Mechanisms of Urinary Incontinence in Peripheral Neurogenic Lesions

Patients with urinary incontinence due to peripheral denervation present a difficult therapeutic problem. Treatment of incontinence itself is generally considered impracticable, and various methods of urinary diversion are usually used in order to preserve renal function.

The results of our work suggest that in many of these patients denervation is incomplete. Electrophysiological studies of the closing muscles in such cases demonstrate the preservation of voluntary control over a varying number of motor units. As a rule, however, these units are not easily available to voluntary activation and are recruited at low innervation frequencies even at maximum effort. Furthermore, the activity can usually only be maintained for limited periods of time. The innervation frequency could always be substantially increased by stimulation of various receptors or afferent nerve fibres which occasionally resulted in effective closure of the bladder. These effects were also obtainable in cases of peripheral neurogenic incontinence (e.g. due to spina bifida) associated with complete sensory loss in lower sacral segments where patients were unaware of their remaining voluntary control.

It is suggested therefore that in at least some of the patients with urinary incontinence due to involvement of peripheral innervation the disability results in part from defective conscious and reflex sensory feedback and insufficient supraspinal control. The latter seems to be a particularly vulnerable function being a relatively recent phylegenetic achievement. The findings of this study suggest that it can be significantly supported by externally controlled and appropriately programmed afferent inflow.