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Review Article

Neurogenic Urinary Retention

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Abstract: This review article on neurogenic urinary retention is divided into three main sections. The first covers the neuroanatomy of the bladder and urethral sphincters, developing the peripheral innervation as well as the spinal cord organization and the cortical and subcortical brain control of micturition. The second discusses the main central and peripheral neurological lesions and diseases causing urinary retention. The last section gives an updated view of the neurophysiological techniques which are now available to test the central and peripheral pathways controlling micturition.

Keywords: Neurology; Urinary retention

The inability to urinate is a common problem encountered by urologists and urogynecologists. Retention of urine is usually due to mechanical obstruction of the urethra, especially in men, but may also result from detrusor failure, failure of sphincter relaxation or incoordination of the detrusor and urinary sphincters.

The aim of this article is to discuss the neuroanatomy of the bladder and urethral sphincters, the various etiologies of neurogenic retention and the new neurophysiological approaches to its understanding.

Neuroanatomy of the Bladder and Urethral Sphincters [1]

The neuroanatomy of the bladder and urethral sphincters is presented in three parts: the peripheral innervation, consisting of both autonomic and somatic innervation, the spinal cord organization, and the cortical and subcortical brain control.

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Peripheral Innervation

Autonomic Innervation of the Bladder:

Parasympathetic efferent nerve supply. The bladder muscle is diffusely and richly supplied with cholinergic efferent nerve fibers: each efferent axon delivers the acetylcholine transmitter to a location within a muscle fascicle. From the site of each varicosity transmitter, molecules diffuse in all directions, exciting whichever smooth muscle cells the transmitter reaches in sufficient quantity [2,3]. This release of transmitter within a group of muscle cells is a mode of innervation ideally suited to the bladder, as it provides a gradient of muscle cell excitabilities with the possibility that excitability thresholds (varicosity – muscle cell distances) are automatically decreased by mechanical distension of the bladder [1]. The cell bodies of these parasympathetic fibers lie either within the pelvic inferior hypogastric plexus or within the vesical plexus [4] situated principally in the adventitia of the bladder base. These postganglionic fibers are supplied by preganglionic fibers with cell bodies in Onuf's sacral nuclei which consist of bilateral symmetrical groups of anterior horn cells, making up a distinct nucleus in the S2 and S3 spinal segments, and including both autonomic and somatic components [5]. There is clear evidence that acetylcholine liberated by the parasympathetic efferent neurons are responsible for detrusor contraction during micturition [6].

Sympathetic efferent nerve supply. Noradrenergic terminals are concentrated mainly at the bladder neck [7]. Some adrenergic endings are also found throughout

the bladder in small numbers [8] and may correspond to the sympathetic innervation of blood vessels [9]. It is suggested that sympathetically mediated inhibition of the bladder depends on indirect inhibition of the excitatory parasympathetic supply within the ganglia of the pelvic plexus, where both types of cell bodies are present [10]. This suggests a sympathetic–parasympathetic interaction for the regulation of vesical musculature [11]. The preganglionic fibers have cell bodies in the intermediolateral grey areas of the thoracic and lumbar segments T10–L2. Noradrenaline, acting as a transmitter for sympathetic neurons can have excitatory or inhibitory effects, according to the predominant receptor type. In some mammalians, alpha receptor sites producing contraction in response to noradrenaline binding have been shown to predominate in the bladder base, while beta receptors producing relaxation are predominant in the vault [1].

Parasympathetic and sympathetic afferent nerve supply. Except for some sparse pacinian corpuscles, vesical afferent axons terminate as free nerve endings [2,12]. These are mainly mechanoreceptors acting as tension receptors responding to detrusor distension and contraction [13]. Because individual receptors have different thresholds, the afferent system can transmit pressure status over a range of pressures by recruiting different receptors at different levels of pressure. Vesical afferents in the thoracolumbar nerves become active only during marked bladder distension [14], and their transection has no effect on voiding [15]. Lumbar afferents are said to convey conscious touch from neck mucosa, distension and pain [16]. Some individuals may have thermal sensation in the bladder; others do not [17]. Vesical afferents in the sacral nerves are silent when the bladder is empty and begin firing at small bladder volumes [15]. At volumes beyond two-thirds of the micturition threshold, sacral afferents participate in the reflex inhibition of wall tension [15]. Sacral afferents convey conscious touch and pain from the mucosa and a sense of bladder fullness [18], leading to the perceptions of desire, urgency and emptying. They are then essential for normal micturition.

Autonomic Innervations of the Urethral Smooth Muscle: Sympathetic and parasympathetic efferent and afferent fibers innervate the urethra. Cholinergic stimulation produces smooth muscle contraction, producing the shortening and widening of the urethra during micturition, along the detrusor contraction. Adrenergic axons also innervate the internal smooth muscle, which has predominantly alpha-excitatory adrenergic receptors [19].

Somatic Innervation of the Striated Urethral Sphincter: The external urethral sphincter consists of periurethral and intramural predominantly fast-twitch striated muscle fibers [20]. It is innervated by the motor fibers that come from the dorsal components of Onuf's sacral nuclei [5].

Spinal Cord Organization

Spinal reflexes within the spinal cord have been reported [21]. Bladder–pelvic nerve afferents activate interneurons that ascend to synapse on sympathetic preganglionic neurons that inhibit detrusor and facilitate internal sphincter excitability [15,22]. Urethral, and probably detrusor, tension receptors activate interneurons that synapse on parasympathetic preganglionic neurons to facilitate detrusor contraction [22]. Additional interneurons inhibit somatic efferent neurons to the external urethral sphincter [23]. Pudendal nerve afferents activate interneurons that inhibit preganglionic neurons to the detrusor, and excite somatic neurons to the external urethral sphincter [23]. Interneurons and projection neurons decussate, so that efferent neurons in the spinal cord are bilaterally activated, complementing the peripheral nerve decussation and the bilateral innervation of the bladder and urethra.

As far as spinal cord pathways are concerned, it seems that there are one ascending and three main descending pathways. The ascending pathway [6] consisting of afferent fibers coming from the vesical tension and urethral mucosal receptors is located superficially in the lateral funiculus. Projection neurons also participate in the ascending pathway via the spinothalamic tract for conscious perception [23]. The descending pathways come from the mid-brain, pons and medulla oblongata, and descend in the reticulospinal tract to excite the preganglionic neurons to the detrusor and inhibit the sphincters, to inhibit neurons of the striated sphincter, and to inhibit detrusor neurons and excite those to the sphincters for continence [6]. Nevertheless, all the different paths from and to the bladder are not yet fully understood.

Cortical and Subcortical Brain Control

The cortical localization of the micturition area is on the medial surface of the cerebral hemisphere, in the paracentral lobule just anterior to the central sulcus [24]. This localization is consistent with that of the motor representation of the pelvic muscles on the cortex, and has been confirmed in humans by transcutaneous electrical and magnetic stimulation of the motor cortex [25,26]. The cortical localization of the sensory input from these organs is in the adjacent sensory cortex, also on the medial surface of the hemisphere [27]. The subcortical brain regions which are correlated to the bladder function include the thalamus [28], the basal ganglia [29] and the mesencephalic–pontine–medullaris reticular formation [6]. The anterior vermis and the fastigial nucleus are also concerned with micturition.

The thalamus is the main relay from the afferent ascending pathways from the bladder and urethral receptors. The basal ganglia influence detrusor contraction [29]: in animals, the electrical stimulation of selected nuclei results in detrusor areflexia, whereas

selected destruction results in detrusor hyperexcitability.

The micturition center is located in the reticular formation of the mid-brain, pons and medulla oblongata [6]. Spontaneous detrusor reflex contractions are associated with neuronal activity in the pontine detrusor motor nucleus. In animals, ablation of the pontine detrusor area results in a permanent abolition of micturition. The afferent input from the pelvic floor have synapses with Purkinje's cells of the anterior vermis of the cerebellum, which themselves influence the fastigial nucleus which has connections with the pontine micturition center [30].

Neurogenic Urinary Retention

Urinary retention resulting from a neurological etiology is less common from obstructive causes in men [31], but seems to be one of the first known causes in females [32]. The causes of neurogenic urinary retention may be divided into three main categories: cortical and subcortical lesions, spinal cord lesions above the conus medullaris, and lesions of the conus medullaris and motor and sensory nerves to the bladder.

During normal micturition, the smooth and striated urethral sphincters relax before or at the onset of detrusor muscle contraction, due to a reflex inhibition of sympathetic and somatic spinal neurons by descending pathways from the brain [23].

When a lesion occurs above the spinal micturition center (L2 vertebral level, S2–S4 spinal segments), urinary retention in general is due to detrusor–sphincter dyssynergia. This dyssynergia results from an involuntary increase in urinary sphincter tone during attempted voiding, so that the detrusor muscle contracts against a closed urethral sphincter, and urine flow is poor or absent [33]. This incoordination between detrusor and sphincters may be recognized by the simultaneous presence of bursts of EMG activity in the striated urethral sphincter, and the high intravesical pressure due to detrusor contraction [34]. These simultaneous recordings are required for the accurate recognition of this disorder. Detrusor–sphincter dyssynergia is important because it leads to an increase in intravesical pressure, with persistant residual urine and risk of recurrent urinary infection. Moreover, there is a risk of hydronephrosis and chronic renal failure if untreated, e.g. by alpha-blocking agents, sphincterotomy, intermittent catheterization or sacral stimulation [35]. Although detrusor–sphincter dyssynergia is said to be due to neurological diseases, some cases appear to be idiopathic and the cause of this micturition disorder is not known. When the neurological lesion affects the conus medullaris or the pelvic nerves, the loss of sensation of bladder fullness leads to detrusor muscle distension, muscle atony, and poor and inefficient contraction. The result is a flaccid neuropathic bladder with increased capacity and large amounts of residual urine, which favors urinary infections. It is important to realize, however, that mixed peripheral and central lesions are not uncommon, that incomplete lesions also occur [36], and that detrusor areflexia may also occur with central lesions [37].

Cortical and Subcortical Lesions

Andrew and Nathan [38] were the first to describe disturbances of micturition in lesions affecting the anterior frontal lobes and nowhere else in the cortex [38,39]. They described 3 patients out of 9 with frontal tumors who had retention of urine, but only frequency of micturition, urgency and incontinence were described in the 7 out of 50 consecutive frontal tumors without fits in the Maurice-Williams series [39]. Strangely enough, excision of the tumor cured the symptomatology, but frontal uni- or bilateral lobectomies typically gave rise to no disorder of micturition [39]. In more recent studies analyzing voiding disorders in patients with acute and chronic cerebrovascular accidents (CVA) [37], the majority of the patients had uninhibited relaxation of the sphincters during involuntary bladder contraction. Nevertheless, 7 patients out of 33 had poor bladder contractions. Detrusor areflexia has been described during the initial phase of a CVA, followed by detrusor hypereflexia, but here only 1 demonstrated evidence of recovery, the others persisting in the underactive state. Interestingly, 3 out of the 6 patients with poor bladder contraction had both involvement of the basal ganglial and the frontal lobe. Further studies are needed, studying pure focal destructive lesions in a chronic state to try to solve the complex problem involved in predicting bladder dysfunction induced by cortical lesions. Nevertheless, bladder function prior to the CVA will never be known, and the high prevalence of involuntary bladder contractions in the normal elderly [40] will always bias the study.

While a unilateral lesion of the putamen does not affect micturition [41], even mild bilateral putamen lesions are associated with incontinence [42]. To the author's knowledge, retention has not been reported in lesions of the putamen, globus pallidus or caudate nucleus.

In Parkinson's disease [43] the incidence of bladder dysfunction was found to range between 37% and 71%. Parkinson's disease (PD) is a degenerative loss of pigmented neurons, particularly in the dopamine-rich substantia nigra [44] but also in dopaminergic autonomic pathways. Detrusor hyperreflexia is found in 70% of the patients [43] with inappropriate relaxation of the sphincter producing urgency and incontinence. Nevertheless, sphincter bradykinesia (11%), which is a failure of the perineal floor to relax rapidly before detrusor contraction, and pseudodyssynergia (7%) are more characteristic of PD, and may be responsible for obstructive voiding symptoms. Detrusor areflexia associated with non-relaxation of the perineum is also observed. In progressive autonomic failure and multiple system atrophy, urinary incontinence seems to be the

rule [45]. Multiple sclerosis (MS) is a disease affecting the central nervous system and producing demyelinization of the white matter of the cortex, brain-stem and spinal cord above or at the conus medullaris. As the lesions are multifocal and most of the time progressive, all types of micturitional abnormalities are found in 50%–80% of patients [46], and may be variable from one time to another [47]. Obstructive symptoms such as difficulty in initiating micturition, elevated postvoid residuals and retensive syndromes are seen in about 20% of MS patients [47]. Bladder dysfunction may be the sole initial symptom in 2% [46]. Detrusor hyperreflexia with vesico–sphincter dyssynergia is commonly seen, but detrusor areflexia associated with non-relaxing sphincter, or non-relaxing sphincter alone, may also occur [47]. However, as is well known, there is no correlation between bladder symptoms and a particular combined cystometry–EMG finding [48,49].

Spinal Cord Lesions Above the Sacral Micturition Center

Injury is the most frequent damage to the spinal cord. Inadequate voiding can be caused by several factors, including detrusor–sphincter dyssynergia, fixed scarred bladder neck, or excess of alpha-adrenergic activity at the bladder neck leading to bladder-neck dyssynergia and autonomic dysreflexia [50]. Following spinal cord injury, for a period of 1 day to usually about 3–4 weeks, all deep-tendon reflexes below the level of injury are absent: this is referred to the 'spinal shock'. The bladder is areflexic during this phase and careful periodic catheterization to prevent bladder overdistension and atony is mandatory. The reflex bladder activity returns about 3–4 weeks after injury. Preventing the association of upper and lower spinal cord injury [51], the final outcome depends in a great part upon the severity of the cord damage. Incomplete lesions of central cord type [52] usually spare long-loop fibers to the bladder.

Complete spinal transection with lesions above the conus has varying degrees of detrusor–sphincter dyssynergia. Such patients do develop reflex bladders, but voiding is inadequate. Also complete lesions above the midthoracic region are invariably associated with autonomic dysreflexia [50].

Transverse myelitis is an uncommon inflammatory process with an acute, subacute or chronic course [53]. Although the most common etiology is viral, all types of infectious agents may produce transverse myelitis [54], including spirochetes such as *Borrelia Burgdorferi* [55]. Most patients experience acute urinary retention at the beginning [56], and detrusor–sphincter dyssynergia is commonly found. Urinary disturbances may persist despite complete neurological recovery [56].

All types of spinal cord lesions may produce urinary disturbances, including intraspinal or compressive primary and secondary tumors, infarction, cervical spondylosis, spinal epidural abscess and spinal cord hematoma. Although approximately 40% of patients with spinal cord compression will have bladder disturbances, it is unusual for acute urinary retention to be the chief complaint or presenting symptom [31].

Cauda Equina and Pelvic Nerve Lesions

Cauda equina lesion due to disc prolapse is a well known cause of acute urinary retention [57–59], which may also be the sole clinical manifestation [60]. Despite claims that bladder paralysis should be surgically treated within 2 days of the onset of symptoms [58,59], there is no evidence in the literature that emergency surgery has any bearing upon the degree of clinical recovery [61].

Urinary disturbances due to lumbar spinal stenosis have a more insidious course and are less well recognized. Half of the patients described by Sharr et al. [62] were referred initially to the urologist, the spinal symptoms being discovered secondarily, as it may be limited to backache only. Urological symptoms include recurrent urinary infection due to an atonic bladder, incontinence or episodes of acute retention. These symptoms may be intermittent [62], as cauda equina claudication is typically brought on by exertion. Cervical and lumbar spinal stenosis may both coexist and have to be looked for, especially when detrusor hyperflexia is found on urodynamic evaluation [63]. Although in most of the tumors of the conus medullaris, bladder disturbances rarely appear early, urinary presentation of cauda equina lesions without neurological symptoms have been described [64], and then have always to be kept in mind. Pinprick sensation of the saddle area, anal sphincter tone, anal reflex and bulbocavernosus reflex always have to be assessed carefully. Malformation of the cauda equina such as tethered cord, or meningomyelocele may also be responsible for bladder dysfunction, and may be associated with spina bifida occulta [65,66]. Viral infection of the sacral roots may produce a benign transient urinary retention in healthy young patients [67,68]. This sacral myeloradiculitis may be due to any neurotropic virus, although herpes virus is the most frequently encountered and should always be looked for over the perineum, but also on the cervix of female patients and in the rectum [69]. A lumbar puncture leads to the diagnosis by showing lymphocytosis [67]. Polyneuropathy may produce urinary retention, especially when the autonomic fibers are affected, such as in the Guillain–Barré syndrome [70] or in diabetes mellitus. Diabetes mellitus is certainly the most common non-traumatic reason for neurogenic paralytic bladder retention in elderly patients [31]. Acute painful urinary retention has been recently described in AIDS peripheral neuropathy [71]. Pelvic nerve damage may result from pelvic fracture, abdominoperineal resection, panproctocolectomy, radical hysterectomy, pelvic neoplasm and pelvic radiotherapy. These result in a loss of the voiding reflex by destruction of the detrusor preganglionic parasympathetic neurons, but

also by the interruption of myelinated fibers supplying the striated external urethral sphincter.

Urinary retention is also due to idiopathic detrusor failure [72], or to a loss of urethral sphincter relaxation associated with an abnormal 'pseudomyotonia' type of EMG activity [73].

If the neurological causes of urinary retention are numerous, spinal cord diseases such as multiple sclerosis and disk prolapse, as well as diabetes mellitus are most frequently encountered in females [32].

Neurophysiological Approach to Neurogenic Urinary Retention

Urodynamic investigations are now widely used by most urologists to study vesicourethral function, and include cystometry, urinary flow rate, urethral pressure profile and integrated pelvic floor muscle EMG. In recent years, neurophysiological techniques have been developed and now contribute to a better understanding of the neural control of micturition and of the pelvic floor innervation.

Perineal Motor Nerve Terminal Latencies

The terminal motor latency of the perineal nerves mesures the function of the distal part of the nerve supply of the periurethral striated sphincter muscle. This measurement is accomplished using a special glove designed by Swash, which consists of a distal stimulating electrode and a proximal recording one (Fig. 1). The perineal terminal motor latency can be recorded by stimulating the pudendal nerve at the ischial spine and recording the contraction of the periurethral sphincter using an intraurethral electrode mounted on a Foley catheter [74]. The pudendal terminal motor latency can be recorded directly by the proximal electrode of the glove, which is placed at the level of the external anal sphincter [74] (Fig. 1). Normal values for the mean right and left perineal and pudendal motor latencies are 2.4 ms ± 0.2 ms and 2.1 ms ± 0.2 ms respectively [75,76].

Clinical Applications: Increased pudendal and perineal nerve terminal motor latencies have been found in anorectal and urinary incontinence [77]. This double incontinence seems to be initiated mainly by childbirth by vaginal delivery [78], although this is not the only cause of the syndrome [79]. To the author's knowledge, increased terminal motor latency has not been described in urinary retention.

Sacral Reflex Latencies

Sacral reflex latencies are obtained by stimulating the dorsal nerve of the penis or clitoris and recording with EMG electrodes the contraction of the bulbocavernosus muscle (bulbocavernosus reflex) [80], or of the anal sphincter (anal reflex) [81], or with electrodes inserted in the external urethra (urethral reflex) [82]. The most commonly used is the bulbocavernosus reflex in men and the pudendo-anal reflex in female patients (Fig. 2).

Clinical Applications: Sacral reflexes assess the integrity of the somatic afferent and efferent myelinated nerves innervating the pelvic floor, as well as the polysynaptic

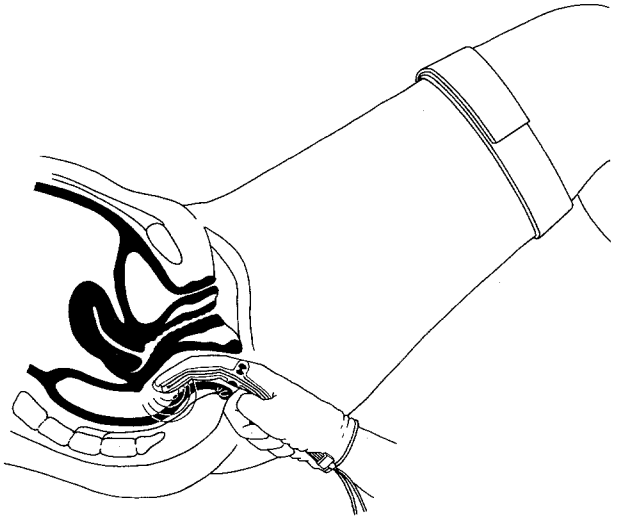

Fig. 1. Intrarectal pudendal nerve stimulation using a Dantec–St Mark's pudendal electrode (13L40, Dantec, Denmark) [74].

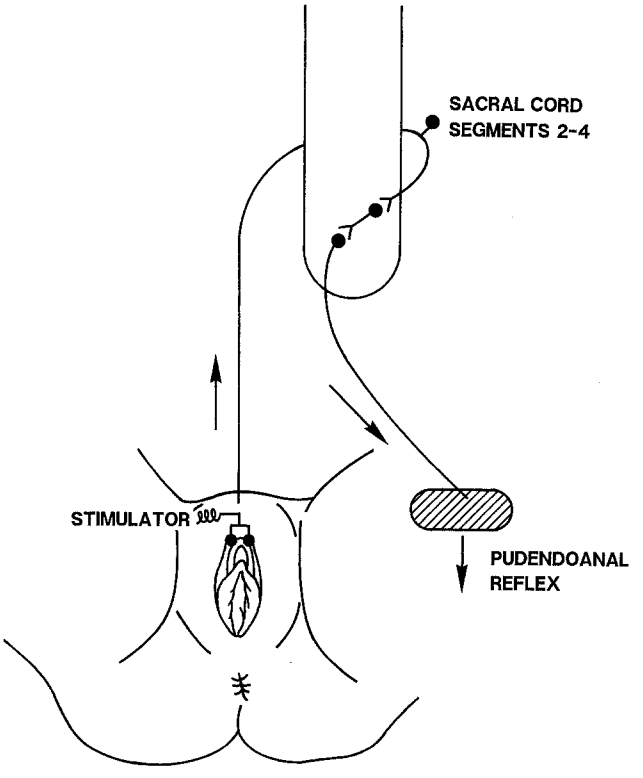

Fig. 2. Electrically elicited pudendoanal reflex in female.

connections in the S2, S3, S4 spinal segments [80,83]. Although theoretically promising, sacral reflex latencies are often misleading in clinical practice, as they could be normal in axonal neuropathies [82], or may be also strictly normal when the small unmyelinated fibers of the autonomic nerves are predominantly affected, such as in diabetic neuropathies [84]. Increased bulbocavernosus latencies have been also found without any peripheral or spinal causes [85].

Pelvic Floor Muscle Electromyography (EMG)

EMG of the urethral and anal striated sphincters, as well as of the puborectalis muscle, using a concentric needle could easily be performed [86]. These muscles show a continuous tonic contraction which could be increased during voluntary contraction or in reflexly controlled contractions, as in the cough reflex, or could cease completely during straining at stool. Normal amplitudes and durations of individual motor units have been determined for both urethral [87] and anal [86] sphincters.

Clinical Applications: EMG is a powerful investigative technique in patients with neuromuscular disease, particularly in the evaluation of disorders in which the nerve supply of muscles is damaged, whether from disease of the spinal nerve roots or peripheral nerves. Urethral sphincter EMG is interesting in the assessment of neurogenic bladder dysfunction [88] in which anal sphincter EMG has to be avoided [89,90]. Urethral sphincter EMG has been proved to be of great interest in the differential diagnosis of Parkinsonism [91]. Abnormal EMG activity has been found in the urethral sphincter of women with persistent urinary retention [92,93]. Although the innervation of the external urethral sphincter and external anal sphincter depends on the pudendal nerves, a dissociation in their activity in neurogenic bladder dysfunctions has been shown [89,94]. In detrusor–external sphincter dyssynergia, the use of an intraurethrally positioned ring electrode may facilitate routine recording of urethral electromyography. The use of coaxial needle electrodes needs an experienced electromyographer and sometimes is not practical, because urine and saline may interfere with the EMG signal [95]. Nevertheless they are highly recommended for detailed studies [34,90]. Perineal surface electrodes must be avoided, as they could reflect volume-conducted from distant muscles and are therefore not selective enough to provide information about very small muscles such as the external urethral muscle.

Somatosensory Evoked Potentials

The peripheral and central conduction time in the pudendal sensory afferent pathway is assessed by stimulating the dorsal nerve of the penis in men [96] and of the clitoris in women [97], and recording the cortical evoked potentials over the scalp [98,99] (Fig. 3). Urinary bladder stimulation may also evoke cortical potentials [100–102].

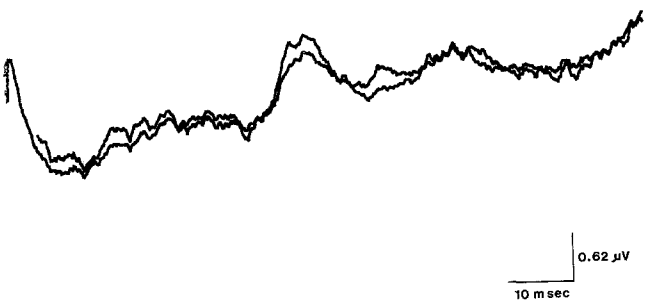

Fig. 3. Pudendal evoked response (PER) in woman.

Clinical Application: Central conduction time from pudendal or bladder stimulation is of clinical importance in the diagnosis of bladder and sexual dysfunction, especially in spinal cord diseases.

Cortical and Spinal Stimulations

Central motor pathways to the striated sphincters have been studied with transcutaneous electrical stimulation of the brain and spinal cord [103], but the technique was painful. Now, transcutaneous magnetic stimulators allow non-painful stimulation of the spinal cord and brain, and the measurement of peripheral and central motor conduction times to the pelvic floor muscles [104,105]. Motor potentials were recently recorded from the detrusor smooth muscle fibers after magnetic stimulation of the sacral cord [106].

The clinical applications seem to be promising, especially when performed in conjunction with other neurophysiological investigations [83]. This technique offers the possibility of quantitatively investigating the central nervous system in disorders such as multiple sclerosis or stroke.

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