



## Letters to the Editor

### What is the optimum fluid intake in male patients with spinal cord injury and neuropathic bladder?

During the seventies and mid-eighties, male patients with spinal cord injury and neuropathic bladder often underwent division of external urethral sphincter and sometimes, bladder neck resection as well,<sup>1</sup> so that they were established successfully on penile sheath urinary drainage. These patients were advised to drink plenty of fluids 'to flush the kidneys' and 'to prevent urinary infection'. Many patients continue to follow this advice, and are happy with the outcome.

During the past 10–15 years, intermittent catheterisation has been recommended as the ideal method of managing the neuropathic bladder. Spinal cord injury patients who choose to manage their neuropathic bladder this way are advised to 'restrict' their fluid intake. The proponents of this regime claim that by restricting the fluid intake and thereby reducing the total urine output to say 1.5 litres in 24 h, a person will be able to manage their bladder by performing catheterisation four to five times a day, and remain continent. If they drink more fluids, they may have to perform more frequent catheterisations, or they may leak urine between catheterisations. This is likely to interfere with their social activities. These arguments appear reasonable and many persons with spinal cord injury follow this advice by restricting the fluid intake to about 2000 ml a day or even less.

Spinal cord injury patients who are performing intermittent catheterisation are often prescribed oxybutynin by mouth or intravesically<sup>2</sup> so that they can avoid urine leaks between catheterisations. When these patients complain of dry mouth which is a side effect of oxybutynin, artificial saliva preparations are prescribed (eg Luborant oral spray) and they are advised to avoid drinking more fluids to quench their dry mouth.

Another reason put forward against drinking plenty of water is that a tetraplegic subject may become more susceptible to hyponatremia if water intake is high.<sup>3</sup>

Now, Michaud and associates<sup>4</sup> demonstrated in a prospective study of 47 909 men, that a high intake of fluids was associated with a reduced risk of bladder cancer in men after control of potential risk factors. The multivariate relative risk of bladder cancer was 0.51 (95% confidence interval: 0.32–0.80) in persons consuming total fluid intake of >2531 ml/day in contrast to those with a total daily intake of <1290 ml/day ( $P=0.004$ ). The daily consumption of 1440 ml (six or more cups) of water was associated with a substantial reduction (51%) in the risk of bladder cancer as compared with the risk among participants who consumed less than 240 ml (1 cup) per day). When fluid intake was modelled as a continuous variable, the risk of bladder cancer decreased by 7% for every increment of 240 ml in daily fluid intake. Thus a high intake of water may reduce the risk of bladder cancer by about 50% on average. These authors concluded that a generous intake of fluid is sensible, because it can also reduce the risk of kidney stones.

Patients with spinal cord injury are at increased risk for developing urinary infection, kidney stones, and bladder

cancer.<sup>5</sup> Should we continue to advise patients with spinal cord injury to restrict their fluid intake? The arguments against extrapolating the findings of Michaud to the spinal cord injury patients are:

- (1) More than 90% of the cases of bladder cancer in this study were transitional cell carcinoma. But, among spinal cord injury patients who developed bladder cancer and in whom adequate data were available regarding tumor pathologic findings, transitional cell carcinoma was seen in only 55%, and squamous cell carcinoma accounted for 33% of cases.<sup>6</sup>
- (2) The results of the study reported by Michaud and associates are consistent with the urogenous-contact hypothesis which associates the development of bladder cancer with prolonged exposure to carcinogens in urine.<sup>7</sup> But in spinal cord injury patients, other mechanisms may be operating for the pathogenesis of bladder cancer. For example, local irritation caused by long-term indwelling urinary catheter or bladder stones may be an important predisposing factor for bladder cancer.<sup>5</sup>

A prospective study of fluid intake and occurrence of urinary infection, urinary stones, and bladder cancer is warranted in spinal cord injury patients with neuropathic bladder. Until we get results of this prospective study, should we adopt a seemingly simple way to reduce the risk of bladder cancer: drink more fluids?

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### The applied neuropathology of human spinal cord injury

I have read with great interest the recent article of Kakulas.<sup>1</sup> I agree with his notion that ‘it is now accepted that a central pattern generator in the lumbar cord exists and becomes isolated following injury’, although his reference to this statement is rather questionable as it is concluded in this editorial comment<sup>2</sup> that the aforementioned ‘...studies do not absolutely prove the existence of a central pattern generator in man...’.

I disagree, however, with the notion within the same paragraph of the review<sup>1</sup> that ‘changing the synaptic inputs from above leads to disinhibition of motor neurons ... and that, ‘paroxysmal reflex contractions ... are the origin of spasms’. As shown in several studies<sup>3,4</sup> (for review see<sup>5</sup>), there is no increase, but rather a decrease in activity of motor neuron activity during spinal locomotion in man, i.e. in complete paraplegic patients. Furthermore, there is a dissociation between flexor reflex activity and muscle spasms when followed from spinal shock to spasticity. While frequency and severity of spasms increase, flexor reflex amplitude decreases over weeks after a spinal cord injury (unpublished observations). Obviously, other factors, such as secondary changes of mechanical muscle fibre properties (for reviews, see<sup>6,7</sup>) contribute to muscle spasms.

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### In reply to Professor Dietz

I have now read Professor Dietz’s letter with interest. His expansion of the points made in my review are quite welcome and I do not have any special comment to make apart from this.

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