Bladder and bowel dysfunction following neurological disease

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Abstract

The lower urinary tract (bladder and urethra) has two roles: storage of urine and emptying at appropriate times. Detrusor muscle contraction is predominantly controlled by parasympathetic influence, while sphincter muscles are controlled by sympathetic and pudendal nerves. The pontine micturition centre is responsible for coordinated activity of the detrusor and sphincters during the storage and voiding phases. Lesions anywhere in the nervous system can cause lower urinary tract dysfunction. Information obtained from history-taking and supplemented by a bladder diary form the cornerstone of evaluation. Ultrasonography is used to assess the degree of bladder emptying and to assess upper tract damage. Urodynamic tests, with or without simultaneous fluoroscopic monitoring, assess detrusor and bladder outlet function. Impaired voiding is most often managed by clean intermittent self-catheterization and should be initiated if post-void residual urine is greater than 100 ml. Storage symptoms are most often managed using antimuscarinic medications. Other options include desmopressin or detrusor muscle injection of botulinum toxin type A. Patients with neurological disease commonly have bowel complaints and may alternate between constipation and incontinence. Understanding of the underlying mechanism is crucial for effective management.

Keywords anticholinergic; bladder; botulinum toxin; desmopressin; detrusor; detrusor–sphincter dyssynergia; oxybutynin; pons; sphincter; urodynamics

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Bladder disorders

The bladder in health and neurological disease

The lower urinary tract (bladder and urethra) has two roles: storage of urine and voiding at appropriate times.[1] To regulate this, a complex neural control system acts like a switching circuit to maintain a reciprocal relationship between the reservoir function of the bladder and the sphincteric function of the urethra (Figure 1). The pontine micturition centre controls this system and receives input from higher centres, particularly the medial aspects of the frontal lobes. Disruption of the spinobulbospinal micturition reflex pathway results in impaired storage and voiding.

Storage phase: the bladder is in the storage phase 99.8% of the time and throughout this time the intravesical pressure never rises beyond 10 cmH_2O. This is achieved by inhibition of parasympathetic activity and an active process of relaxation of the detrusor resulting from bladder compliance. During this phase, sympathetic- and pudendal-mediated tonic contraction of the sphincters ensures urinary continence.

Lesions that affect the spinal or suprapontine micturition pathways can affect this phase, resulting in storage symptoms such as frequency, urgency and urge incontinence. Urodynamic studies demonstrate detrusor overactivity, whereby filling the bladder results in a spontaneous rise in intravesical pressure (Figure 2).

Voiding phase: the first event during voiding is relaxation of the pelvic floor muscles and external and internal urethral sphincters, which is achieved by inhibition of pudendal and sympathetic activity. This is followed some seconds later by parasympathetic-mediated detrusor contraction. This coordinated activity requires intact neural pathways between the bladder and the pontine micturition centre.

Voiding dysfunction is seen typically in patients with spinal cord lesions, who complain of voiding symptoms such as hesitancy, straining, slow and interrupted stream, and possibly retention. The loss of coordinated activity results in detrusor–sphincter dyssynergia, characterized by simultaneous contraction of the external urethral sphincter and detrusor muscle. This can result in incomplete bladder emptying and abnormally high pressures in the bladder. The lack of normal parasympathetic drive from the descending bulbospinal pathways results in poorly sustained detrusor contraction and can contribute to incomplete emptying. Importantly, more than 50% of patients are unaware of incomplete emptying and often find that they have to micturate within minutes of supposedly emptying their bladder (double voiding). Incomplete emptying of urine results in reduced functional capacity of the bladder, thereby exacerbating storage symptoms.

Causes for neurogenic lower urinary tract dysfunction

The lower urinary tract is commonly involved in neurological disease (Table 1).[2] In patients with myelopathy, detrusor overactivity correlates with extent of paraparesis. As sacral outflow to the bladder originates caudal to the nerves to the lower limbs, patients with upper motor neurone signs in the legs generally will have detrusor overactivity as well. This is most clearly seen in

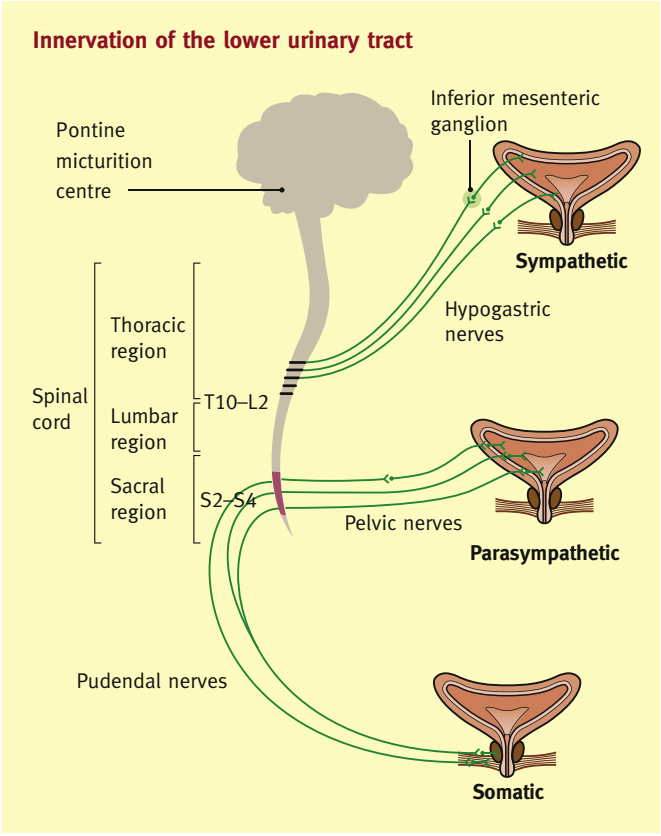

Figure 1

multiple sclerosis. Lower motor neurone disturbance in patients with polyradiculopathy or peripheral neuropathy results in reduced or absent detrusor contractions and impaired sphincter relaxation. Patients have reduced sensation of bladder fullness, inability to initiate micturition voluntarily and bladder distension to the point of overflow incontinence.

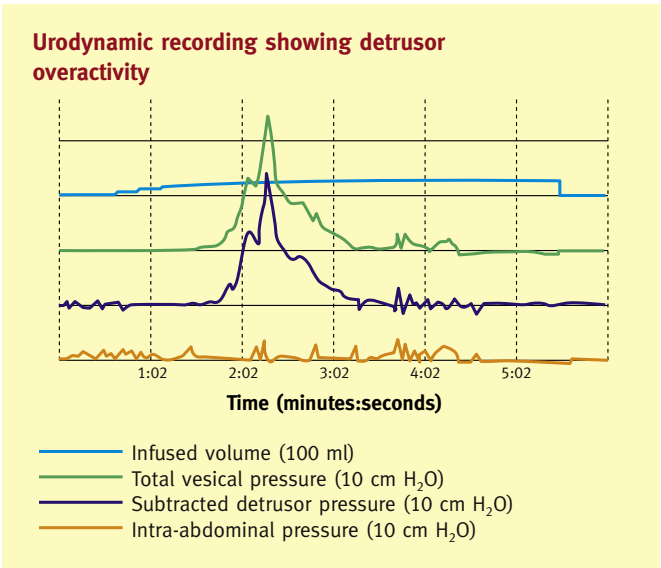

Figure 2

Neurological causes for lower urinary tract dysfunction

Suprapontine	Stroke
	Degeneration: Parkinson's disease, multiple system atrophy, dementia with Lewy bodies
	Hydrocephalus, normal pressure hydrocephalus
	Cerebral palsy
	Neoplasm
	Trauma
Suprasacral (infrapontine)	Demyelination: multiple sclerosis, transverse myelitis
	Trauma
	Degenerative disc disease
	Neoplasm: metastatic, primary
	Vascular: arteriovenous malformations
	Infections: tropical spastic paraparesis (HTLV-I)
Infrasacral	Spinal dysraphism
	Cauda equina: arachnoiditis, lumbar intervertebral disc prolapse
	Peripheral neuropathy: diabetes mellitus
	Iatrogenic — pelvic or retroperitoneal surgery

Table 1

Patients with spinal cord injury and spinal dysraphism are prone to develop upper tract renal disease due to raised intravesical pressure and may develop vesico-ureteral reflux and hydronephrosis. However, for reasons that are still unclear, this seldom occurs in those with progressive neurological disease, and bladder symptoms should be regarded as medically manageable.

Evaluation

History-taking forms the cornerstone of evaluation.[3–5] Information obtained from history can be supplemented using a bladder diary, which records information regarding time and volume of each voiding, output, incontinence and episodes of urgency (Figure 3). However, the extent of incomplete bladder emptying cannot be predicted from the history or clinical examination, and it is important to estimate the post-void residual urine using ultrasonography or catheterization.[6] In patients known to be at risk of upper tract disease, ultrasonography should be performed periodically.

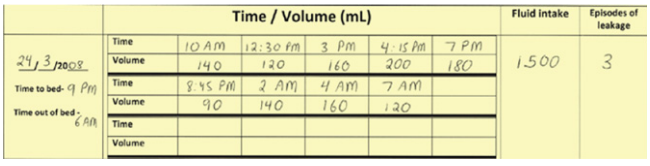

Figure 3 Bladder diary over 24 hours demonstrating daytime and nighttime frequency, low-volume voids and incontinence; this is the pattern seen in detrusor overactivity.