Spinal Cord (2008) 46, 164

www.nature.com/sc

LETTER TO THE EDITOR

Reply to Dr JR Silver's letter

Spinal Cord (2008) **46,** 164; doi:10.1038/sj.sc.3102143; published online 11 December 2007

A numbered level of paralysis facilitates calculations, as noted in the text, and has the advantage of more precise statistical analysis. ASIA A and B patients were grouped to retain sufficient numbers for statistical analysis, testing the effect of the level of paralysis on renal function. There were no sacral lesions (Table 1).

The concern that creatinine clearance is unreliable can be answered as follows:

24-h urine collections have been managed by the patients in this survey for many years with instructions from the nursing staff in preparation for annual examinations. It is believed that with this experience, the accuracy is quite high. Data about urinary drainage were not collected. The type of urinary drainage may ultimately affect renal function, but it cannot be a reliable indicator of renal function in the individual patient. A direct measure of renal function in the creatinine clearance is more cogent.

Undoubtedly, many of the patients had chronic pyelonephritis. To accept, for the purposes of the argument, that all patients with a creatinine clearance of less than $50\,cm^3\,min^{-1}$ had salt wasting due to pyelonephritis, there were 12 such patients in the cohort reported. Three of them were paralyzed higher than the median level, T4, and nine were paralyzed at lower levels, $P = 0.13$. Salt wasting due to pyelonephritis, if it occurred, was not overly represented at the higher levels of paralysis where salt wasting was more common. Had these patients been removed from analysis, the correlation of salt wasting with higher levels of paralysis would have been greater. It is suspected that both neurogenic and nephrogenic salt wasting occurs in these patients with the former developing at the higher levels of paralysis.

The statement that creatinine clearance is normal in paraplegics applies to acute injury as referenced,[1] not to the chronic paraplegic patients reported.

There was no greater sodium output at the higher levels than at the lower ones (Table 2), although there was greater sodium output at the higher levels relative to creatinine clearance. The kidney was not conserving sodium as expected to compensate for a reduced blood pressure and renal blood flow at the higher levels of paralysis.

I would like to add that the attention that Dr Silver has brought to his experience in acute spinal cord injury presents an interesting contrast to the current study in chronic spinal cord injury. Acutely paralyzed tetraplegic subjects (1–5 days post-injury) retain both water and salt,[2,3] while chronically paralyzed tetraplegic subjects retain water but lose salt.[4] An explanation is conceivable. Although both acute and chronic tetraplegic subjects are stimulated to retain salt and water because of hypotension, the chronic tetraplegic patient has lost ability to respond to the stimulus to retain salt. It is suggested that the sympathetic innervation to the renal tubules permitting sodium retention has been impaired in the chronic tetraplegic. This impairment may manifest some time after the first few days of paralysis when chemical transmitters in the centrally denervated sympathetic neurons are exhausted.[5]

JH Frisbie
Spinal Cord Injury Service (128), Boston Healthcare Center, West Roxbury, MA, USA

References

1 Doggart JR, Guttmann L, Silver JR. Comparative studies on endogenous creatinine and urea clearances in paraplegics and tetraplegics. *Paraplegia* 1966; **3**: 229–242.
2 Silver JR, Doggart JR. Reduced sodium output following acute spinal injury. *Spinal Cord* 2004; **42**: 191–198.
3 Silver JR, Doggart JR, Burr RG. The reduced urinary output after spinal cord injury—a review. *Paraplegia* 1995; **33**: 721–725.
4 Frisbie JH. Salt wasting, hypotension, polydipsia, and hyponatremia and the level of spinal cord injury. *Spinal Cord* 2007; **45**: 563–568.
5 Jacobsen EB, Fristad I, Heyeraas KJ. Nerve fibers immunoreactive to calcitonin gene-related peptide, substance P, neuropeptide Y, and dopamine beta-hydroxylase in innervated and denervated oral tissues in ferrets. *Acta Odontol Scand* 1998; **56**: 220–228.