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Letters to the Editor

Autonomic dysreflexia during urodynamics.
A Giannantoni *et al. Spinal Cord* 1998; 36: 756–760

This paper is of great interest but unfortunately the way the results are presented gives rise to problems that need resolving.

In Patients and Methods they say 'Twenty-three patients had a lesion at the cervical level, the other 25 at the dorsal (above T6). Yet in Table 1, 24 patients are said to have cervical lesions and 24 to have thoracic lesions.

Table 1 states that disease duration in Group A (without autonomic dysreflexia) was 41.9 ± 66, and in Group B 57.8 ± 75.6. It would have been more meaningful to have given the medians and ranges. The data is probably highly skewed, that is to say, durations less than 41.9 are widely spread out.

Again, they say there is no difference in the disease duration between the two groups (Table 1). Statistically, there is no difference, because the scatter of the data is so wide that overlap is considerable but it would seem that the ones who developed autonomic dysreflexia (Group B) developed it later on, at 57.8 as against 41.9 months (Group A). This is what one would expect clinically.

What the data does show and which the authors state but not in the Conclusion is that the cervical patients had a bigger increase in blood pressure as well as a higher incident of AD than the thoracic patients which is unremarkable.

In the section Patients and Methods, I do not understand what they mean by: 'We decided to respect the usual conditions of filling and voiding of the patients in real life (for example in patients performing clean intermittent catheterization we did not overcome the volumes usually achieved with catheterization).' This is incomprehensible.

The most interesting observation that they make is that three patients were examined while in the spinal shock phase, two of whom had a history of autonomic dysreflexia. We need to know what were their criteria for spinal shock. They do not specify how soon after the injury this occurred and whether their tendon reflexes were present.

They again refer to it on p 759 (2nd column, paragraph 1): 'this could also explain the finding of symptoms and signs of autonomic dysreflexia in patients with detrusor areflexia due to spinal shock. In these patients autonomic dysreflexia could be the first sign of spinal shock recovery.' They just say that they had detrusor areflexia due to spinal shock. There are, of course, other causes of areflexia, i.e. over distension.

They are postulating that autonomic dysreflexia can occur during the stage of spinal shock. This is against the conventional wisdom which postulates that in the stage of spinal shock autonomic activity is abolished.

I, too, have made previous observations showing that autonomic activity is present in the state of spinal shock and in 1971[1] I made observations on the blood pressure, forearm and hand blood flows and heart rate on 15 tetraplegic patients in the stage of spinal shock. I found that the autonomic reflex of inspiratory vasoconstriction was preserved and one patient at 7 days after spinal injury showed autonomic hyperreflexia when 1600 ml of urine was retained inadvertently in his bladder. Subsequent investigations by cystometry to 350 ml on that patient and three others while in a state of spinal shock showed a small but significant increase in blood pressure which rose up to 20 mmHg systolic which is in keeping with Giannantoni *et al*'s findings of a graduated response.

Matthias *et al*[2] looked at the problem in 1979 studying five tetraplegic patients in the stage of spinal shock. They stimulated the bladder by percussion and found a small rise in blood pressure.

Since my original observations in 1971 I have found two further patients showing early autonomic dysreflexia and two additional cases in the literature (Head and Riddoch[3]). Taken in their paper, it would seem that there is well documented evidence now of autonomic activity in the stage of spinal shock.

The cases that I found and those described by Head and Riddoch[3] occurred when the bladder activity was heightened because of trauma to the urethra or retention of large volumes of urine since in the stage of spinal shock the bladder is atonic and can fill to a much larger capacity than normal and without any detrusor contractions occurring.

At a later stage both the bladder capacity becomes smaller and detrusor contractions occur. Detrusor contractions are a potent stimulus for autonomic dysreflexia. No doubt the contraction of the bladder and the pulling open of the bladder neck causes stimuli of the receptors.

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In reply to Dr J Silver

Autonomic dysreflexia during urodynamics. A Giannantoni *et al. Spinal Cord*, 1998; 36: 756–760

We do appreciate the comments of Dr Silver about our paper.

With regard to the first point raised by Dr Silver, the correct distribution of the patients is 23 with cervical lesion and 25 with thoracic one. We regret the typing error in Table 1.

We agree that the lack of significant difference in the disease duration is probably due to the wide scatter of the data and that generally speaking patients who developed autonomic dysreflexia developed it later on; nevertheless, this is not always true. In our series there were three patients who developed autonomic dysreflexia soon after the lesion, while still in the spinal shock phase.

The finding of great severity of autonomic dysreflexia in the cervical patients is not new[1] and has only been confirmed by our data; in our opinion this was not one of the most important results and that is why we did not state it in the conclusions.

In describing bladder capacity during cystometry the ICS has recommended the following terms[2]: 'maximum cystometric capacity (the volume at which the patients feel that they can no longer delay micturition) is difficult to define if the patient's sensation is absent or reduced. In deciding how far to fill the bladder in these conditions the urodynamicist should be guided by evidence of the functional bladder capacity from the frequency-volume chart'. Thus, as with other authors[3], we stopped filling when a detrusor contraction occurred or at volumes normally achieved with catheterization, which better reproduce the usual pattern in daily living.

With reference to the neurological features of the patients in the shock phase, we did not give any particular information because they all satisfied the criteria for spinal shock: patients showed a picture of flaccidity with absence of the tendon reflexes and of the bulbocavernosus reflex[3]. These three patients have been evaluated at a mean time from trauma of 3.3 months (minimum 2, maximum 5); two of the three have been re-evaulated 2 months later and again showed detrusor areflexia and the absence of the bulbocavernosus reflex. We are satisfied that in these patients detrusor areflexia was not linked to bladder overdistension because none of the patients had a history of acute urinary retention. As we stated in the discussion, the presence of autonomic dysreflexia in the shock phase may indicate a partial preservation of the autonomic system or may be the first sign that the shock phase is resolving. Following these data we would like to suggest that one of the most remarkable aspects in the evaluation of the patients in the shock phase is the urodynamic evaluation.

Dr Silver highlighted pioneer works in this field in agreement with our findings not reported in our paper because of brevity and because an exhaustive review of the literature was beyond the purposes of our work.

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