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JERRY G. BLAIVAS

MS Bladder

Evaluation of Urinary Bladder Symptoms in Multiple Sclerosis

The physiology of micturition has remained a semimystery in the minds of many neurologists. Disturbances of bladder function occur only infrequently in most neurologic diseases, with the important exceptions of spinal cord injuries and multiple sclerosis. Thus, few neurologists gain a great deal of experience in dealing with them either diagnostically or therapeutically. The symptoms of bladder dysfunction are, naturally, completely nonspecific and are determined by the location of the lesion or lesions. In order to understand the diagnostic significance of the symptoms, it is necessary to obtain a reasonably clear understanding of the various anatomic and physiologic components of normal and abnormal excretory and storage functions. The same background information is required for initiating intelligent management—a process that is not easily separable from diagnosis.—CMP

Bladder function abnormalities occur at some time during the course of multiple sclerosis (MS) in approximately 50 to 80 percent of patients.[1–9,53] In 10 to 12 percent of MS patients, they form part of the initial symptom complex and comprise the sole initial complaint in approximately 1 to 2 percent.[9]

Strict diagnostic criteria must be established to evaluate the integrity of the neurologic pathways innervating the lower urinary tract. This is especially important because bladder symptoms are so prevalent in the general population. The mere presence of such symptoms is not sufficient evidence of a neurologic lesion, however. A detailed history and physical examination as well as urodynamic and electrophysiologic studies must substantiate the diagnosis of MS in a patient suspected of having the disease. Since the most common cause of urinary bladder symptoms is urinary tract infection, no patient should be subjected to more sophisticated testing until urinary tract infection has been excluded by urinalysis and appropriate cultures.

SYMPTOM ANALYSIS

Although most textbooks classify urinary bladder symptoms as being either obstructive or irritative in nature, we find it more convenient to consider them as related to problems of urinary storage, to problems of bladder emptying, or to problems of both storage and emptying (Table 1).[4,10] Symptoms that occur during the storage phase are either irritative or due to incontinence. Irritative symptoms include urinary frequency, urgency, urge incontinence, dysuria, suprapubic pain, a constant awareness of the urge to void, and urethral pain. The classic symptoms of bacterial cystitis also include dysuria, frequency, and urgency. Nevertheless, as many as 60 percent of women with these symptoms do not have bacterial infections.[11] Conversely, many patients with bacterial cystitis do not have dysuria or any pain; their only symptom may be frequency, urgency, or incontinence. Moreover, MS patients who already have neurogenic lower urinary tract symptoms may develop bacterial cystitis without any significant change in the symptoms. Progression to pyelonephritis may be insidious or may be heralded by sudden fever, flank pain, and systemic symptoms.

Urinary incontinence may be characterized in several ways. Patients may feel a severe urge to void and, if unable to reach a bathroom in time, void uncontrollably. This is called urge incontinence and is most often caused by detrusor hyperreflexia (involuntary bladder contractions). Other patients have no awareness of the urge to micturate, but when an involuntary contraction occurs, they suddenly and uncontrollably start to void. Depending upon the precise neurologic lesion, they may or may not be able to interrupt the stream once it has begun. In some patients interruption of the stream is accomplished by voluntarily contracting the external urethral sphincter which, on a reflex basis, abolishes the involuntary detrusor contraction and aborts micturition. In other patients this reflex is impaired, and even though the stream can be voluntarily interrupted, the involuntary detrusor contraction persists and incontinence ensues.[12]

Stress incontinence is characterized by the involuntary loss of urine during sudden increases in intra-abdominal pressure, such as occur during coughing, sneezing, and engaging in physical activity. It is most often

TABLE 1.

- Filling/Storage Disorders
- Bladder causes
  - Involuntary detrusor contractions
    - Detrusor hyperreflexia (due to suprasacral neurologic lesions)
    - Detrusor instability
      - Due to outlet obstruction
      - Due to inflammation
      - Idiopathic
  - Pain during bladder filling
    - Cystitis
    - Neurologic disorders (hyperesthesia)
    - Idiopathic/psychologic
  - Small bladder capacity
    - Fibrosis of bladder wall (radiation, turberculosis, interstitial cystitis)
    - Idiopathic/psychologic
- Outlet causes
  - Stress incontinence (due to poor transmission of intra-abdominal pressure to the urethra)
  - Sphincter malfunction
    - Post operative damage
    - Neurologic (sympathetic and parasympathetic denervation)
- Emptying Disorders
- Bladder causes
  - Neurogenic impairment (thoracolumbar or sacral lesions)
  - Myogenic impairment
  - Psychogenic
- Outlet causes
  - Anatomic obstruction
    - Vesical neck contracture
    - Prostatic (benign prostatic hyperplasia, cancer)
    - Urethral valves or structure
  - Functional obstruction
    - Vesical neck dyssynergia
    - External sphincter dyssynergia
- Filling/Storage and Emptying Disorders
- Detrusor-external sphincter dyssynergia
- Impaired detrusor contractility/sphincter dysfunction
- Bladder outlet obstruction/involuntary detrusor contractions
- Involuntary detrusor contractions/sphincter dysfunction

The Diagnosis of MULTIPLE SCLEROSIS

Edited by:

Charles M. Poser, M.D.

Lecturer on Neurology
Harvard Medical School
Attending Neurologist
Beth Israel Hospital
Formerly, Professor of Neurology
Boston University School of Medicine
Boston, Massachusetts

DONALD W. PATY, M.D., F.R.C.P. (C)

Professor and Head, Division of Neurology, Director of Multiple Sclerosis Research Program, University of British Columbia, Vancouver, British Columbia, Canada

LABE SCHEINBERG, M.D.

Professor of Neurology and Rehabilitation Medicine, Albert Einstein College of Medicine, New York, New York

W. IAN MCDONALD, F.R.C.P.

Professor of Clinical Neurology, Institute of Neurology, University of London, The National Hospital, Queen Square, London, England

GEORGE C. EBERS, M.D.

Associate Professor, Department of Clinical Neurological Sciences, University of Western Ontario, Director, Multiple Sclerosis Clinic, University Hospital, London, Ontario, Canada

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Joan Crawford Poser, M.A.

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an anatomic defect due to relaxation of the pelvic floor musculature with descent of the urethra, but it may occasionally be caused by extensive denervation of the bladder neck.[13,14] Overflow incontinence is usually described as a dribbling loss of urine due to severe bladder outlet obstruction. However, many patients are unable accurately to describe their incontinence and merely report that they find themselves wet. This not only occurs in patients with neurologic disorders, but also in neurologically normal persons.

Symptoms of impaired bladder emptying include difficulty initiating the urinary stream (hesitancy), diminished size and force of the stream, double voiding, postvoid dribbling, and total urinary retention. These symptoms are most often due to either bladder outlet obstruction or impaired detrusor contractility.

It has been documented that the relationship between urinary bladder symptoms and the underlying pathophysiology is inexact. In a series of 425 consecutive patients evaluated prospectively, only 45 percent of those who complained of urinary storage symptoms were actually observed to have an underlying pathophysiology consistent with those symptoms. Of patients with emptying symptoms, 75 percent had either bladder outlet obstruction or impaired detrusor contractility, but both storage and emptying disturbances were noted in only 54 percent of patients who complained of those symptoms.[15] In MS patients the correlation between symptoms and urodynamic observations was equally poor.[4,5]

Because of the disparity between urinary bladder symptoms and the underlying abnormality, we believe that persistent symptoms should serve more to alert the physician to the need for further urodynamic studies than to establish a specific diagnosis. The details of these studies will be discussed. Before undergoing any urodynamic studies, all patients with urinary bladder symptoms should first obtain a clear voided urine specimen for urinalysis and culture to exclude urinary tract infection. Unfortunately, many MS patients are unable to produce voluntarily a midstream sample either because of incontinence or urinary retention, so that these patients must be catheterized.

Vesicourethral Physiology

A detailed discussion of the anatomy and physiology of the lower urinary tract is beyond the scope of this chapter. Our purpose is to present a simplified overview of the normal mechanisms of bladder and sphincteric function.

The bladder and its sphincters function as a single physiologic unit whose main purpose is the storage and timely expulsion of urine (Fig. 1). Although many of the details of the anatomy and physiology remain controversial, it is generally agreed that the primary continence mechanism in both sexes is the smooth muscle of the vesical neck and proximal urethra. The urinary stream can be voluntarily interrupted by contracting the striated periurethral musculature or external sphincter, but this muscle plays only an ancillary role in preserving continence and, when the proximal smooth muscle is deficient, the external sphincter is generally incapable of maintaining continence.

Vesicourethral Innervation

Voiding Pathways

It is generally agreed that the micturition reflex is not a simple sacral cord reflex. Rather, it is integrated in the rostral brainstem in an area designated as the pontine micturition center (PMC).[12,16–19] Afferent pelvic parasympathetic stimuli resulting from bladder filling ascend in the lateral white matter of the spinal cord and synapse in the PMC.[20] Descending pathways from the PMC synapse with neurons of the hypogastric (sympathetic), pelvic (parasympathetic), and pudendal (somatic) nuclei.[18,21–23] The result of these neurologic events is a micturition reflex: relaxation of the external urethral

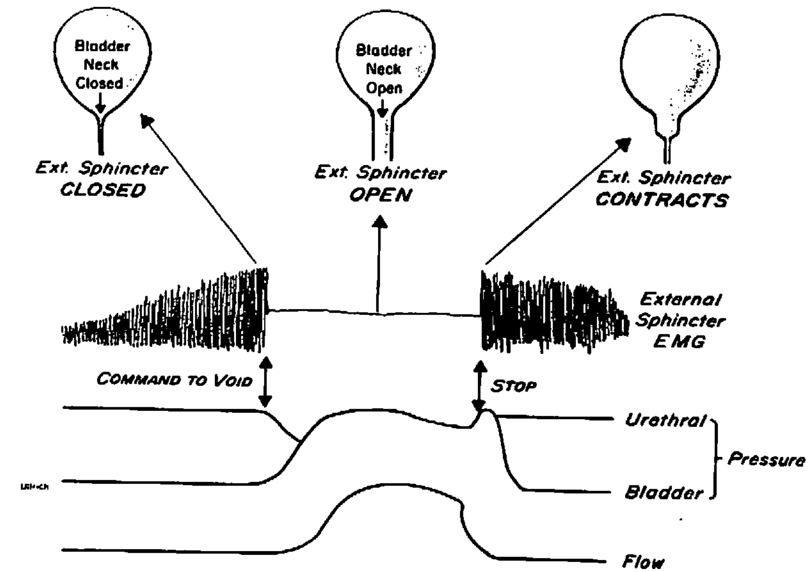


FIG. 1. Physiology of micturition. The primary continent mechanism in both sexes is the smooth muscle of the proximal urethra (bladder neck). As the bladder fills with urine, detrusor pressure remains fairly constant. Continence is preserved because urethral pressure remains greater than bladder pressure and the bladder neck remains closed. There is a gradual increase in electromyographic activity (EMG) of the striated external urethral sphincter. Voluntary micturition is heralded by the sudden and complete relaxation of the external urethral sphincter accompanied by a fall in urethral pressure. The detrusor contraction is marked by a rise in detrusor pressure. As voiding occurs the entire proximal urethra becomes isobaric with the bladder. Voluntary interruption of the stream is accomplished by contraction of the external urethral sphincter that occludes the urethra in its membranous portion.

sphincter, a fall in urethral pressure, initiation of detrusor contraction, opening of the vesical neck, and coordinated micturition.[12] The micturition reflex may be initiated or abolished at will (Fig. 2).

The sacral micturition center is located in the second through fourth segments of the sacral spinal cord and consists of the pelvic and pudendal reflex arcs.[19,24] The pelvic nerve is the major motor nerve to the bladder. Stimulation of the efferent pelvic nerve results in detrusor contraction. The pudendal nerve is the major motor and sensory nerve to the striated external urethral sphincter. Stimulation of its efferent fibers results in contraction of the external urethral sphincter. Micturition initiated by the sacral micturition center is uncoordinated or dyssynergic and is characterized by contraction (instead of relaxation) of the external urethral sphincter during voiding.[12,25]

Storage Pathways

The principal storage pathways are the suprapontine, frontopudendal, sympathetic, and pelvipudendal.[12,17–19]

Suprapontine neurologic pathways emanate from the frontal cortex, thalamus, hypothalamus, basal ganglia, limbic system, cerebellum, and other parts of the central

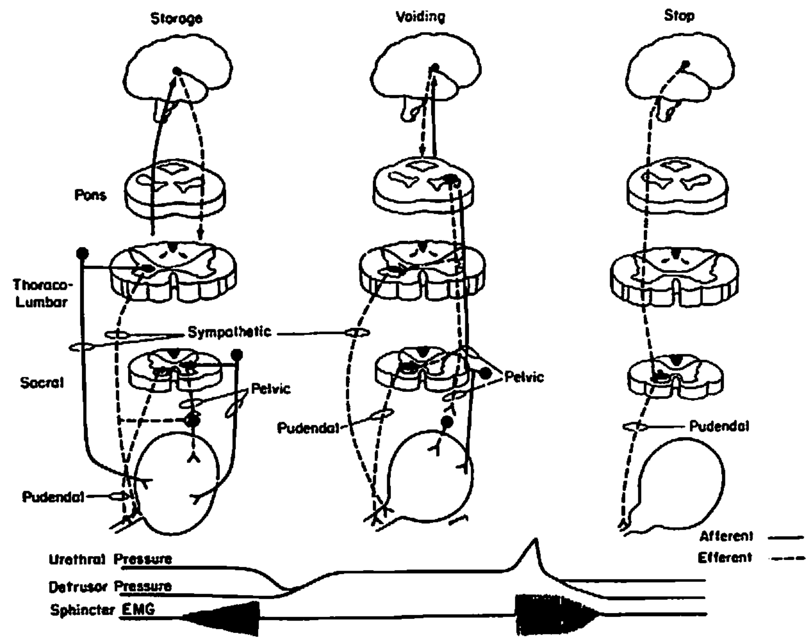


FIG. 2. Summary of major neurologic pathways involved in voiding. Storage: bladder distension results in afferent pelvic nerve discharge. After synapse in the pudendal nucleus efferent pudendal nerve impulses result in contraction of the external urethral sphincter. At the same time afferent sympathetic discharges traverse the hypogastric nerve. After synapse in the sympathetic nuclei efferent firing causes inhibition of transmission at the postganglionic parasympathetic neuron, which inhibits detrusor contraction, and increased tone at bladder neck. Net effect is that urethral pressure remains greater than detrusor pressure, facilitating urine storage. Voiding: afferent pelvic nerve discharges ascend in spinal cord and synapse in PMC. Descending efferent pathways cause inhibition of pudendal firing, which relaxes external sphincter; inhibition of sympathetic firing, which opens bladder neck and permits postganglionic parasympathetic transmission; and pelvic parasympathetic firing, which causes detrusor contraction. Net result is that relaxation of the external sphincter causes a decrease in urethral pressure, followed almost immediately by detrusor contraction, and voiding ensues. Stop: voluntary interruption of urinary stream. Descending corticospinal pathways emanate from the motor cortex synapse in pudendal nucleus, resulting in contraction of the external urethral sphincter. Urethral pressure increases above detrusor pressure, interrupting the stream.

nervous system that modulate the activity of the PMC and permit the voluntary control of micturition. Direct connections between the motor cortex and the pudendal nucleus (probably via the corticospinal tract) allow voluntary control of the external urethral sphincter.[12,17–19]

The role of the sympathetic nervous system has only recently been clarified.[18,21,22,26–29,36] During bladder filling, sympathetic receptors in the bladder wall send afferent discharges via the hypogastric nerve to the sympathetic neurons in the thoracolumbar portion of the spinal cord.

Efferent sympathetic firing has two important effects. First, via an alpha-mediated receptor, tonic contraction of the smooth muscle of the proximal urethra is maintained. Second, via another alpha receptor, transmission of excitatory impulses from the preganglionic to the postganglionic parasympathetic neurons is inhibited. Continence is maintained by helping to keep the vesical neck closed and by inhibiting parasympathetic stimulation of the detrusor.

The major neural pathway for storage, centered in the sacral cord, is the pelvipudendal reflex. During bladder filling, afferent pelvic nerve stimuli (probably via an interneuron) cause increased efferent activity in the pudendal nerve, which makes the external sphincter increase its tone.

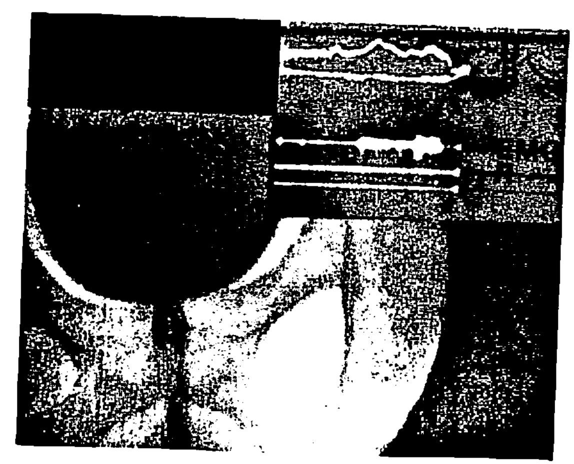
Effect of Neurologic Lesions on Vesicourethral Function

Neurologic Lesions Above the Pons

Neurologic lesions above the pons characteristically result in detrusor hyperreflexia.[12] Since the pontine micturition reflex pathways are intact, micturition proceeds normally from a physiologic standpoint (sphincter relaxation and detrusor contraction), but voluntary control is absent (Fig. 3). Depending upon the site and nature of the neurologic lesion, the patient may appreciate the involuntary detrusor contraction as an urge to void, or he may be totally unaware of it. Once aware of the contraction, he may be able to contract the external urethral sphincter voluntarily and interrupt the stream if the frontal cortex to pudendal nucleus pathway is intact (Fig. 4). If this pathway is not intact, he will be unable to contract the external urethral sphincter voluntarily. In some patients interruption of the stream by contraction of the external urethral sphincter causes abolition of the voluntary detrusor contraction, whereas in others the contraction continues unabated and the patient becomes incontinent once the external urethral sphincter fatigues.

The mere presence of involuntary detrusor contractions is not indicative of a neurologic lesion. They may be seen in patients with bladder outlet obstruction, such as benign prostatic hypertrophy,[30,31] or they may be idiopathic. Accordingly, the clinician should exercise caution when attributing involuntary detrusor contractions to a neuro-

FIG. 3. Detrusor hyperreflexia due to a neurologic lesion above the brainstem. Coordinated micturition is characterized by complete relaxation of the external urethral sphincter EMG, sustained detrusor contraction, and complete opening of the vesical neck. The only abnormality is the fact that the micturition reflex occurs without the patient's control. U: urethral pressure; B: intravesical pressure; R: rectal pressure; EMG: sphincter electromyography.



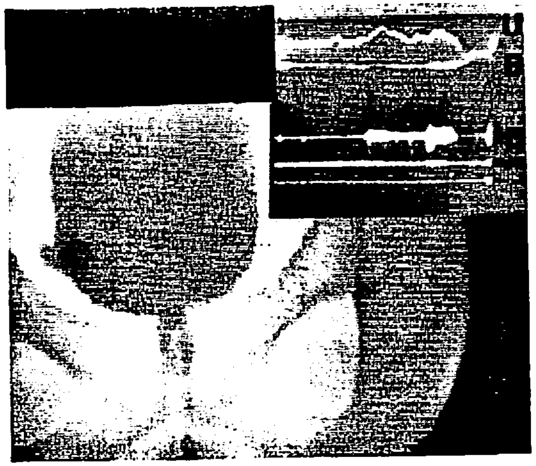


FIG. 4. Detrusor hyperreflexia due to a suprapontine neurologic lesion that does not interfere with the motor cortex to pudendal nucleus pathway. A micturition reflex is initiated involuntarily. Once the patient is aware of voiding, he voluntarily contracts the external sphincter (increased EMG activity at far right of tracing) and interrupts the stream. U: urethral pressure; B: intravesical pressure; E: sphincter electromyography.

logic lesion unless there is historical or physical evidence to corroborate this impression.

Neurologic Lesions That Interrupt the Pontine-Sacral Micturition Pathways

Neurologic lesions interrupting the pontine-sacral axis result in uncoordinated micturition.[12,25] This uncoordinated micturition may vary from frank detrusor-external sphincter dyssynergia (DESD) at the one extreme to low-magnitude, brief duration, involuntary detrusor contraction at the other. DESD is characterized by an involuntary contraction of the external urethral sphincter during an involuntary detrusor contraction (Fig. 5).[12,25,32,33] In its classic forms, DESD is pathognomonic of a neurologic lesion interrupting the pontine-sacral axis.[25] Nonetheless, several cystometrogram or electromyogram (EMG) abnormalities and artifacts may simulate DESD. These will be discussed in more detail later.

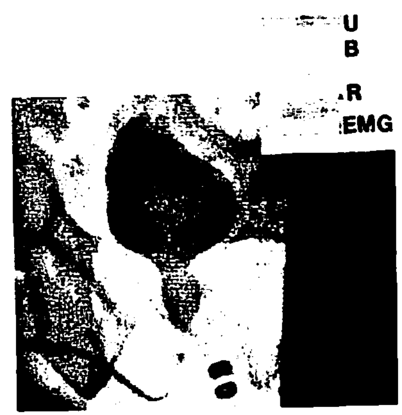


FIG. 5. Detrusor-external sphincter dyssynergia due to interruption of the PMC. During the involuntary detrusor contraction, there is complete obstruction at the membranous urethra because of contraction of the external urethral sphincter. U: urethral pressure; B: intravesical pressure; R: rectal pressure; E: sphincter EMG.

Neurologic Lesions Interrupting the Sacral Micturition Center

Complete abolition of the parasympathetic and pudendal reflex pathways results in detrusor areflexia and absence of voluntary or involuntary contractions of the external urethral sphincter. When the sympathetic pathways are intact, the vesical neck and proximal urethra remain closed. These patients have urinary retention because of the competent (vesical neck) sphincter, but may have overflow incontinence.[12,24,34,35] Cystometric studies document detrusor areflexia and absence of sensation. However, after approximately 6 to 12 months, patients usually develop a hypertonic detrusor characterized by a steep rise in the tonus limb of the cystometrogram.[34,37] Perianal sensation is absent, the bulbocavernosus reflex is abolished, anal sphincter tone is diminished, and voluntary anal control is lost. In the absence of a myopathic disorder, these observations are pathognomonic of a neurologic lesion involving the sacral micturition center.

There are few data concerning lesions that selectively involve the afferent or efferent sacral neurologic pathways, but McGuire[38] has suggested that isolated neurologic lesions of the posterior (afferent) limb of the parasympathetic nerve results in detrusor areflexia and a hypotonic bladder, that is, a bladder than accepts large volumes with little, if any, rise in detrusor pressure. A selective lesion of the anterior (motor) route usually leads to an areflexic, hypertonic detrusor.[38]

Neurologic Lesions Interrupting Sympathetic Pathways

Sympathetic neurologic lesions are exceedingly difficult to document in human beings; accordingly, most of the lesion data have been obtained in the cat.[18,21,22,26–29,36] The human data have been obtained principally in the following circumstances: studies of patients who have sustained spinal cord

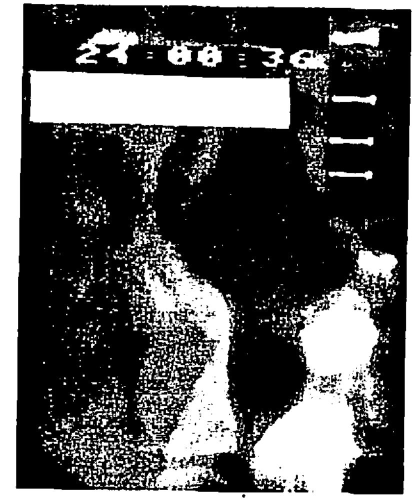


FIG. 6. Open vesical neck due to sympathetic decentralization. During bladder filling, without a rise in intravesical pressure, the entire proximal urethra is visualized and is isobaric with the bladder. B: intravesical pressure; R: rectal pressure; E: sphincter electromyogram.

infarction[14] or who have undergone abdominoperineal resection of the rectum;[39] pharmacologic studies,[40] and histochemical studies.[41] The preponderance of evidence suggests that sympathetic injury results in loss of the proximal (vesical neck) sphincteric mechanism characterized by diminished proximal urethral closure pressure and an open vesical neck (Fig. 6).

Diagnostic Methodology

Because of the relatively poor correlation between urinary bladder symptoms and underlying pathophysiology, it is necessary to perform physiologic tests of bladder and sphincteric function in order to determine whether there is a neurologic lesion that might account for the patient's symptoms. Again, before urodynamic testing all patients should have a urinalysis and culture. When infection is present (colony counts greater than 10^5/ml), it should be treated with appropriate antibiotics, but if persistent infection occurs despite treatment, it may be necessary to proceed with the urodynamic studies while the patient continues to be treated with antibiotics. Microscopic hematuria demands complete urologic investigation, including intravenous pyelogram, cystoscopy, and urinary cytology in order to exclude occult neoplasm.

Cystometry

Cystometry is the graphic representation of bladder pressure as a function of volume.[42] However, much more diagnostic information may be obtained from a cystometric examination than the numerical values of pressure, volume, and capacity. If the main goal of urodynamic investigation is to reproduce the patient's symptoms while making physiologic observations and measurements, the cystometric examination is the prototype. The patient voids, a catheter is placed in the bladder, and postvoid residual urine is measured. Infusion of fluid (water or saline) or carbon dioxide is begun at a constant predetermined rate up to 300ml/min. During bladder filling, the patient is instructed to try neither to void nor inhibit micturition, but to report all sensations to the examiner. At slow rates of fill, detrusor pressure remains almost constant as the bladder accomodates to increasing volume by increasing wall tension at the expense of pressure (Fig. 7A). At more rapid filling rates, the viscoelastic properties of the bladder result in an increasing slope of the cystometric curve.[43] The volume at which subjects normally first experience sensations of bladder filling, first urge, and severe urge to void are variable and serve as only a subjective guideline. However, if the patient experiences his usual symptoms, such as urgency, pain, or incontinence, the physiologic measurements of pressure and volume serve as objective data to explain the etiology of the symptoms.

A sudden rise in intravesical pressure may be due either to a detrusor contraction or to increased intra-abdominal pressure from cough or straining. Simultaneous measurement of intra-abdominal pressure via a rectal balloon catheter is useful for making this distinction (Fig. 7B). When the sudden rise in intravesical pressure is interpreted by the patient as an urge to void, the presence of an involuntary detrusor contraction is confirmed. Although most involuntary detrusor contractions are greater than 15cm water, they may be of less magnitude, and some patients actually void voluntarily with no measurable rise in detrusor pressure (Fig. 7C). Any detrusor contraction that is not volitional is an involuntary detrusor contraction. Once it has begun, the patient may or may not be aware of it and may or may not be able to abort it. When an involuntary detrusor contraction occurs in a patient with a diagnosable neurologic condition known to be associated with abnormalities of bladder innervation, and condition is termed detrusor hyperreflexia. The same cystometric observations in a patient whose neurologic examination is normal is called detrusor instability.

A steep rise in intravesical pressure during filling is termed detrusor hypertonia and is usually indicative of either chronic inflammation with scarring or parasympathetic neuronal damage. The absence of detrusor contraction during cystometry is not necessarily abnormal and is encountered in up to 50 percent of otherwise normal women. It has been our experience that the most common misconception about cystometry is the misinterpretation of a curve demonstrating an acontractile bladder. Unless there are other neurologic observations to suggest lower neuron disease, we consider such a cystometric curve to be normal.

Because of the difficulty in diagnosing subtle lower motor neuron disease affecting the bladder, it has been suggested that the bethanechol denervation supersensitivity test may be used to distinguish neurologic from non-neurologic causes of bladder acontractility during cystometry.[44,45] It is our opinion that the false positive rate of this examination as currently performed precludes its use in patients whose neurologic examination is normal.[46,47]

Electromyography

EMG of the external urethral sphincter should always be performed with simultaneous cystometry.[42,48–50] It has two main applications in urodynamic testing. First, it can be used to detect whether the perineal floor muscles are contracting or relaxing at any given instant. Second, when individual motor unit action potentials are observed, the integrity of these muscles and their nerve supply can be assessed. On the basis of standard EMG criteria, combined with clinical observations, the innervation of the external urethral sphincter (and by implication, the bladder) may be classified as normal, upper motor neuron lesion, lower motor neuron lesion, or mixed lesion.[48] It should be remembered, however, that the pelvic floor consists not only of the external urethral sphincter, but also of the bulbocavernosus and ischiocavernosus muscles, the external anal sphincter, and the levator ani. In MS there may be a different degree of dysfunction in these muscles,[3,51] and multiple sampling sites may be necessary.

Since the EMG detects striated-muscle activity, which is under complete voluntary control, it is impossible to evaluate EMG activity unless the examiner is certain of what the patient is trying to do. For example, if a patient experiences an involuntary detrusor contraction and attempts to abolish it by contracting the external urethral sphincter, the EMG response would be considered normal, as in Figure 4. However, if the sphincter contracts involuntarily, as in Figure 5, a diagnosis of DESD can be made.

Urethral Pressure Profile

Although we believe that determination of the urethral closure pressure profile is not necessary for routine urodynamic investigation, it may be somewhat helpful in diagnosing denervation of the proximal urethral segment, including the bladder neck.[13,14,39] The urethral pressure profile is performed by slowly withdrawing a catheter while measuring the pressure at intervals along the urethra.[42] Normally, the entire proximal half to two-thirds of the urethra has a pressure considerably greater than the bladder pressure, this length being designated as the functional urethral length. In patients with denervation, the proximal urethral segment remains isobaric with the bladder. With the single exception of this observation and that of very low urethral pressures (that is, less than 20cm of water), there seems little of diagnostic value in the urethral pressure profile.

Voiding Cystourethrography

The radiographic visualization of the lower urinary tract during both storage and voiding is an extremely useful diagnostic procedure, especially when performed with fluoroscopic monitoring. When bladder outlet obstruction has been diagnosed from urodynamic studies, the site and nature of the obstruction are usually apparent radiographically. The presence or absence of vesicourethral reflux may be detected and the diagnosis of sympathetic decentralization may be suggested by noting an open vesical neck during bladder filling. There is usually a characteristic radiographic appearance for DESD, prostatic obstruction, and urethral strictures.

Combined Studies

The simultaneous measurement of multiple urodynamic parameters is the most foolproof, artifact-free method of diagnosing lower urinary tract conditions. Synchronous/video/pressure/flow/EMG studies are currently employed, however, at only a few diagnostic centers throughout the world.[42,52]

Sacral Evoked Responses

A recent development in electrophysiologic testing is the measurement of the latency of

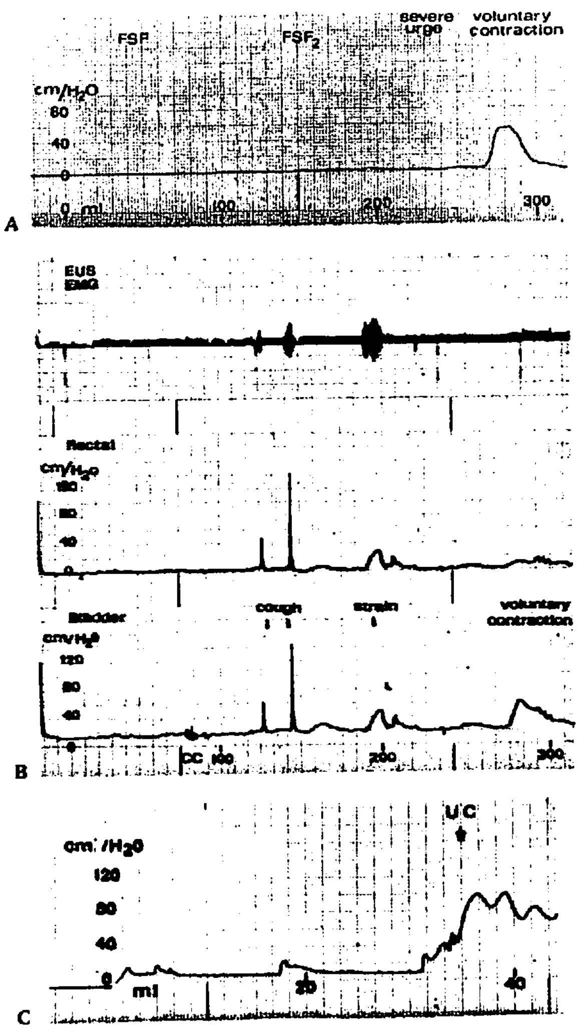


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FIG. 7. A: Normal cystometrogram. Note little rise in detrusor pressure during bladder filling. B: Simultaneous rectal and intravesical pressure with sphincter EMG. Note that each increase in intra-abdominal pressure is transmitted to the bladder and might stimulate a detrusor contraction, if both pressures were not being measured. C: Detrusor hyperreflexia. UC: involuntary detrusor contraction.

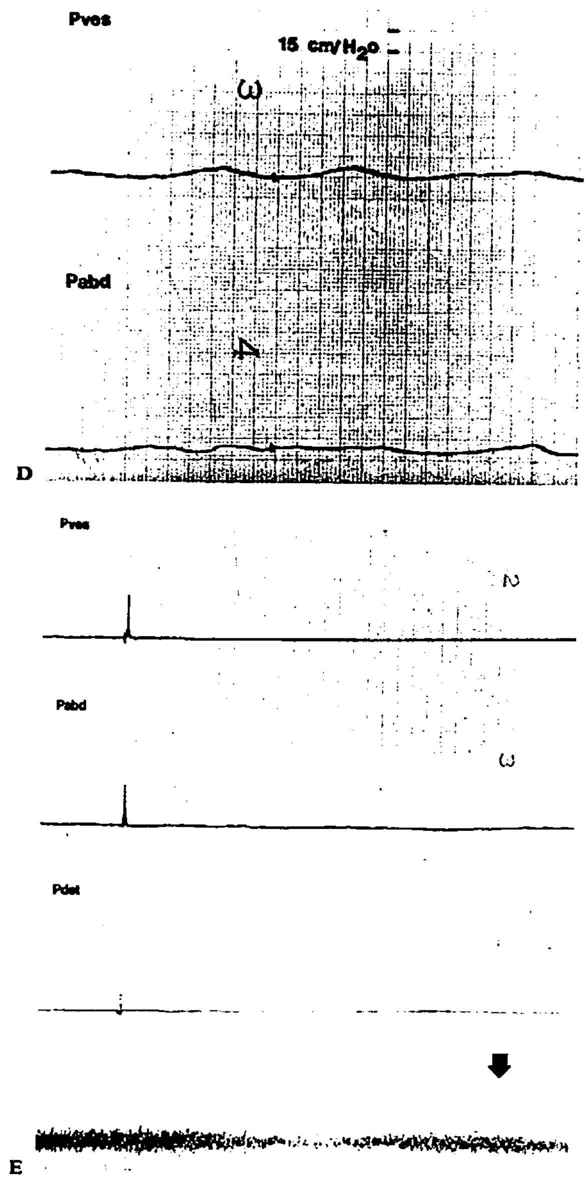

FIG. 7. D: Low-magnitude detrusor contraction. E: Absence of voluntary detrusor contraction. This is not considered abnormal unless there are other observations on physical examination to suggest lower motor neuron disease.

the bulbocavernosus reflex.[54-59] Clinically, the bulbocavernosus reflex is elicited by briskly squeezing the glans, penis, or clitoris and observing or feeling the reflex contractile response of the external anal sphincter or bulbocavernosus muscle. Alternatively, the reflex may be stimulated by pulling the balloon of a Foley catheter against the vesical neck. The bulbocavernosus reflex is present in almost all normal men and in approximately 50 percent of patients with an incomplete lower motor neuron lesion.[60,64] Since it is difficult to grade the reflex clinically, measurement of the bulbocavernosus reflex latency offers a more quantitative means of evaluating the sacral reflex arcs.

When one side of the penis is stimulated electrically, there is a bilateral contractile response in the bulbocavernosus muscle.[54,56,58] The sensory limb of the reflex is probably mediated by small diameter myelinated pudendal nerve afferent fibers whose conduction velocity is approximately 20m/sec.[56] The efferent limb is conducted by pudendal motor fibers whose conduction velocity is 50 to 60m/sec.[59] Preliminary data suggest that the reflex is polysynaptic, traversing at least several spinal cord segments.[55] A normal response is seen in Figure 8.

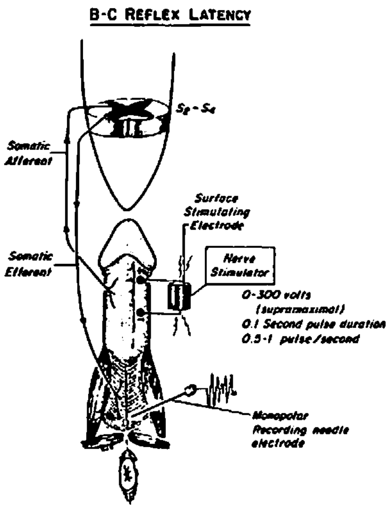

FIG. 8. Measurement of the latency of the bulbocavernosus reflex.

Somato-sensory Evoked Potentials

Another recent application of electrophysiologic techniques to the lower urinary tract is the use of somatosensory evoked potentials.[61,62] The neuroanatomic pathways involved in somatosensory stimulation are of somatic origin and, hence, traverse the spinal cord in the dorsal column, whereas those for the micturition reflex are in the lateral horn.[20] Accordingly, it is possible for a neurologic process, such as a plaque of MS, to involve one pathway and spare the other. In order to overcome this obstacle, several investigators have begun preliminary investigations in eliciting evoked potentials by stimulating the vesical neck, proximal urethra, and bladder.[63]

DIAGNOSTIC CRITERIA IN MULTIPLE SCLEROSIS

Diagnostic Formulation

On the basis of a carefully obtained history, physical examination, and urodynamic studies as outlined, the following abnormalities are considered to be directly caused by neurologic deficits.

Detrusor-External Sphincter Dyssynergia

The diagnosis of DESD indicates the presence of a neurologic lesion interrupting the pontine-sacral micturition pathways.[12,25] Special caution should be exercised when diagnosing DESD to be certain that it is not being mimicked by voluntary attempts to suppress micturition, straining to void (with a reflex increase in EMG activity) and EMG artifacts caused by a variety of mechanical and electronic problems.

Electromyographic Abnormalities

The presence of spontaneous potentials in the form of positive sharp waves or fibrillation, the absence of any electrical activity, or an increased number of polyphasic potentials, are indicative of a neuropathic or myopathic process. Often EMG examination of the perineal musculature is insufficient to distinguish myopathy from neuropathy, and further EMG and clinical testing, including muscle biopsy, may be necessary to make the proper diagnosis.

Prolongation of Bulbocavernosus Reflex Latency

Prolongation of the bulbocavernosus reflex latency is indicative of a neuropathy involving the pudendal nerve. This technique, however, does not distinguish peripheral from central neurologic lesions.

Prolongation of Genitocerebral Evoked Response

Although the diagnostic utility of the genitocerebral evoked response is in its infancy, and normal values have not been well established, sufficient data are available to suggest that abnormalities of the evoked response will be definitive for establishing the presence of neurologic lesions.

Physical Observations Indicative of a Sacral Neurologic Lesion

The absence of the bulbocavernosus reflex in the male is indicative of a neurologic lesion involving the second, third, or fourth sacral segments of the spinal cord. Approximately 50 percent of men with incomplete lower motor neuron lesions have a detectable reflex, and the reflex is present in only about 70 to 80 percent of normal women.[60] The absence of perianal sensation, lax anal sphincter tone, and the inability to contract and relax the external anal sphincter voluntarily are further evidence of a neurologic lesion. Nonetheless, the inability to contract and relax the external anal sphincter voluntarily is not by itself considered to be an objective neurologic abnormality, as many patients simply do not comply with the examiner's request because of embarrassment.

Observations Compatible with, But Not Diagnostic of, Neurologic Lesions

Involuntary Detrusor Contractions

Any suprasegmental neurologic lesion may result in detrusor hyperreflexia. However, non-neurologic conditions may also be associated with involuntary detrusor contractions. Approximately 60 percent of men with prostatic obstruction have detrusor instability, as do 5 to 10 percent of normal women who undergo provocative cystometry. Thus, the presence of involuntary detrusor contractions may not be used as objective evidence for a neurologic lesion unless it is associated with DESD.

The Absence of Voluntary Detrusor Contraction During Cystometry

The inability to generate a voluntary detrusor contraction during a cystometric examination is not in and of itself abnormal. Approximately 25 to 60 percent of normal women do not have a detrusor contraction during cystometry, and at least 5 to 10 percent of normal men fail to evince one. This is believed to be caused by psychic inhibition because of the embarrassing and unfamiliar setting in which the examination is performed. Detrusor areflexia is defined by the International Continence Society as an acontractible bladder due to a neurologic lesion. However, they offer little advice on distinguishing a neurologic from a psycho-

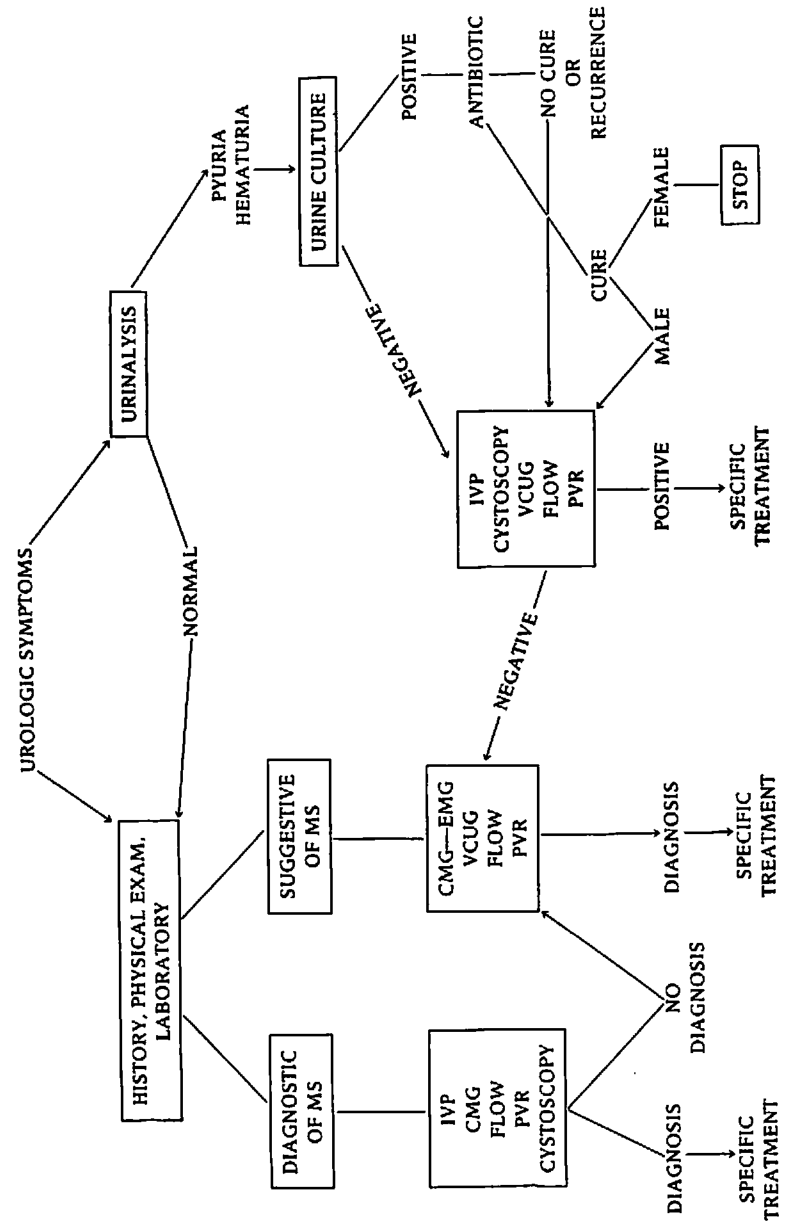

FIG. 9. Algorhythm for the diagnostic evaluation of bladder symptoms in MS. See text for details. IVP: intravenous pyelogram; VCUG: voiding cystourethrography. PVR: postvoid residual urine; CMG: cystometry. EMG: electromyography of the external urethral sphincter.

logic or myogenic cause of failure of detrusor contraction. At present, associated neurologic observations must be relied upon, such as those of sacral denervation, to make a diagnosis of detrusor areflexia.

POSITIVE BETHANECHOL DENERVATION SUPERSENSITIVITY TEST

As originally described by Lapides and coworkers[45] and modified by Glahn,[44] a positive bethanechol denervation supersensitivity test is reportedly indicative of a neurologic lesion involving the bladder. However, in several series the false positive rate varied from 10 to 50 percent.[44,46,47] We consider a positive bethanechol test suggestive, but not diagnostic, of a neurologic lesion.

SENSORY ABNORMALITIES

Since women with psychogenic urinary retention usually deny any bladder sensation and since the exact volumes at which the usual sensory parameters occur are highly variable from patient to patient, it is difficult to ascribe changes in bladder sensation to neurologic abnormalities. A suggested urodiagnostic algorhythm is illustrated in Figure 9.

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