

# Neurogenic bladder

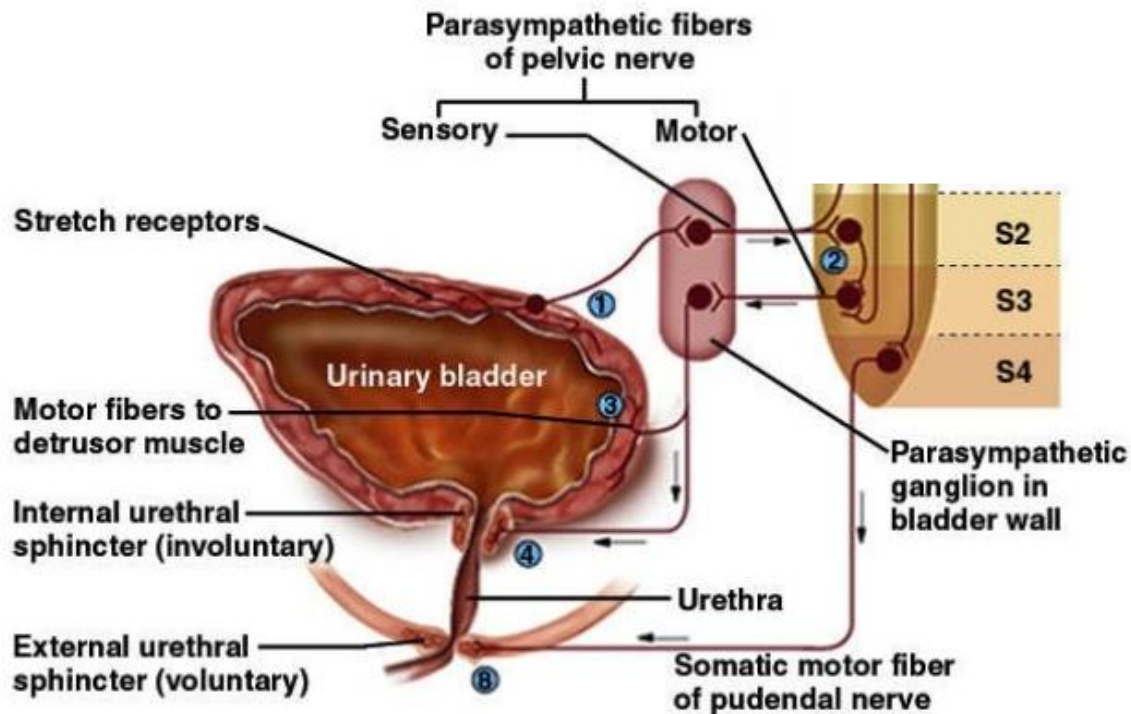
# Introduction

Neurogenic bladder refers to bladder dysfunction due to neurological injury.

This includes:

- 1) lesions above the pontine micturition center producing an **uninhibited bladder**.
- (2) lesions between the pontine micturition center and sacral spinal cord (eg. traumatic spinal cord injury or multiple sclerosis involving cervicothoracic spinal cord) producing an **upper motor neuron bladder**.
- (3) sacral cord lesions that damage the detrusor nucleus but spare the pudendal nucleus producing a **mixed type A bladder**.
- (4) sacral cord lesions that spare the detrusor nucleus but damage the pudendal nucleus producing a **mixed type B bladder**.
- (5) **lower motor neuron bladder** from sacral cord or sacral nerve root injuries.

# Infantile Micturition reflex



# Mature micturition reflex

## Cortical center

Second frontal gyrus(paracental lobule)  
Inhibitory to pontine center

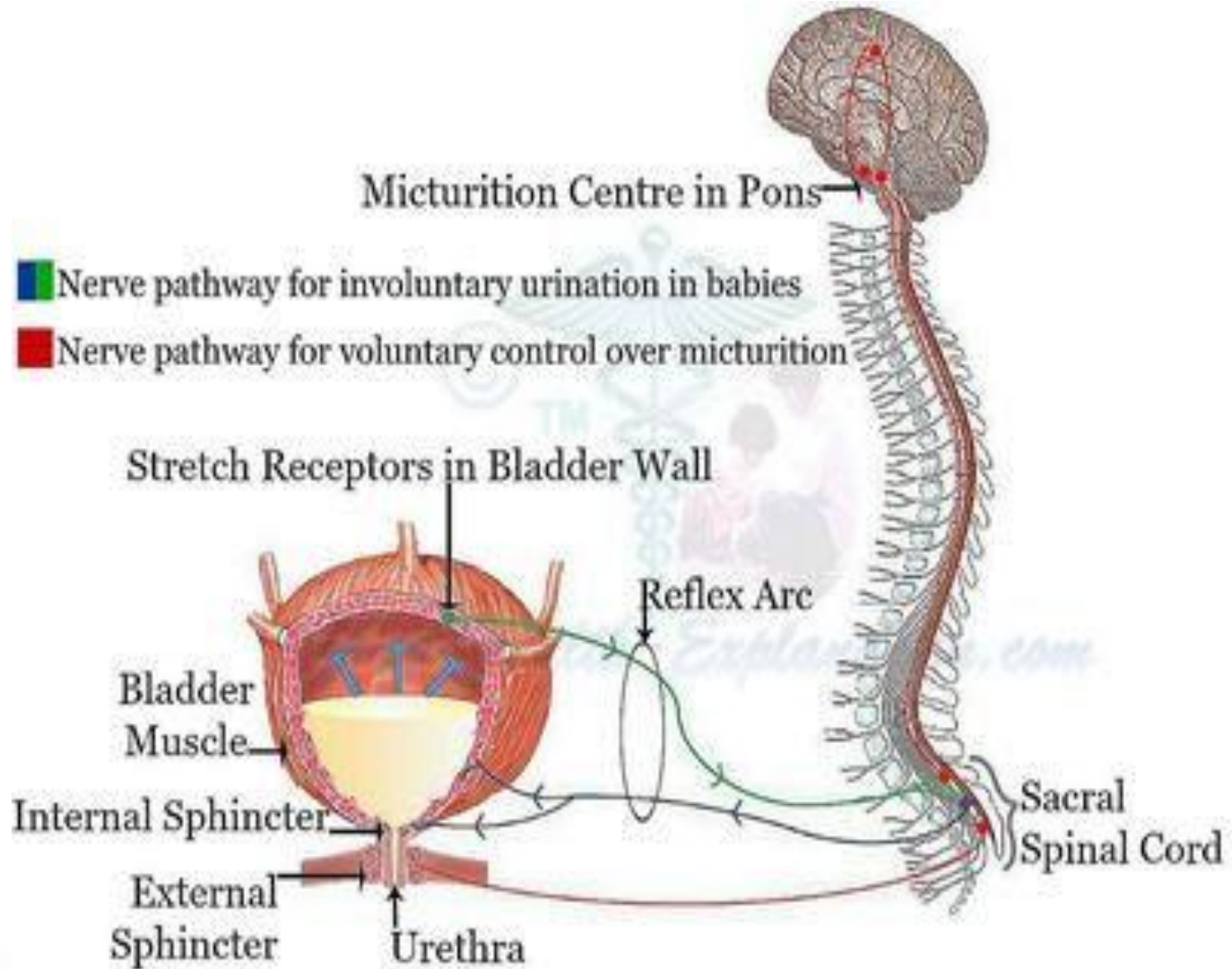
## Brain stem center

Pons- Barrington nucleus  
Facilitatory to micturition

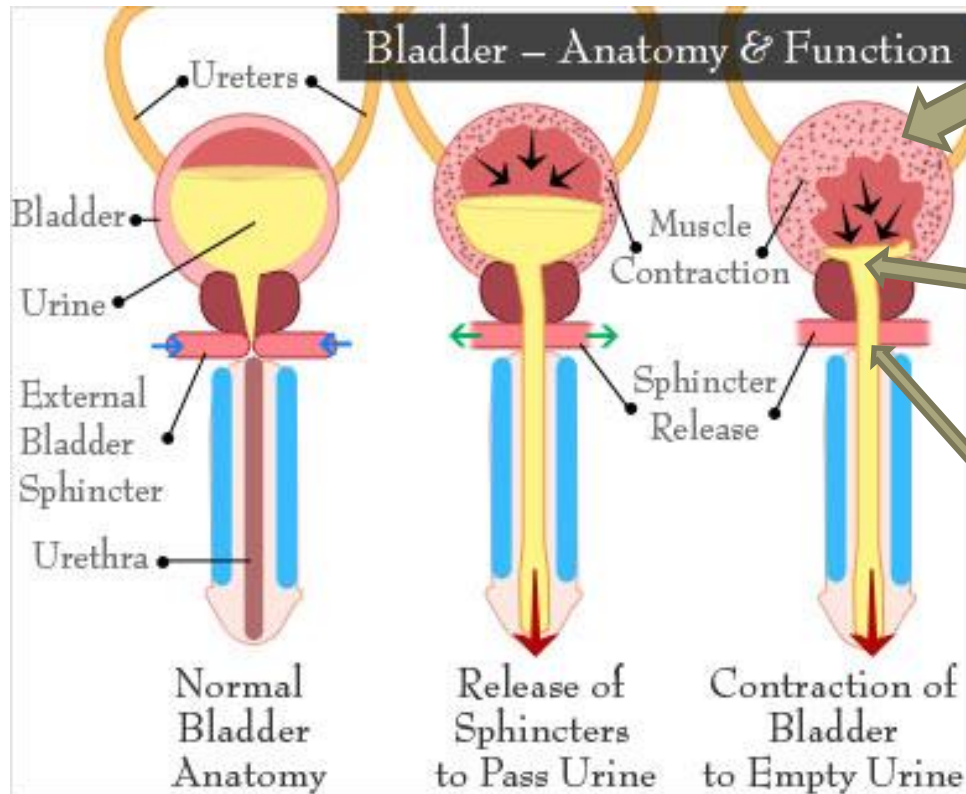
## Sacral spinal cord (Onuf nucleus)

Parasympathetic (S2,S3,S4)  
Reflex evacuation

# Mature micturition reflex



# Normal anatomy

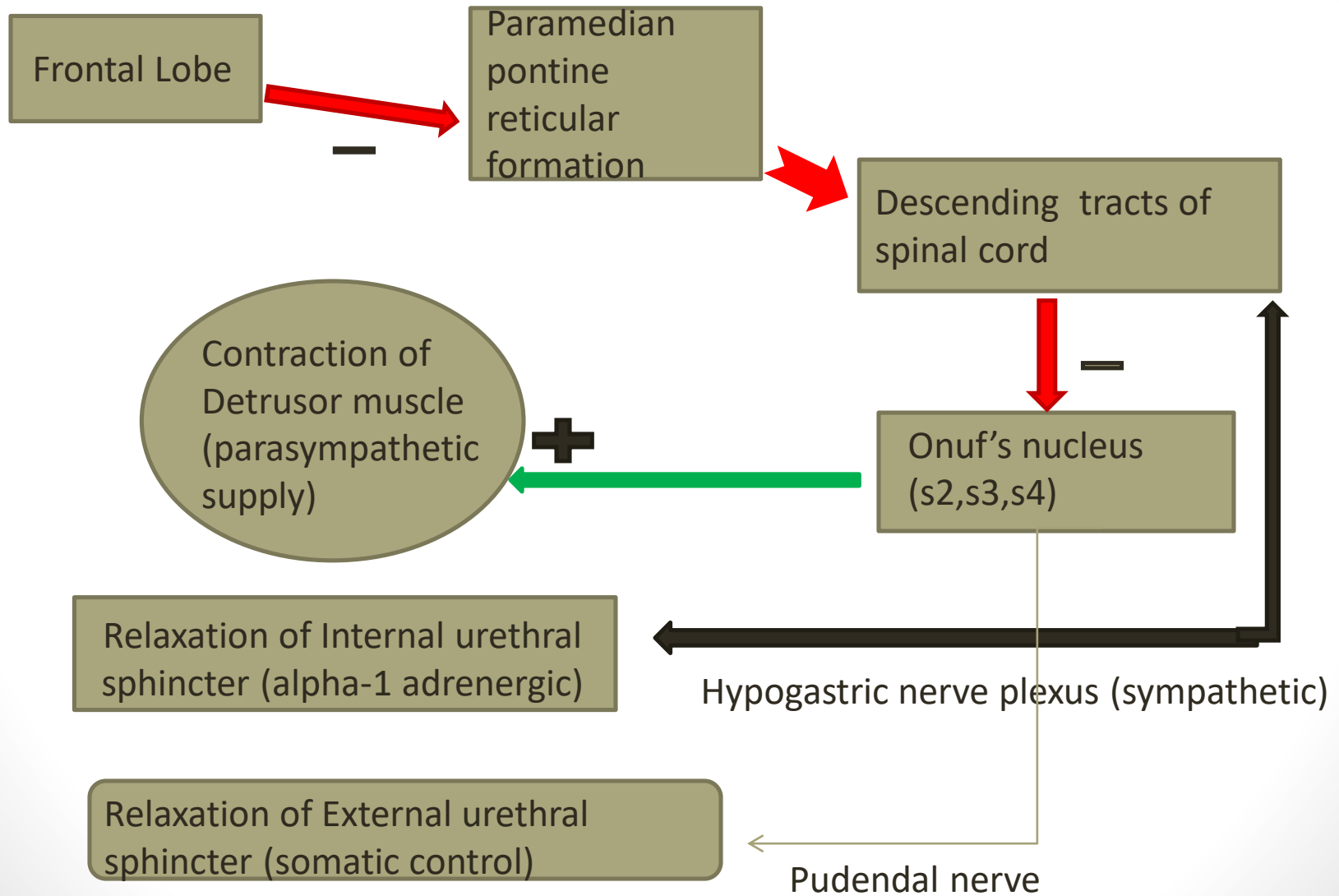


M3- Muscarinic receptors

Alpha 1 adrenergic receptor

Pudendal nerve (somatic motor efferent)

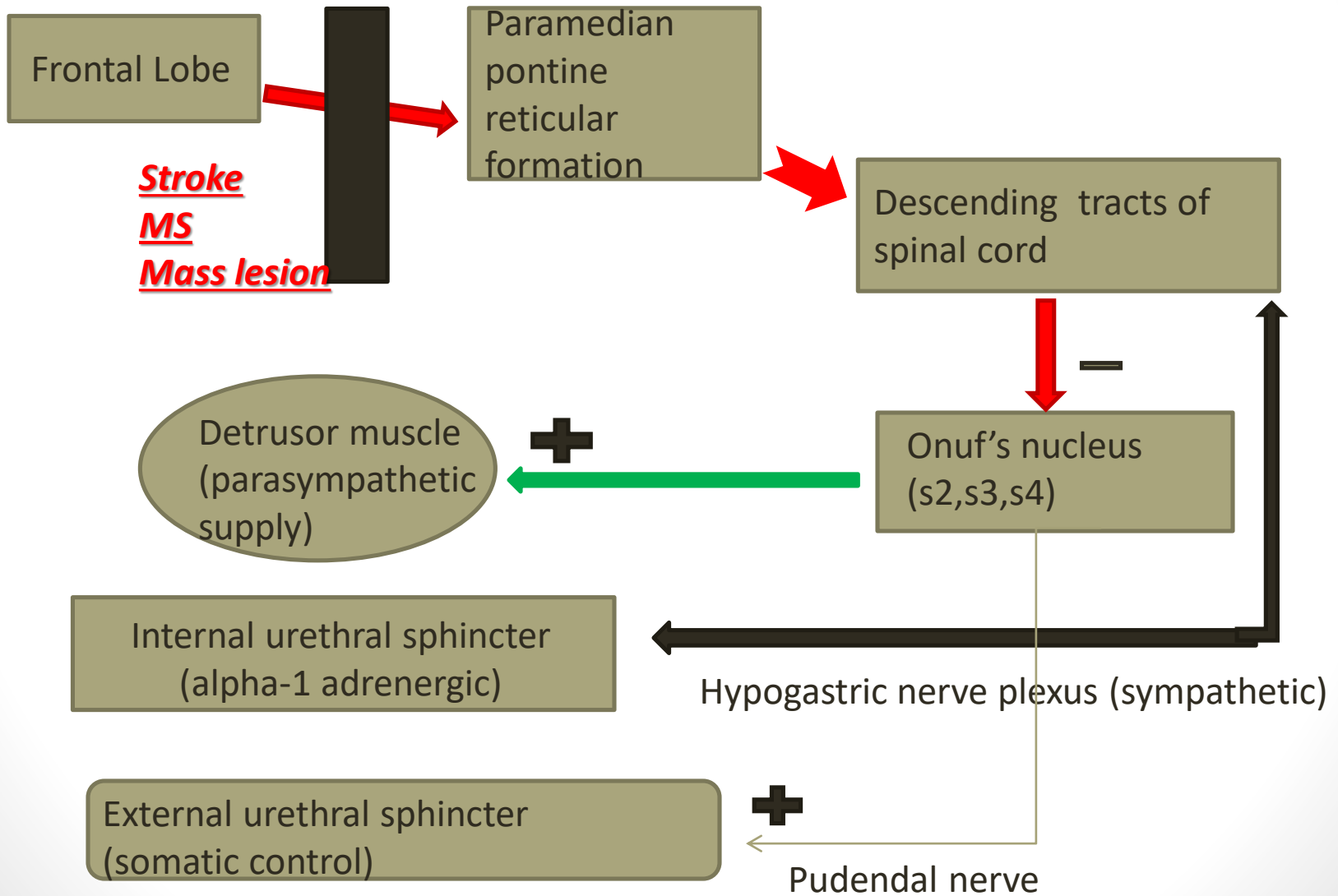
# Micturition neuro-physiology



# Levels of lesions



# Uninhibited bladder



# Causes

Most common causes include:

- Anterior circulation Stroke
- Multiple Sclerosis
- Intracranial space occupying lesions
- Cerebral palsy
- Fronto-temporal dementia
- Alzheimers disease
- Normal pressure hydrocephalus

# Pathogenesis

- The uninhibited neurogenic bladder or **infantile bladder** is characterized by uncontrolled contractions of bladder smooth muscle.
- In the infant up to two to three years of age, filling of the bladder stimulates proprioceptive endings in the bladder wall which in turn elicit a micturition reflex and resultant detrusor contraction.
- In the **adult** an **acquired defect** in the corticoregulatory tract may result in an uninhibited bladder.

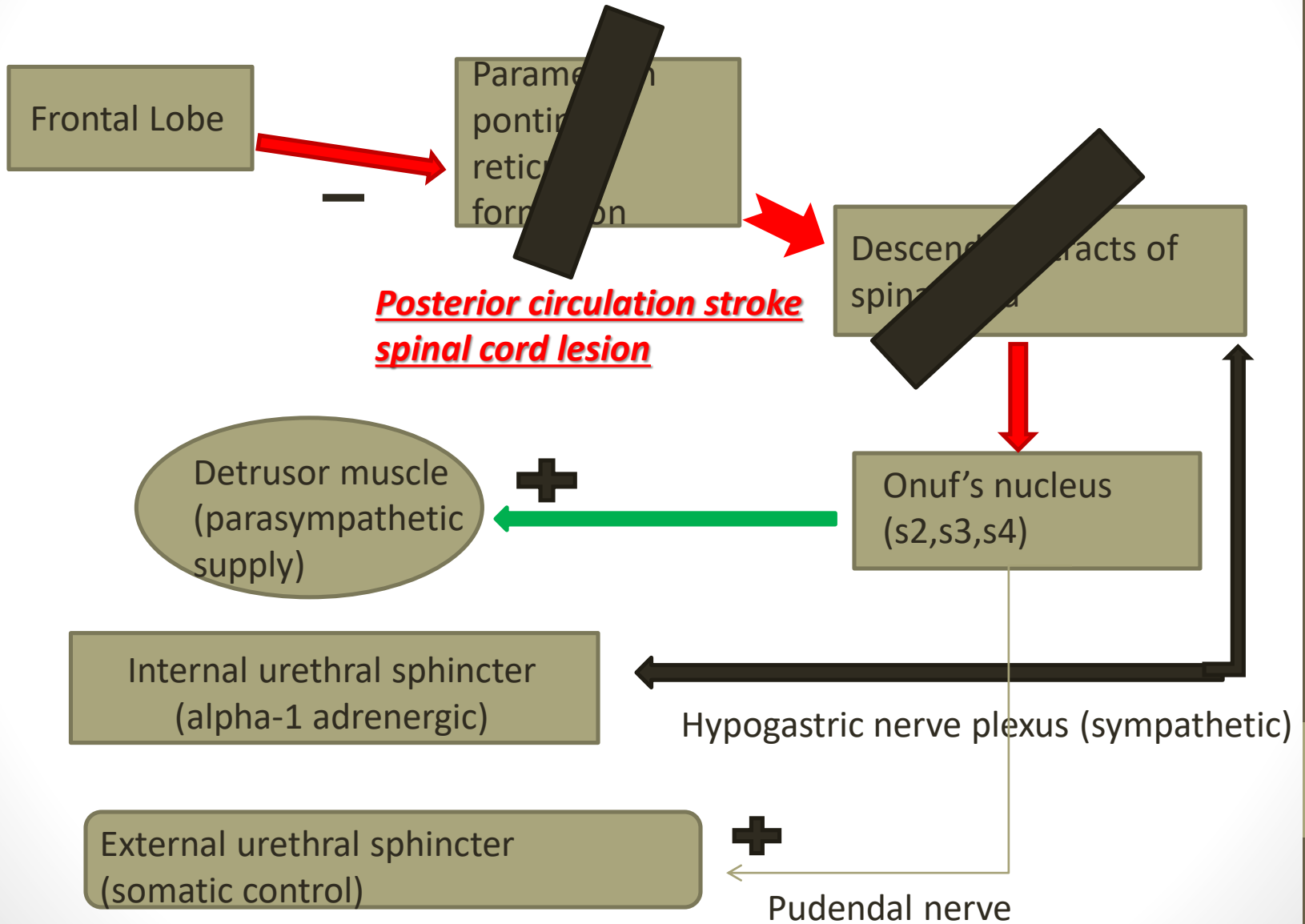
# Symptoms

- Acute onset of urge incontinence, increased urinary frequency and enuresis.
- Inappropriate urination (no social inhibition)

# Complications

- Social withdrawal: due to inappropriate social behavior
- UTI: due to frequent leaking of urine and poor hygiene

# Spastic neurogenic bladder



# Causes

Most common causes in adults include:

- cord injury (excluding sacral spine)
- Posterior circulation Stroke
  
- In children:
- Sacral agenesis
- Spina bifida (Myelomeningocele)

# Pathogenesis

- Pontine center regulates voiding by synergizing the contraction of detrusor muscles and relaxation of the urethral sphincter.
- Thus pontine lesions (and spinal cord lesions above the sacral spinal segments) are characterized by detrusor-sphincter dyssynergia (DSD)



# Detrusor-sphincter dyssynergia (DSD)

- DSD is described as asynchronous contraction of the detrusor muscles and urethral sphincter.
- Simultaneous contraction of the detrusor muscles and urethral sphincter leads to very high internal bladder pressure.
- If the detrusor pressure exceeds sphincter pressure incontinence occurs else urinary retention occurs.

# Symptoms

- Lower abdominal pain, abdominal spasms
- Increased urgency, frequency of urination, leaking of urine (**urge incontinence**)
- **Urinary retention**
- Difficulty voiding voluntarily
- Patient will need to strain (Valsalva manoeuvre) or compress the lower abdomen (Crede manoeuvre) to pass urine.

# Differential Diagnosis

For increased urgency and frequency:

- Urinary tract infection
- Urethral obstruction (initial stages)

For Urinary retention:

- Anti-muscarinic drugs (TCA's, Typical Anti-psychotics, First generation anti-histamines)
- Alpha-1 adrenergic agonists
- Urethral obstruction (late stages)

# Spinal Shock

- Seen in the first 2-3 weeks after a CNS insult
- All spinal reflexes will be suppressed
- Patients may present with acute urinary retention with no urinary urgency or abdominal pain
- The risk is highest during the first 48 hours
- Thus all patients immediately after a CNS insult need urgent urinary catheterization.

# Acute complications of DSD

# Clinical scenario

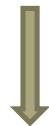
A known case of spastic quadriparesis residing in a nursing home presents to the ER complaining of headache, blurry vision, vomiting and chest pain. Patient has altered sensorium. Non contrast CT brain shows no acute intracranial bleed. EKG shows T wave inversion and ST depression in leads V4,V5 and V6. Cardiac troponins are negative. Patient has an indwelling Foley catheter



Inspection of his nursing home medical records shows that total urine output in the last 36 hours has been 100ml



Abdominal palpation reveals non tender cystic swelling in lower abdomen



## **Vitals:**

**PR = 180/min**

**BP = 230/109 mm Hg**

# Autonomic Hyperreflexia

- Acute life threatening complication seen in case of spinal lesions above T-6 spinal level.
- Occurs due to disinhibition of sympathetic nervous system.
- Any noxious stimulation (pain, inflammation, bladder distention, bowel distention) can lead to excessive sympathetic outflow.
- **Should be suspected in any patient with cord injury above T-6 spinal level if SBP increases by > 40mm Hg from baseline**

# Typical patient presentation

Patients with autonomic hyperreflexia can present with:

- Tachycardia
- Arrhythmias
- Hypertensive urgency/ emergency
- Cold extremities
- **Above the level of cord injury patient will have:**

**Flushing**

**Diaphoresis**

**This can be attributed to parasympathetic supply remaining intact above the level of spinal cord lesion**



# Prevention

- All cases of spinal cord injury above T-6 spinal level must have regular BP monitoring. Any rise in SBP >40mm Hg above baseline is indicative of autonomic hyperreflexia.
- There must be:
- Regular fluid input and urine output monitoring
- Regular enemas to prevent bowel over-distention
- Early detection and treatment of any infection

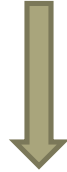
# Management

- 1) If indwelling foley catheter is in place irrigate the catheter with 20ml saline to check for blocks
- 2) enema to relieve bowel over-distention
- 3) Emperic antibiotics if WBC count is elevated
- 4) Anti-hypertensive drugs to be given if SBP >150 mm Hg
- **DONOT use Beta-blockers**
- Nitrates, nifedipine or sildenafil can be used to control BP
- 5) Monitor for and manage end-organ damage if present

# Long term Complications of DSD

# Pathogenesis of post renal failure

Bladder hypertonicity produces hypertrophy of the detrusor muscle.

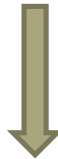


The normal oblique course of the ureter through the detrusor wall at the ureterovesicular junction is compromised to allow vesicoureteral reflux.



Bilateral hydronephrosis occurs

Chronic backflow and stasis of urine



Recurrent Pyelonephritis occurs

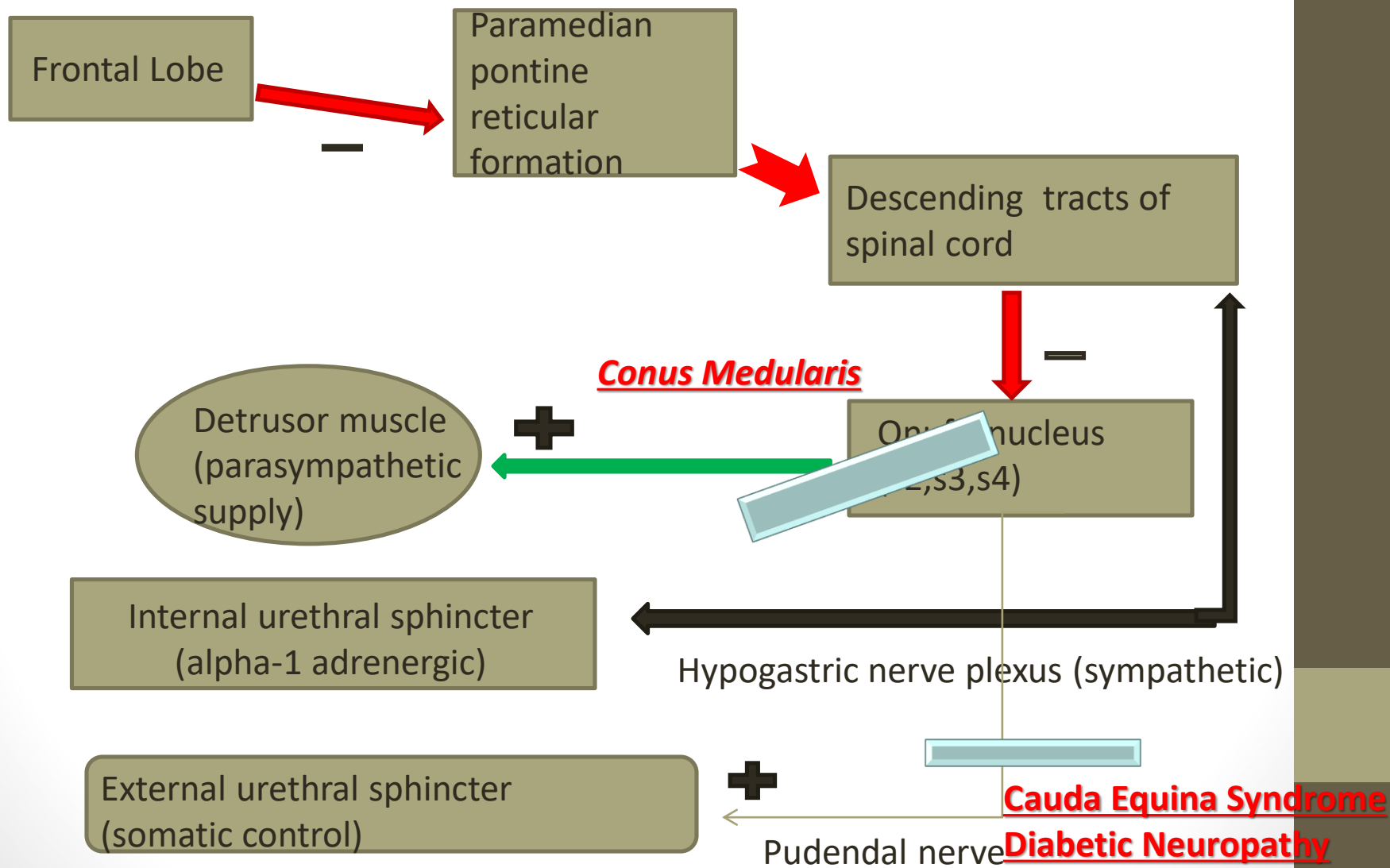
End stage complication



Post Renal failure

# Flaccid bladder

# Flaccid Neurogenic Bladder



# Causes

Most common causes include:

1) **Sacral spine/nuclear lesions**: Conus Medullaris syndrome, traumatic injury to sacral spinal segments

2) **Spinal shock**

3) **Lower motor Neuron lesions**:

- May include compressive neuropathy (cauda equina syndrome)
- Demyelination neuropathies (Diabetic Neuropathy)
- Iatrogenic causes: Damage to pudendal nerve during pelvic procedures

# Clinical symptoms

Overflow incontinence is the main presenting symptom. Other symptoms include abdominal distention and bloating sensation.

Patients will present with:

- Difficulty initiating stream of urine
- Involuntary dribbling of urine
- No pain on urination, no urgency



# Differential diagnosis

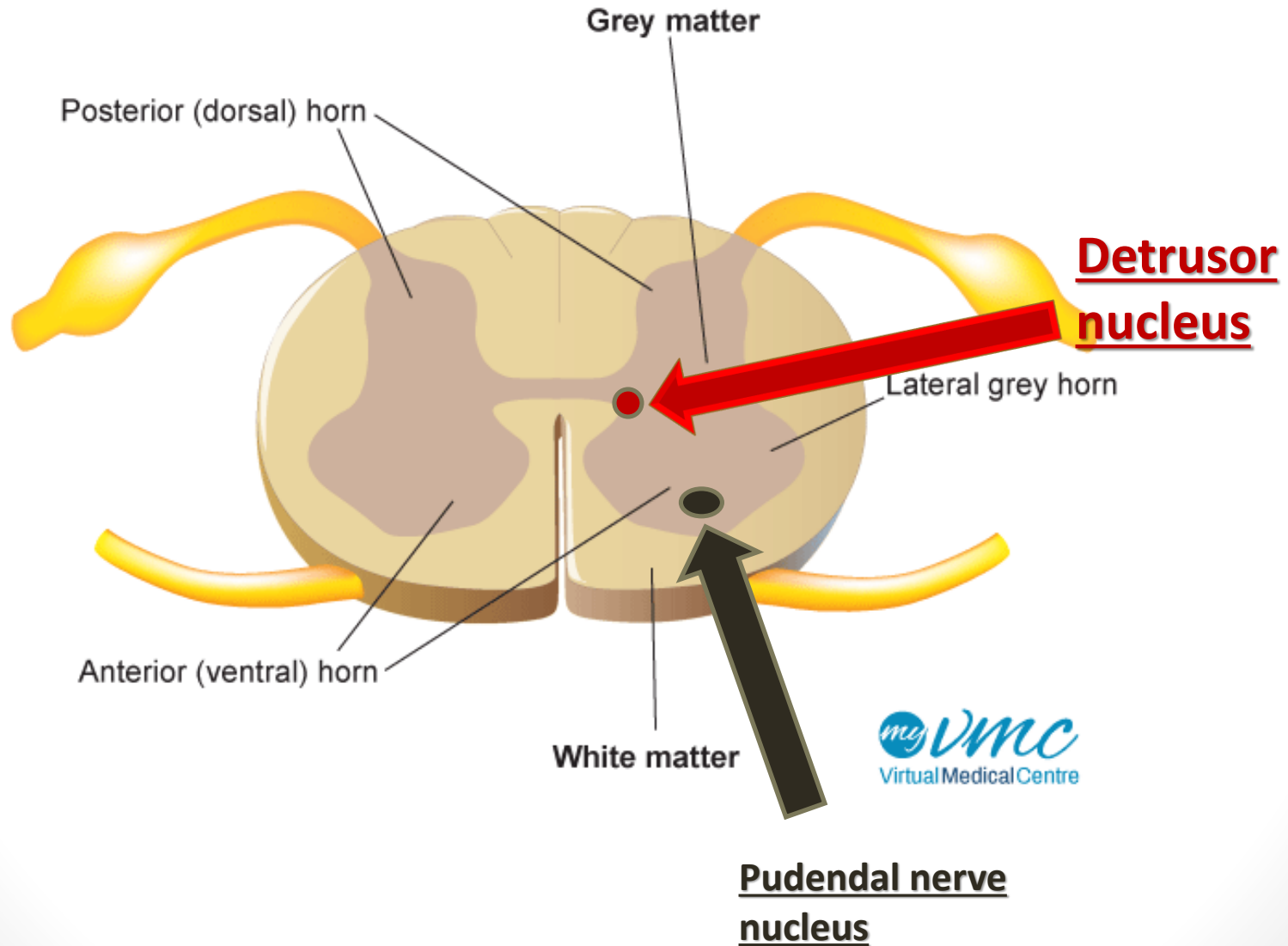
Other common causes of over flow incontinence are:

- BPH
  - Pelvic organ prolapse (POP)
  - Epidural analgesia
  - Spinal anesthesia
- 
- BPH and POP can be differentiated from flaccid bladder by the presence of abdominal pain and urgency in the former

# Complications of flaccid bladder

- 1) Recurrent UTI's
- 2) Bladder calculi due to stagnation of urine
- 3) Maceration and secondary infection of skin of the groin
- 4) vaginitis/cervicitis leading to Pelvic Inflammatory Disease
- 5) Myogenic bladder: chronic overdistention of bladder leads to permanent detrusor muscle damage leading to irreversible distended bladder

# Onuf nucleus (S2,S3,S4) anatomy



# Mixed type A neurogenic bladder

- **The more common of the mixed type bladders**
- **Detrusor nucleus damaged:** detrusor nucleus damage renders the detrusor flaccid (also referred to as detrusor areflexia)
- **Pudendal nucleus intact:** the intact pudendal nucleus is spastic producing a hypertonic external urinary sphincter
- The bladder is distended and has low pressure due to detrusor hypotonia
- Spastic external sphincter produces **urinary retention** and incontinence is uncommon.
- The detrusor pressure is low **so upper urinary tract damage does typically not occur**

# Mixed type B neurogenic bladder

- **Detrusor nucleus intact:** the bladder is spastic due to the disinhibited detrusor nucleus.
- **Pudendal nucleus damaged:** external urethral sphincter is flaccid
- Therefore bladder volume is low (**no urinary retention**)
- Due to flaccid external urethral sphincter **incontinence occurs.**

# Overview of clinical features

Type of neurogenic bladder	Bladder volume	Internal bladder pressure	Risk of kidney damage	Incontinence/retention
Uninhibited bladder	normal	normal	low	Incontinence (social disinhibition)
Spastic bladder	low	high	high	Retention and/or Urge incontinence
Flaccid bladder	high	low	low	Overflow incontinence
Mixed type A	high	low	low	Retention
Mixed type B	low	low	low	Overflow incontinence

# Diagnosis

# Diagnosis of neurogenic bladder

Steps of diagnosis	Testing modalities	Used to detect
1) Identify type of bladder dysfunction: (Spastic, flaccid, mixed)	1) Postvoidal residual volume 2) Cystometrography 3) Peak urinary flow rate testing 4) Pressure flow video studies	1) Bladder volume 2) Bladder pressure 3) Urethral sphincter pressure 4) Detrusor sphincter dyssynergy
2) Etiology of bladder dysfunction	1) Urine analysis/culture 2) CBC with differential count 3) HbA1c 4) USG KUB 5) CT/MRI brain 6) MRI spine	1) Presence of UTI 2) Risk of Diabetic neuropathy 3) Urethral outflow obstruction 4) CNS lesion



# Diagnosis of neurogenic bladder

- **Diagnostic tests:**

**1) Postvoid residual volume :** Postvoid residual volume plus voided volume estimates total bladder capacity and helps assess bladder proprioception.

**A volume <50 mL is normal; < 100 mL is usually acceptable in patients > 65 but abnormal in younger patients; and > 100 mL may suggest detrusor underactivity or outlet obstruction.**

**2) Cystometrography:** pressure-volume curves and bladder sensation are recorded while the bladder is filled with sterile water; provocative testing (with bethanechol) is used to stimulate bladder contractions.

# Diagnostic tests

- **Peak urinary flow rate testing:** with a flow meter is used to confirm or exclude outlet obstruction in men.

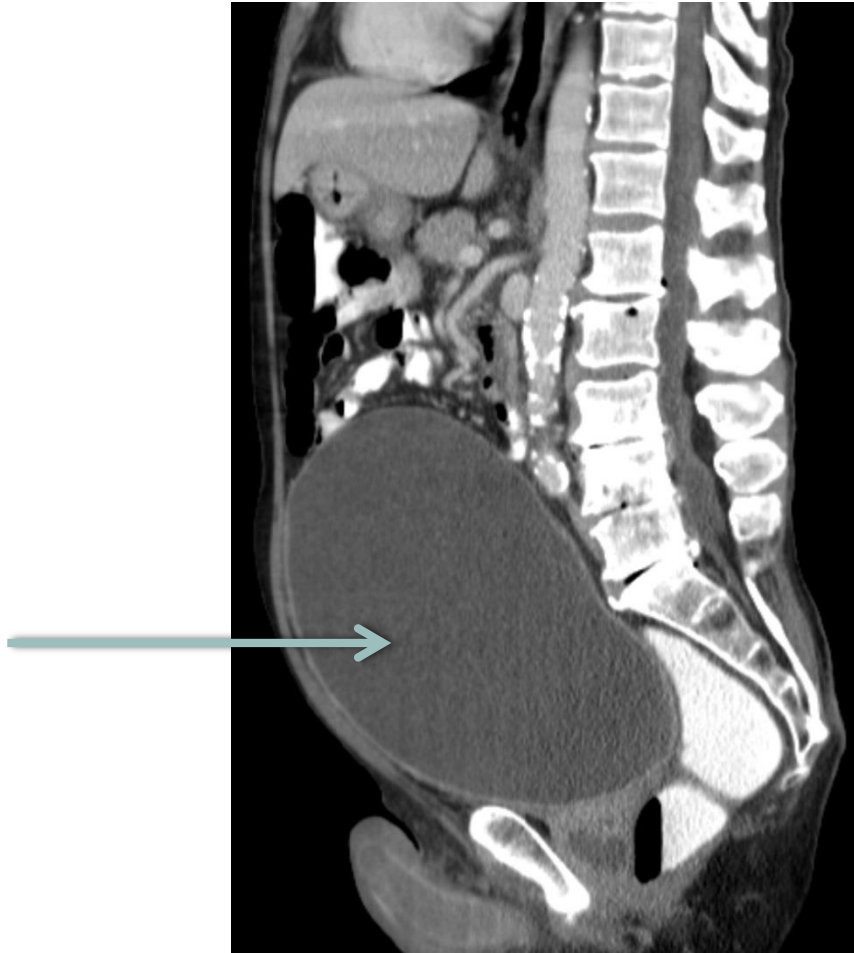
Results depend on initial bladder volume, **but a peak flow rate of  $< 12$  mL/sec with a urinary volume of  $\geq 200$  mL and prolonged voiding suggest outlet obstruction or detrusor underactivity.**

**A rate of  $\geq 12$  mL/sec excludes obstruction and may suggest detrusor overactivity.**

- **Pressure-flow video studies:** done with voiding [cystourethrography](#), can correlate bladder contraction, bladder neck competency, and detrusor-sphincter synergy,

# CT imaging

Detrusor  
muscle  
hypotonia  
causing  
bladder  
distension



# Treatment of neurogenic bladder

- Goals of therapy:
  - 1) achieve or maintain continence
  - 2) prevent upper urinary tract damage
  - 3) minimize risk of UTI's
  - 4) prevent bladder overdistention

# Treatment of uninhibited neurogenic bladder

Treatment is primarily behavioral therapy which involves:

- 1) Fluid intake time table
- 2) Trained timed voiding: patient is trained to trigger voiding by performing Crede maneuver or valsalva manoeuvre at a pre-fixed time and place

# Treatment of spastic neurogenic bladder

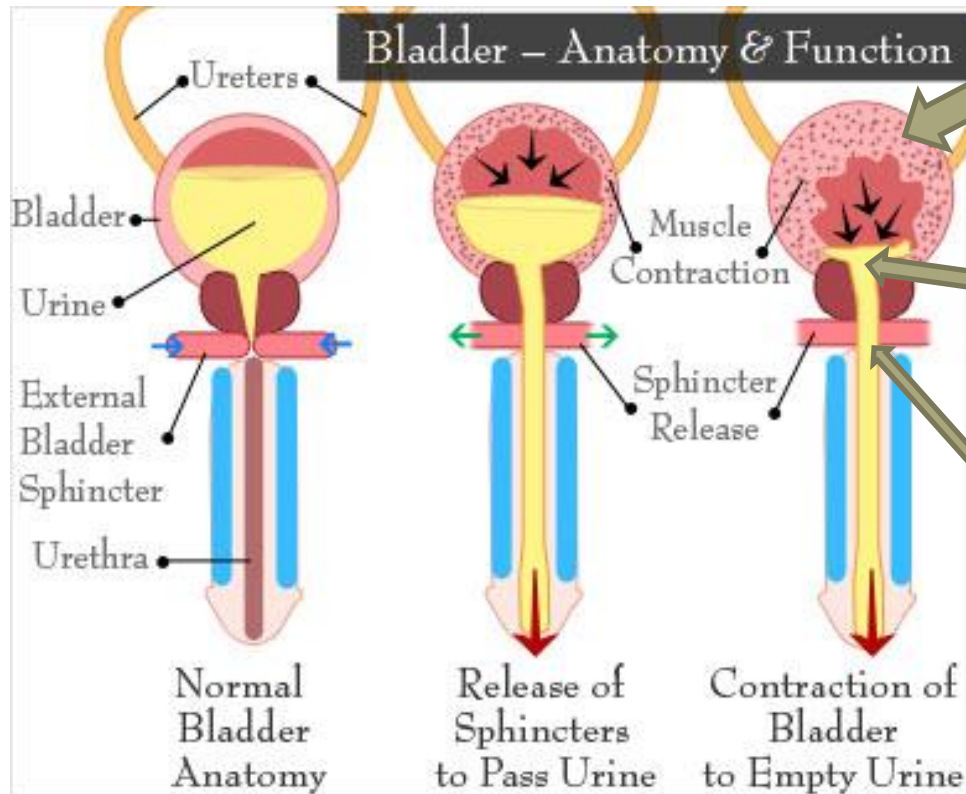
## 1. Non Pharmacological

- a) Fluid intake time table
- b) Voiding stimulation techniques (Crede technique, valsalva)

## 2. Medications:

- First line agent: **anti-muscarinic** (oxybutynin, tolterodine)
- Trospium is a M-3 specific anti-muscarinic agent that has lower ADR profile than oxybutynin
- Adjuvant agents: **alpha 1 adrenergic blocker** (prazosin), **Tri-cyclic antidepressant** (imipramine)
- **Alpha-2 agonists** (clonidine) can be used for internal urethral sphincter spasm
- Benzodiazepines can be used for external urethral sphincter spasm

# Normal anatomy



M3- Muscarinic receptors

Alpha 1 adrenergic receptor

Pudendal nerve (somatic motor efferent)

# Treatment of spastic neurogenic bladder

- 3. **Minimally invasive techniques:**
  - A) clean intermittent self catheterization
  - B) Injection of BOTOX into detrusor muscle and internal urethral sphincter to relieve spasm.



# Refractory cases of urinary retention

- **Invasive Surgical techniques:**
- sacral nerve root stimulation
- Last resort measure: Enterocystoplasty, which is the anastomosis of the bladder to the ileum allowing urine to drain into the bowel. This is done as a last resort measure incase post-renal failure is imminent.

# Treatment of flaccid neurogenic bladder

- **Flaccid bladder:** Treatment of flaccid bladder often involves invasive modalities of management and is associated with more complications than spastic bladder.
- Non invasive modalities:
- **1) condom catheter**
- **2) cholinergic agonists : (Urecholine)** this modality has many contraindications such as
  - **Bronchial asthma, COPD: bronchospasm**
  - **Cardiovascular disease: heart block**
  - **Parkinsons disease: increase in tremors**

# Treatment of flaccid neurogenic bladder

- Invasive modalities:

Indwelling Foley's catheter	Suprapubic catheter
Lower risk of 1) UTI 2) Bladder calculi	Lower risk of 1) Urethral damage 2) Urethral strictures

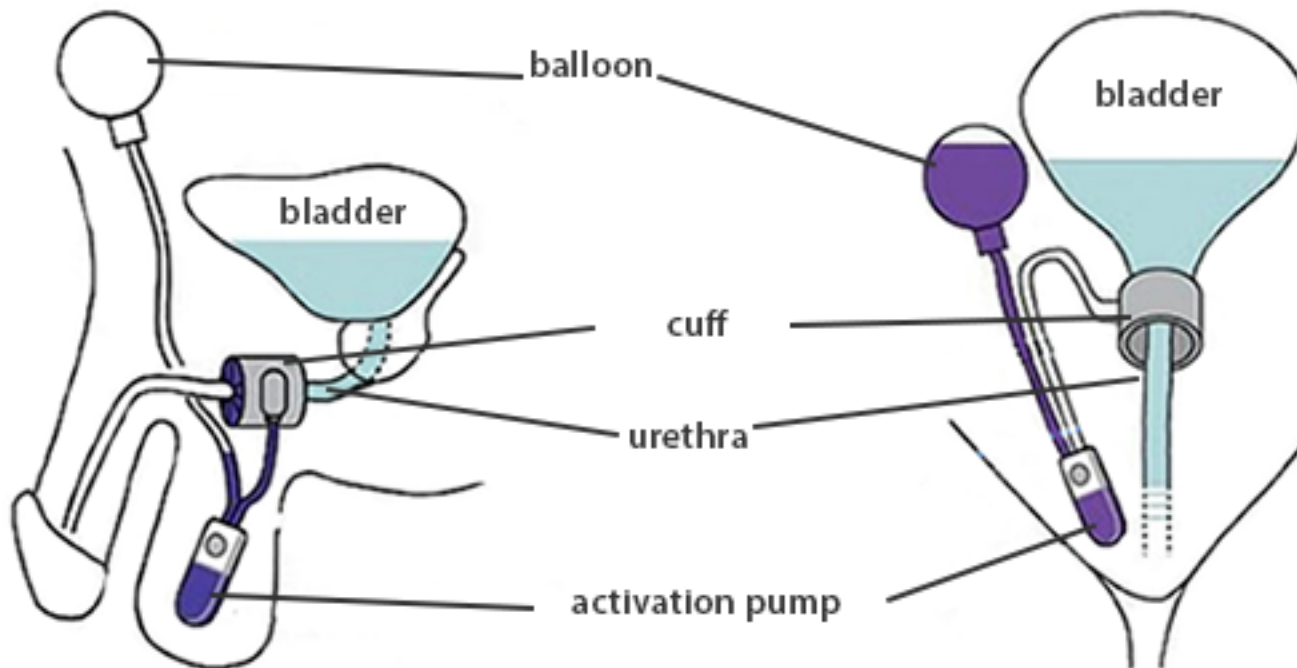
Common precautions to be taken:

- 1) High fluid intake (>3L per day) to avoid catheter block calculi formation
- 2) 11g of dietary citrate per day to alkalinize the urine and reduce precipitation of calcium and magnesium salts

# Surgical measures for flaccid bladder

- **Artificial implantable urinary sphincter** : Gold standard modality for treatment of flaccid bladder

*Implantation of an artificial sphincter to a man (left) and a woman (right).*



# References

- 1) Dorsher P.T., Mcintosh P.M, (2012) Neurogenic bladder, *Advances in Urology*,doi:10.1155/2012/816274
- 2) Koyuncu, E., & Ersoz, M. (2017). Monitoring development of autonomic dysreflexia during urodynamic investigation in patients with spinal cord injury. *The Journal of Spinal Cord Medicine*, 40(2), 170–174.  
<http://doi.org/10.1179/2045772315Y.00000000049>