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LETTER TO THE EDITOR

JH Frisbie Salt wasting, hypotension, polydipsia, and hyponatremia and the level of spinal cord injury

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Physicians have been concerned with the regulation of salt and water handling by spinal injury patients since 1917 when Gordon Holmes drew the attention of Harvey Cushing to the oliguria experienced by soldiers in the First World War. Harvey Cushing attributed it to a derangement of the adrenal cortex.

Since that time, it has attracted much attention and research. Dr Frisbie has contributed an idiosyncratic paper in an attempt to elucidate the problem. There are many queries about his methodology. He draws attention to the level of spinal cord injury in the title and postulates that the nerve supply of the kidney is significant in regulating salt and water balance. Unfortunately, in his clinical data, he presents a method of classification of the patients 1–25 that does not follow accepted terminology. He groups together patients A and B, thus grouping complete lesions, with incomplete lesions, which makes it impossible to determine those patients who have a denervated kidney. Furthermore, he goes from 1 to 25. Were there any patients with sacral lesions, that is, with Cauda Equina lesions? At the very least they could have served as controls!

The study relies substantially upon the collection of a single 24-h collection of urine by the patients prior to coming to the hospital. Such a collection of urine is notoriously inaccurate and is one of the most difficult estimations to make. It has been abandoned by many investigators and even under hospital conditions.

There is no information at all about the patients' renal condition. Did they have catheters to collect the urine? Did they have persistent infection? If they had catheters, was the bladder expressed? What was the status of the upper renal tract? Did they have pyelonephritis? The endogenous creatinine clearance is substantially normal in a paraplegic patient.[1] It is fascinating that the one piece of information that he does rely on is the creatinine clearance and he states in Table 1 that this varied from 8 to 263 $cm^3\,min^{-1}$.

In Figure 5, there are approximately 10 patients with a creatinine clearance of less than 50 $ml\,min^{-1}$, and 2 of these are below 8 $ml\,min^{-1}$. Why is this expressed in the table as $cm^3\,min^{-1}$ and in the figure as $ml\,min^{-1}$? If the collection is accurate and the figure is to be believed, then surely these patients warrant further investigation to determine if they were suffering from chronic pyelonephritis or renal failure, a likely cause of impaired sodium handling in paraplegic patients. In any event, they should be excluded from the study of the nerve supply, as inclusion of these patients could not give information on the effect of nerve supply to the kidney.

Excretion of salt by a normal kidney is notoriously labile. Exposure to a very hot climate and the state of hydration of the patient will dramatically alter the salt excretion, which can be reduced almost to zero under extreme climatic conditions. The difficulties in monitoring sodium metabolism have been recognised over the years. There is a well-documented way of studying salt that has to be adhered to if the results are to be evaluated. In order to demonstrate if there are changes, patients have to receive a controlled intake of sodium. This is a standard procedure that has to be done in hospital under controlled conditions in order for the patients to start off at a stable base line. They receive a controlled sodium diet of 20 mmoles daily plus a NaCl tablet 196 $mmol\,day^{-1}$.[2] This had to be continued for many days in order to reach a state of equilibrium. Frisbie recognized this in his paper in 1997,[3] by loading some of his patients with salt.

He states in his discussion.

In addition impaired sodium conservation has been found at the higher levels of paralysis. This association could not be explained by sodium intake which is not related to the level of paralysis. How does he know this? The patients were out patients, he has no means of knowing what diet they were on before they came to hospital and how they collected this 24 h specimen. This statement is speculative.

I have been interested in this problem of water and sodium handling since 1956 and began to subject the patients 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 on a full-time research grant 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.[1] The much more difficult problem of salt and water handling entailed salt loading by two different means, infusion and tablets, and unfortunately the studies were incomplete. I could not publish them and as I subsequently carried out full-time clinical work I had no opportunity to fill in the gaps but I knew that there was a problem.

I am well aware of the problem that Dr Frisbie alludes to and published the paper with many reservations in 2004.[2] It did appear that the patients with high cord transaction did have problems with water and salt handling but the mechanism was not elucidated. Unfortunately, such studies could not be repeated today for demographic and ethical reasons. To collect together such patients so soon after injury would be impossible and there would be reservations in carrying out such loading experiments.

I do agree that the problem is important and when I carried out a survey of spinal units I found that they were admitting at least one patient with a cervical cord transection every 2–3 months with pulmonary oedema caused by over transfusion at the receiving hospital. It was still occurring in 2001.[2]

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