REVIEW OF NEUROGENIC BLADDER IN MULTIPLE SCLEROSIS

DANIEL H. PIAZZA, M.D.
ANANIAS C. DIOKNO, M.D.

From the Section of Urology, Department of Surgery, University of Michigan Medical Center, Ann Arbor, Michigan

ABSTRACT — Neurologic involvement of the urinary bladder and urethral sphincter in multiple sclerosis has been known for some time. Thirty-one patients with a proved diagnosis of multiple sclerosis were evaluated urologically for symptoms of urinary incontinence, retention, or urinary tract infection. On initial presentation, 27 (74 per cent) were found to have neurogenic bladders of which 23 (85 per cent) were of the uninhibited type. Electromyography of the periurethral striated muscle revealed vesicosphincter incoordination in 9 of the 19 patients studied. Approximately one-half of the patients with uninhibited bladders had uncoordinated sphincters. Modalities of treatment are discussed.

The occurrence of neurogenic bladder disorder in multiple sclerosis has been known for some time. The type of lesion producing the bladder defects can be as varied as the disease itself and can produce many different types of neurogenic bladder dysfunction. Theoretically any type of neurogenic bladder and sphincter abnormality is possible. We have, therefore, attempted to review those patients with multiple sclerosis who have been studied urodynamically in an attempt to characterize their bladder lesions.

Material and Methods

Patient charts were screened carefully to determine the accuracy of diagnosis of multiple sclerosis. Only those patients who had substantial evidence of multiple sclerosis following a thorough evaluation by the department of neurology were included in the study. Patients diagnosed as "probable" or "possible" multiple sclerosis were not included in the analysis. The indication for urologic evaluation included urinary retention, urinary incontinence, or recurrent urinary tract infections. Urologic evaluation consisted of a thorough history and physical examination, complete urine analysis, intravenous pyelography, cystometry, and cystoscopy. Electromyography of the periurethral striated muscle (external sphincter) was performed on the majority of patients as part of their initial urologic studies.

The technique of cystometry and external sphincter electromyography has been described previously. The classification of the type of neurogenic bladder used was that of Nesbit and Lapides. The types of external sphincter abnormality observed on electromyography were based on the classification of Diokno and Koff.

Results

Thirty-one patient charts were reviewed. There were 14 males ranging in age from twenty-six to sixty-three years and 17 females from twenty-eight to sixty-five years of age. The cystometric evaluation showed that 23 patients had uninhibited neurogenic bladders, 1 motor paralytic, 1 reflex bladder, 3 normal, 1 decompensated, and 2 were classified as mixed upper and lower motor neuron bladders.

The electromyographic study performed at the time of cystometry revealed that of the 15 patients with uninhibited neurogenic bladders, 8 had coordinated sphincter activity and 7 had uncoordinated activity. The patients with the
reflex neurogenic bladder and mixed type of neurogenic bladder had uncoordinated electromyographic activity with detrusor contraction. Two patients with normal cystometric evaluation showed a normal coordinated external sphincter. Of the 15 patients with uninhibited neurogenic bladder undergoing combined cystometry and electromyography, over one-third presented with urinary tract infections while the remainder presented with irritative symptoms of frequency, urgency, or urge incontinence.

Comment

A majority of patients with multiple sclerosis who have neurogenic bladder dysfunction will present with urinary retention, urinary incontinence, or recurrent urinary tract infection. Any patient with multiple sclerosis who presents with this symptomatology should have complete urodynamic evaluation.

Cystometry is the single most important test in identifying normal and abnormal function. Cystometric examination should include assessment of residual urine, exteroceptive and proprioceptive bladder sensation, and integrity of the sacral reflex arc through the bulbocavernous reflex. The cystometrograph should delineate the bladder capacity, intravesical pressure, presence or absence of uncontrolled detrusor contractions, and the ability to contract the bladder voluntarily. Bethanechol (Urecholine) and methantheline (Banthine) tests should be performed if there is doubt in the cystometric diagnosis.

When cystometry is combined with external sphincter electromyography, the neurologic and functional status of the external sphincter can be assessed. Denervation may be identified, and sphincter coordination or uncoordination with detrusor contraction can be assessed. It should be emphasized that when performing electromyography, the external urinary sphincter should be sampled and that needle electromyography is preferable to that of surface electromyography. Our observation and that of others have shown that in the presence of bladder dysfunction, dissociation of activity can occur between the anal and urinary sphincter.1,4

We prefer to use the Nesbit-Lapides classification of neurogenic bladder dysfunction because it gives us an immediate idea of the bladder dysfunction. When cystometry is combined with sphincter electromyography, additional information regarding the function of the external sphincter is obtained. Coordinated sphincter relates to the ability of the sphincter to relax voluntarily or involuntarily in the presence of detrusor contraction. Uncoordinated system suggests poor relaxation or even increased spasticity in the presence of detrusor contraction.

Our study showed that the most frequent type of bladder observed on initial presentation in multiple sclerosis is the uninhibited or infantile type. The results also showed that about one half of these patients have uncoordinated sphincters. These observations explained why these patients are incontinent yet may present with elevated residual urine. It appears that in patients in this group uncontrolled contractions develop in response to distention or bladder stretch. The intravesical pressure generated by the detrusor contraction is strong enough to overcome the intraurethral pressure despite the lack of sphincter relaxation. Incontinence results with incomplete emptying. In general these patients can generate voluntary detrusor contractions, but because of spasticity of the external sphincter or lack of relaxation, voiding is accomplished under very high pressure resulting in incomplete emptying.1,4

Treating this group of patients with anticholinergic agents alone will be hazardous. This will only lead to increasing residual urine or urinary retention.

The treatment of these patients may be simplified by the use of intermittent self-catheterization for most cases to provide adequate emptying.8 An anticholinergic agent is used as an adjunct to suppress the uninhibited contractions and prevent urge incontinence. Uninhibited contraction which generates high intravesical pressures may perpetuate lower urinary tract infection if not suppressed with anticholinergics even though residual urine is removed by intermittent catheterization.7

Those patients with uninhibited neurogenic bladder and coordinated sphincter are characterized by urinary frequency and urge incontinence. This group does not have residual urine unless they have decompensated bladder from previous retention or urinary overdistention in the past. Because of normal coordination, they can be treated safely with anticholinergic agents without fear of urinary retention.

The motor paralytic and the decompensated bladder are usually associated with urinary retention. These patients are also best managed by intermittent self-catheterization. With this
technique, the patient may regain his own bladder function. It is suggested that repeat urodynamic studies should be performed in several weeks especially if the neurologic status has improved.

Urinary tract infection is often the clue that lower urinary tract dysfunction is present. Since altered dynamics of the bladder and sphincter will reduce the ability of the bladder to prevent active infection from being eradicated once it is introduced, this should be taken as a sign that further urologic evaluation is necessary. It is known that the types of neurogenic bladder in multiple sclerosis can vary according to the status of the multiple sclerosis itself. Although we did not study the progression or changes of the bladder dysfunction in individual patients, we have certainly observed this phenomenon in individual cases. Since dysfunction can revert to normal or to another type of bladder dysfunction, it is important to keep the treatment as simple, conservative, and easily reversible as possible. Destructive and irreversible procedures such as external sphincterotomy and supravesical urinary diversion should be relegated to the end stage irreversible cases of multiple sclerosis and only after all other types of conservative therapy have been tried and have failed.

The goal of urodynamic evaluation of multiple sclerosis patients is to make the patient comfortable and socially acceptable by improving continence or providing inoffensive means of emptying urine from the bladder. The ultimate objective is to protect the patient's kidneys from damage by preventing recurrent infection and avoiding the long range problems associated with urinary diversion.

Since it has been documented that neurogenic bladder findings may be the first indication of progressive neuromuscular disease, likewise any patient with known neuromuscular disease (multiple sclerosis, etc.) should be studied urologically at the onset of symptoms or the findings of urinary tract infection. Prompt diagnosis and treatment of neurogenic bladder dysfunction in these patients may prevent later complications of renal failure, calculus disease, pyelonephritis, and sepsis.

Ann Arbor, Michigan 48109
(DR. PIAZZA)

References