Urinary bladder dysfunction

Bladder dysfunction due to lesions in the central or peripheral nervous systems. Clinical manifestations differ based on the location of the lesion.

Symptoms can include overflow incontinence, frequency, urgency, urge incontinence and retention. The risk of serious complications (eg, recurrent infection, vesicoureteral reflux, autonomic dysreflexia) is high.

Bladder management is vital to protect the kidneys from obstruction and subsequent loss of renal function.

Neurogenic bladder

Neurogenic bladder.

Overactive bladder

Overactive bladder.

Specific injuries affecting the bladder

Urinary bladder dysfunction affects a large number of women and men and can have a serious impact on one’s daily life.

Over 50% of patients do not have classic presentations or symptoms.

1. supraspinal (lesions above the brainstem): loss of centrally mediated inhibition of the pontine voiding reflex. Voluntary inhibition of micturition is lost. Coordination of detrusor filling and contraction with smooth and striated urinary sphincters is intact, allowing maintenance of normal bladder pressures with a low risk of high-pressure renal damage. Patients have detrusor overactivity without detrusor-sphincter dyssynergia. Detrusor hypertrophy is less pronounced. Symptoms: urinary frequency or urgency, incontinence, and nocturia [1)].

If sensory pathways are interrupted unconscious incontinence occurs (insensate incontinence AKA incontinence of the unawares type). Voluntary bladder emptying may be maintained and timed voiding together with anticholinergic medications is used in management. Areflexia may sometimes occur.

2. complete (or near complete) spinal cord lesions:

a) suprasacral (lesion above the S2 spinal cord level, which is ≈ T12/L1 vertebral body level in an adult): loss of innervation to the pontine micturition center results in reflexive voiding modulated by the sacral voiding center (located in the conus medullaris). [2].

Etiologies: spinal cord injury, spinal cord tumors, transverse myelitis.

● Initially following spinal cord injury, there may be spinal shock. During spinal shock the bladder is acontractile and areflexic (detrusor areflexia); sphincter tone usually persists and urinary retention is the rule (urinary incontinence generally does not occur except with overdistention). This requires catheter drainage (intermittent or indwelling) due to retention until the spinal shock resolves typically within 6 months [3].

● after the spinal shock subsides, most develop detrusor hyperreflexia → involuntary bladder contractions without sensation (automatic bladder), smooth sphincter synergy, but striated dyssynergy (involuntary contraction of the external sphincter during voiding which produces a functional outlet obstruction with poor emptying and high vesical pressures which is transmitted to the kidneys and may result in loss of renal function). Bladder fills and empties spontaneously due to reflexive voiding [4].

Bladder hypertrophy occurs when contraction against a closed sphincter and bladder storage pressure increases. Patients have DO with DSD. Management goals: Decrease bladder pressures and preserve renal function, usually with pharmaceuticals and intermittent catheterizations. The frequency of bladder drainage is determined by urodynamic pressures to ensure urine volumes consistent with safe storage pressures

b) infrasacral lesions(lesions below the S2 spinal cord level): includes injury to conus medullaris, cauda equina, or peripheral nerves (formerly referred to as lower motor neuron lesions). Etiologies: large HLD, trauma with the compromise of the spinal canal, or peripheral nerve injuries (traumatic or iatrogenic with pelvic surgery). Detrusor areflexia usually ensues, and does not have voluntary or involuntary bladder contractions. Reduced urinary flow rate or retention results, and voluntary voiding may be lost. Overflow incontinence develops. Usually associated with loss of bulbocavernosus (BCR) and anal wink reflexes (preserved in suprasacral lesions, except when the spinal shock is present and perineal sensory loss. NB: Up to 20% of neurologically normal patients do not exhibit a BCR [5]

3. specific disease processes

a) herniated lumbar disc: most consist initially of difficulty voiding, straining, or urinary retention. Later, irritative symptoms may develop

b) spinal stenosis(lumbar or cervical): urologic symptoms vary, and depend on the spinal level (s) involved and the type of involvement (e.g., in cervical spinal stenosis, detrusor hyperactivity or underactivity may occur depending on whether the involvement of the micturition neural axis is compression of the inhibitory reticulospinal tracts or myelopathy involving the posterior funiculus)

c) Cauda equina syndrome usually produces urinary retention, although sometimes incontinence may occur (some cases are overflow incontinence) urgency incontinence, and nocturia.

d) Peripheral neuropathies: such as with diabetes,usually produce impaired detrusor activity

e) neurospinal dysraphism: Most myelodysplastic patients have an areflexic bladder with an open bladder neck. The bladder usually fills until the resting residual fixed external sphincter pressure is

exceeded and the leakage occurs

f) Multiple sclerosis: 50–90% of patients develop voiding symptoms at some point. The demyelination primarily involves the posterior and lateral columns of the cervical spinal cord. Detrusor hyperreflexia is the most common urodynamic abnormality (in 50–99% of cases), with bladder areflexia being less common (5–20%). Patients have DO with DSD without upper tract injury or loss of compliance

g) tethered cord: Urologic complaints are present on initial presentation 30–70% of the time. The most common urologic symptoms are urgency and incontinence. Urodynamic findings show 19 surgical correction20 DO with DSD. Urinary dysfunction improves in more than half, but not all patients, after Etiologies of urinary retention: 1. bladder outlet obstruction (a brief differential diagnosis list is presented here) a) urethral stricture: retention tends to be progressive over time b) prostaticenlargementinmales: ● benign prostatic hypertrophy (BPH) & prostate cancer: retention tends to be progressive over time ● acute prostatitis: onset of retention may be sudden c) women: bladder or vaginal prolapse which can produce a urethral kink d) obstructingthrombusfromhematuria(clotretention) e) bladder calculi f) bladder or urethral foreign bodies g) urethral cancer: rare 2. detrusor areflexia or hypotonia a) spinal cord lesion ● trauma ● tumor ● myelomeningocele b) caudaequinasyndrome(p.1254) c) medications: anticholinergics, narcotics d) diabetesmellitus(autonomicneuropathy) e) herpes zoster at the level of the sacral dorsal root ganglia21 (p 967) 3. postoperative urinary retention (POUR): occurs in ~ 4% of all surgeries, and 20–40% in neurosur- 22,23 Urinary retention gical patients after general anesthesia. position (eg BPH) along with anesthetic. Propofol, narcotics, benzodiazepines, inhaled anesthetics, and local intrathecal and epidural have all been shown to impact bladder contraction and coordination of micturition. POUR should be managed with CIC or indwelling catherization along with alpha blockers (see below) in men. Voiding trial may be done as soon as postoperative day 1 to avoid prolonged catheterization but keeping the Foley for 3–4 days has been shown to nificant difference in other studies. sequela of bladder distention Evaluation of bladder function Urodynamics (UDS) POUR may persist > 1 week. Preoperative use of Urgent intervention is recommended to avoid long term Felt to be secondary to combination of patient predis- 23 alpha blockers in at risk patients has shown protective against POUR in some studies, but not sig- 24 Usually combined with X-ray (cystometrogram [CMG]) or fluoro (videourodynamics). Measures intravesicular pressures during retrograde bladder filling through a urethral catheter, usually com- bined with sphincter electromyography. Assesses intravesical pressures during filling and voiding. Objectively assesses detrusor muscle at time of sensation to void. Most importantly, assesses bladder compliance, bladder storage pressures and risk for long term upper tract deterioration. Bladder pres- ment of a neurogenic bladder. UDS can also be used in the neurologically intact patient to determine 26 decrease need for replacement of the catheter. 25 storage of urine, there is a high risk of progressive CKD. Routine UDS can help ensure safe manage- sures: < 40 cm H20 is the cut off for safe storage pressures. If bladder pressure > 40 cm H20 during if urinary retention is secondary to obstruction versus bladder areflexia.

Types

Overactive bladder, incontinence, obstructions of the urinary tract, and urination difficulties due to neurological conditions or spinal cord injury.

An accurate assessment of the underlying causes of bladder dysfunction is key to alleviating its symptoms. We offer state-of-the-art diagnostic tests, including on-site video urodynamic evaluation. All video urodynamic studies are performed and interpreted by a urologist rather than a technician, increasing the accuracy of the testing and making for a more valuable study.

Lower urinary tract (LUT) dysfunction is a common sequela of neurological disease, resulting in symptoms that have a pronounced effect on quality of life. The site and nature of the neurological lesion affect the pattern of dysfunction. The risk of developing upper urinary tract damage and renal failure is much lower in patients with slowly progressive non-traumatic neurological disorders than in those with spinal cord injury or spina bifida; this difference in morbidity is taken into account in the development of appropriate management algorithms. Clinical assessment might include tests such as uroflowmetry, post-void residual volume measurement, renal ultrasound, (video-)urodynamics, neurophysiology, and urethrocystoscopy, depending on the indication. Incomplete bladder emptying is most often managed by intermittent catheterisation, and storage dysfunction by antimuscarinic drugs. Intradetrusor injections of onabotulinumtoxinA have transformed the management of neurogenic detrusor overactivity. Neuromodulation offers promise for managing both storage and voiding dysfunction. An individualised, patient-tailored approach is required for the management of LUT dysfunction associated with neurological disorders [6].

Bladder dysfunction in Parkinson's disease

Bladder dysfunction in Parkinson's disease.

Treatment

Urinary bladder dysfunction treatment.

1)
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