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Letter to the Editor

Spinal shock revisited: a four-phase model

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I enjoyed reading the meticulous and thoughtful review entitled 'Spinal shock revisited: a four-phase model' by JF Ditunno, JW Little, A Tessler and AS Burns[1]

The authors were kind enough to quote my own study,[2] suggesting that:

> One case report suggested that autonomic disreflexia may occur as early as 7 days after a complete injury, but the general time course of its development requires further study.

This is correct but in fact, I observed two cases of autonomic disreflexia, both in response to bladder distension from a blocked catheter, one after 7 days and one after 11 days I also recorded two further cases in the literature appearing at 22 days and 31 days for traumatic catheterisation. I believed that there was autonomic activity occurring at a very early stage. I carried out a further study by carrying out a cystometrogram in the first patient. Systolic blood pressure increased by 20% but no symptoms were precipitated. Further cystometrograms on three other patients in spinal shock showed small increases in pressure but it clearly would have been unethical to carry out the cystometrogram beyond the stage of 350 ml filling.[3]

Mathias *et al*[4] carried out similar investigations. They relied on the bladder being distended by natural filling and the stimulus was percussion of the bladder. The mean change in blood pressure was from 130/58 to 138/60 mmHg.

I conclude that there is a reduction rather than abolition of spinal autonomic activity in the state of spinal shock. This was supported by the observations of Rossier *et al*.[5] They also carried out studies in patients in spinal shock and found that bladder filling was accompanied by elevation of resistance in the bladder neck due to increased sympathetic activity in the smooth muscle component of the entire urethra. This pressure decreased after the administration of an alpha-adrenergic blocking agent in two patients. This was at a time when the striped muscle component was in abeyance.

In the state of spinal shock, the autonomic activation of the bladder by the parasympathetic nerves is abolished. The bladder is atonic[5–7] and can fill to a much larger capacity without any detrusor contractions occurring. Destruction of the pontine micturition centre or interruption of the neuraxis below the pons by spinal cord transection causes the immediate elimination of the micturition reflex and the slow development of involuntary, uncoordinated, spinal mechanisms that mediate autonomic voiding in paraplegic patients and animals.

The sympathetic innervation of the bladder is not considered of prime importance in the motor side of the voiding. However, there are sympathetic receptors at the bladder neck whose distribution is significant and serve as receptors for autonomic dysreflexia from bladder filling. The act of normal micturition is initiated by cortical control. The brain receives afferent signals from the bladder and urethra through the posterior columns, spinothalamic and spinocerebellar tracts and descending motor pathways travelling in the corticospinal and reticulospinal tracts.

In all the patients whom I studied, the stimulus was clearly supramaximal and was not produced as an experimental situation.

A distinction must be made between physiological filling of the bladder by urine, which is slow and a speedy infusion when the bladder is filled during a cystometrogram. These will produce different effects. The slow filling takes much longer to elucidate reflex contractions whereas the rapid filling during a cystometrogram produces reflex contractions at a much smaller volume.

I agree that this is clearly a subject that requires further study.

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