the question of whether hyponatraemia per se or its excessively rapid correction is responsible for neurological damage; either central pontine myelinolysis or cerebral oedema and hypoxic damage. The meticulous clarification by Swales repays careful reading. New, but not clarifying, information is available describing the development of fatal diabetes from untreated hyponatraemia [5] and the importance of the speed of onset in the management of hyponatraemia [3], if necessary by urgent measures such as continuous arteriovenous haemofiltration before liver transplantation [8]. Time will tell whether the sex differences which result in increased morbidity from hyponatraemia in female rats apply also in the human female [6].

Less controversy surrounds the maintenance of potassium balance in the perioperative period, but a sound update is provided by Vaughan.

Recommendations concerning appropriate fluids for resuscitation are beginning to be questioned now that high molecular weight starch solutions are available and hypertonic saline solutions are finding a place. Anxieties raised by the occurrence of acute hyperosmolality after administration of sodium bicarbonate solutions and its relation to high mortality and brain damage should make us question the potential adverse effects of hyperosmolar fluid loading with hypertonic saline on cerebral haemodynamics. It is likely that hypertonic saline exerts reflex and venoconstrictor effects in addition to osmotic effects which may be beneficial in resuscitation of hypovolaemic patients. Nevertheless, the advice of Mattar [9] that “the hypovolaemic patient needs volume not a quick fix” is apt.

On the question of preventing postoperative oliguria, more questions are raised by our conventional answers and we are repeatedly faced with the difficulties of assessing body fluid status from limited access to major body fluid compartments.

Erdmann brings an excellent perspective into the question of calcium usage in resuscitation by bringing his own work to challenge our current (and recently revised) recommendations.

Long may we continue to examine this major area of our practice, explode old myths and explore innovations.

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REFERENCES


