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CORRESPONDENCE

Re: A study of predictors for hyponatraemia in patients with cervical spinal cord injury

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Song et al. [1] have addressed the problems of hyponatraemia in patients with cervical spinal cord injuries. They admit to limitations in the study; it was a retrospective study and they did not measure the oral intake of fluid.

They suggested that the impairment of the autonomic nervous system and inappropriate secretion of antidiuretic hormone after cervical spinal cord injury might lead to an increased loss of serum sodium. They quote Nakao et al. who found that patient's daily urine volume was associated with serum sodium, and as their urine volume increased serum the sodium levels decreased [2]. They suggested that this could have resulted from the function of sodium reabsorption being damaged after the cervical spinal cord injury and this damage might have led to increased wasting of sodium and water.

There has been interest in the regulation of salt and water balance in patients who have sustained a spinal injury since 1915 when Gordon Holmes drew the attention of Harvey Cushing to the oliguria experienced by soldiers in the First World War [3]. Harvey Cushing attributed it to a derangement of the adrenal cortex.

I have been interested in this problem of water and sodium handling since 1956 and began to subject patients with acute traumatic spinal injuries to water loading tests in which I showed that there was avid water retention in the acute stage of injury [4]. I pursued my studies for 3 years at Stoke Mandeville Hospital and demonstrated in the first instance that the creatinine clearance in the early stage after spinal injury was normal, that is the level of filtration rate was not affected by the level of cord transection, thus the glomerulo-filtration rate was independent of the nerve supply to the kidney [5]. The much more difficult problem of investigating salt and water handling entailed salt loading by two different means, infusion and tablets [6].

A total of 53 studies were carried out on 52 patients between 1962 and 1964. In all, 49 patients were studied during the first 10 days after injury: a control group of 39 patients received no supplementary sodium, two received NaCl infusion, two NaCl tablets orally, and six received NaCl infusion followed by NaCl tablets orally. Four patients were studied for more than 15 days after injury (one of these had been studied in the acute stage); they received NaCl infusion and one received in addition NaCl tablets for four subsequent days. The results showed that the patients with high cord transection did have problems with water and salt handling.

In all patients, urinary sodium excretion was minimal on day 2 and increased thereafter. On days 2–6, it was significantly lower in patients with a complete transection of the cervical cord than in patients with lower lesions. In the early studies, nine patients excreted less than 40% of the administered load within 24 h. In four of these patients, excretion was 10% or less. In the later studies, three of the four patients excreted at least 80% of the infused Na+ on the same day.

In contradiction to the suggestion of Song et al., there clearly was sodium retention, not sodium wasting. It is not due to changes in the innervation of the kidney but to compensation for sympathetic insufficiency, blood pressure being maintained by the secretion of aldosterone, vasopressin, and other hormones. The decreased urinary output seen acutely after cord transection is not due to renal failure and the patient's condition can be made dangerously worse by attempts to create a diuresis.

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