

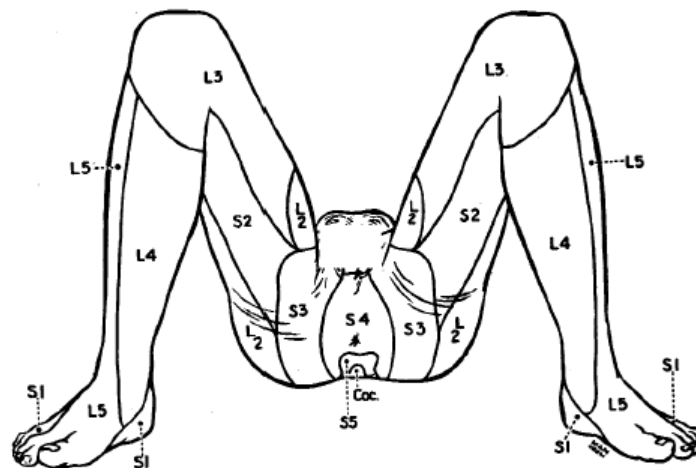
Neurogenic bladder

Preservation of renal function comes first

Anatomy revision

Parasympathetic motor (S2/3/4)	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 (S2/3/4)	Lateral funiculus Onuf's nucleus medial anterior horn Unconscious rhabdosphincter tone

Spinal cord ends 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)

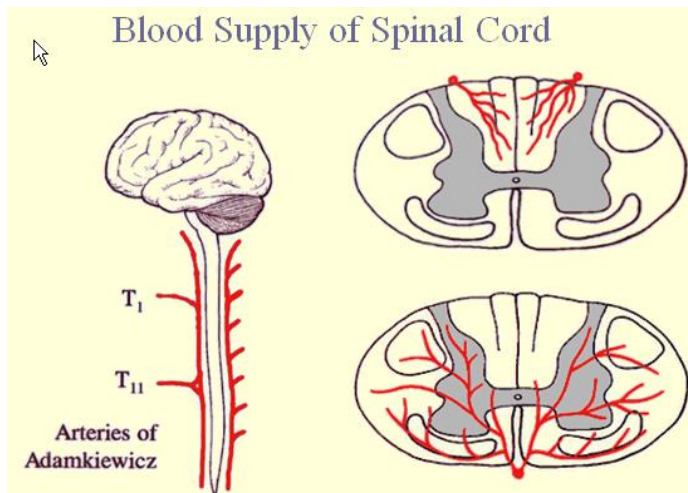
Cremaster	L1/2
Knee	L3/4
Ankle	S1/2
Bulbocavernosus	S3/4
Anal	S5

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)

Expected behavior of the bladder and external sphincter in neurologic disease		
Location of lesion	Clinical examination	Likely urodynamic findings
Suprapontine	Abnormal neurologic examination specific to condition	Detrusor hyperreflexia Synergic striated sphincter
Spinal/suprasacral	Muscle spasm, brisk reflexes	Detrusor hyperreflexia
	Positive digital anal reflex	Detrusor sphincter dyssynergia
	Positive bulbocavernosus reflex	Normal compliance
Conus or infrasacral	Negative digital anal reflex	Areflexic or underactive detrusor
	Negative bulbocavernosus reflex	Low compliance with open bladder neck Urethral sphincter incompetence

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

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 UI exacerbadated 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 bulbocavernosus 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:

- (i) Vagal outflow causing bradycardia
- (ii) 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.

Treatment of established autonomic dysreflexia

- Remove stimulus (i.e. draining bladder)
- Sit up (lessen chance of hypertensive brain injury)
- GTN spray/buccal suscard
- Sublingual nifedipine or captopril
- IV labetolol

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

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 manoeuvre). 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

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 compliance and
 paralysis of pelvic floor
 Spontaneous recovery in 85%; reassure and teach ISC

(iv) Diabetes mellitus

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)

Lapides 1972

Indicated for residual volumes > 100 ml

Good eyes and hands required

Careful counselling and assessment required – CNS

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 signalling 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:

	Roots		
	S2	S3	S4
Erection	+++	+	0
Bladder	++	+++	++
Bowel	0	+	+++

SARS

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