IMJ

Official Journal of the Irish Medical Organization

October 2003 Volume 96 No. 9

IMJ Commentary

Paediatric Intravenous Fluid Regimens Need Review

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Duke and Molyneux[1] have recently called into question the current intravenous fluid guidelines for children. The recommended maintenance rates 100mls/Kg for the first 10Kg, 50mls/Kg for the next 10Kg and 20mls/Kg for body weight over 20Kg are based on data produced by Winters[2] in 1973. There are now concerns that it may be excessive particularly in ill children who have impaired free water excretion. A second consideration is the use of hypotonic solutions with 0.18% or 0.2% saline being generally employed. The authors point out that the use of hypotonic maintenance fluid is based on a child's normal requirements; with 0.18% Saline in 5% Dextrose he will receive 3mmol/Kg Sodium and 3.5mgs/Kg/min Glucose. The emerging concern is that this type of fluid in these amounts runs the risk of causing hyponatraemia particularly in the ill child with reduced free water excretion. Primate studies show that in the rhesus monkey model of pneumococcal sepsis free water clearance decreased to 17% of baseline values during the first 9 hours of infection. Using hypotonic fluids may be particularly hazardous in developing countries where facilities to measure serum sodium concentrations are unavailable.

Paediatrics has long been at the forefront of research about fluid therapy, many of its innovations being subsequently adopted by Medicine and Surgery. The early Paediatricians particularly those in the US were stimulated by the high infant mortality from infantile gastroenteritis. Their fundamental studies showed that gastroenteritis was a combination of acid/base and water/salt problems.

In 1920 John Howland and his Johns Hopkins team documented that infants suffering from severe diarrhoea had associated acidosis. He

noted that children with severe gastroenteritis had the same breathing pattern as described by Kussmaul in relation to diabetics. In 1930 the St. Louis paediatrician Alexis Frank Hartmann recommended the use of sodium lactate in solutions in addition to sodium chloride because in this way more alkali than chloride was administered. However despite these rescue fluids mortality from diarrhoea remained high.

The great breakthrough was made by the New Haven paediatrician Daniel Cade Darrow[3] who found that there was the considerable loss of 25% of the body potassium during acute diarrhoea. Despite the great fears about potassium that existed at the time, Darrow in 1946 started infusing dehydrated children with potassium over [4-8] hour periods. The results were remarkable. The infant mortality from diarrhoea was reduced from 20% to 5%. His courage earned him the name 'Daring Darrow'. James Gamble[4], a Harvard paediatrician in the 1940s, showed the importance of the sodium ion for the maintenance of the extracellular fluid volume and that it constituted 20% of the body weight. He repeatedly stated that to maintain a stable extracellular space only three substances are necessary; water, sodium and chloride ions. Gamble also introduced the concept of the anion gap.

These fluid and electrolyte developments were complemented by the work of Poul Astrup[5]. Astrup's interest in blood gases was generated by the 1952 Copenhagen polio epidemic and the development of the first intensive care units. Subsequently in collaboration with the Radiometer he developed methods and equipment for the measurement of blood gases.

Duke and Molyneux have revisited some of these concepts particularly impaired free water excretion which is encountered in many acute severe illnesses. Hyponatraemia occurs in as many as 40% of these children. An abrupt fall in serum osmolarity of 5mmol/l decreases osmotic pressure difference between the capillary lumen and the brain interstitium by 95mmHg. This favours water accumulation in brain cells. In a randomised trial of fluid management in bacterial meningitis facial oedema, as a marker of excess fluid

administration, was associated with more deaths and handicap (RR 2.5). When hyponatraemia occurs there is need to correct it slowly by not more than 12mmol/l per 24 hours. The evidence suggests that the neurological complication of pontine and extrapontine myelinolysis is a consequence of the rapid correction of hyponatraemia rather than hyponatraemia itself.[6]

The points being raised are also relevant for adults. A study of post-operative adults showed that those receiving 0.18% saline at 3 litres per 24 hours had a median fall in serum sodium at 24 h and 48 h of 5.4 mmol/l and 7.1mmol/l respectively whereas the serum sodium did not fall in those receiving 0.9% saline

The two authors propose that that 0.9% saline with 5% Dextrose at reduced infusion rates (70% of maintenance) results in a lower frequency of hyponatraemia, seizures and adverse neurological sequelae.

These findings are thought provoking. Units should review their maintenance fluid practice, particularly if they are experiencing hyponatraemia.

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Editor

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