Neurogenic bladder

Preservation of renal function comes first

Anatomy revision

Parasympathetic motor (S234)
- Lateral funiculus
- Lateral grey horn
- Pelvic nerves (nervi erigentes)
- Contraction of detrusor
- Inhibition of Onuf’s nucleus

Sympathetic motor (T10-L2)
- Lateral funiculus
- Lateral grey horn
- Hypogastric nerves
- Relaxation of detrusor
- Contraction of bladder neck

Somatic motor (S234)
- Lateral funiculus
- Onuf’s nucleus medial anterior horn
- Unconscious rhabdosphincter tone

Spinal cord ends at at L1/2 vertebral junction. S2/3/4 sacral cord segments typically opposite T12. Therefore T11 lesions suprasacral, L1 lesions infrasacral, and T12 lesions variable

Reflexes (main levels)
- Cremaster: L1/2
- Knee: L3/4
- Ankle: S1/2
- Bulbocavernous: S3/4
- Anal: S5

Terminology (ICS)

- Overactive bladder – syndrome characterised by urgency with or without urge incontinence, usually with frequency and nocturia
- Detrusor (bladder) overactivity – UDS diagnosis only; characterised by involuntary contractions during filling
- Detrusor hyperreflexia – not a recommended phrase; replaced by neurogenic lower urinary tract dysfunction (NLUTD)
Classification systems
Multiple classification systems proposed
Two most commonly utilised are;
(i) Location of defect
   Sacral or infrasacral
   Suprasacral
   Pontine or suprapontine
(ii) Functional outcome (i.e Madersbacher – recommended by EAU)
   Detrusor (overactive, normoactive or underactive)
   Sphincter (overactive, normoactive or underactive)

A. Suprapontine lesions
Failure of higher centre control leads to frequency, nocturia, urgency and urge incontinence. UDS show bladder overactivity with co-ordinated sphincter. Many older men may have co-existent bladder outflow obstruction – essential that UDS unequivocally diagnoses BOO before any surgery contemplated. Common causes of suprapontine neurogenic bladder:
(i) Dementia
   Poorly understood neurological condition
   UDS in dementia
Neurogenic bladder

40% normal
40% bladder
15% SUI
5% retention with overflow
Bladder retraining impractical
Anticholinergics may exacerbate confusion (try trospium)
Surgical intervention for BOO poorly tolerated (pulled catheter etc.)
Timed voiding can help

(ii) Parkinson’s disease
Basal ganglia disease – degeneration of substantia nigra
Relative dopamine deficiency vs. Ach
Dopamine believed to inhibit bladder contractions centrally
Urge and UUI exacerbated by poor mobility
UDS useful to exclude BOO, otherwise anticholinergics

(iii) Multiple-system atrophy
Aka Shy-Drager syndrome
Middle aged males
Autonomic neuropathy, esp basal ganglia, lateral horn cells and
Onuf’s nucleus
Postural HT, impotence and urinary incontinence
UDS – bladder overactivity and paralysed sphincter
No effective treatment: anticholinergics ineffective; catheterise when necessary

(iv) CVA
Acute phase a/w urinary retention; recovery characterised by bladder overactivity. UDS typically OAB with synergistic striated and smooth sphincter, occasionally normal. Typically urgency and urge incontinence. Basal ganglia and thalamus lesions tend to have preservation of levator function, therefore no incontinence

(v) Brain tumours
Variable
Posterior fossa tumours a/w retention of urine

(vi) Cerebral palsy
Typically normal tracts and normal voiding

B. Suprasacral lesions
Preserved spinal reflex arc; disruption of descending inhibition and pontine co-ordination. UDS typically show bladder overactivity with detrusor-sphincter dyssynergia. Usually normal compliance

(i) Suprasacral spinal cord injury
Initial injury characterised by spinal shock
Spinal shock
Withdrawal of inhibitory/excitatory influences 2’ injury
Variable period (hours to weeks) characterised by hypocontractility, flaccidity and areflexia
Typically painless urinary retention with absent bulbocavernous and anal reflexes
Return of function often characterised by spasticity (bladder overactivity) and return of bulbocavernous and anal reflexes. Typically 6 months to 2 years.

**Detrusor sphincter dyssynergia**
- Failure of rhabdospincter to relax during bladder contraction
- Initially no voiding, but bladder contraction strengthens to allow some leakage – voiding in staccato fashion
- Seen in 70-100% of suprasacral spinal cord lesions
- Risk factor for upper tract deterioration (see below)

**Autonomic dysreflexia**
- Normally ascending nerve fibres carrying pain/distension signals send slips to sympathetic outflow and adrenal medulla in particular, preparing host for ‘fight or flight’. Causes tachycardia, hypertension, headache, flushing, sweating and piloerection.
- Peripheral baroreceptors trigger central parasympathetic response which act in 2 ways:
  1. Vagal outflow causing bradycardia
  2. Descending inhibitory outflow to sympathetic nerve cell bodies, tempering sympathetic response
- In lesions above T6 spinal inhibition does not occur, leading to uncontrolled hypertension. Vagal outflow is preserved explaining bradycardia
- May be anticipated and painful stimuli avoided if possible. Spinal anaesthesia better vs. GA.

**Risk factors for upper tract deterioration**
- DSD
- Low (poor) compliance < 20ml/cm water
- VUR on video UDS
- High DLPP > 40 cm water

Hydronephrosis seen in ~ 20% of spinal injuries patients at 15-20 yrs (Hackler 1977)

(i) **Spinal bifida (myelomeningocele)**
- See paediatric neuropathy
- Classification (Rickwood and Thomas; Mundy)
  - Suprasacral (discoordinated voiding; aka contractile bladder)
  - Retained sacral conus reflexes (anocutaneous (S5); bulbocavernosus (S3,4))
  - Detrusor hyperreflexia
  - DSD
  - No sphincter paralysis

*Tom Walton January 2010*
Neurogenic bladder

Sacral (acontractile bladder)
 Absent sacral conus reflexes
 Often poorly compliant bladder
 High pressure (retainers) or low pressure (wetters)
dependent on intrinsic EUS tone (static sphincteric
obstruction)
 NB. Paralysis of EUS means that intrinsic tone may be
overcome by external pressure – ‘expressible bladder’
pathognomonic for neuropathic bladder

There may be an intermediate type characterised by detrusor
overactivity and a weak external sphincter similar to Shy-Drager

(ii) Multiple sclerosis
 Demyelinating disorder characterised by white plaques in CNS/cord
Commonest cord segment involved = cervical
Young adults; females > males; Northern latitudes
Relapsing remitting in 85%; 80-100% have LUTS
Typically detrusor overactivity and DSD. Irritative LUTS and impaired
bladder emptying
DSD appears safe in MS* – very low risk of upper tract deterioration.
OK to teach ISC and discharge
* Holds true for all forms of progressive neurological disease

C. Sacral and infrasacral lesions
Usually characterised by bladder acontractility and paralysis of urethral
sphincter. Residual tone at BN and EUS (? sympathetically mediated)
overcome at varying degrees of bladder filling. Bladder emptied by abdominal
strain or fist (Crede manouvre). Dependent upon DLPP (no contraction) upper
tract may be at risk. If DLPP > 40cm water risk of upper tract deterioration.
Examination reveals absent bulbocavernous and anal reflexes usually with
flaccid areflexic legs.

(i) Sacral cord (conus) injury
 Acontractile bladder with paralysed sphincter
 Often poor bladder capacity +/- poor compliance – no cycling
 Mx = improve capacity and re-inforce/close sphincter

(ii) Lumbar disc herniation
 Typically L4/L5 or L5/S1 interspaces
 Central disc protrusion a/w cauda equina syndrome
 Cauda equina syndrome
 Acute back pain, saddle anaesthesia, painless
 urinary retention and defaecation problems
 Distended bladder, sensory loss, absent bulbocavernous and
 anal reflexes, lower limb flaccidity and areflexia
 Give IV dexamethasone 16mg, arrange urgent MRI and refer to
 spinal surgeons
 UDS findings most commonly normal compliance, areflexia and
 pelvic floor denervation

(iii) Pelvic surgery
 Typically AP-resection, anterior resection and hysterectomy
Neurogenic bladder

NLUTD reported in up to 60% post-op; of these 15% permanent
Due to damage to hypogastric and pelvic nerves
UDS shows reduced sensation; hypocontractility, poor complianace and paralysis of pelvic floor
Spontaneous recovery in 85%; reassure and teach ISC

(iv) Diabetes mellitis
Chronic hyperglycaemia results in autonomic and PN demyelination
Diabetic NLUTD may be predicted by the presence of peripheral neuropathy: 75-100% of patients with sacral dermatome paraesthesia have bladder dysfunction. Sacral reflexes may be reduced
UDS shows impaired sensation, hypocontractility and high PVR

(v) Other
Herpes zoster VZV; sacral dermatome vesicles + AUR; transient
Guillain-Barre Sporadic, autoimmune, usually transient
Fowler’s syndrome Idiopathic retention in young females
a/w polycystic ovary syndrome
Raised resting EUS tone on EMG
Failed relaxation of sphincter during voiding
indicated by raised urethral pressure profile during voiding
Hormones, topical nitrates and BoTox ineffective;
SNS promising

Management
Life expectancy of a paraplegic normal; tetraplegic a/w 20% reduction. QOL normally distributed as for general population
Principles:
Urodynamics mandatory
Preservation of renal function priority
Surgery avoided unless absolutely necessary
Most patients adequately managed with anticholinergics and CISC

(i) Detrusor overactivity
Conservative
Bladder training
Timed voiding
Sheath/catheter
Pharmacological
Anticholinergics
Intravesical capsaicin +/- LA
Intravesical resiniferatoxin
Intravesical botulinum toxin
Surgical
Sacral nerve stimulation (dorsal root)
Posterior rhizotomy and anterior root stimulation (SARS)
Clam ileocystoplasty
Detrusor myomectomy
Ileal conduit urinary diversion

(ii) Detrusor underactivity
No role for parasympathomimetics (distigmine bromide a/w risk of cholinergic crisis)
Clean intermittent self catheterisation (CISC)

(iii) Sphincter overactivity
Clean intermittent self catheterisation
? Botulinum Toxin
Transurethral sphincterotomy

(iv) Sphincter underactivity
Pads, sheath or catheter
Ephedrine or pseudoephedrine (not duloxetine)
Bulking agents
Autologous sling/ TVT
Artificial sphincter
Bladder neck closure and mitrofanoff
Clean intermittent self catheterisation (CISC)

Transurethral sphincterotomy
Indications
Failed first-line therapy (anticholinergic/CISC) due to:
High voiding pressure /DSD
Hydronephrosis
VUR
Severe autonomic hyperreflexia
Typically performed under spinal to reduce risk of autonomic hyperreflexia
Long incision at 12 o’clock from bladder neck to bulbar urethra – can be difficult. Patient then managed by condom catheter

Sacral nerve stimulation
Anterior and posterior rami of sacral nerves emerge separately from the anterior and posterior sacral foraminae.
2 types:
Posterior stimulators
Medtronic ® InterStim Sacral Nerve Stimulation (SNS) System™, Minnesota, USA
Interfere with spinal reflex arcs by electrical modification at S3 posterior sacral foramina.
Used to suppress detrusor overactivity - identical to those for used for severe OAB
Useful for patients with incomplete suprasacral spinal cord lesions with some preserved sensation
Also appears to be effective for patients with Fowler’s syndrome (non-obstructive urinary retention) – improves detrusor-sphincter co-ordination, possibly by reducing protective afferent signally from pelvic floor
Anterior root stimulation
Sacral anterior root stimulation (SARS)
Pioneered by Giles Brindley (Finetech-Brindley technique)
Electrodes applied intrathecally to anterior nerve root – much more extensive procedure involving laminectomy
Usually combined with posterior rhizotomy in patients with complete suprasacral spinal cord lesions
Posterior rhizotomy (sacral deafferentation) abolishes bladder reflex, diminishes autonomic dysreflexia, improves capacity, reduces DSD, improves bowel continence, but loss of reflex erections, reflex ejaculation and bowel emptying
Different nerve roots for different somatic motor loss:

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Very effective, but high morbidity, high reoperation rate and implant dysfunction