

Detrusor Hyperreflexia in Neurogenic Bladder Disorders Caused by Localized Partial Lesions of the Spinal Cord and Cauda Equina

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In 70 patients studied by cystometry, detrusor hyperreflexia seen with localized partial lesions of the spinal cord and cauda equina could be differentiated into two types. In one type, in which the hyperreflexia was of a reflex nature and bladder compliance was unchanged, the detrusor contractility was described as *compliant hyperreflexic*. In the other type, in which the contractions were of a rhythmic nature and accompanied by reduced compliance, the contractility of the detrusor was described as *noncompliant hyperreflexic*. The compliant hyperreflexic contractions correlated well with signs of upper motor neuron disturbance, the noncompliant hyperreflexic contractions with lower motor neuron disturbance.

KEY WORDS: Neurogenic bladder; Localized partial lesions; Spinal cord; Detrusor hyperreflexia; Neurological deficits

In a normal cystometrogram, in which intravesical pressure is recorded continuously during a constant rate infusion of fluid into the bladder, there is a limited but steady increase in detrusor pressure of not more than 15 cmH₂O on reaching maximum cystometric capacity. This phenomenon, which was previously described as *bladder tonus* or *accommodation*, is now measured in terms of detrusor compliance. Detrusor compliance, C , is defined as the change in detrusor volume, ΔV , for unit

change of detrusor pressure, ΔP , and is expressed as $C = \Delta V/\Delta P$ [1].

Sudden increases in detrusor pressure that occur during filling are described in terms of detrusor contractility. The normal detrusor contracts with an increase of detrusor pressure only during voiding; the contraction is voluntarily initiated and if necessary can be voluntarily suppressed. Any increase of detrusor pressure that cannot be voluntarily suppressed is defined as detrusor overactivity. Detrusor overactivity without any obvious neurological deficit is called *detrusor instability*, and detrusor overactivity in neurological disorders is called *detrusor hyperreflexia*. Whether detrusor instability is synonymous with detrusor hyperreflexia is not known at present [1]. Detrusor hyperreflexia can be either spontaneous or provoked by rapidly filling the bladder, altering posture, coughing, jumping, walking, or stimulating the skin of the perineum or hypogastrium. However, detrusor hyperreflexia as defined above does not refer to any particular site of the lesion or to any particular neurological deficit.

It was the purpose of this study to examine types of detrusor hyperreflexia in relation to the site of the lesion and in relation to the neurological manifestations in patients with localized, confirmed, partial lesions of the spinal cord or cauda equina with a view toward differentiating the various types of hyperreflexia that occur.

Materials and Methods

Methods, definitions, and units conform to the standards recommended by the International Continence Society, except where specifically stated otherwise [1].

Urodynamic studies were performed in 70 patients, 52 men and 18 women between 19 and 76 years of age, all of whom had localized lesions of the spinal cord or cauda equina demonstrated by myelography and confirmed at surgery. The studies were undertaken irre-

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spective of the presence or absence of symptoms of urinary disturbances.

Cystometry was carried out with the patient in the supine position by infusing normal saline at room temperature at a constant rate of 15 mL/min via a nylon periurethral catheter, 1.0 mm external diameter (Portex Ltd.), using a pump to generate the necessary pressure head. The intravesical pressure was recorded via a similar catheter lying alongside the first and connected to an external pressure transducer. Intraabdominal pressure was simultaneously recorded via a fluid-filled balloon catheter in the rectum, and the detrusor pressure was recorded by electronically subtracting the intraabdominal pressure from the intravesical pressure. The patient was requested to cough or change position from time to time to provoke detrusor hyperreflexia.

The patients were then classified as having cervical lesions, dorsal lesions, lesions of the conus medullaris, or lesions of the cauda equina. Those with lesions between D-10 and L-2 were classified as having lesions of the conus medullaris. There were 19 patients with cervical lesions, 15 with dorsal lesions, 23 with lesions of the conus medullaris, and 13 with lesions of the cauda equina. They were then reclassified in relation to their neurological manifestations. All 34 patients with cervical and dorsal lesions had pyramidal weakness of the limbs with brisk reflexes and extensor plantar responses, and the lesions were classified as pure upper motor neuron lesions. The 13 patients with lesions of the cauda equina had flaccid weakness of the limbs with diminished tone and reflexes and segmental sensory impairment, and the lesions were classified as pure lower motor neuron lesions. The 23 patients with lesions of the conus medullaris had mixed neurological features, but 2 had predominantly upper motor neuron signs and 21 predominantly lower motor neuron signs. Thus, 36 patients could be classified as having upper motor neuron signs and 34 as having lower motor neuron signs.

The type of detrusor hyperreflexia was then assessed in relation to the site of the lesion and correlated with the neurological manifestations of upper or lower motor neuron disturbance.

Results

Detrusor hyperreflexia was seen in 38 patients (54.3%); it was most common in patients with lesions of the conus medullaris (86.9%) and least common in those with lesions of the cauda equina (15.4%) (Table 1).

Two distinct types of hyperreflexia were seen. In one type, sudden increases in detrusor pressure occurred during filling. They ranged from 20 to 80 cmH₂O, lasted 2-3 seconds, and often occurred spontaneously but could

Table 1. Distribution of Patients With Detrusor Hyperreflexia By Site of Lesion

| | Cervical | Dorsal | Conus | Cauda equina | Total |
|------------------------|------------------------|--------|-----------|--------------|-----------|
| No. of patients | 19 | 15 | 23 | 13 | 70 |
| Detrusor hyperreflexia | 10 (52.6) ^a | 6 (40) | 20 (86.9) | 2 (15.4) | 38 (54.3) |

^aValues in parentheses indicate percentages.

be provoked by coughing, sitting up, or pressing on the lower abdomen (Figure 1). They were sometimes accompanied by a desire to void, sometimes by incontinence, and could not be suppressed by the patient. Detrusor pressure returned to the precontractile level at the end of each contraction, the increase in the baseline detrusor pressure being minimal. The baseline pressure remained less than 15 cmH₂O above the starting pressure throughout. As the compliance of the bladder was normal, this type of hyperreflexia was called *compliant hyperreflexia*.

In the second type of detrusor hyperreflexia there were rhythmic increases in detrusor pressure varying from 10 to 50 cmH₂O and lasting 3-5 seconds (Figure 2). At the end of each contraction the baseline pressure did not return to its precontractile level, resulting in an overall increase in the baseline pressure of 40 to 50 cmH₂O. As compliance of the bladder was reduced, this type of hyperreflexia was called *noncompliant hyperreflexia*. The noncompliant hyperreflexia was rhythmic and seemed to be independent of provoking maneuvers.

The detrusor hyperreflexia seen in cervical and dorsal lesions was only of the compliant hyperreflexic type, and that seen in lesions of the cauda equina was only of the noncompliant hyperreflexic type (Table 2). Of the two patients with lesions of the conus medullaris and predominantly upper motor neuron signs, one had a compliant hyperreflexic bladder and the other a noncompliant hyperreflexic bladder. Of the 21 patients with lesions of the conus medullaris and predominantly lower motor neuron disturbances, 14 had noncompliant hyperreflexic bladders and 4 had compliant hyperreflexic bladders. Thus, 17 of the 18 patients with detrusor hyperreflexia associated with upper motor neuron lesions had compliant hyperreflexia, and 16 of the 20 patients with detrusor hyperreflexia associated with lower motor neuron lesions had noncompliant hyperreflexia. These relationships between compliant hyperreflexia of the detrusor and signs of upper motor neuron involvement and between noncompliant hyperreflexia and signs of lower motor neuron involvement in localized lesions of the spinal cord and cauda equina are statistically highly significant ($p < 0.005$).

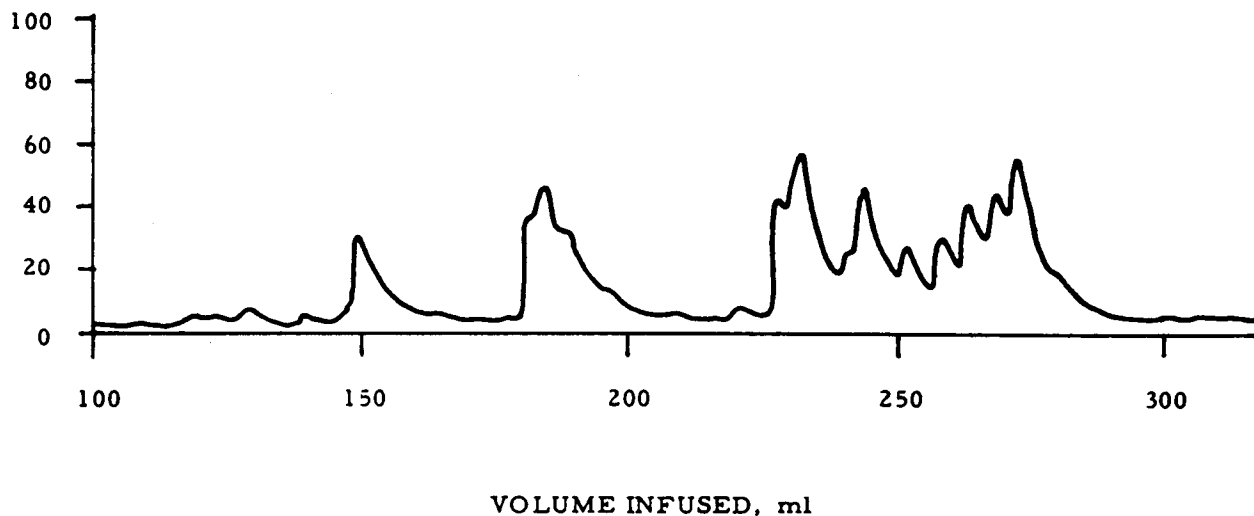


Figure 1. *Compliant hyperreflexia: Pattern of detrusor pressure during cystometry in a patient with a neurofibroma at the T-6 level showing hyperreflexic contractions of the detrusor but no alteration of the baseline pressure.*

Discussion

Detrusor hyperreflexia occurring in humans with lesions of the spinal cord has been well known since Head and Riddoch [5] described automatic reflex bladder function in such patients. Denny-Brown and Robertson [2] clearly demonstrated by cystometrography the reflex detrusor contractions occurring in patients with upper motor neuron lesions and also indicated the rhythmic nature of the contractions in patients with lesions of the cauda equina. The pathogenesis of these rhythmic contractions was thought to be myogenic by Sherrington [7], who demonstrated that they occurred in the isolated cat blad-

der. Elliot [3], however, showed that the rhythmic contractions could be abolished by dissection of the autonomic ganglia from the feline detrusor and suggested a neural basis for the contractions. Norlen [6] showed that spontaneous rhythmic contractions of the detrusor in patients with lower motor neuron disturbances could be abolished by α -adrenergic blocking agents such as phenoxybenzamine and suggested that these contractions result from the influence of the sympathetic nervous system on the α -adrenergic receptors in the parasympathetically denervated bladder.

Figure 2. *Noncompliant hyperreflexia: Pattern of detrusor pressure during cystometry in a patient with marked L-5-S-1 spondylolisthesis, showing rhythmic contractions of the detrusor with progressive increase of the baseline pressure, hence reduced compliance.*

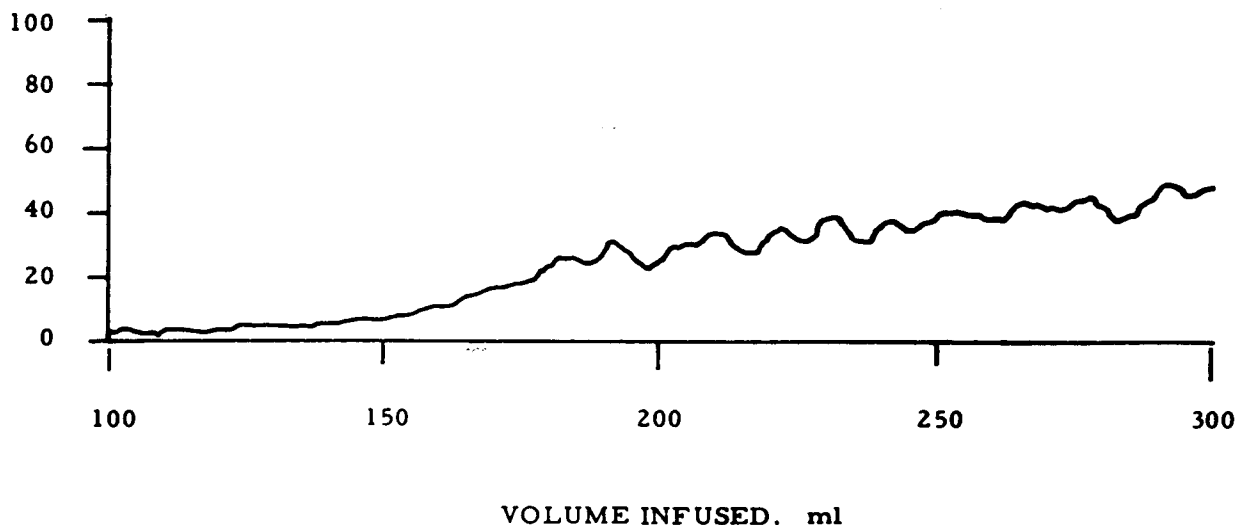


Table 2. Distribution of Patients with Detrusor Hyperreflexia in Relation to Neurological Disturbance and Site of Lesion

| | Cervical and dorsal | | Conus medullaris | | Cauda equina | |
|----------------------------|--------------------------|-------------------------|----------------------------------|----------------------------------|-------------------------|--------------------------|
| | Total upper motor neuron | Pure upper motor neuron | Predominantly upper motor neuron | Predominantly lower motor neuron | Pure lower motor neuron | Total lower motor neuron |
| No. of patients | 36 | 34 | 2 | 21 | 13 | 34 |
| Compliant hyperreflexia | 17 (94.4) ^a | 16 (100) | 1 | 4 (22.2) | 0 | 4 (20) |
| Noncompliant hyperreflexia | 1 (5.6) | 0 | 1 | 14 (77.8) | 2 | 16 (80) |
| Total | 18 (100) | 16 (100) | 2 (100) | 18 (100) | 2 (100) | 20 (100) |

^aValues in parentheses indicate percentages.

Thus, lower motor neuron disturbances cause a type of overactivity of the detrusor different from that caused by upper motor neuron disturbances, in which the detrusor overactivity is of a reflex nature. Furthermore, in upper motor neuron lesions the detrusor compliance is normal; reduced compliance is encountered only in lower motor neuron lesions [4].

Analysis of the pattern of detrusor hyperreflexia in the patients in this study has demonstrated the two types of detrusor hyperactivity described. Furthermore, the compliant hyperreflexic type of detrusor hyperactivity, which can be provoked by external stimuli and therefore has characteristics of a reflex phenomenon, correlates well with signs of upper motor neuron disturbance, in which such bladder function is known to occur. Similarly, the noncompliant type of detrusor hyperreflexia, which has a rhythmic nature, correlates well with signs of lower motor neuron involvement, in which such contractility has been described. As the present definition of detrusor hyperreflexia does not differentiate between the two types of overactivity, any description of the urodynamic functions in current terms would be unhelpful in differentiating lower motor neuron from upper motor neuron disturbances of the bladder, even when such differences exist. When detrusor hyperreflexia exists in patients with localized partial lesions of the spinal cord and cauda equina, identification of the detrusor overactivity as compliant hyperreflexic or noncompliant hyperreflexic should serve to differentiate bladder disturbances caused by upper motor neuron lesions from those caused by lower motor neuron lesions.

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References

1. Bates CP, Bradley WE, Glen ES, Melchoir H, Rowan D, Sterling AM, Sundin T, Thomas D, Torrens M, Turner-Warwick R, Zinner NR, Hald T. Fourth report on the standardisation of terminology of lower urinary tract function. *Br J Urol* 1981;53:333-5.
2. Denny-Brown D, Robertson GE. The state of the bladder and its sphincters in complete transverse lesions of the spinal cord and cauda equina. *Brain* 1933;56:397-462.
3. Elliot TR. The innervation of the bladder and urethra. *J Physiol (Lond)* 1907;35:367-445.
4. Gunasekera WSL, Richardson AE, Seneviratne KN, Eversden ID. Significance of detrusor compliance in patients with localized partial lesions of the spinal cord and cauda equina. *Surg Neurol* 1983;20:59-62.
5. Head H, Riddoch G. The automatic bladder, excessive sweating and some other reflex conditions in gross injury of the spinal cord. *Brain* 1917;40:188-263.
6. Norlen L. The autonomic bladder. A clinical and experimental study. *Scand J Urol Nephrol Suppl* 1976;36:1-28.
7. Sherrington CS. Notes on the arrangements of some motor fibres in the lumbosacral plexus. *J Physiol (Lond)* 1892;13:621-772.