# PREDICTING LOWER URINARY TRACT DYSFUNCTIONS IN PATIENTS WITH SPINAL CORD INJURY

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ABSTRACT — The results of combined cystometry and perineal electromyography were reviewed retrospectively in 75 consecutive, traumatic spinal cord-injured patients to predict lower urinary tract dysfunctions. In patients with vertebral spinal injuries at vertebral level T7 or above a reflex neurogenic bladder eventually developed. In those with vertebral level injuries T11 or below a lower motor neuron bladder dysfunction developed. Injuries at the vertebral levels T8, T9, and T10 represent a gray zone; and, depending on adjacent soft tissue injury, in these patients an upper or lower motor neuron bladder dysfunction developed. The transition from spinal shock (areflexia) to reflex neurogenic (hyperreflexia) bladder occurred at different times in different patients and could not be correlated to level of injury or its severity. The periurethral striated muscle was generally denervated if a lower motor neuron bladder dysfunction existed, however, rarely, dissociation may occur. When a reflex neurogenic bladder existed, sphincter dyssynergia was present 68 per cent of the time. This also could not be correlated with time after injury, level of injury, or severity of injury

We have previously advocated the importance of early and frequent urodynamic evaluation in the management of the patient with spinal cord injury.[1] Although we still support the importance of such evaluation, some clinically useful predictions regarding the lower urinary tract dysfunctions after spinal cord injury can be made. This article illustrates the limitations of predicting the lower tract dysfunctions as they relate to time after injury, level of injury, and severity of injury.

## Material and Methods

Seventy-five consecutive, traumatic spinal cord-injured patients were reviewed retrospectively. The results of combined cystometric and perineal electromyography were tabulated in those patients whose injuries involved vertebral levels C2 through L5. Evaluations were performed between one week and thirteen years after the time of spinal injury.

Cystometry was performed using the carbon dioxide ($CO_2$) cystometer, and the bladder dysfunction was classified according to the original

work by Lapides.[2,3] A spinal shock (areflexia) bladder was diagnosed when sensation was absent, detrusor contractions were absent, and when the bethanechol chloride (Urecholine) supersensitivity test was negative (Fig. 1). When sensation and detrusor contractions were absent but the bethanechol chloride supersensitivity test was positive, an autonomous neurogenic (areflexia) bladder was diagnosed (Fig. 2). A reflex neurogenic (hyperreflexia) bladder was diagnosed when sensation was absent and involuntary detrusor contractions occurred (Fig. 3). Comparisons to the normal cystometrogram can be made by referring to Figure 4.

Electromyography (EMG) of the periurethral striated muscle was done using the TECA TE-4 electromyograph with 37 mm. Teflon-coated monopolar needles. The specific techniques and classifications used were those as described by Diokno and co-workers.[4,5] In the female, the needle was placed in the fold just adjacent to the urethral meatus. In the male, the needle was placed in the perineum, and, with a finger in the rectum, directed toward the apex of the
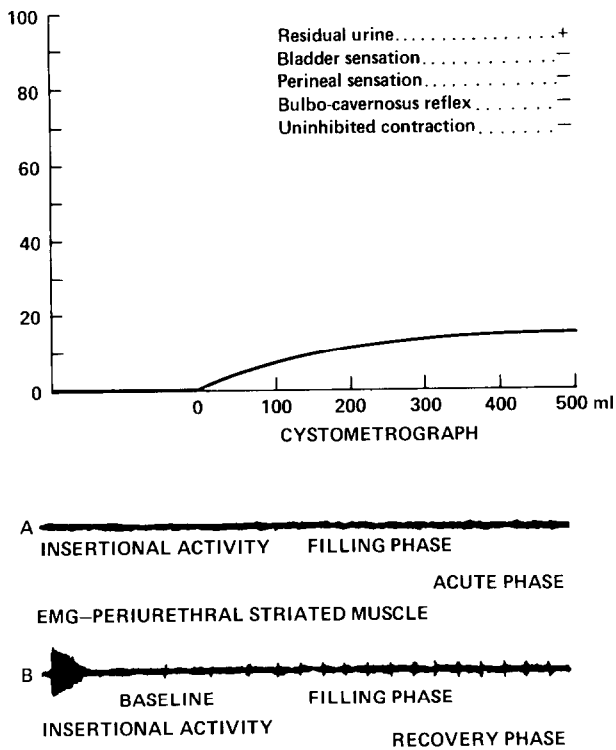
FIGURE 1. *Cystometrogram of spinal shock bladder with periurethral electromyography in both acute and recovery phase.*



FIGURE 2. *Cystometrogram of autonomous neurogenic bladder with periurethral electromyography showing both complete and incomplete denervation.*

prostate. With minimal practice, these techniques can be mastered. Denervation was diagnosed when no electromyogram activity could be found, or, when positive waves or fibrillations occurred. Electromyography in the recovery phase was defined as EMG activity in the face of a spinal shock bladder (Fig. 1). Sphincter dyssynergia (uncoordinated EMG) was defined by a change or an increase in EMG activity during a reflex detrusor contraction, while sphincter synergia (coordinated EMG) showed decreased electromyogram activity during a reflex detrusor contraction (Fig. 3). Normal electromyography of the periurethral striated muscle consisted of normal motor units with good voluntary relaxation and contraction (Fig. 4).

## Results

The cystometric results were analyzed by dividing them into three groups. Group I included the initial cystometrograms of all patients (75 studies from one week to thirteen years after injury). Group II included all cystometrograms (40) done at ten weeks or earlier, and Group III included all cystometrograms (53) done at four months or earlier. The specific results of these cystometric evaluations can be found in Table I. When lower motor neuron bladders are eliminated, it can be seen that a greater percentage of bladders become reflex
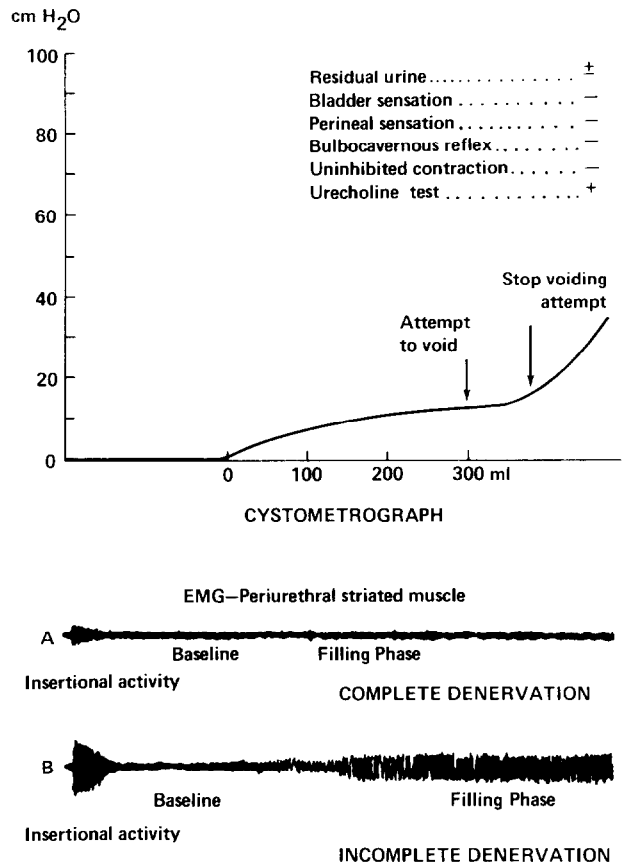
neurogenic bladders as time progresses (i.e., from ten weeks to four months).

Of the 29 patients who had spinal shock bladders initially, 10 later had documentation of reflex neurogenic bladders. The average halfway point between the two evaluations was 6.3 months (some of the later evaluations were delayed). The longest lasting spinal shock bladders were documented at seven, nine, ten, and thirteen months. These patients had injuries of different spinal levels and severity.

None of the 30 patients who initially presented with reflex neurogenic bladders had subsequent documented cystometric change. The earliest reflex neurogenic bladders were documented at one, three, four, and six weeks. These patients also had injuries of different spinal levels and severity. Forty-one patients in Group I eventually carried the cystometric diagnosis of reflex neurogenic bladder. All patients evaluated after thirteen months with a vertebral level injury T7 or above had a reflex neurogenic bladder.

All 15 patients with evidence of lower motor neuron bladder injury (autonomous, motor paralytic, and mixed neurogenic bladders) had
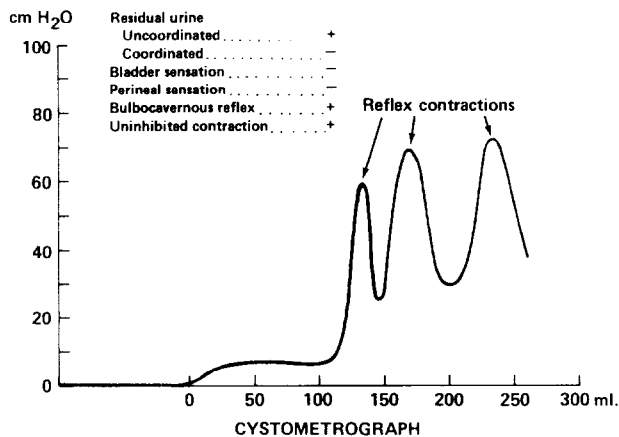
cm H₂O — $cm\ H_2O$

Residual urine
Uncoordinated . . . . . . . . +
Coordinated . . . . . . . . . . . . —
Bladder sensation . . . . . . . . . —
Perineal sensation . . . . . . . . . —
Bulbocavernous reflex . . . . . . . +
Uninhibited contraction . . . . . . +

Reflex contractions

CYSTOMETROGRAPH

I. Coordinated bladder and periurethral striated muscle

Baseline | Filling phase | Involuntary relaxation

Insertional activity

II. Uncoordinated bladder and periurethral striated muscle

A — UNCOORDINATED PERIURETHRAL STRIATED MUSCLE — Involuntary contractions

B — CLONIC PERIURETHRAL STRIATED MUSCLE

C — TONIC PERIURETHRAL STRIATED MUSCLE

FIGURE 3. *Cystometrogram of reflex neurogenic bladder with periurethral electromyography showing synergia (coordinated) and dyssynergia (uncoordinated).*

Residual urine . . . . . . . . . . . . . 0 ml
Bladder sensation
Hot and cold . . . . . . . . . . . +
Perineal sensation . . . . . . . . . +
Bulbocavernous reflex . . . . . . . +
Uninhibited contraction . . . . . . . —

cm H₂O — $cm\ H_2O$

Stop voiding on command
Voiding on command aga
Voiding on command

CYSTOMETROGRAPH

EMG—PERIURETHRAL STRIATED MUSCLE

Baseline | Filling phase | Voiding phase | Post void
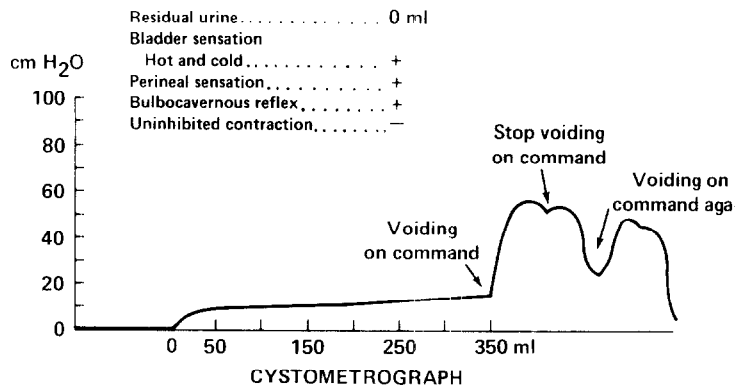
Insertional activity

FIGURE 4. *Normal cystometrogram with normal periurethral electromyography.*

injuries at the vertebral level T11 or below. Of 7 patients with injuries at vertebral levels T8 to T10, there was 1 spinal shock, 3 reflex neurogenic bladders, 2 autonomous, and 1 motor paralytic neurogenic bladder.

Seventy-four of the 75 patients in Group I had electromyography of the periurethral striated muscle. At initial evaluation, 28 patients had EMG in the recovery phase (spinal shock

TABLE I. *Cystometric results*

| Bladder Dysfunction | | Evaluation* | | |
| --- | --- | --- | --- | --- |
| | | Group I (75 Patients) | Group II (40 Patients) | Group III (53 Patients) |
| | Spinal shock | 29 | 18 | 23 |
| Upper motor neuron bladders | Reflex neurogenic bladder | 30 | 11 | 19 |
| | Uninhibited neurogenic bladder | 1 | 0 | 0 |
| Lower motor neuron bladders | Autonomous neurogenic bladder | 10 | 8 | 8 |
| | Motor paralytic neurogenic bladder | 4 | 3 | 3 |
| | Mixed neurogenic bladder | 1 | 0 | 0 |

*Group I, initial evaluation (one week to thirteen years); Group II evaluated ten weeks or earlier; Group III, evaluated four months or earlier.

TABLE II. *Results of electromyography of periurethral striated muscle*

| EMG Result | Initial Evaluation (74 Patients) | Comments | Final Results on 41 Patients with Reflex Neurogenic Bladders |
|---|---|---|---|
| Recovery phase | 28 | 9 eventually became uncoordinated and 1 became coordinated when reflex neurogenic bladders were documented | 0 |
| Denervated | 13 | 12 had injuries T8 or lower | 0 |
| Coordinated (synergia) | 12 | No change at further evaluations | 13 |
| Uncoordinated (dyssynergia) | 21 | 4 eventually became coordinated | 28 |

bladder with EMG activity), 13 patients had denervated EMG, 12 patients had coordinated EMG, and 21 patients had uncoordinated EMG (Table II).

Twelve of 13 patients with denervated EMG were associated with vertebral level injuries T8 or lower. Of the 28 patients with EMG in the recovery phase, 9 were later documented to change to uncoordinated and 1 to coordinated EMG when reflex neurogenic bladders were documented. Documented change from uncoordinated EMG to coordinated EMG occurred in 4 patients. The final documented EMG on the 41 patients with reflex neurogenic bladders revealed 13 to be coordinated and 28 to be uncoordinated. Neither the level nor severity of the injury was helpful in predicting sphincter synergia or dyssynergia.

## Comment

In patients with a vertebral level injury T7 or above a reflex neurogenic bladder eventually develops; and in patients with a vertebral level injury T11 or lower a lower motor neuron bladder dysfunction (usually autonomous neurogenic bladder) develops. Upper or lower motor neuron bladder dysfunction may develop in patients with vertebral level injuries T8, T9, and T10.

The timing of the transition from spinal shock bladder (areflexia) to a reflex neurogenic bladder (hyperreflexia) was variable. This occurred as early as one week and as late as thirteen months after injury. The timing of this transition could not be correlated with either level of injury or severity (completeness) of injury. Generally this transition seemed most likely to occur between three and six months after injury.

A patient with a vertebral level injury T7 or above who begins to void spontaneously can be assumed to have a reflex neurogenic bladder. His management, though, must include knowl-
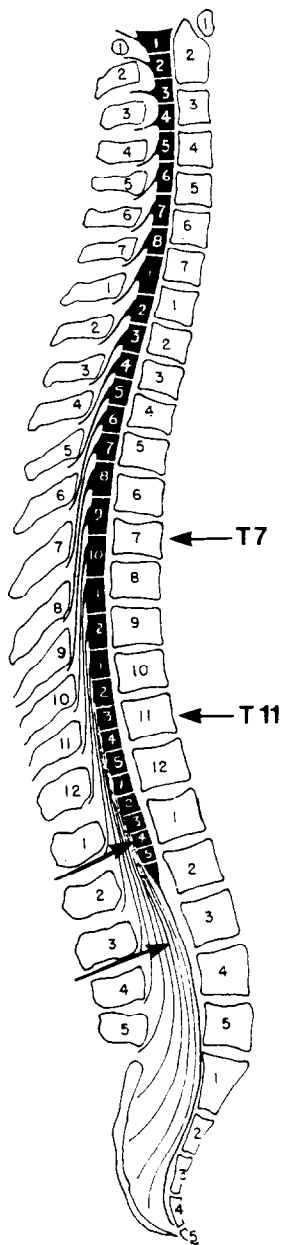
edge of his periurethral striated muscle activity at the time of emptying. This is of critical importance since reflex emptying with external sphincter dyssynergia will cause increased intravesical pressure and a myriad of possible complications.[6,7]

Most injuries below the T8 vertebral level will lead to denervation of the periurethral striated muscle (92 per cent). This denervation generally was associated with lower motor neuron bladder denervation, although rarely, dissociation may occur. This means that the bladder ($S_2$–$S_4$) may be denervated while the periurethral striated muscle (also $S_2$–$S_4$) may be innervated.

Initial evaluation of the periurethral striated muscle revealed 36 per cent (12 of 33) to be coordinated to reflex contractions. Documented change from uncoordinated EMG to coordinated EMG occurred in 4 patients and could not be correlated to time after injury, level of injury, or severity of injury. Of 28 patients with EMG initially in the recovery phase, 9 were later documented to change to uncoordinated and one to coordinated EMG after reflex neurogenic bladders developed. Thirty-two per cent of the patients (13 of 41) who eventually had documented reflex neurogenic bladders had coordinated EMG. It was impossible to correlate coordination of the periurethral striated muscle with time after injury, level of injury, or severity of injury.

If an established spinal cord-injured patient who voids reflexly has been managed without any difficulties (infection, stones, pyelonephritis, renal insufficiency), it may be assumed that he has a reflex neurogenic bladder with coordinated EMG and consequently requires no further urologic evaluation. However, since the majority of reflex neurogenic bladders will have sphincter dyssynergia (68 per cent in this study), we routinely evaluate these patients

FIGURE 5. *Injury to vertebral level T11 will affect sacral roots 2–4 with adjacent soft tissue injury of just one or two vertebral bodies. T7 injury would have to extend soft tissue injury to five or six vertebral bodies before affecting critical nerve roots.*

enough to cause fracture or dislocation. An injury to T7 would have to extend adjacent soft tissue injury to five or six vertebral bodies before the critical nerve roots were affected. Since this much soft tissue injury is unlikely, an upper motor neuron bladder dysfunction occurs. It is the vertebral levels T8, T9, and T10 which fall into a gray zone. Depending on the amount of caudal soft tissue injury and whether the nerve roots S2, S3, and S4 are affected, an upper or lower motor neuron bladder dysfunction will exist. Similar logic can be used to explain the denervation of the periurethral striated muscle (also innervated S2, S3, and S4) seen in some spinal-injured patients.

Previous experience (including patients outside this study group) has shown that wider areas of soft tissue spinal cord injury result from vertebral gunshot wounds, abscesses, and vascular spinal cord injuries. More localized spinal cord injury occurs from tumor and vertebral body dislocations.

More difficult to explain is the reason for sphincter coordination or dyssynergia accompanying a reflex neurogenic bladder. According to Bradley's neural loop theory, loop III is composed of afferent sensory axons from the detrusor muscle coursing to the pudendal motor neurons (which control the periurethral striated muscle).[8] The fact that high spinal injuries may disrupt this loop and cause dyssynergia implies that other factors are operative. Yet unidentified internuncial nerves or reorganization of the sacral reflex arc eventually may help explain what causes sphincter coordination or dyssynergia.

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before complications ensue. This approach is not mandatory, and some may choose to wait for a first complication (usually infection) before embarking on a urodynamic evaluation. One must be cautious in delaying evaluation since the first complication may be a septic episode. After any urologic problem has arisen, evaluation definitely should not be delayed.

It is not difficult to explain why injuries to the vertebral level T11 and below result in lower motor neuron denervation, while injuries to T7 or above result in upper motor neuron bladder dysfunction. Figure 5 shows that injury to vertebral level T11 will begin to affect the sacral nerve roots S2, S3, and S4 (innervation of the bladder) if an extension of inflammation, hemorrhage, and scarring of one or two vertebral bodies occurs. Such soft tissue injury is not unusual since the initial injuries were severe