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Hyponatraemia after orthopaedic surgery

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Ignorance of the effects of hyponatraemia after surgery is widespread—and damaging

Iatrogenic injury is an unfortunate reversal of the physician's role. To cause the death or brain damage of a patient has to be the physician's worst transgression, particularly if the causes are well known, simple, and reversible. Each is true of acute postoperative hyponatraemia, but, despite repeated warnings, the condition remains common. According to a recent estimate based on prospective and retrospective studies, 20% of women who develop symptomatic hyponatraemia die or suffer serious brain damage, totalling 10 000-15 0000 cases every year in the United States and Western Europe.[1]

An elderly female friend of ours is a classic example. Some months ago she underwent a routine knee replacement operation. Before the operation her blood sodium concentration was 134 mmol/l—borderline hyponatraemia—attributable to her long term use of thiazide diuretics. After the operation she vomited frequently and received 6 litres of 5% dextrose saline over two days before passing into a coma. Her blood sodium concentration measured on the second day after surgery was 115 mmol/l, but electrolyte disturbance was disregarded by the orthopaedic doctors as a potential cause of coma until the medical team were called the next day. Sodium concentrations were restored to 134 mmol/l over five days, leaving our friend with mild—but permanent—cognitive impairment. The hospital concerned "apologises unreservedly" but confessed ignorance about the risks of hyponatraemia after joint replacement surgery.

Although the literature is full of similar examples, too many orthopaedic surgeons seem unaware of the dangers of hyponatraemia or its characteristic neurological symptoms. Perhaps the reason lies partly in the scatter of relevant publications: most of the articles are published in journals dedicated to neurology, urology, and acute care; only a handful of reports refer specifically to orthopaedic surgery[2–4]; and neither the Royal College of Surgeons nor the British Orthopaedic Association publishes guidelines. Many articles focus on tightly defined issues, such as the association between thiazide diuretics and hyponatraemia,[2] to the exclusion of a more general overview. As a result, four fundamental problems have arisen: clinicians fail to recognise patients at high risk of hyponatraemia; disregard the dangers of routine infusions of hypotonic fluids; confuse early symptoms of hyponatraemia with postoperative sequelae; and attribute the serious neurological symptoms of hyponatraemic encephalopathy to other conditions such as stroke.

Postoperative hyponatraemia is provoked by surgical stress, which causes a syndrome of inappropriate antidiuretic hormone in almost everyone, often promoting water retention for several days.[5 6] Women are more affected than men, as a result of their smaller fluid volume and other sex related hormonal factors.[5] Premenopausal women and children are prone to brain damage at sodium concentrations as high as 128 mmol/l. Postmenopausal women do not usually become symptomatic until sodium concentrations have fallen below 120 mmol/l, although normal symptoms can occur at higher levels if the rate of change is rapid.[5–7] Importantly, normal ageing impairs fluid homoeostasis and therefore increases the risk of major perturbations in sodium and water balance, especially severe hyponatraemia.[7] The risk of hyponatraemia among elderly people is compounded by chronic diseases and long term medications. In particular, many women requiring orthopaedic surgery also take thiazide diuretics to control hypertension.[2] Thiazides are well known to induce mild hyponatraemia and have been linked to the rapid onset of serious postoperative complications.[2 3]

Women at risk of hyponatraemia are imperilled by routine infusions of isotonic dextrose. Patients recovering from surgery metabolise glucose almost immediately, so "isotonic" dextrose infusions are in effect hypotonic. Since the 1950s numerous reports have linked hypotonic infusions with death or permanent brain damage in postoperative patients.[1] Recent authoritative reviews warn against routine infusions of dextrose,[1 6 8] even stating explicitly: "the rationale for using hypotonic fluids in postoperative patients is difficult to discern and has no place in the modern practice of medicine."[1] Volumes as low as 3-4 litres over two days may cause convulsions, respiratory arrest, permanent brain damage, and death in women who were healthy before admission.[5 8] Most of these cases go unrecognised and are ascribed to conditions such as stroke, arteriovenous malformation, subarachnoid haemorrhage, or herpes encephalopathy, even when blood sodium concentrations are known.[8]

Early symptoms of hyponatraemia (such as weakness, nausea, vomiting, and headache) can be distinguished from postoperative sequelae on the basis of sodium concentrations. Timing also helps discrimination: many patients tolerate surgery without

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complications, being able to talk, walk, and eat before symptoms of hyponatraemic encephalopathy develop.[1] Treatment is simple and should be prompt: the risk of not treating acute cerebral oedema far exceeds the small risk of osmotic demyelination from treatment.[1 6] Fluid infusions should be restricted to normal or hypertonic saline and sodium concentrations monitored every two hours.[1 5 6] The aim is to raise serum sodium by 1-2 mmol/l per hour (depending on the severity of neurological symptoms) until symptoms resolve.[1 6] A loop diuretic such as frusemide (furosemide) may be used to enhance free water excretion and hasten the restoration of normal sodium concentrations.[1 6] Iatrogenic hyponatraemia is inexcusable. It is time that doctors woke up to the risks.

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Dietary management of hepatic encephalopathy

Too many myths persist

Myths are difficult to dispel and may delay good evidence based clinical practice. This is illustrated well by a paper in this week's issue on the dietary management of hepatic encephalopathy in patients with cirrhosis (p 1391).[1] Protein restriction in symptomatic patients with hepatic encephalopathy has been the cornerstone of treatment since the 1950s,[2] yet there is no evidence that it has any clinical benefit.

Hepatic encephalopathy is a syndrome of impaired mental status and abnormal neuromuscular function which results from major failure of liver function. Important factors contributing to it are the degree of hepatocellular failure, portosystemic shunting, and exogenous factors such as sepsis and variceal bleeding.[3] The pathogenesis of the syndrome is still uncertain, although current hypotheses include impaired hepatic detoxification of ammonia absorbed from the gut[4] and an increase in aromatic amines, which are precursors for false transmitters in the brain—for example, octopamine—and which alter the balance between neuronal excitation and neuronal inhibition.[5] Furthermore, increased expression of benzodiazepine receptors in hepatocellular failure suggests that the γ-aminobutyric acid-benzodiazepine inhibitory neurotransmitter system may be implicated in the development of hepatic encephalopathy.[6]

Protein restriction as a treatment conveniently began with 20 g protein/day and, with clinical recovery, 10 g increments were introduced every 3-5 days, as tolerated by the patient, to a limit of 0.8-1.0 g/kg body weight[3]; this was considered sufficient to achieve a positive nitrogen balance. This practice continues despite evidence showing that patients with stable cirrhosis have a higher protein requirement than normal, around 1.2 g/kg dry body weight to remain in positive balance.[7]

Protein energy malnutrition, defined by anthropometric criteria, may occur in 20-60% of patients with cirrhosis depending on the severity of the liver disease.[8] It is a common finding, with causative factors which include anorexia, nausea, malabsorption, and a hypermetabolic state. Intake may be further reduced by use of unpalatable low protein diets, already restricted in sodium and fluid.

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In 1997 the European Society for Parenteral and Enteral Nutrition published consensus guidelines recommending that the daily protein intake in patients with liver disease should, if possible, be around 1.0-1.5 g/kg depending on the degree of hepatic decompensation.[7] The guidelines also recommended that in patients who were intolerant of dietary protein 0.5 g protein/kg should be used transiently and that the remainder of their requirements should be achieved by giving branched chain amino acids.[9] However, not all studies agree on the use of branched chain amino acids.[10] Furthermore, aggressive enteral nutritional support of patients with alcoholic liver disease accelerates improvement without exacerbating hepatic encephalopathy.[11] Taking smaller meals more often and eating a late evening meal also improve nitrogen balance without exacerbating hepatic encephalopathy.[12] This may also be achieved with vegetable protein as opposed to animal proteins.[13]

The dilemma for the clinician arises in patients with acute hepatic encephalopathy, where increasing protein intake may worsen the condition in 35% of patients.[4] Use of branched chain amino acids may improve nitrogen balance but without producing any clinical improvement in the encephalopathy.[9] However, there is no consensus about the rate at which dietary protein should be reintroduced and at what clinical stage this is appropriate—the key points for the clinician.

Soulsby and Morgan provide recent evidence of perpetuation of the myth of protein restriction in patients with encephalopathy and, perhaps more alarmingly, that this therapy is used in patients with cirrhosis who have no neuropsychiatric impairment.[1] We

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