Lack of Relationship of Conus Reflexes to Bladder Function after Spinal Cord Injury

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Summary—A series of 20 patients with acute complete suprasacral cord lesions underwent serial urodynamic assessment of vesicourethral function and serial measurement of sacral reflex latency times (SRL) and reflex threshold throughout a follow-up period of 42 to 83 weeks (mean 50).

No correlation was found between any pattern of SRL latencies or reflex thresholds and subsequent bladder behaviour. The reproducibility of sacral reflex latencies was found to be poor (mean variation of serial measurements from initial reading 21%) and could not be explained on the basis of "dynamic" neurological recovery. Studies using the bladder as a stimulus site were unreliable. The value of SRL studies in detecting subtle neurophysiological changes is discussed.

Following a traumatic injury to the spinal cord we expect to observe a pattern of bladder activity which depends on the level of the neurological lesion (Head and Riddoch, 1917). During the phase of "spinal shock" we repeatedly examine the patient for evidence of return of both the anal skin reflex and the glans bulbar reflex, or the "conus reflexes". When they are present we predict with some confidence that reflex bladder activity will subsequently recover.

This observation implies the existence of a "sacral micturition reflex", analogous to the clinical conus reflexes, recovery of which is required in order for reflex bladder activity to occur. Such a simplified concept, however, fails to explain certain anomalies. Firstly, the speed of bladder recovery varies greatly from patient to patient, even when conus activity is the same (Arnold et al., 1984). The bladders of some patients with apparently normal conus reflexes never regain reflex activity. Secondly, in patients with similar patterns of neurological recovery and, in particular, similar conus reflexes, there are enormously varying degrees of detrusor–sphincter co-ordination, resulting on the one hand in an efficiently emptying reflex bladder and, on the other, a high pressure dyssynergic voiding system which is hazardous to upper tract function (Blaivas et al., 1981; Siroky and Krane, 1982; Wyndaele, 1985).

The aim of this project has been to examine the relationship between sacral afferent and reflex activity, when assessed by neurophysiological techniques, and the patterns of bladder and sphincter function which are seen following an acute spinal cord injury.

Patients and Methods

Twenty patients with acute complete suprasacral cord lesions, admitted over a 12-month period, were studied. They included 18 males and 2 females (age range 15–45 years, median 45). There were 9 cervical lesions and 11 injuries to the thoracic cord. Although all had complete motor and sensory loss at the time of admission, 3 patients with cervical fractures subsequently developed a degree of sensory sparing within the sacral root distribution.

Recovery phase

Immediately upon admission to the Spinal Injuries Unit, and subsequently during the recovery period (the period of immobilisation which usually lasts for approximately 6 to 10 weeks), patients were
regularly examined for evidence of motor or sensory recovery below the lesion. Simple 2-channel cystometry was performed every other week, to detect the approximate time when bladder contractions first occurred in response to bladder filling. Sacral reflex latency times and reflex thresholds (see below) were performed at least twice during this initial period.

Established phase
Once the bony lesion was stable and the patient was mobilised, video-urodynamic studies (see below) were performed for a detailed assessment of vesicourethral function and SRL studies were repeated, both at least twice during a follow-up period of 42 to 83 weeks (mean 50).

Sacral reflex latency studies
This technique is well documented (Siroky and Krane, 1982; Galloway et al., 1985). A number of theoretical neural pathways associated with micturition were assessed in this study. Electrical stimuli were applied, independently and in turn, to the dorsal nerve of the penis (bipolar surface electrode), the proximal urethra and the bladder mucosa (separate bipolar ring electrodes mounted on the shaft and tip of a 14F Foley catheter).

Electromyographic responses were recorded from either a concentric monopolar needle electrode (Disa 13L50), inserted into the superficial part of the external anal sphincter in women and into the bulbocavernous muscle in men, or the urethral ring electrode lying within the distal urethral sphincter mechanism which was reconnected to an input channel on the EMG pre-amplifier. Reflex latency times and reflex thresholds were measured for each combination of stimulation and recording site.

The equipment used for all neurophysiological tests was the Dantec Neuromatic 2000M (band width 20 Hz–2 kHz); 200 µs pulses were delivered at a rate of 3 Hz. Measurements were made from averaged responses, from trains of up to 100 stimuli applied at supramaximal level (3 x threshold level).

Videourodynamics
Combined filling and voiding cystometrograms, with simultaneous radiological screening and video recording, were used to assess vesicourethral function in detail, once the patient could be easily transferred to the X-ray department. Our usual technique for studying neurogenic bladder dysfunction was used (Thomas, 1979).

Vesicourethral function was assessed according to the recommendations of the International Continence Society. Bladders were recorded as being either hyper-reflexic, hypocompliant or acontractile in behaviour. Detrusor sphincter dyssynergia (DSD) was assessed radiologically and in relation to changes in bladder pressure and recorded as being either absent, early (lasting less than 30 s), prolonged (showing repetitive contractions of the sphincter unit in association with detrusor contraction), or terminal (occurring in the terminal phase of voiding, i.e. preventing complete bladder emptying). Bladder emptying was regarded as efficient when the residual urine was less than 100 ml in the absence of vesicoureteric reflex. Thus emptying was recorded as being good, bad or absent. When emptying was bad, this was assessed as being due to either DSD or poorly sustained detrusor contractions or both.

Results
Figure 1 shows how, in the majority of patients, clinically detectable conus reflexes reappeared within 8 days of the injury and 9 of these were recorded in the first 24 h. It is possible that these reflexes had never disappeared. Only 1 patient had electrically detectable reflexes before they were present clinically (35 weeks). All except 3 patients subsequently developed normal somatic reflexes although, even in these 3, the conus reflexes had returned (24 h, 35 days and 80 days).

Bladder function
The time taken for detrusor activity to recur varied from 4 to 37 weeks and showed no relationship with the level of the neurological lesion (Fig. 2). All but 3 patients eventually developed hyper-reflexic bladder activity. Nine patients had efficiently emptying hyper-reflexic bladders and 6 had hyper-reflexia
with poor emptying due to DSD; 3 had poor emptying due to poorly sustained detrusor contractions and 3 had persistently acontractile bladders after a minimum of 15 months from injury.

Sacral reflex latencies and thresholds

When recordable, the latency values were normal in 84 to 93% of recordings (Table 1). Normal values had been previously established for our technique from a group of 20 healthy volunteers and corresponded to all previously reported normal ranges. Reflexes were unrecordable in 5 to 16% of studies. Only 2 recordings in the entire series had a prolonged latency time. Thus there was little evidence from these data of any continuing sacral neuropathy once conus reflex activity had recurred. Reflexes resulting from stimulation of the bladder fundus were found to be unreliable.

An attempt was made to correlate neurophysiological findings with urodynamic findings. Both the eventual outcome for bladder and sphincter behaviour and the changes which had occurred during the follow-up period were correlated with the patient's age, neurological level of the injury, the time of appearance of clinical conus lesions and subsequent patterns of normal, abnormal or absent sacral reflex latencies and threshold recordings. No correlation was seen between any of these sets of parameters, other than to reaffirm that the appearance of clinical conus reflexes predicts bladder recovery reliably (85% of patients).

Although most latency values were within normal limits, serial recordings were found to vary from initial recordings, at the time of admission, by between 18 and 25% (Table 2). There was no temporal relationship of these latency values to the time elapsed since injury and thus these figures reflect the poor reproducibility of these tests and not any dynamic neurological recovery occurring over this period. The mean variations for reflex threshold were 88% for the dorsal nerve threshold and 97% for the posterior urethral threshold.

Discussion

Sacral reflex latency studies have been enthusiastically applied by urologists as an “accurate and sensitive” means of detecting occult sacral neuropathy (Siroky et al., 1979; Galloway et al., 1985; Fidas et al., 1987 a and b). However, doubt has been cast on their value in specific clinical situations (Paquin et al., 1985; Benvenuti et al., 1987; Desai et al., 1988). Since the incidence of abnormal reflexes in the normal population is unknown (no large study of normal controls has been reported), the significance of these abnormal responses is uncertain. It could represent, on the one hand, a variant of normal or, on the other, a localised anatomical or physiological disruption of reflex pathways within the sacral cord or abnormal modulation of sacral reflex activity from a supraspinal level. Even in spinal cord injured patients with apparently complete lesions, supraspinal influence on segmental reflex activity has been shown to occur (Cioni et al., 1985). Such modulation may be of fundamental importance in determining
patterns of reflex activity, but has not been investigated.

This study of 20 patients with acute spinal cord injuries has demonstrated that the tests are of little value in spinal cord injury since they reveal no more about the predicted recovery of either somatic or bladder reflex activity than does a simple clinical examination of the conus reflexes.

SRL studies almost certainly test the integrity of myelinated neural pathways and a common "pool" of neurones at cord level. Fowler (personal communication) pointed out that any reflex from the perineum to the spinal cord, and back, with a latency of less than 100 ms cannot possibly be conducted via autonomic nerves, which are all slow conducting fibres. Furthermore, the lack of reproducibility within the group who were studied serially, albeit recovering from neurological injury, raises doubt as to value of SRL tests in detecting subtle neurophysiological changes.

No correlation has yet been confirmed between any currently measurable neurophysiological parameter and patterns of bladder and sphincter behaviour following spinal cord injury. Mundy (1987) suggested that the recovery of bladder function is dependent not on the recovery of existing reflex pathways but on the establishment of new pathways through existing (but unused) synapses, possibly left vacant after loss of the long routed micturition reflexes (Torrens, 1985). Inevitably this would result in variable complexity of these newly established pathways and some may never develop at all. This could account for the various patterns of detrusor sphincter co-ordination and also explain why a number of patients with suprasacral lesions never regain bladder activity. Although there is no evidence for this hypothesis, in this light it would seem obvious that the integrity of a simple somatic reflex, such as is tested clinically with a conus reflex or neurophysiologically by SRL, has little relevance to the sort of complex pathways that may become established to permit reflex bladder action.

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