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URODYNAMIC PATTERN CHANGES IN MULTIPLE SCLEROSIS

SEBASTIAN J. CIANCIO, STEVEN E. MUTCHNIK, VICTOR M. RIVERA, AND TIMOTHY B. BOONE

ABSTRACT

Objectives. Multiple sclerosis (MS) causes neurologic symptoms to change over time. Voiding dysfunction is common in patients with MS, and few studies have examined the changes in urodynamic patterns in these patients over time. The purpose of this study was to examine the frequency and nature of urodynamic pattern changes in patients with MS who underwent two or more urodynamic studies.

Methods. Twenty-two patients (7 men and 15 women) with well-documented MS were referred to one urologist (T.B.B.) for evaluation of lower urinary tract symptoms. All patients had undergone two or more urodynamic evaluations during a 14-year period for persistent or new symptoms, and a retrospective comparison was made among the urodynamic test results.

Results. Overall, 12 (55%) of 22 patients experienced a change in their urodynamic patterns and/or compliance during a mean follow-up interval of 42 ± 45 months between the urodynamic studies. Most patients initially had urodynamic patterns showing detrusor hyperreflexia, detrusor external sphincter dyssynergia, or detrusor hypocontractility. Fourteen (64%) of the 22 patients studied had the same or worsening of the same symptoms and 8 (36%) of 22 had new urologic symptoms. Six (43%) of 14 patients with no new symptoms and 6 (75%) of 8 with new symptoms had significant changes found with follow-up urodynamic testing.

Conclusions. A significant proportion of patients with MS with and without new urinary symptoms will develop changes in their underlying urodynamic patterns and detrusor compliance. Therefore, urodynamic evaluations should be repeated at regular intervals in symptomatic patients to optimize clinical management, reduce complications, and better enable these patients to manage their neurogenic bladder dysfunction. UROLOGY **57:** 239–245, 2001. © 2001, Elsevier Science Inc.

Multiple sclerosis (MS) is a disabling neurologic condition characterized by exacerbations and remissions with associated changing signs and symptoms. The pathologic hallmarks of MS are the demyelinating plaques in the white matter of the central nervous system. These plaques eventually affect the myelinated nerve tracts that mediate voiding and produce voiding dysfunction in more than 80% of patients.[1] Urodynamic findings in patients with MS include detrusor hyperreflexia, detrusor external sphincter dyssynergia, and detrusor hypocontractility.[2,3] Neurologic patterns in patients with MS are known to change with time, but the incidence and manner of the changing urodynamic patterns have rarely been studied over time. In one series, Wheeler *et al.*[4] examined 18 patients retrospectively and found evidence of urodynamic change in 55%. The purpose of this study was to examine the frequency and nature of the urodynamic pattern changes in patients with MS who underwent two or more urodynamic studies.

From the Scott Department of Urology and Department of Neurology, Baylor College of Medicine, Houston, Texas

Reprint requests: Timothy B. Boone, M.D., Ph.D., 6560 Fannin, Suite 2100, Houston, TX 77030

Submitted: January 26, 2000, accepted (with revisions): September 13, 2000

MATERIAL AND METHODS

During a 14-year period from October 1984 to August 1998, 22 patients with well-documented MS underwent two or more urodynamic evaluations because of new or persistent lower urinary tract symptoms. Seven men and 15 women between 22 and 69 years of age (mean 46 ± 10) were referred to one urologist (T.B.B.) for the urologic evaluation. The average age of the men was 43 ± 13 years (range 22 to 59) and that of the women was 46 ± 9 years (range 35 to 69). MS was characterized clinically by a neurologist (V.M.R.) as relapsing remitting (9 patients), primarily progressive (4 patients), or secondarily progressive (9 patients) at the time of each patient's most recent office visit. Patients were excluded if the diagnosis of MS was in question or if they had bladder stones, marked

0090-4295/01/$20.00
PII S0090-4295(00)01070-0

prostatic enlargement on digital rectal examination, infected urine at the time of the study, urodynamic evidence of bladder outlet obstruction, or a history of benign prostatic hyperplasia or urethral stricture disease.

A history and detailed neurologic and urologic physical examination were performed on all patients. Symptomatic patients underwent an initial urodynamic investigation because of irritative symptoms (14 patients), obstructive symptoms (2 patients), incontinence (7 patients), and/or recurrent urinary tract infections (4 patients). All patients had sterile urine cultures before the urodynamic evaluation. Repeated examinations were performed because of new symptoms or a lack of response to treatment.

The urodynamic evaluations conformed to the International Continence Society standards.[5] The urodynamic technique involved simultaneous measurement of intravesical, intraurethral, and intra-abdominal pressures, electromyography of the external anal sphincter, urinary flow rate measurement, and, in at least one study in all patients, voiding cystourethrography with fluoroscopy (Laborie, Aquarius). A double-lumen 8F catheter was inserted transurethrally into the bladder. Radiographic contrast material was used for cystometry, and fluoroscopy was selectively applied during the study. Contrast was infused at a rate of 10 to 50 mL/min depending on patient tolerance and/or the need for provocative testing.

Fluoroscopy was used to confirm the diagnosis of detrusor external sphincter dyssynergia and to exclude the diagnosis of bladder outlet obstruction secondary to benign prostatic hyperplasia or other non-neurogenic causes. Radiographic imaging also allowed us to evaluate the patients for vesicoureteral reflux and stress urinary incontinence.

The mean interval between the urodynamic studies was 35 ± 35 months (range 3 months to 13 years). Three patients underwent more than two urodynamic studies during the period of investigation, and the intervals between each study were used in calculating the overall mean interval.

Bladder compliance was calculated at the maximum cystometric capacity by dividing the volume change by the change in the detrusor pressure during that change in bladder volume.[6] Detrusor pressure changes associated with hyperreflexia were excluded from the compliance calculations. Bladder compliance was considered abnormal if less than 20 mL/cm H_2O. Detrusor hyperactivity with impaired contractility (DHIC) was considered a subcategory of detrusor hypocontractility. This diagnosis was used when the urodynamic findings consisted of uninhibited detrusor activity on filling with impaired detrusor contractility on voiding, as evidenced by a postvoid residual volume of greater than 50% of capacity, without concomitant obstruction.[7] The diagnosis of detrusor external sphincter dyssynergia was given when patients had electromyographic evidence of sphincteric contractions during involuntary detrusor contractions; this was corroborated using fluoroscopy when this modality was available (most cases).

Patient treatment throughout the study period was based on symptoms, urodynamic findings, and patient ability and preferences. The initial management consisted of oral anticholinergic therapy in 14 patients, clean intermittent catheterization in 3, observation in 4, incontinence pads in 1, Foley catheter placement in 1, and bladder neck suspension in 1 patient with genuine stress urinary incontinence. Patients who were not compliant with a given therapy were classified according to the therapy that they chose (eg, observation or incontinence pads).

RESULTS

The results are summarized in Table I, which gives the interval between the urodynamic studies for each patient, and the MS pattern, urodynamic pattern, detrusor compliance, and patient treatment at the time of each urodynamic evaluation.

Overall, 12 (55%) of 22 patients experienced a change in their urodynamic patterns and/or compliance during 42 ± 45 months (range 3 months to 13 years), including 2 patients who experienced a deterioration in detrusor compliance without gross changes in the urodynamic patterns. Of these 12 patients, 5 had relapsing remitting MS, 2 had primarily progressive MS, and 5 had secondarily progressive MS. The MS pattern changed in only 1 patient between urodynamic studies, and this patient experienced neither a reduction in detrusor compliance nor a change in symptoms or urodynamic patterns.

Table II groups the patients on the basis of their initial urodynamic pattern and shows the distribution of the urodynamic patterns on the repeated evaluation. Most patients initially had urodynamic patterns consistent with detrusor hyperreflexia, detrusor external sphincter dyssynergia, or detrusor hypocontractility. Initially, 15 patients (68%) had hyperreflexia, 4 patients (18%) had hypocontractility (including 1 with DHIC), and 3 patients (14%) had normal urodynamic characteristics. Of the 15 patients with initial detrusor hyperreflexia, 5 had detrusor external sphincter dyssynergia. Three (60%) of these 5 had the same pattern on the repeated evaluation, 1 (20%) had hypocontractility, and 1 (20%) had a normal study 40 months after the first but demonstrated hyperreflexia on a repeated evaluation 11 months later. Of the 10 patients with hyperreflexia and synergistic sphincter activity, 6 (60%) continued to have hyperreflexia with synergy, 1 (10%) developed detrusor external sphincter dyssynergia, and 3 (30%) developed hypocontractility, including 1 with DHIC.

Of the 4 patients who initially had detrusor hypocontractility, 1 (25%) remained hyporeflexic, 1 (25%) developed detrusor hyperreflexia, 1 (25%) developed hyperreflexia with detrusor external sphincter dyssynergia (Fig. 1), and 1 (25%) had stable DHIC on both the initial and follow-up examinations. The patient who developed detrusor external sphincter dyssynergia also had vesicoureteral reflux diagnosed with video urodynamic studies. No other patients had vesicoureteral reflux. Of the 3 patients with normal initial studies, 1 (33%) remained normal, 1 (33%) developed detrusor hyperreflexia, and 1 (33%) developed detrusor hypocontractility.

The urologic symptoms in the patients with MS on presentation and at follow-up are described in Table I. Fourteen (64%) of 22 patients studied had the same or worsening of the same symptoms at follow-up and 8 (36%) of 22 had new symptoms of incontinence, obstructive or irritative symptoms,

TABLE I. ***Urodynamic pattern changes in multiple sclerosis***

Pt. No.	Sex	Interval Between Evaluations (mo)	Symptoms	MS Pattern	Urodynamic Pattern	Compliance (mL/cm H_2O)	Management
Patients with Changes in Urodynamic Patterns							
1	Male	Presentation	Urge incontinence	NA	Hypocontractility	17	CIC, anticholinergics
		3	Same	NA	DESD	10	Dose increased
		9	Flank pain	RR	DESD	245	Same
2	Female	Presentation	Hesitancy	PP	Hypocontractility	37	Observation
		49	Urgency	PP	Hypocontractility	66	CIC
		20	Severe incontinence	PP	Hyperreflexia	9	Indwelling catheter
3	Female	Presentation	Urgency	PP	Hyperreflexia	12	Anticholinergics
		33	Incontinence	PP	Hypocontractility	16	Anticholinergics, CIC
4	Female	Presentation	Recurrent UTI	NA	Hyperreflexia	50	Anticholinergics
		151	Same	RR	DHIC	40	Anticholinergics, CIC
5	Male	Presentation	Urge incontinence	RR	Normal	40	Observation
		88	Same	RR	Hypocontractility	73	Anticholinergics
6	Female	Presentation	Nocturia, frequency, urgency	RR	Normal	100	Anticholinergics
		9	Same	RR	Hyperreflexia	48	Anticholinergics
7	Male	Presentation	Recurrent UTI	SP	Hyperreflexia	150	Anticholinergics
		30	Same	SP	DESD	41	Anticholinergics, CIC
8	Male	Presentation	Frequency, nocturia, incontinence	SP	DESD	30	Anticholinergics
		40	Retention	SP	Normal	40	Observation
		11	Urge incontinence	SP	Hyperreflexia	41	Anticholinergics
9	Female	Presentation	Frequency, urgency	SP	Hyperreflexia	45	Anticholinergics
		13	Retention	SP	Hypocontractility	14	Indwelling catheter
10	Female	Presentation	Frequency	NA	DESD	215	Anticholinergics
		4	Retention	SP	Hypocontractility	9	CIC
Patients with Deterioration in Compliance Only							
11	Female	Presentation	Urge incontinence	SP	Hyperreflexia	70	Anticholinergics
		15	Same	SP	Hyperreflexia	16	Bladder augmentation
12	Female	Presentation	Urge incontinence	RR	Hyperreflexia	34	Anticholinergics
		39	Same	RR	Hyperreflexia	12	Anticholinergics
Patients Without Urodynamic/Compliance Changes							
13	Female	Presentation	Severe incontinence	PP	Hyperreflexia	120	Anticholinergics
		19	Same	PP	Hyperreflexia	NA	Cystectomy/conduit
14	Female	Presentation	Severe incontinence	SP	DESD	70	Incontinence pads
		33	Same	SP	DESD	60	Anticholinergics, incontinence pads
15	Male	Presentation	Frequency, urgency, nocturia	SP	DESD	7	Anticholinergics
		1	Same	SP	DESD	10	Anticholinergics
		22	Same	RR	DESD	43	Anticholinergics
16	Male	Presentation	Frequency, urgency, hesitancy	SP	Hypocontractility	43	Observation
		93	Same	SP	Hypocontractility	136	CIC
17	Female	Presentation	Frequency, urgency, enuresis	RR	Hyperreflexia	25	Anticholinergics
		14	Same	RR	Hyperreflexia	40	Anticholinergics
18	Female	Presentation	Recurrent UTI	RR	DHIC	22	CIC
		6	Same	RR	DHIC	22	CIC, anticholinergics
19	Female	Presentation	Urgency, hesitancy	RR	DESD	21	Anticholinergics, CIC
		29	Urge incontinence	RR	DESD	28	Anticholinergics, CIC
20	Male	Presentation	Frequency, urgency	SP	Hyperreflexia	95	Observation
		37	Hesitancy	SP	Hyperreflexia	105	Observation
21	Female	Presentation	Recurrent UTI	PP	Hyperreflexia	15	Indwelling catheter
		24	Same	PP	Hyperreflexia	10	Diaper
22	Female	Presentation	Stress incontinence	RR	Normal	55	Needle suspension
		12	Urge incontinence	RR	Normal	150	Anticholinergics

Key: Pt. No. = patient number; MS = multiple sclerosis; NA = not available; CIC = clean intermittent catheterization; DESD = detrusor external sphincter dyssynergia; RR = relapsing remitting; PP = primarily progressive; DHIC = detrusor hyperactivity with impaired contractility; UTI = urinary tract infection; SP = secondarily progressive.

TABLE II. *Evolution of urodynamic patterns, grouped by initial urodynamic diagnosis*

Initial Diagnosis	Patients (n = 22)	Follow-up Evaluation*				
		Normal	Hypo-contractility	Hyperreflexia/ Synergy	Hyperreflexia/ DESD	DHIC
Normal	3 (14%)	1 (12)	1 (87)	1 (9)		
Hyperreflexia with sphincter synergy	10 (45%)		2 (23)	6 (25)	1 (30)	1 (150)
Hyperreflexia with DESD	5 (23%)		1 (4)	1 (51)	3 (55)	
Hypocontractility	3 (14%)		1 (92)	1 (20)	1 (11)	
DHIC	1 (5%)					1 (6)

Key: Abbreviations as in Table I.
** Numbers in parentheses are mean interval (in months) between studies.*

or flank pain. Of the 14 patients with no new symptoms, 6 (43%) had significant urodynamic changes on follow-up, including 4 with a change in urodynamic patterns, and 2 with a reduction in compliance at capacity. Six (75%) of the 8 patients with new symptoms had a significant change in their urodynamic patterns and three of these patients had changes in both patterns and compliance on follow-up.

On the repeated urodynamic evaluation at a mean of 28 ± 26 months after the first study, a total of 5 patients were noted to have a significant reduction in bladder compliance. Initially, all 5 patients had a compliance measurement greater than 20 mL/cm H_2O (mean compliance 80 ± 77). On follow-up, all patients experienced a decrease in compliance to less than 20 mL/cm H_2O (mean 12 ± 3). Three of these patients had a change in symptoms and a change in their urodynamic patterns, but 2 patients had no change in their symptoms or patterns.

As previously stated, patient management consisted of oral anticholinergic therapy, intermittent catheterization, observation, incontinence pads, Foley catheter placement, or surgery. All the patients who had a change in their urodynamic patterns or in compliance were offered additional therapy as indicated based on the new findings. Patients with compliance changes did not appear to have any predisposing factors or management peculiarities compared with other patients before the onset of compliance changes. One patient underwent augmentation cystoplasty for a high-pressure, low-capacity bladder when pharmacologic therapy failed to restore safe storage pressures.

COMMENT

MS is a disabling neurologic condition affecting approximately 1 in 1000 Americans.[8] The disease has protean neurologic manifestations and follows a varying clinical course. MS is characterized by focal demyelinating lesions that can occur at different levels in the central nervous system, resulting in genitourinary system dysfunction. The etiology of MS is unknown, but current reports favor an autoimmune origin involving central nervous system antigens.[9] The variable character and location of the plaques in the central nervous system, along with the associated edema, account for the changes in both the neurologic and urologic features of the disease over time. For example, suprasacral plaques will cause varying degrees of detrusor hyperreflexia with associated signs and symptoms, and sacral plaques will result in detrusor hypocontractility and, possibly, pudendal neuropathy.[10–12] Neurogenic bladder dysfunction is a debilitating quality-of-life issue affecting most patients with MS. Collectively, urologic symptoms become the most socially disabling and embarrassing aspect of the disease, affecting more than 80% of patients with MS.[13–15] Irritative voiding symptoms and incontinence were the predominant initial complaints in our series, consistent with previous reports.[3,13,16,17]

Our urodynamic data are comparable with those of other reported series.[4,10,11,13] Initially, 68% of our patients had hyperreflexia, 23% had detrusor external sphincter dyssynergia, 18% had hypocontractility, and 14% had normal urodynamic patterns. Fifty-three percent of the patients with hyperreflexia underwent a change on the repeated evaluation (40% had a change in the urodynamic pattern and/or compliance and 13% had a change in compliance alone), as did 50% of patients with hyporeflexia. Most patients with detrusor external sphincter dyssynergia (three of five) continued to have the same diagnosis on the follow-up examination, consistent with previous data showing that once it develops, it usually persists.[4]

A review of the published reports revealed few studies that directly address the range of urodynamic changes experienced by patients with MS over time. In the largest series, Schoenberg and Gutrich[18] performed repeated urodynamic evalua-

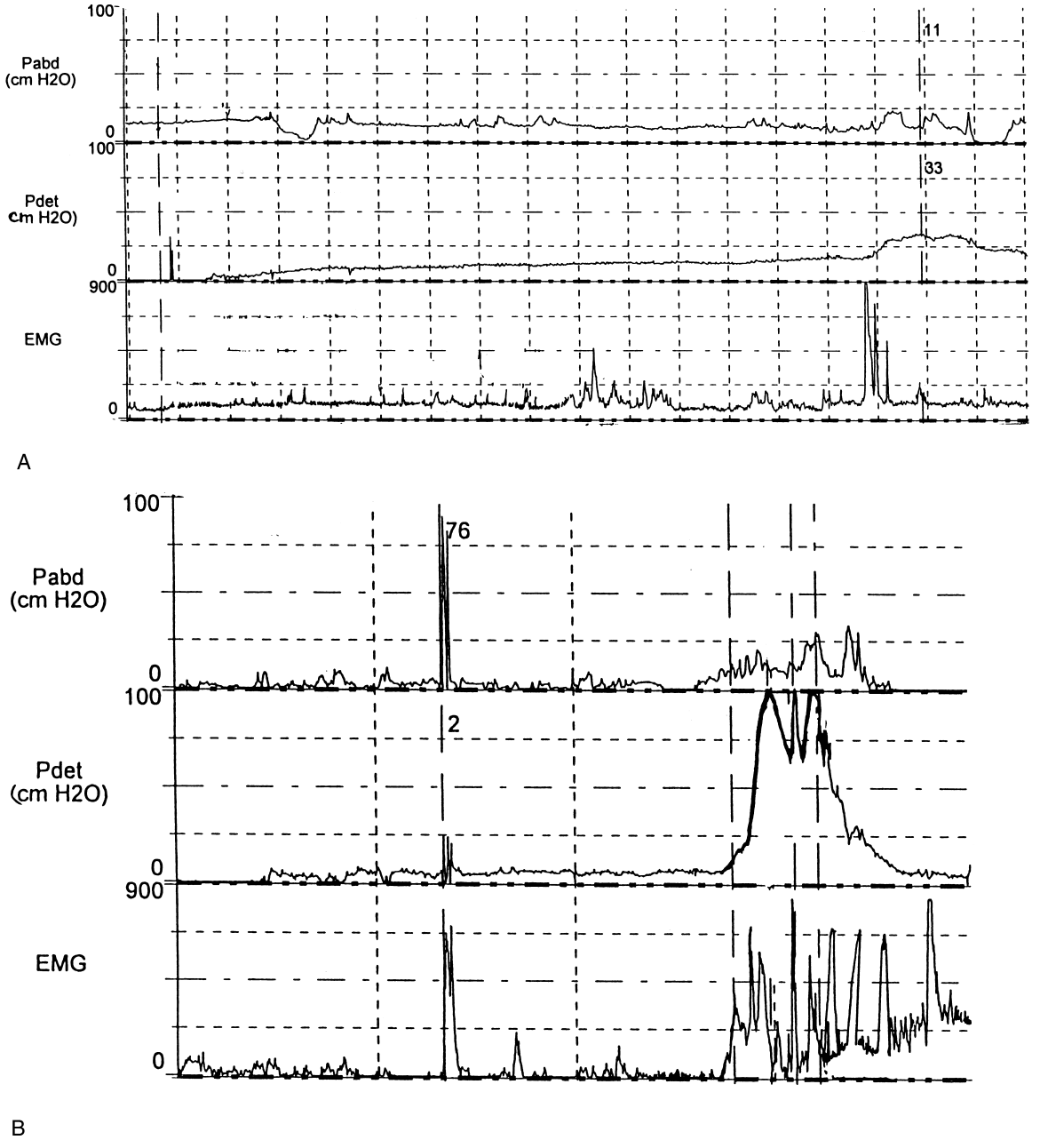

FIGURE 1. *Two urodynamic studies 3 months apart from the same patient (patient 1) showing urodynamic change from (A) detrusor hypocontractility to (B) detrusor external sphincter dyssynergia. Pabd = abdominal pressure; Pdet = subtracted detrusor pressure; EMG = electromyography.*

tions on 33 symptomatic patients during a 2.5-year period and found differences in 12, all of whom changed from having detrusor hypocontractility to having detrusor hyperreflexia. Wheeler *et al.*[4] found temporal changes in the urodynamic patterns in 55% of 18 patients in a retrospective study. Goldstein *et al.*[1] found changes in 4 of 9 patients undergoing repeated urodynamic evaluations. Blaivas and associates[17] reported on repeated cystometry in 6 patients with changing or persistent symptoms and found changes from detrusor hyperreflexia to detrusor areflexia in all. Piazza and Diokno[16] and Summers[19] also mentioned anecdotally that urodynamic patterns can change over time in patients with MS. Our purpose was to compare the urodynamic patterns in patients with MS who had undergone more than one study because of new or persistent lower urinary tract symptoms and to assess the trends over time with respect to both symptoms and urodynamic patterns. Our results showed changes in the pattern in 45% of 22 patients with new or persistent symptoms and changes in the pattern and/or compliance in 55%.

It has been reported that specific urologic prob-

lems in patients with MS cannot be effectively diagnosed and treated on the basis of the symptoms alone.[17] Kraus *et al.*[20] demonstrated a lack of correlation between symptom and quality-of-life scores and bladder dysfunction determined urodynamically. We add to this concept the idea that changes in symptoms are not necessary for there to be changes in bladder and urethral function. In our series, 43% of patients with no new urologic symptoms developed a change in the urodynamic pattern and/or compliance on the follow-up examination. This finding emphasizes that a urodynamic evaluation is necessary to properly treat the patient with MS and voiding symptoms.

Previously, it has been suggested that the evaluation of patients with MS should be limited to physical examination and postvoid residual urine measurement,[21] although most investigators support the importance of urodynamic assessment in directing therapy.[14,16,22,23] Interestingly, Sirls *et al.*[24] looked at the evaluation and treatment of 113 patients with MS and concluded that a limited evaluation of these patients is sufficient, without the routine use of electromyography. We believe that electromyography provides more precise diagnostic information and identifies the subgroup of patients with MS and detrusor external sphincter dyssynergia who have been noted to have an accelerated complication rate and a higher degree of treatment failure.[2,24,25]

A prospective analysis is required to address whether there are any significant predictive factors for neurourologic change in patients with MS and to confirm our data regarding urodynamic alterations without symptom progression.

Neuropathic bladder and urethral dysfunction secondary to MS appear to be unpredictable and dynamic. MS itself can behave in a similarly unpredictable manner, yet no correlation was found between the changes in the urodynamic patterns and the changes in the underlying MS pattern. Without being able to rely on symptoms to evaluate and treat vesicourethral dysfunction in these patients, it is clinically prudent to repeat urodynamic evaluations in symptomatic patients, even in patients who have persistent but not necessarily new symptoms. Appropriate management not only improves their quality of life, but may prevent complications in some patients, especially men with MS and detrusor external sphincter dyssynergia.[25]

CONCLUSIONS

A significant proportion of patients with MS both with and without new genitourinary symptoms will develop changes in their underlying urodynamic pattern and bladder compliance, often without changes in the MS pattern. Therefore, urodynamic evaluations should be repeated at regular intervals in symptomatic patients to optimize clinical management, reduce complications, and better enable these patients to cope with their lifelong disability.

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EDITORIAL COMMENT

In my opinion, the conclusions the authors have reached are exactly correct. The clinical implication is that patients with MS do experience changes in their voiding pattern, and thus the dictum has been (and remains) that one should avoid invasive or irreversible procedures designed to affect the voiding dysfunction in these patients unless absolutely necessary. Doubtless, all who treat such patients would agree with this. The authors have collected a good deal of data in the process of putting together this report. There are at least two caveats with respect to the data themselves:

1. If medication was instituted at the time of the first evaluation and not discontinued before the second evaluation (not mentioned in the article), it may be that any difference in urodynamic pattern has resulted from the treatment and not the disease.

2. One of the golden rules of urodynamic evaluations is that if the study does not reproduce the clinical symptoms, it is not an optimal study. Thus, in someone who complains of urge incontinence or frequency and urgency, if the urodynamic study shows only "hypocontractility," it may well be that under other circumstances (eg, ambulatory urodynamic assessment) the urodynamic study would have revealed quite different findings.

These potential problems notwithstanding, I would agree with the authors' conclusions that urodynamic evaluations should be repeated at regular intervals in these patients, but my primary purpose in doing so would be to assess the presence of risk factors that would prompt a change in therapy, and not necessarily just to notice a change in the urodynamic pattern in a laboratory urodynamic study. The real question, then, is, in how many patients did the new appearance of risk factors on the subsequent urodynamic evaluation prompt a change in therapy?

Alan J. Wein, M.D.
Division of Urology
University of Pennsylvania Health System
Philadelphia, Pennsylvania

PII S0090-4295(00)01071-2