Bladder dysfunction in neurosurgical patients: anatomy, diagnosis and management

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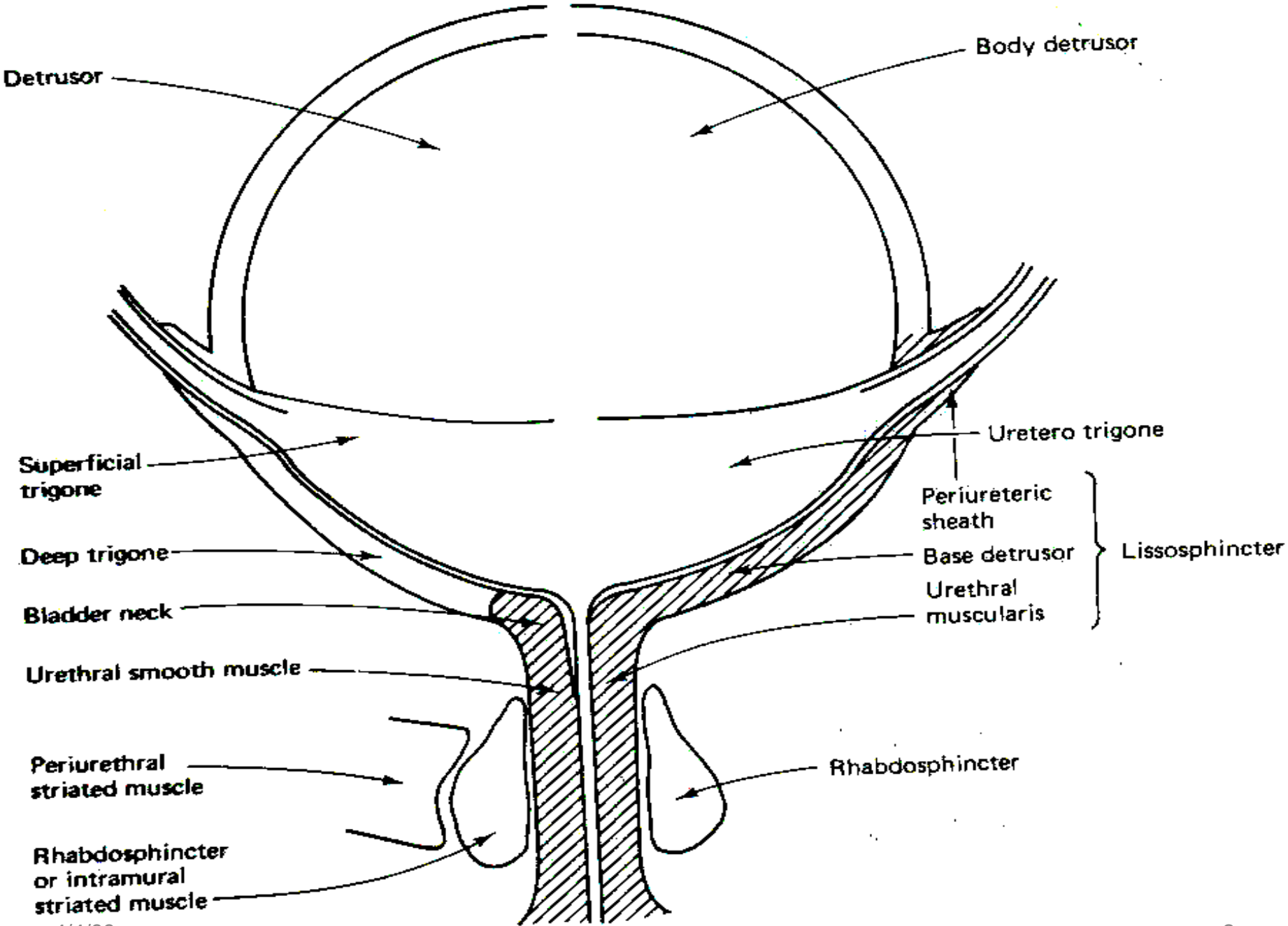
Bladder anatomy

- Hollow organ with 3 layers

  inner mucosa

  middle smooth muscle detrusor

  outer adventitia
- Detrusor muscle fibres are arranged in 3 layers- inner and outer longitudinal and middle circular
- Trigone is triangular region formed by ureteral orifices and bladder outlet


Detrusor
Body detrusor
Uretero trigone
Superficial trigone
Periureteric sheath
Deep trigone
Base detrusor
Lissosphincter
Bladder neck
Urethral muscularis
Urethral smooth muscle
Periurethral striated muscle
Rhabdosphincter
Rhabdosphincter or intramural striated muscle

Bladder anatomy

- Bladder is involved primarily in compliant distension during gradual ureteral filling
- Low pressure filling and storage of urine occurs
- Generation of efficient detrusor contraction causes voiding

Sphincteric mechanisms

- Proximal/internal (**smooth muscle sphincter**)- formed by circular smooth muscle fibers at bladder neck and proximal prostatic urethra(m)
- Distal/external (**striated sphincter**)-
  -- *Slow twitch* fibers responsible for baseline tonic activity
  -- *Fast twitch* responsible for intermittent reflex and voluntary contractions
- Striated sphincter is under partial voluntary control

(note-removal of proximal sphincter as in TURP doesn't produce incontinence)

Neuroanatomy of bladder control

Cortical control

- Antero medial frontal lobes involved in voluntary initiation of micturition and inhibition of reflex voiding
- Sends tonically inhibitory signals to detrusor
- To avoid voiding till an appropriate location is found
- Has connection with pons; and corticospinal fibres synapse with motor nuclei controlling striated sphincter

Pontine micturition centre

- Located at pontomesencephalic reticular formation (Barrington nucleus)

Coordinating the activities of the urinary sphincters and the bladder

Mediated by reticulospinal tracts which synapse with detrusor and sphincter motor centre in sacral cord

Affected by emotions, some people may experience incontinence when they are excited or scared

Ability of the brain to control the PMC is part of the social training that children experience during growth(3-4yrs)

Spinal control

- Somatic (S2-S4)
  - Pudendal nerves(Excitatory to external sphincter)
- Parasympathetic (S2-S4)
  - Pelvic nerves(Excitatory to bladder, relaxes sphincter)
- Sympathetic (T10-L2)
  - Hypogastric nerves to pelvic ganglia
  - Inhibitory to bladder body, excitatory to bladder base/urethra

Sympathetic system

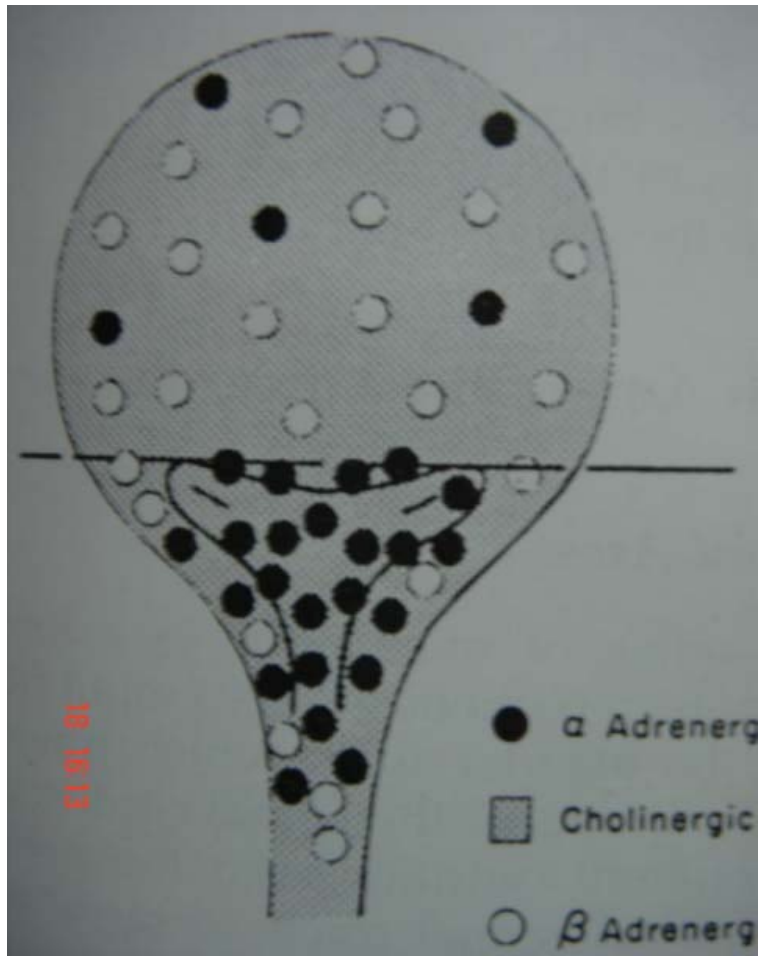
Adrenergic

α_1

Trigone, bladder neck, urethra
Maintain continence by contraction of bladder neck smooth muscle

β_2

Bladder neck and body of bladder
Inhibitory when active to
Relax bladder neck on void
Relax bladder body for storage



Sympathetic system

Normally, bladder and the internal urethral sphincter primarily are under sympathetic control

-Bladder can increase capacity without increasing detrusor resting pressure
-Stimulates the internal urinary sphincter to remain tightly closed
-Inhibits parasympathetic stimulation
-Micturition reflex is inhibited

Parasympathetic system

Immediately preceding parasympathetic stimulation, sympathetic influence on the internal urethral sphincter becomes suppressed so that the internal sphincter relaxes and opens

Pudendal nerve is inhibited → external sphincter opens → facilitation of voluntary urination

Somatic system

- Regulates the actions of voluntary muscles
  - External urinary sphincter
  - Pelvic diaphragm
- Innervation is via the pudendal nerve
  - Originates from the nucleus of Onuf at the ant horn of S 2-4
  - Activation of the pudendal nerve causes → contraction of the external sphincter and the pelvic floor muscles
  - Neuropraxia of pudendal may occur with….
    - Difficult or prolonged vaginal delivery, causing stress urinary incontinence

Bladder filling and urine storage require:

- Accommodation of increasing volume at low IV pressure, app. Sensation
- Bladder outlet closed at rest
- Absence of involuntary bladder contraction

Bladder Emptying / voiding:

- Coordinated contraction of bladder smooth muscle
- Concomitant lowering resistance at sphincters
- Absence of anatomic obstruction

Urine storage reflexes

During the storage of urine, distention of the bladder produces low-level vesical afferent firing

stimulates the sympathetic outflow in the hypogastric nerve to the bladder outlet (the bladder base and the urethra) and the pudendal outflow to the external urethral sphincter

Sympathetic firing also inhibits contraction of the detrusor muscle

A

Voiding reflexes

- During voiding, bladder-afferent firing in the pelvic nerve activates spinobulbospinal reflex pathways (shown in blue) that pass through the pontine micturition centre.

- This stimulates the parasympathetic outflow to the bladder and to the urethral smooth muscle (shown in green) and inhibits the sympathetic and pudendal outflow to the urethral outlet (shown in red).

Bradleys loops

The central connections of the bladder sphincter unit have been funtionally grouped together into ‘circuits’ the so called Bradleys loops

Loop I Pathways integrating the cerebral cortex, basal ganglia, thalamus and the cerebellum with the pontomesencephalic reticular formation

Loop II Afferents from the bladder stretch receptors travelling via the dorsal roots and posterior column to the ponto-mesencephalic reticular formation

Efferents from here to the sacral motor neurons controlling the Detrusor and the striated sphincter

Bradleys loops

Loop III Local loops between the bladder stretch receptors , sacral segments and thence to the detrusor and sphincters

Loop IV Descending pathways from the motor cortex to the sacral anterior horn cells innervating the striated sphincter

Neurogenic bladder dysfunction

- This has been classified by Lapides into :

1. Uninhibited bladder
2. Reflex neurogenic bladder
3. Sensory paralytic bladder
4. Motor paralytic bladder
5. Autonomous bladder

Uninhibited bladder

- Reflex voiding without voluntary control
- Coordinated activity of the sphincter and the detrusor
- Seen in suprapontine lesions
- Filling upto a normal threshold of 300-350 ml Outlet resistance and filling pressures are normal

Reflex neurogenic bladder

- Seen in lesions of the spinal cord above the sacral levels
- Automatic voiding on reaching a threshold of 150- 200 ml
- Incoordinated sphincter and detrusor activity
- High outlet resistance and early upper tract changes
- Classic sphincter EMG shows cresendo-decresendo pattern with detrusor contraction

Autonomous & sensory/motor paralytic bladder

- From a clinical point of view the separation of these three categories is artificial and they often have a considerable degree of overlap
- The usual picture is of an areflexic , capacious bladder with high compliance and large residual volumes
- Seen in lesions of the sacral cord , autonomic efferents and the afferents

Neurological evaluation

- General h/o
  - Congenital, traumatic, metabolic, degenerative
  - SCI, surgery, PD, MS, CVA, Drugs, family history
- Genitourinary h/o
  - Voiding habits, h/o UTI, reflux, stones
- Urinary s/o
  - LUTS – voiding, storage
- Associated neurological symptoms
  - Visual, sensory, motor, gait
- Sexual and bowel dysfunction

Symptomatology

- *Urgency* and frequency:
- Exaggeration of normal sensation of need to void occurring at lower volume threshold, usually accompanied by increased frequency
- Feeling of need to void is combined vesical and somatic sensation localized in perineum and transmitted along sacral afferents
- Abnormalities may be due to
  - Changes in bladder wall
  - Abnormal motor impulses causing detrusor contraction
  - Injuries to sensory pathways

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Symptomatology

- Suprapontine lesions can have urgency and frequency despite good capacity and no detrusor instability
- Increased frequency may be there in detrusor areflexia with urinary retention
- Dysuria: painful sensation of micturition

Symptomatology

- *Hesitancy:*

  (difficulty in beginning the voiding reflex)

  cortical/ corticospinal lesions (voluntary control of striated sphincter function is lost)

  outlet obstruction

  impaired detrusor function

Symptomatology

Incontinence

- involuntary loss of urine per urethra
- SUI (decreased outlet resistance)
- Urge incontinence (detrusor instability with intact sensation)
- Overflow incontinence(frequent dribbling due to impaired detrusor function,bladder fills to the point when P_{ves}>Urethral closing pressure)
- Total incontinence (ectopic ureter/fistula)

Symptomatology

- Post void residual urine(decreased bladder contractility/outlet obstruction or both)-clinically by palpation/percussion, radiologically

  by ultrasound and by catheterization
- Clinical examination
  - Motor – tone, bulk
  - Sensory
  - Reflexes
    - DTR, babinski, BCR (S2-S4) → absent in 15% neurological intact females, anal reflex(S2-S5)

- Laboratory investigation – urinalysis and cultures, renal function tests

Symptomatology

- Radiological evaluation
  - Upper urinary tract

    USG, IVP, Renal scan

    Poor compliance, SSD → 6mthly FU by USG (recurrent infections, change in symptoms, hematuria)
  - Lower urinary tract

    VCUG – anatomical, VUR, diverticula, stones

    Radionuclide cystogram → functional
- Endoscopic examination – not routinely indicated
  - Stricture, diverticula, stones, hematuria

UDS

- Urodynamic examination – 3 important principles
  - Study that doesn't duplicate patient's symptoms is not diagnostic
  - Failure to record an abnormality does not rule out its existence
  - Not all abnormalities detected are clinically significant
  - **Indications**
    - All patients of neurogenic bladder
      - nature of detrusor, sphincter
      - Prognosis, management

Cystometry

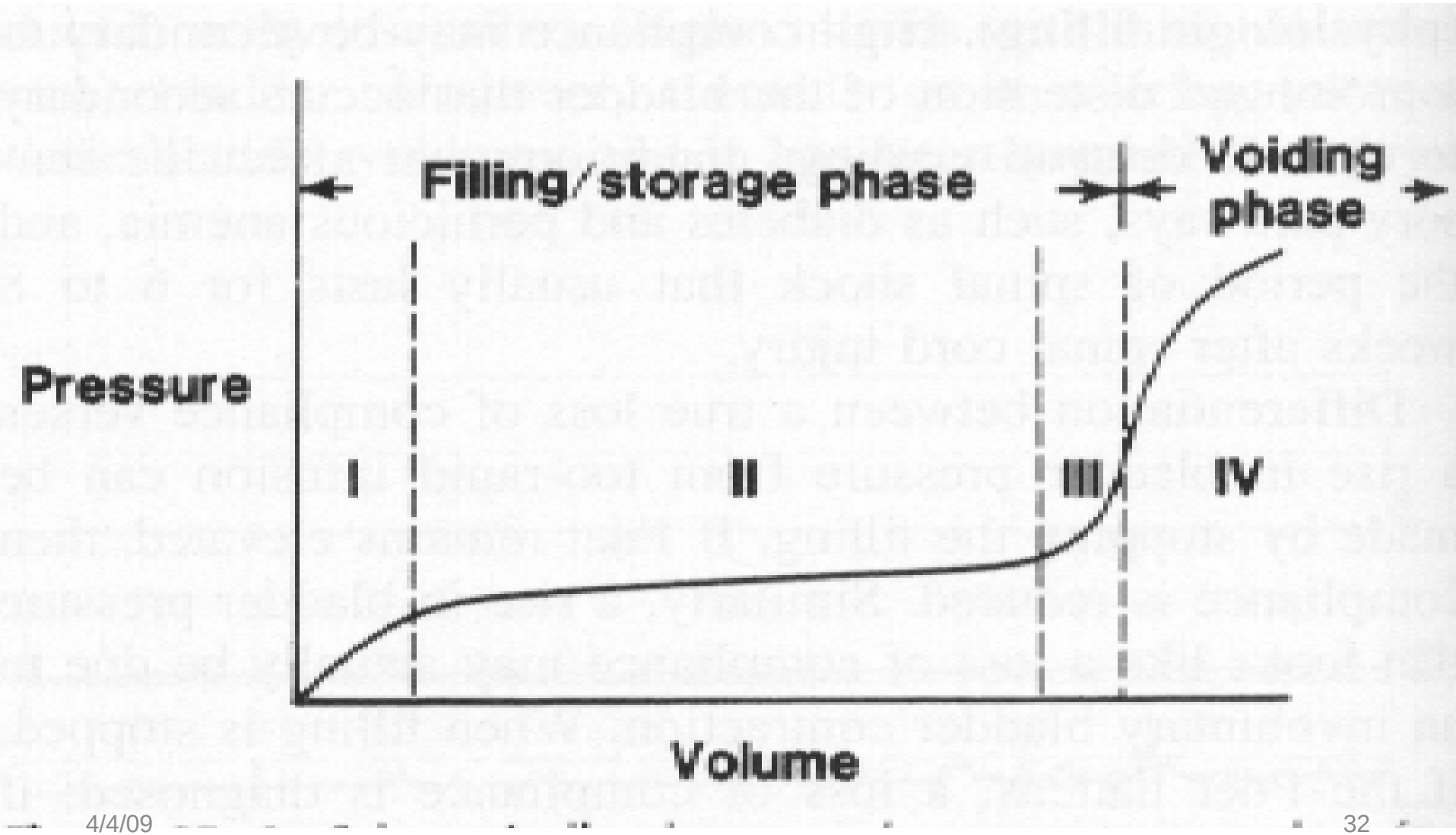
- Measurement of intravesical pressure during different phases of bladder function
- Filling cystometry examines the bladder during filling and storage
- Bladder sensation, capacity and compliance are tested and presence of involuntary contractions (instability) noted.
- Bladder is filled and patient asked to report the first sensation of filling
- Cystometric capacity is the volume at which patient has strong urge to void and is generally the end point of filling

- Bladder compliance (V/P) is ability to accommodate increasing filling volumes-in normal bladder there is only a few cm pressure change
- Minimal changes in detrusor pressures are present in normal bladder

  Detrusor hyperreflexia- involuntary contraction due to associated neurological disease

  Detrusor instability- in the absence of known neurological disease

Detrusor leak point pressure- required to drive urine across the sphincter mechanism, reflects pressure transmission to upper tracts and subsequent damage


Filling/storage phase
Voiding phase
Pressure
I
II
III
IV
Volume

Evaluation of voiding

- Voiding cystometry comprise simultaneous recording of intravesical pressure, intra abdominal pressure and flow rate during voiding
- Combined with simultaneous recording of sphincter EMG to see for dyssynergia

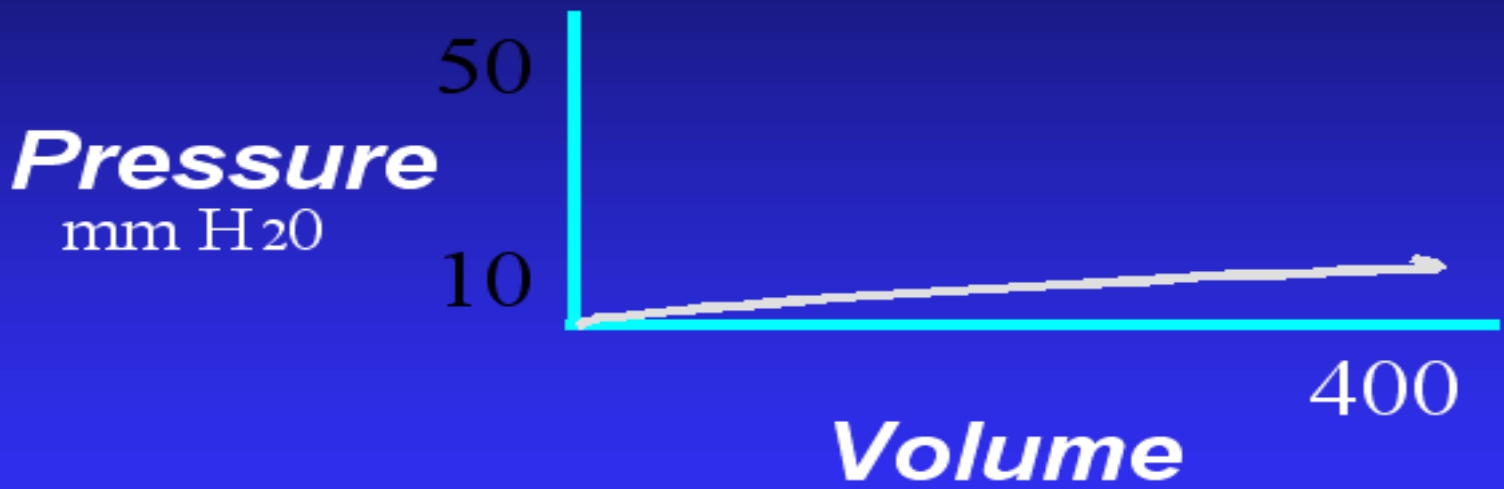
Uroflometry

- Urinary flow reflects final result of micturition process
- Needs integration of detrusor function, bladder neck and striated sphincter opening and urethral conductivity
- Max. flow rate is the most important parameter
- >15ml/s indicates normal function
- <10ml/s indicates infravesical obstruction
- Reliable only when voided volumes is 200-400ml

Pressures

- Rectal pressure = abdominal pressure
- True detrusor pressure = intravesical pressure – rectal (abdominal) pressure
- Normal bladder resting pressure =8 to 40 cm H_20
- Normal compliance is < 15 cm H_20 increase in pressure during filling
- Avg. urethral closure pressure is 60 cm H_20

UDS



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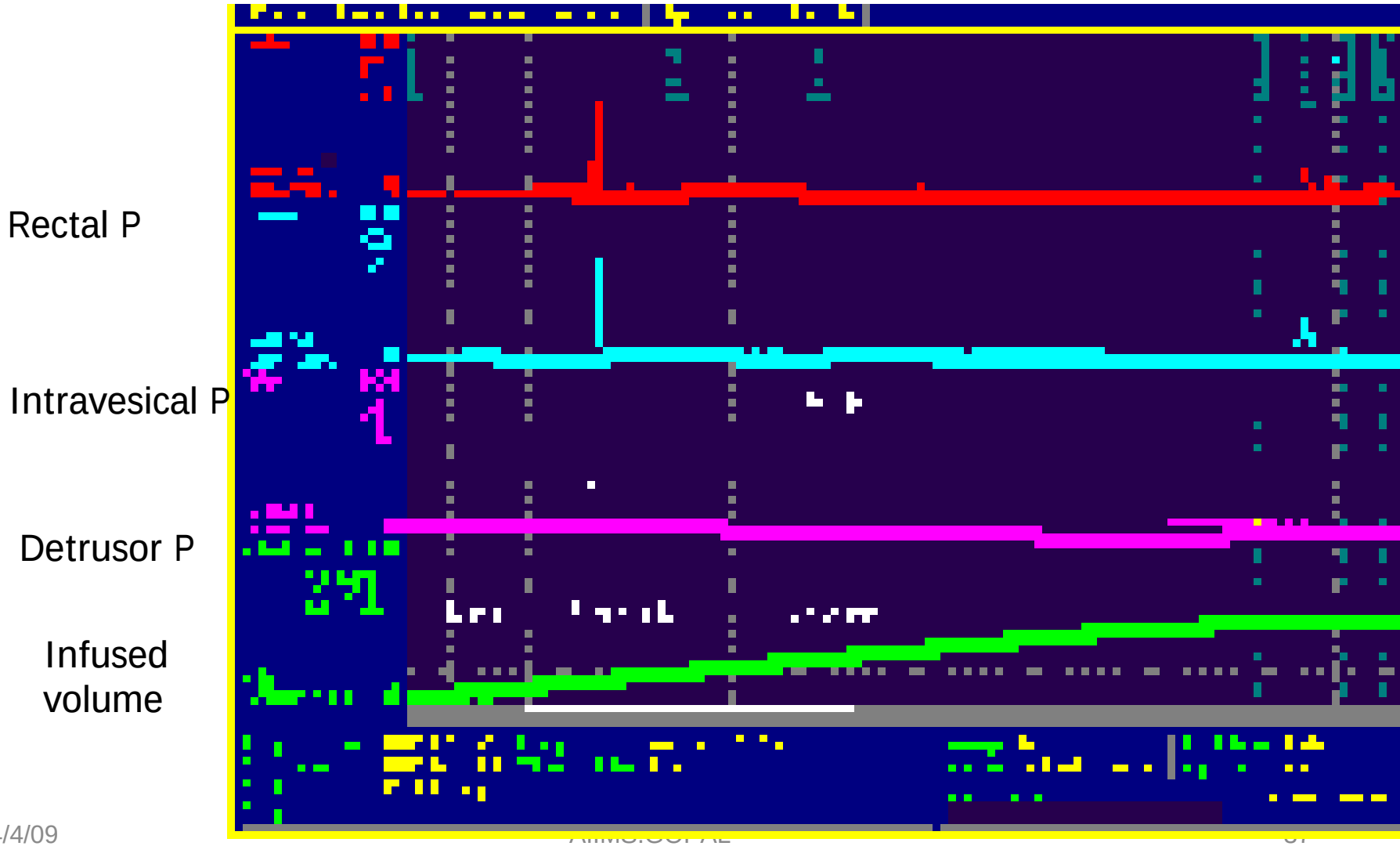
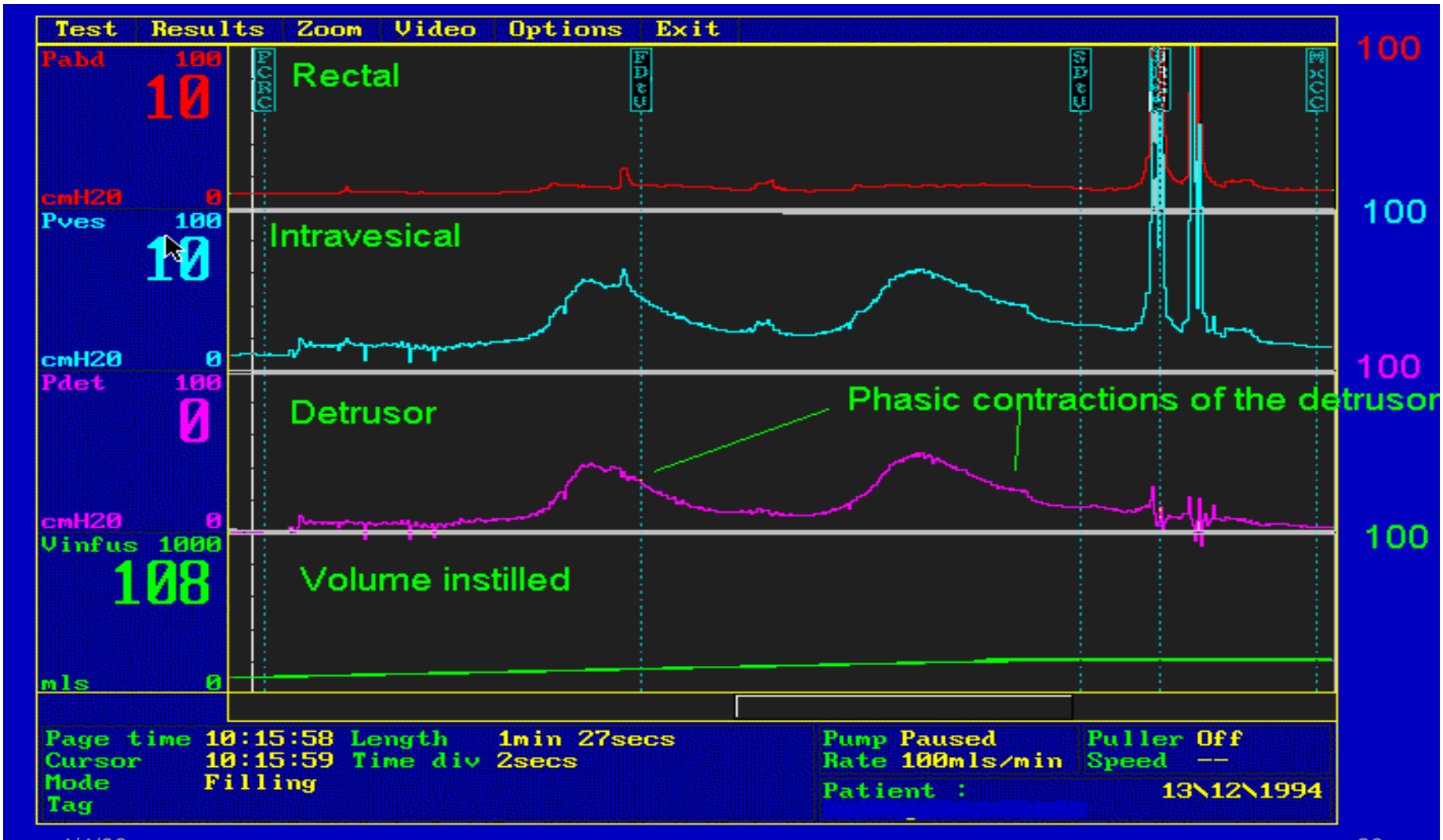


Stable Bladder

Neurogenic Detrusor Hyperactivity



Low Compliance Bladder

Urological dysfunction in specific neurological disorders

Supraspinal lesions:

- lesions above the pontine micturition centre result in impairment of pontine inhibitory influences and manifest by frequency, urgency, detrusor hyperreflexia and occasionally urge incontinence
- Traumatic/vascular or acute injury to frontal lobes cause acute retention followed by chronic period of uninhibited detrusor activity

Supraspinal lesions

- If areas involving voluntary control of ext. sphincter then hesitancy(difficulty initiating voiding)
- If sensation and area of voluntary control is unaffected, urgency and frequency may occur but pt may be able to control urge incontinence by striated sphincter activity.
- Unilateral cortical lesions have only subtle change in voiding behaviour.

Spinal lesions

- Effect of spinal cord lesions depend on their chronology, level and completeness
- **Acute spinal injury(spinal shock)**

  absence of sensation and motor function, loss of voluntary control and sphincter compromise ⟶ painless retention

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Supra sacral lesions(UMN lesion)

- Sensory impairment, detrusor hyperreflexia and detrusor sphincter dyssynergia

- High pressures in upper tracts
- Lesions above T10(sympathetic outflow):

  impairment of pain/sensation/temperature

  dyssynergia between detrusor, striated and smooth sphincter
- Autonomic dysreflexia

Autonomic dysreflexia

- In response to visceral stimuli below injury(particularly bladder and rectal distension)patients experience sweating, flushing, severe headache ,pilorection above injured level and profound hypertension with reflex bradycardia
- When injury is above T6(sympathetic outflow)
- Due to loss of inhibitory control

Supra sacral lesions(UMN lesion)

Injury below lower thoracic cord:

sensory impairment, detrusor hyper reflexia, smooth muscle sphincter synergy and striated sphincter dyssynergia but no autonomic dysreflexia

Sacral lesions

Mixed Neuropathic Bladder (Type A)

A lesion in the conus with damage to **detrusor (para sympathetic) nucleus causes:**

· detrusor hyporeflexia (underactivity) with external sphincter hyperreflexia

· characteristically large volume with overflow incontinence

Mixed Neuropathic Bladder (Type B)

A lesion in conus involving **pudendal (somatic) nucleus causes:**

· detrusor hyperreflexia with external sphincter hypotonia

· small volume,frequency, incontinence

Infrasacral Bladder

- A **Lower Motor Neuron lesion from conus medullaris and/or cauda equina damage**
- areflexia (not atonia) of detrusor (due to post-ganglionic fibres being in bladder wall)
- and areflexia with atonia of pelvic floor muscles
- may have isolated increase in bladder neck/internal sphincter resistance (intact T11- L 2 sympathetics)
- non-contractile bladder with leakage from overflow
- loss of voluntary control of both anal and urethral sphincter, as well as sexual responsiveness

Spinal dysraphism

- Type and severity of dysfunction difficult to predict solely on the basis of level and extent of defect
- Detrusor areflexia/hyporeflexia with/without sphincter dyssynergia
- Loss of compliance due to denervation supersensitivity due to loss of sacral parasympathetic fibres via renervation from intact sympathetic or from damage to bladder musculature from overdistension /rec infection
- Incontinence if present occurs when pressure exceeds fixed outlet resistance
- High pressure >40 cm H20 is dangerous

- 10% of children with myelomeningocele (MMC) will develop a dilatation deformity within the first year of life, and 35% by the 4th year of life
- over 50% of MMC children have a dangerous bladder (having an active sphincter with or without an active detrusor)

JD van Gool et al., Bladder-sphincter dysfunction in myelomeningocele, Eur J Pediatr (2001) 160: 414-420

Cervical myelopathy

- A variable combination of detrusor hyperreflexia and detrusor sphincter dyssynergia is the commonest urodynamic finding

Herniated Disc

Lumbar disc herniation → irritation of the sacral nerves → detrusor hyperreflexia

Acute compression of sacral roots (trauma) → detrusor areflexia.

Treatment of neurourologic dysfunction

- Goals for **bladder management include :**

1. protecting upper urinary tracts from sustained high filling pressures (ie. >40cm water)
2. minimising post-voiding residual volumes to less than 100ml (ideally <50 ml)
3. preventing urinary tract infections
4. avoiding bladder overdistension
5. maintaining continence
6. choosing a technique which is compatible with person's lifestyle

Wein classification

- Patients who fail to empty
  - because of the bladder
  - because of the outlet
- Patients who fail to store
  - because of the bladder
  - because of the outlet

Bladder retention

A) Behavioral methods-

- timed voiding,
- measures to increase intra abdominal pressure(valsalva, crede)-normal outlet resists leakage and hence may cause rise in bladder pressure
- Promotion of reflex contractions(triggered voiding) by stimulation of sacral and lumbar dermatomes

Bladder retention

B) Pharmacologic methods-

- Parasympathomimetic agents-bethanecol(25-50mg oral qid)
- Alpha antagonists- prazocin(1 -4mg tds) inhibits detrusor as well as relaxes smooth muscle of bladder outlet and striated sphincter tone. Has limited role

Bladder retention

Foleys indwelling catheter

- Infections, bladder neck erosions, fistula, malignancy due to chronic irritation

Suprapubic catheters

- Increased risk of stone formation and infection

CIC(clean intermittent catheterization)

- Introduced by lapides(1972),mainstay of treatment of vesicogenic retention
- Soft catheters(Size 10-14 Fr for males and 14-16 for females) are inserted under well lubricated, clean(rather than sterile) conditions
- Interval is determined to prevent leakage
- Asymptomatic bacteriuria in the absence of infection is common but harmless as long as complete voiding and no increase in intravesical pressure are ensured

- Each catheterization should follow the same routine: clean hands, prepare the material needed, clean the meatal region, apply lubrication, and insert the catheter without touching directly the part which goes into the body
- If resources are limited, catheters can be reused for a long time with cleaning and proper storage techniques between catheterizations.

Bladder retention

D)Surgical methods—

- Reduction cystoplasty-resection of dome of bladder as nerves enter from base
- Bladder myoplasty eg LD flap
- Electrical stimulation-Brindley device stimulates ant sacral nerve roots

Outlet retention

A) Pharmacologic-

- Alpha antagonists-assist in the relaxation of smooth sphincters

  Phenoxybenzamine 10-20mg/day, α1, α2

  Prazosin – α1, 2-3mg/day, first dose phenomenon

  Terazosin doxazosin - long acting α1 blockers

  Tamsulosin, alfuzosin - α1 a blocker

- Striated sphincter-diazepam, baclofen

Outlet retention

B) Surgical---

TURP

Transurethral incision of sphincter -in dyssynergia produces continuous incontinence managed with condom catheter

Bladder incontinence

A) Behavioral—aimed at keeping the volume of bladder below the threshold at which problem occurs

fluid restriction

timed voiding

pelvic floor exercises+ bio feedback

Bladder incontinence

B)Pharmacologic

- Anticholinergic-
  tolteradine (1-2mg/day)
  oxybutinin (2.5-5mg tds)
- Tricyclic antidepressants
  imipramine(25-50mg HS)
- Intra vesical capsaican
  decreases sensory input by damaging unmyelinated afferent fibers
- Intranasal/subcut vasopressin
  useful in nocturnal incontinence
- Denervation
  intravesical botulinum

Bladder incontinence

C)Surgical—

- Augmentation (enterocystoplasty/ureterocystoplasty)
- Urinary diversion eg ileal conduit
- Denervation-sacral deafferentation
  - Selective sacral rhizotomy – b/l posterior sacral rhizotomy *(Brindley)*
- S3 sacral electrode placement

Outlet incontinence

A) Behavioral-

kegel exercises(pelvic floor strengthening)

fluid restriction

timed voiding

B)Pharmacologic-

alpha agonist-stimulate bladder neck and proximal urethral smooth muscle

pseudo ephedrine 30-60 mg 6hrly

Outlet incontinence

C) Minimally invasive

- condom catheters

-absorbent pads

- indwelling catheters

Outlet incontinence

D) Surgical

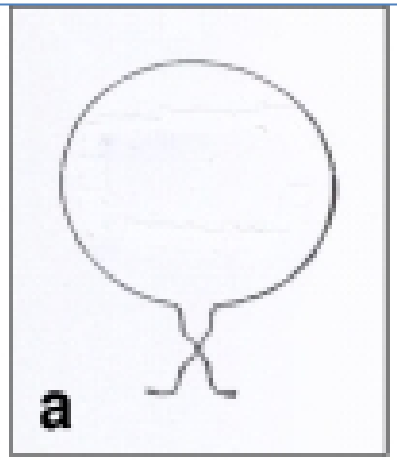
suspension procedures

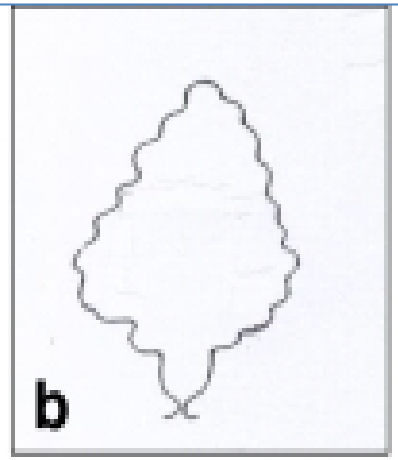
bladder neck reconstruction

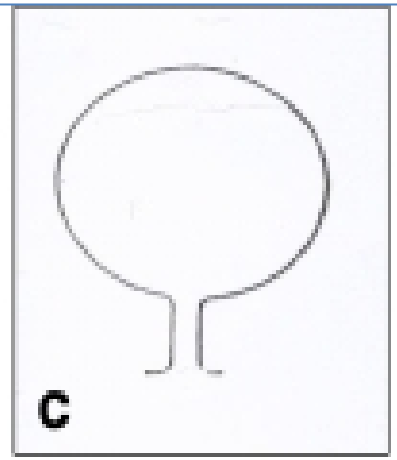
teflon injections in outlet

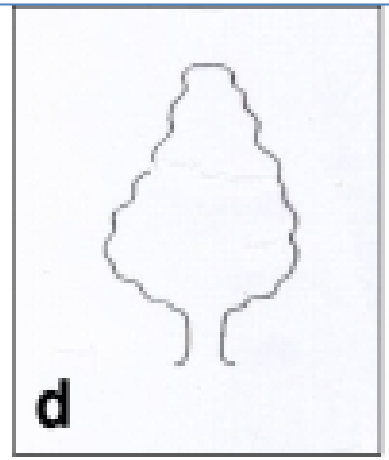
artificial sphincters

In a nutshell









	dysfunction	*consequences*	*management*
a :	sphincter ++ / detrusor --	unsafe, leaking, infections	safe and dry with CIC
b :	sphincter ++ / detrusor ++	DSD, unsafe from birth (reflux, infections, renal damage)	safe and dry with oxybutynin + CIC
c :	sphincter -- / detrusor --	safe but wet	safe and dry with CIC + outlet surgery *cave* detrusorinstability after outlet surgery
d :	sphincter -- / detrusor ++	wet and unsafe	safe and dry with CIC + oxybutynin + outlet surgery

Take home message

- Neurology of the bladder has received insufficient attention to date
- They lead to major morbidity/mortality in SCI and MMC patients
- Simple non operative methods can save patients kidneys and alleviate anxiety
- Recognition of the problem and giving it due importance is the key

Thank you