Neurogenic Bladder (NB)

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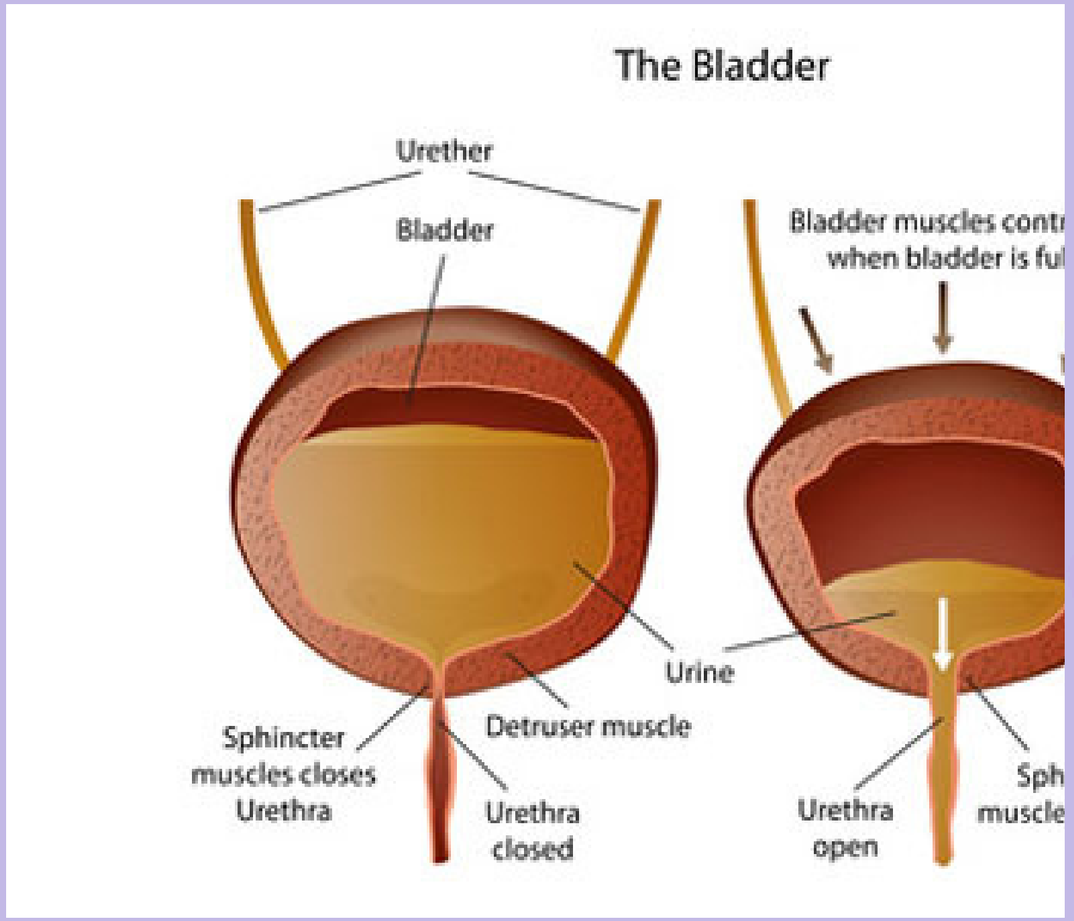
- Neurogenic bladder is a term used to describe lower urinary tract (LUT) dysfunction resulting from a neurologic disease or process.
- Up to 80% of neurological diseases develop Lower Urinary Tract Symptoms (LUTS)
- Accurate diagnosis and proper management of LUT dysfunction in the neurogenic population consist of two main goals: (1) to preserve the safety of the bladder with lowpressure storage and adequate emptying and (2) to maintain a reasonable quality of life in relationship to the bladder.

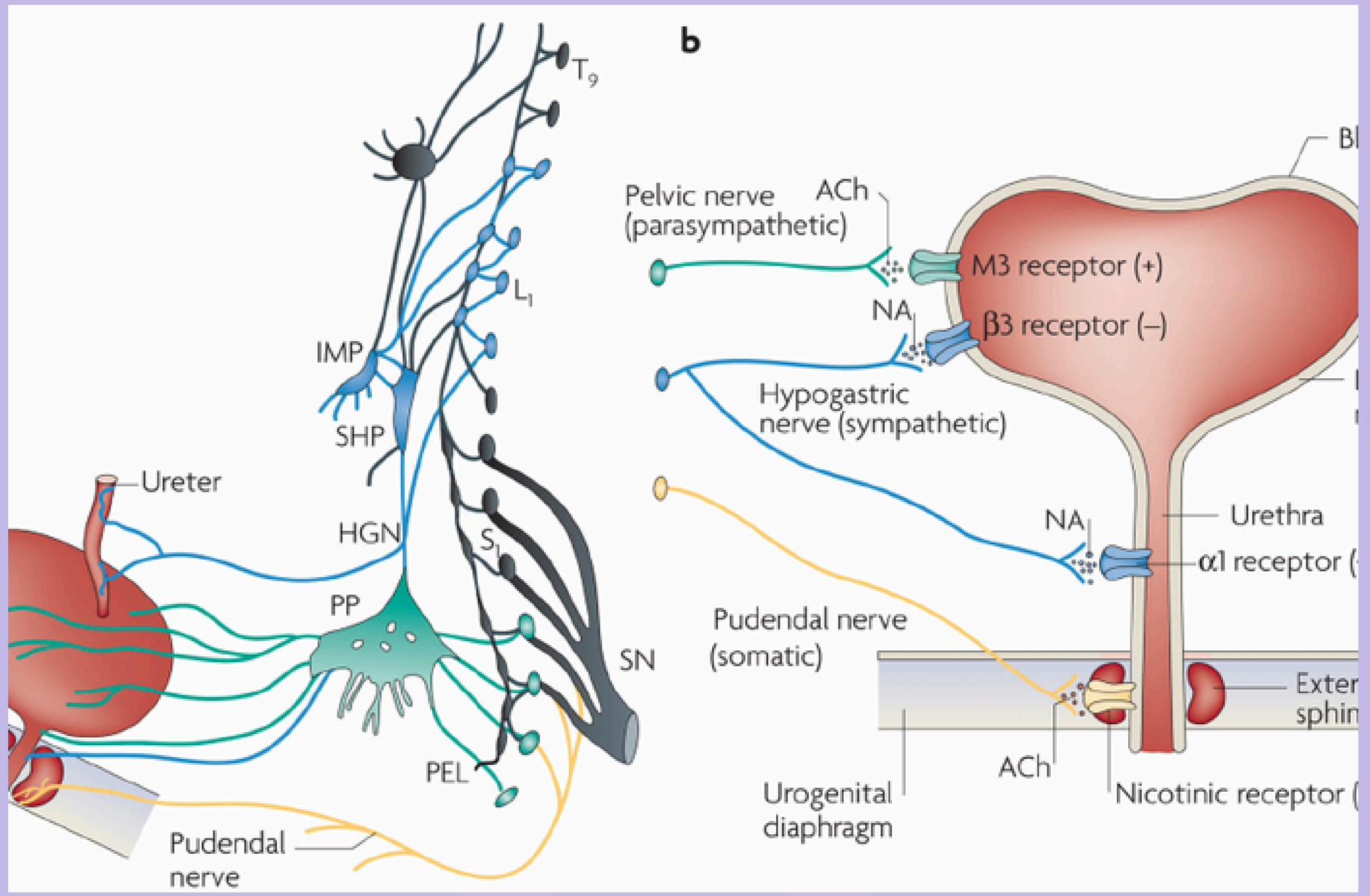
lower urinary tract function

urine storage and micturition depend on the coordination of the bladder, bladder neck ,urethra and urethral sphincter



coordination between the muscles of the lower urinary tract is mediated by neural pathway in the ;
brain
spinal cord
peripheral nerve

NEURAL CONTROL OF THE LOWER URINARY TRACT (LUT)



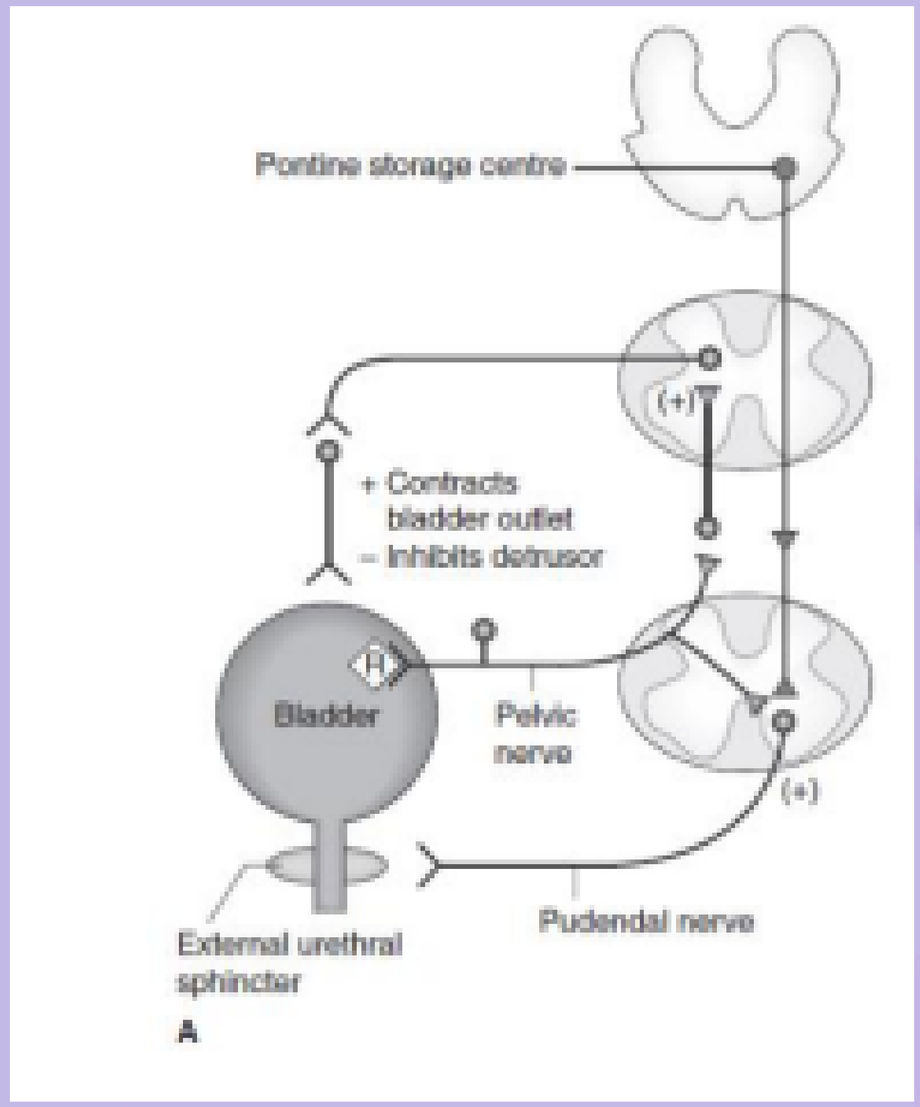
Phase	Storage phase
Parasympathetic	Off
Sympathetic	On
Somatic	On

- Pelvic splanchic nerve (Parasympathetic) signals lead to contraction of the Detrusor (M3 receptors = Stimulatory).
- Hypogastric nerve (Sympathetic) signals lead to Relaxation of the Detrusor (B3 receptors = Inhibitory) and contraction of Internal Urethral sphincter (A1a receptors = Stimulatory).
- Puodendal nerve (Somatic) signals lead to Contraction of the external urethral sphincter (Nicotinic receptors = Stimulatory)

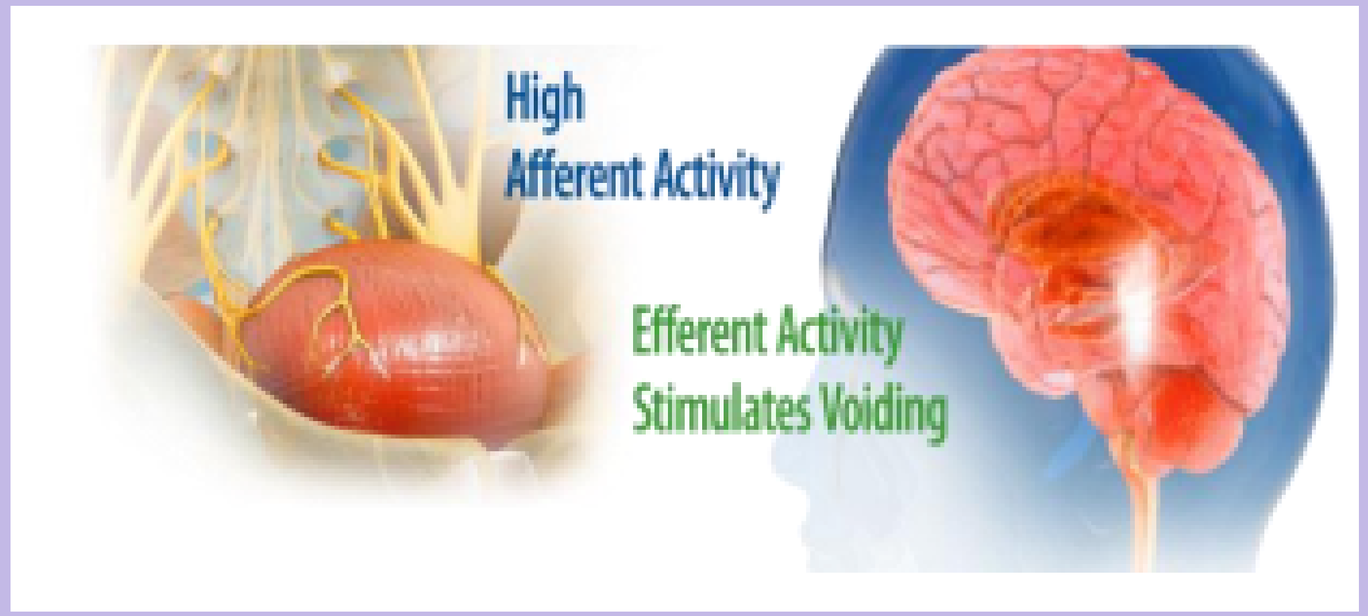
low-level afferent signal are organized in the spinal cord and promote urine storage via if it on signal from the cns

urine storage reflux

- During normal bladder filling, sympathetic (hypogastric nerve) and somatic (pudendal nerve) neural mechanisms mediate the contraction of the internal smooth and external striated urethral sphincters, respectively. As the bladder fills, sympathetic-mediated inhibition of the detrusor allows for the bladder to accommodate increasing volumes at low intravesical pressures



so this reflects is *on*
when we have
“ low intensity signal and ssocially causes”



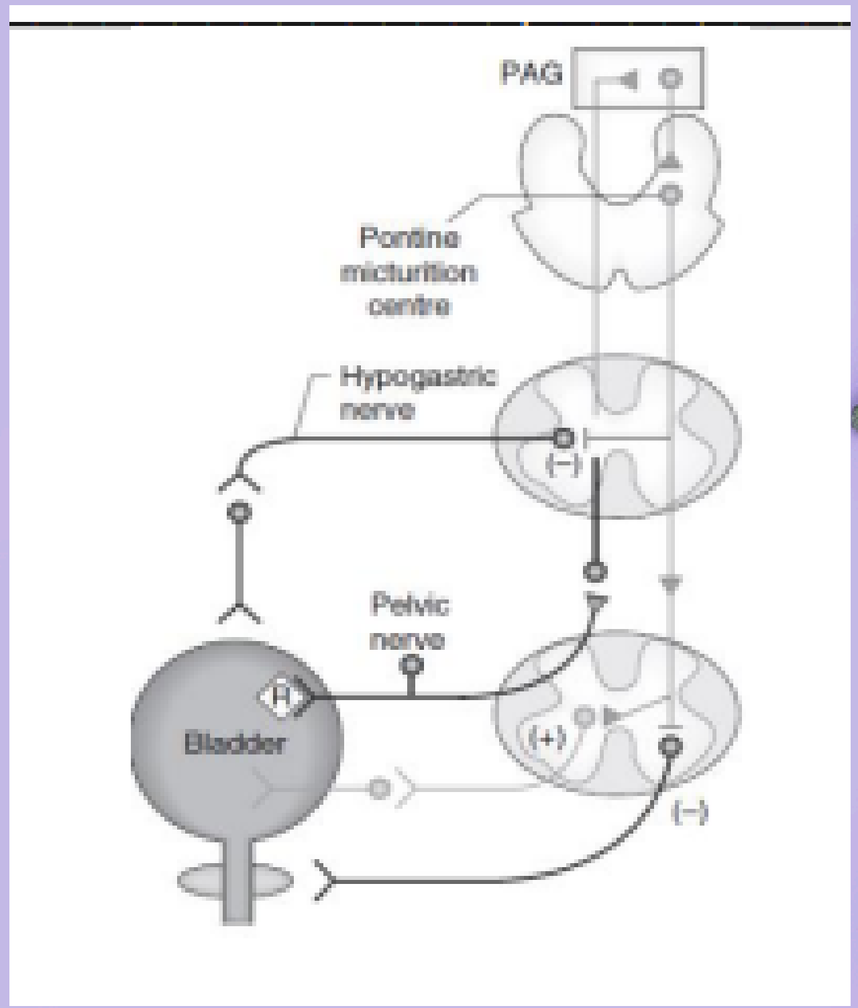
high level afferent signal are stimulated by intravesical pressure resulting in increased activity to the brain

descending efferent pathway then cause voluntary bladder contraction and the flow of urine

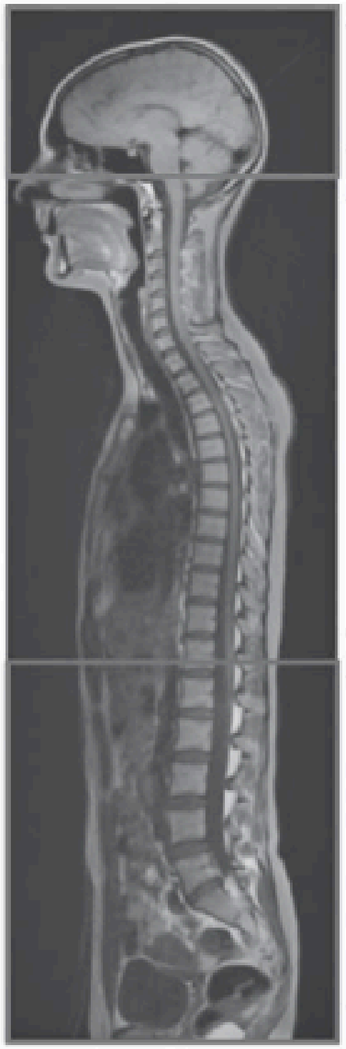
normal voiding is. therefore, a function of a positive feedback mechanism

Urine voiding reflex
A positive feedback loop

- When an individual decides that it is time to urinate, the pontine micturition center (PMC) is released from tonic inhibitory control of the higher cortical and subcortical centers of the brain, initiating the voiding process. The parasympathetic system then switches “on,” stimulating a detrusor contraction and relaxation of the pelvic floor and external and internal urethral sphincters



Nervous system damage and LUT dysfunction



Suprapontine lesion
- **History:** predominantly storage symptoms
- **Ultrasound:** insignificant PVR urine volume
- **Urodynamics:** detrusor overactivity

Upper motor neuron

Storage symptoms : Frequency , Urgency & Nocturia.

Each filling episode of bladder will leave to activation of the pontine center.

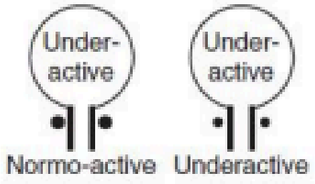
Spinal (infrapontine-suprasacral) lesion
- **History:** both storage and voiding symptoms
- **Ultrasound:** PVR urine volume usually raised
- **Urodynamics:** detrusor overactivity, detrusor-sphincter dyssynergia

Upper motor neuron

Voiding symptoms : Weak stream , Retention , Hesitancy & Dribbling.

Pontine center isn't receiving signals from below.

Sacral/infrasacral lesion
- **History:** predominantly voiding symptoms
- **Ultrasound:** PVR urine volume raised
- **Urodynamics:** hypocontractile or acontractile detrusor



Lower motor neuron

Weak bladder.

History and Physical Examination

- A detailed history should factor in urinary tract symptoms, neurologic symptoms and diagnosis (if known), the clinical course of the neurologic disease, bowel symptoms, sexual function, comorbidities, and use of prescription and other medication and therapies.
- assessment of patient mobility, hand function, cognitive function and social support are also important.
- Other factors to consider are risk and history of urinary tract infections, decubitus ulcers, and other urologic factors that may contribute to LUT dysfunction such as prostate enlargement in men and urethral hypermobility in women.

History and Physical Examination

A general physical examination should include blood pressure measurement, an abdominal examination, an external genitalia examination in males and a vaginal examination if clinically indicated to look for pelvic floor prolapse in women along with a rectal exam to look for fecal loading or alteration in anal tone

. A focused neurological examination is also recommended. This may include assessment of cognitive function, ambulation and mobility, hand function, and lumbar and spinal segment function, including testing sensation and reflexes in the urogenital area.

Investigations

A. Urine Testing
B. Measurement of Renal Function
C. Upper Tract Evaluation
D. Urodynamic Investigations

MANAGEMENT OF NEUROGENIC BLADDER

Management of neurogenic bladder

- **1.Management of Storage Dysfunction:**
  A- Behaveioral and conservative Treatments.
  B- Pharmacotherapy.
  C- Neuromodulation.
  D- Onabotulinumtoxin A
  E- Surgical.

- **2-Management of voiding dysfunction:**
- **A- Medications.**
- **B- Catheterization.**

A- Behavioral and conservative Treatments.

Lifestyle interventions

1. Moderation of fluid intake to 1-1.5 litres per day

2. Avoid alcohol , caffeine

3. Drug regimens avoiding diuretics ,

4. control of chronic cough and constipation ,

5. cessation of smoking .

6. exclusion or treatment of urinary tract infection ,

7. weight reduction is desirable .

Behavioral therapy

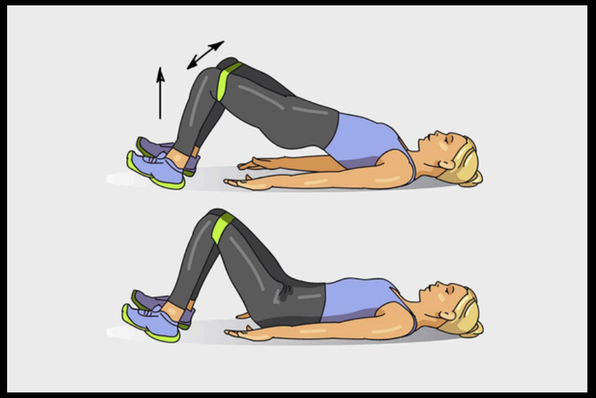
1. Timed voiding

2. Pelvic floor muscle training and exercise (including pelvic floor relaxation) .

3. Delayed voiding .

4. Double voiding .

5. Biofeedback

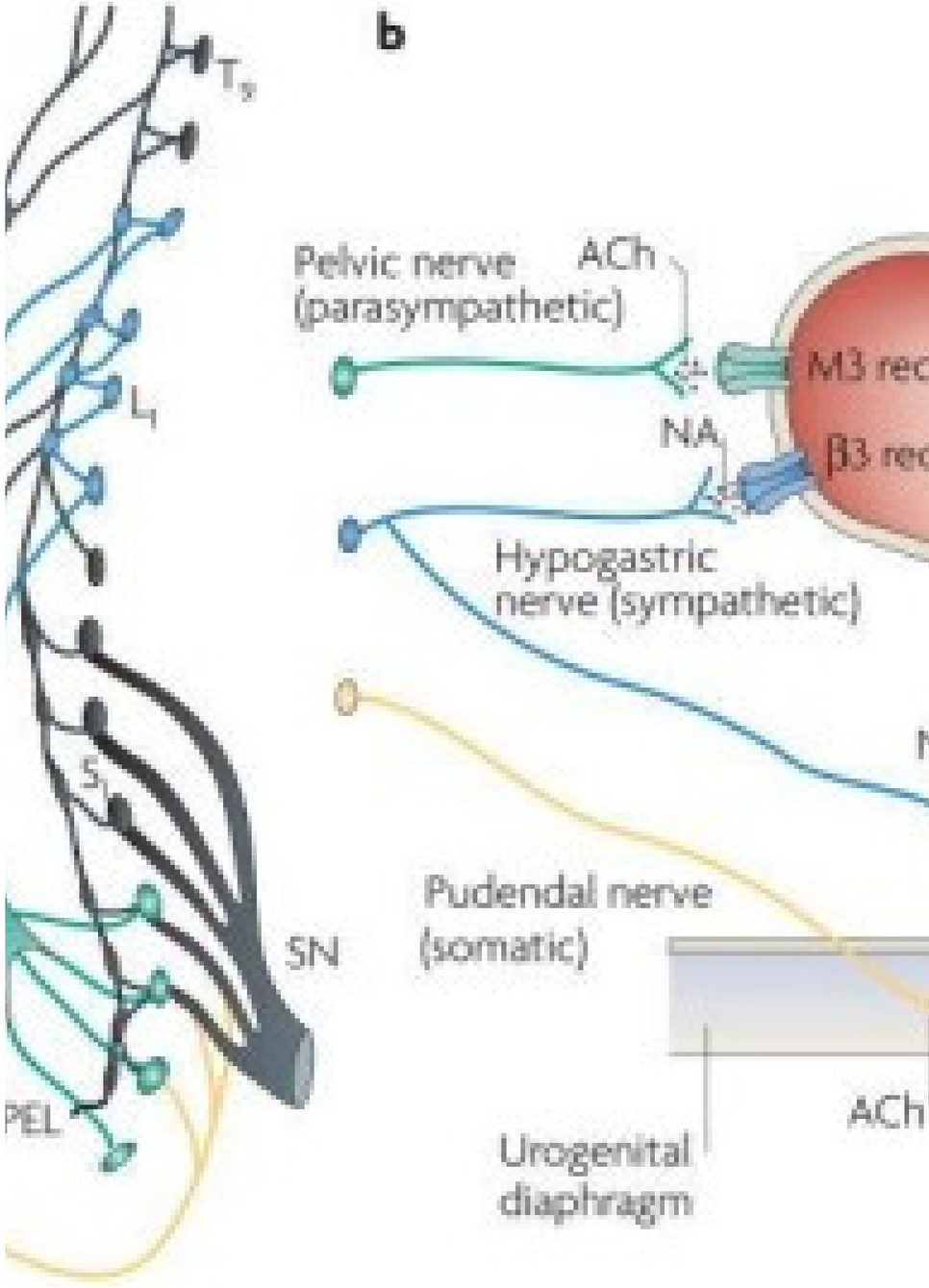


B. Pharmacotherapy
TWO LINE OF MEDICATIONS
Anticholinergic drugs

Anticholinergic medications are the mainstay of pharmacotherapy for individuals wi neurogenic detrusor
Overactivity (level of evidence la) and are considered to be first-line therapy, at times combination with clean intermittent catheterization (CIC).

Anticholinergic medications aim to increase bladder capacity and reduce episodes o urinary incontinence
secondary to neurogenic detrusor Overactivity.

There are two types of muscarinic receptors in the bladder: M2 and M3. M2 recepto are most abundant,
but M5 receptors are functionally more relevant to bladder relaxation.

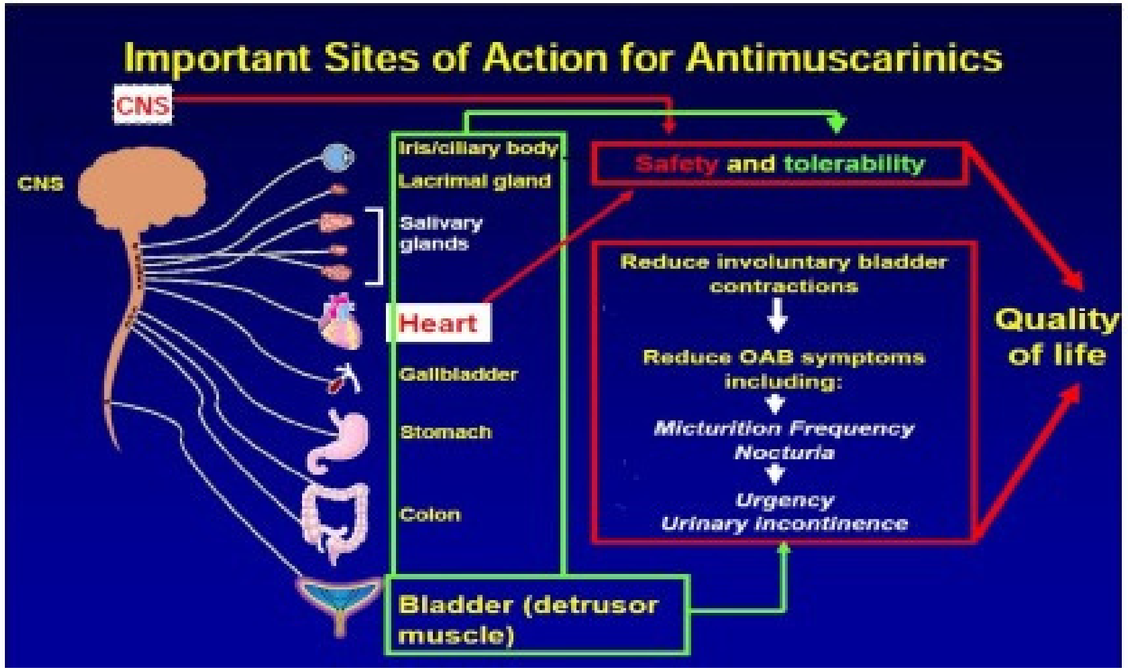


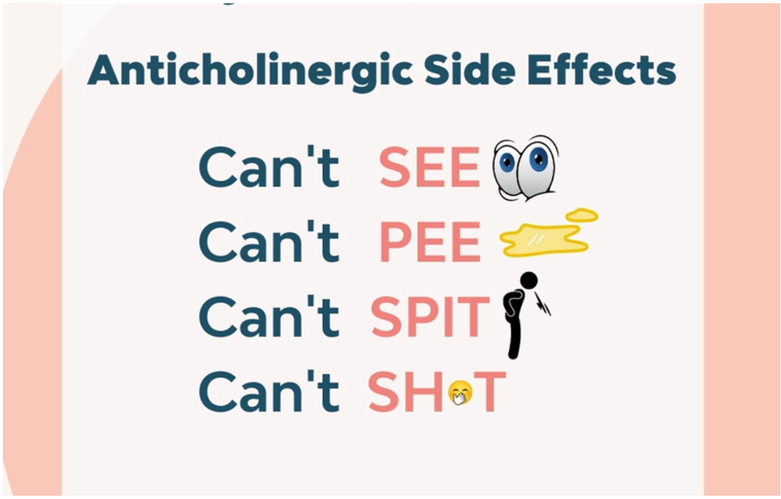
Adverse effects of anticholinergic medications most commonly
include dry mouth, blurred vision, constipation, tachycardia, and confusion, some or all of which may already be present in the
neurogenic patient.

Difficulty emptying the bladder is another potential adverse
event of these medications, which should be considered in any
individual who has an elevated PVR and in individuals with
multiple sclerosis, stroke, or Parkinson's discase.

It is also recommended to monitor PVRS after Starting treatment with an anticholinergic medication and to take into account that these medications can cross the blood-brain barrier, can reduce bladder emptying increasing the risk for urinary tract
infection, and can precipitate or exacerbate constipation.

Oxybutynin (immediate release [IR),extended release [ER]. patch, topical gel),Tolterodine, Solifenacin, Fesoterodine (3r y)
Darifenacin (M3 Selective)
Trospium chloride (4ry) .

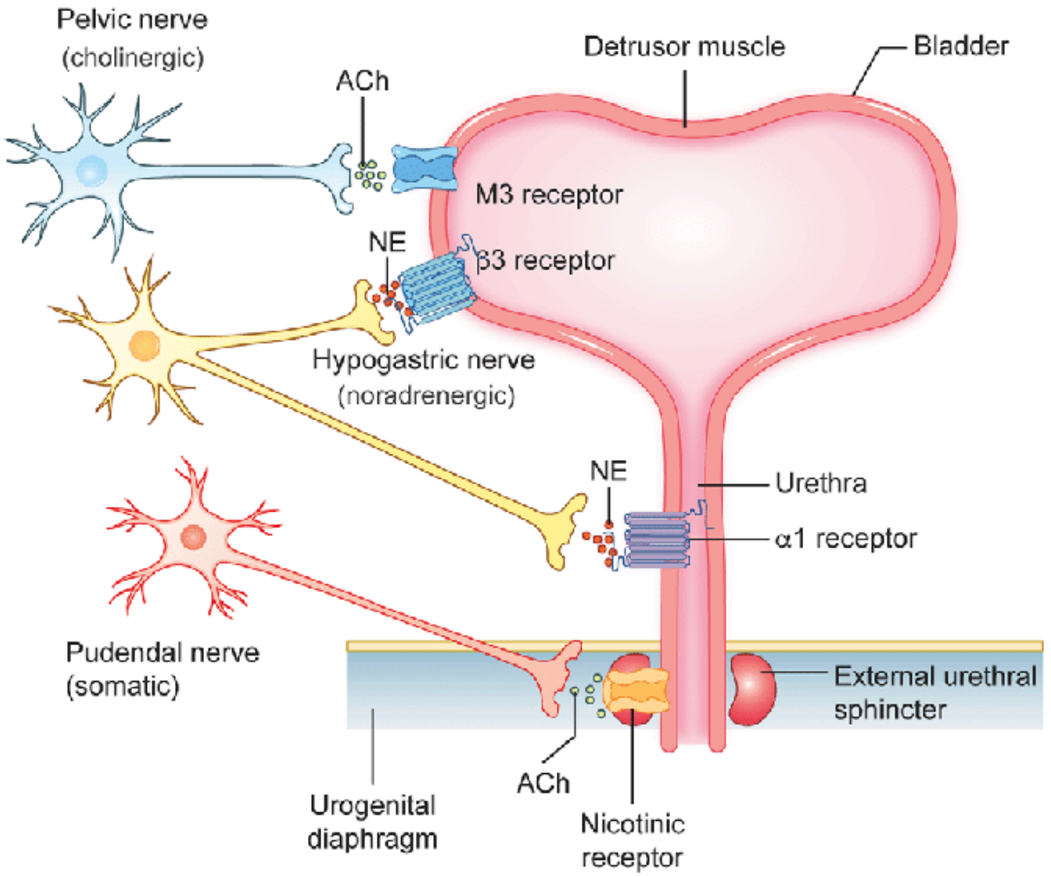




Beta-AR agonists
Over 97% of B-adrenergic receptors in the bladder are of the p3 type, representing the main method of
bladder relaxation in humans.

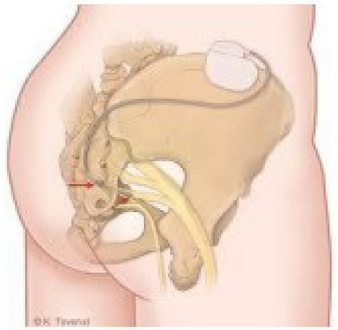
These receptors serve to relax the detrusor muscle, making them an ideal therapeutic target. The selective p3-adrenoceptor agonist mirabegron was approved in by the Food and Drug Association (FDA) in 2012 for
clinical use as an additional medication used to treat overactive bladder symptoms.

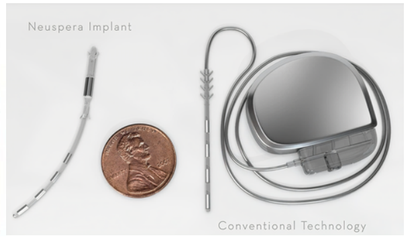
The main side effects of this medication are cardiovascular with a mean rise in blood pressure of up to 2.4 mm Hg and small increases in heart rate .

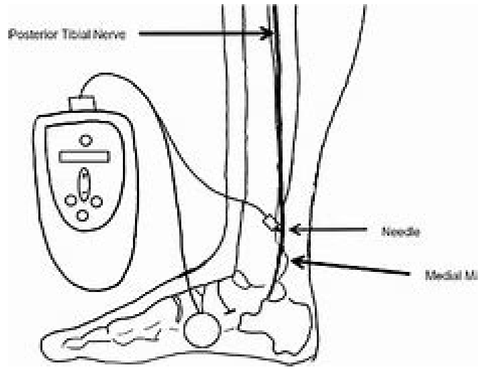


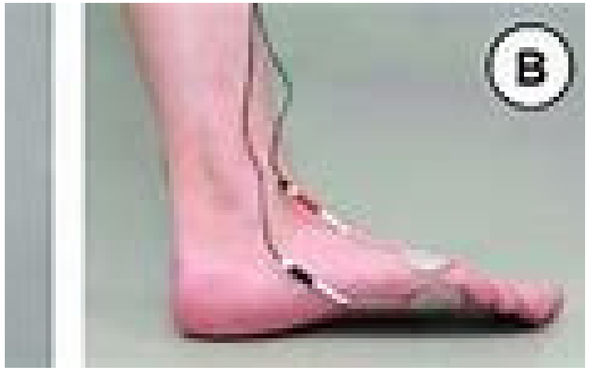
C- Neuromodulation

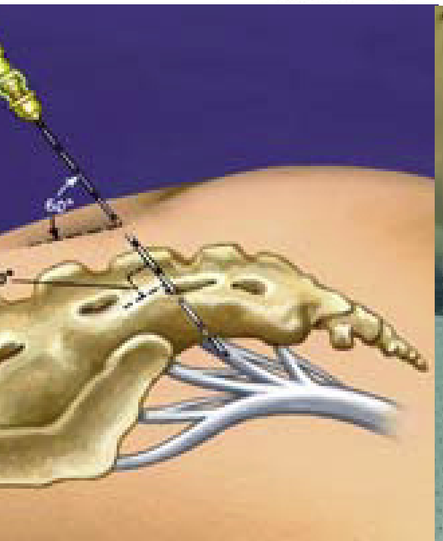
- **Neuromodulation is a well-established third-line treatment for nonneurogenic overactive bladder, but its use in neurogenic bladder is relatively less established. Neuromodulation is currently performed in two varieties:**
  A- sacral neuromodulation (SNM) **B -percutaneous tibial nerve stimulation (PTNS).**

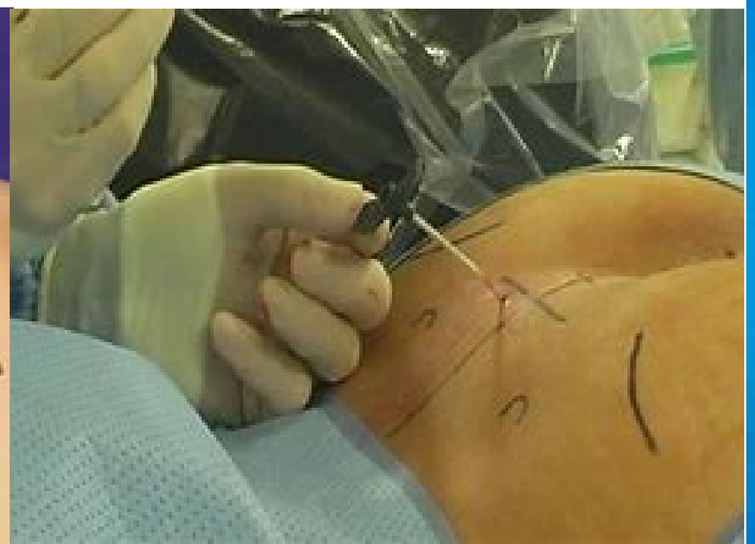








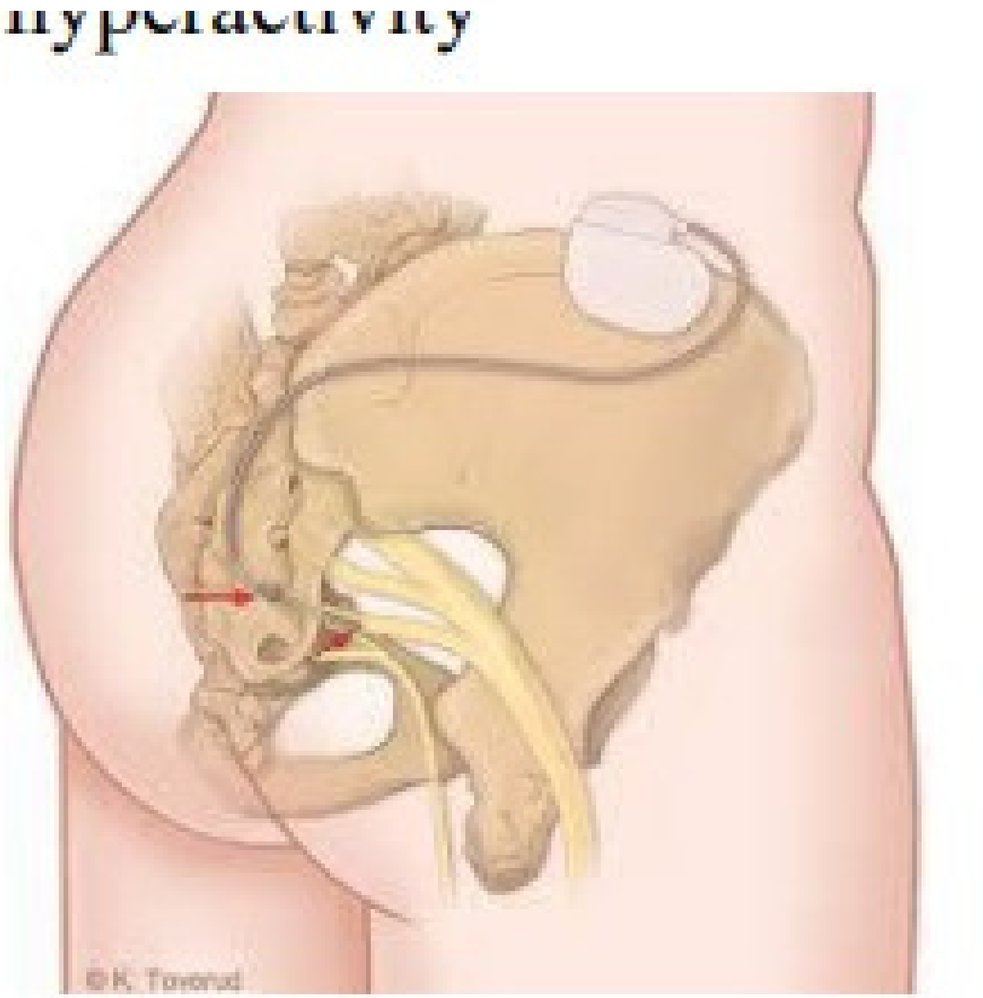




Sacral neuromodulation

Stimulation of the S3 / S4 nerve root by an implanted electrical pulse generator

The stimulator is a small electrical pulse generator , approximately the same size as a cardiac pacemaker , and is commonly implanted in the upper outer quadrant of the buttock .

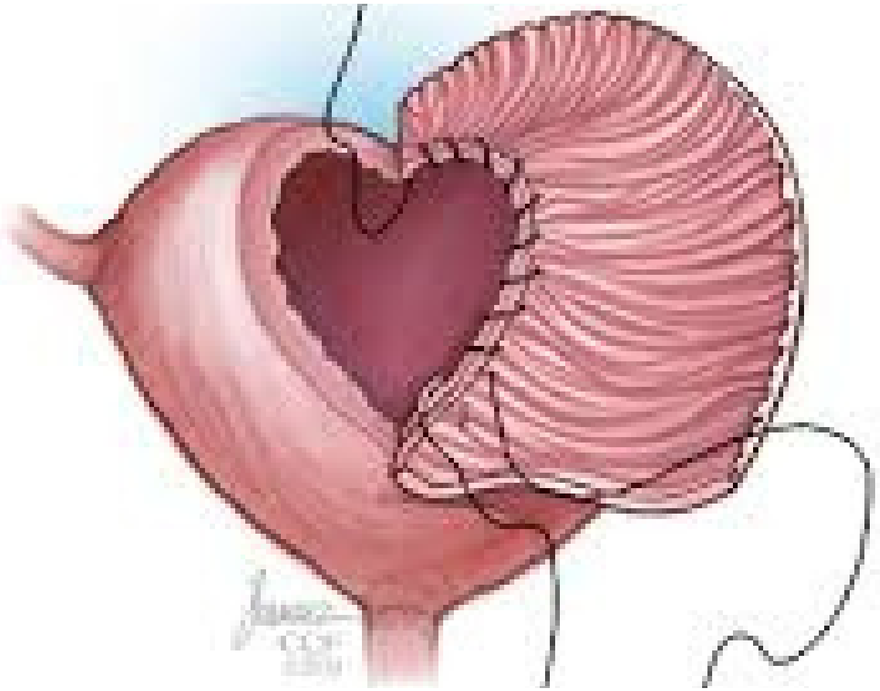


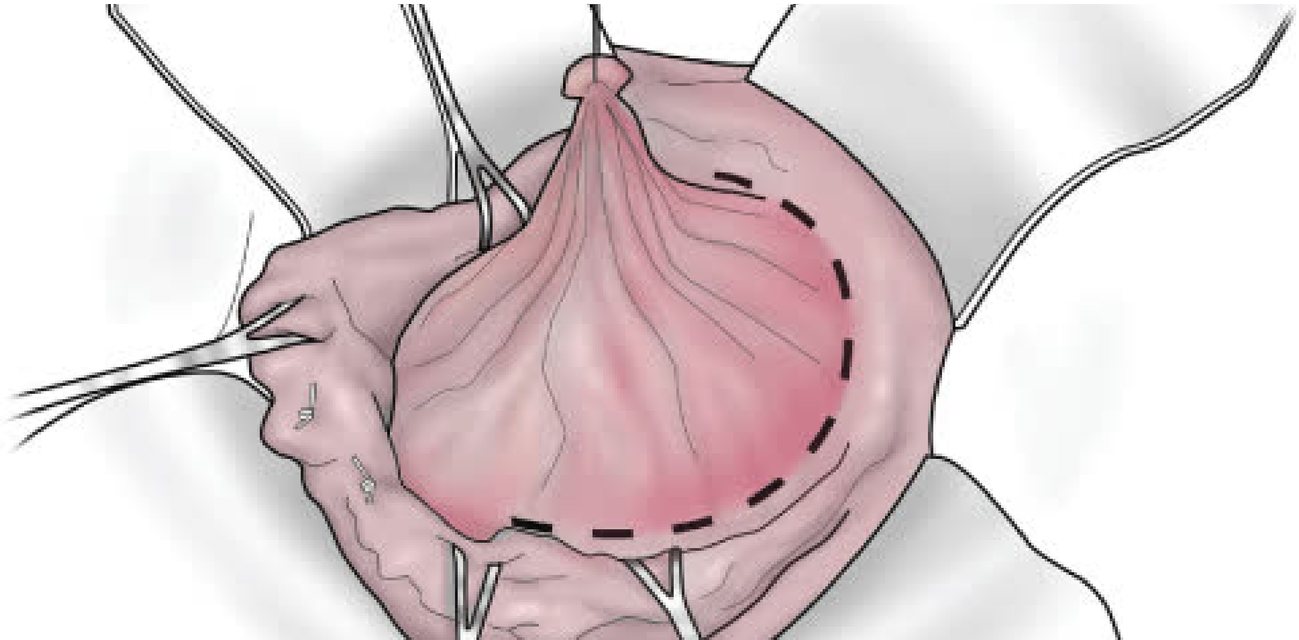
D. Onabotulinumtoxin A

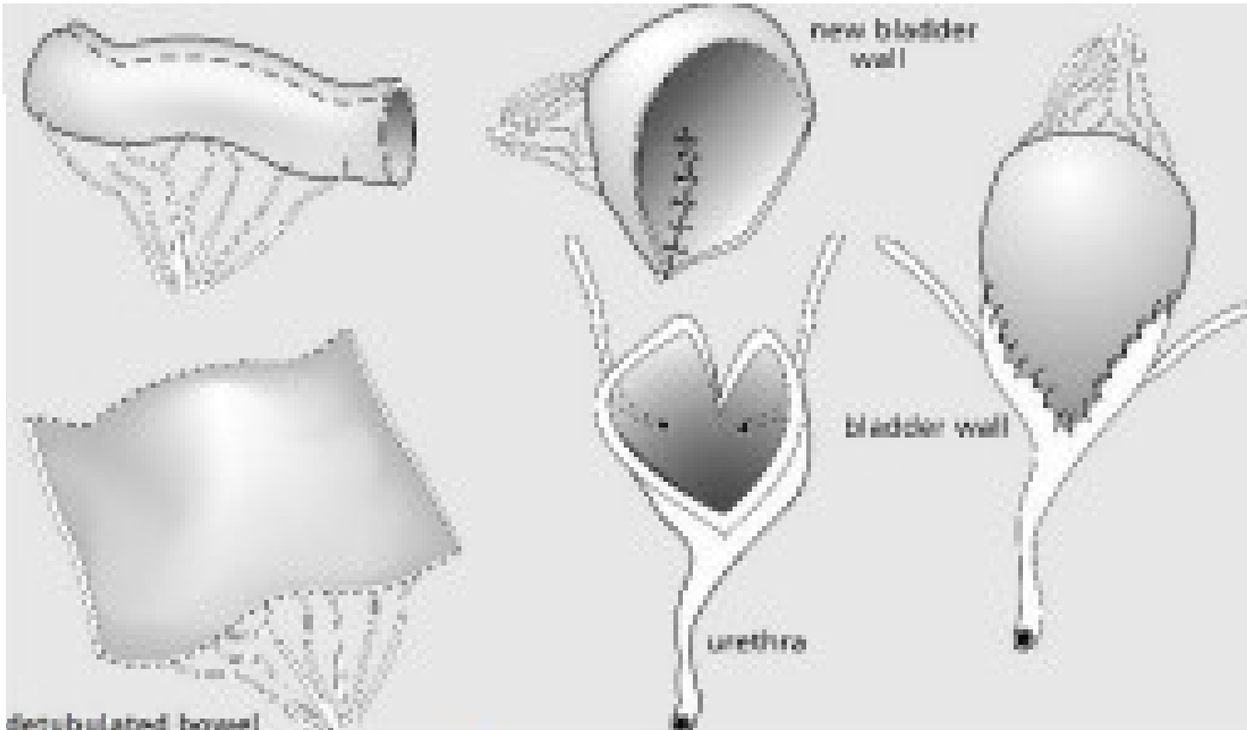
- Onabotulinumtoxin A works by blocking the release of acetylcholine from nerve endings (exocytosis), resulting in the blockage of neural transmission and alteration of afferent sensory input. It is important to consider that these injections may increase the PVR among individuals who void, introducing the need for CIC or other more invasive bladder management strategies.

- E. Surgical :

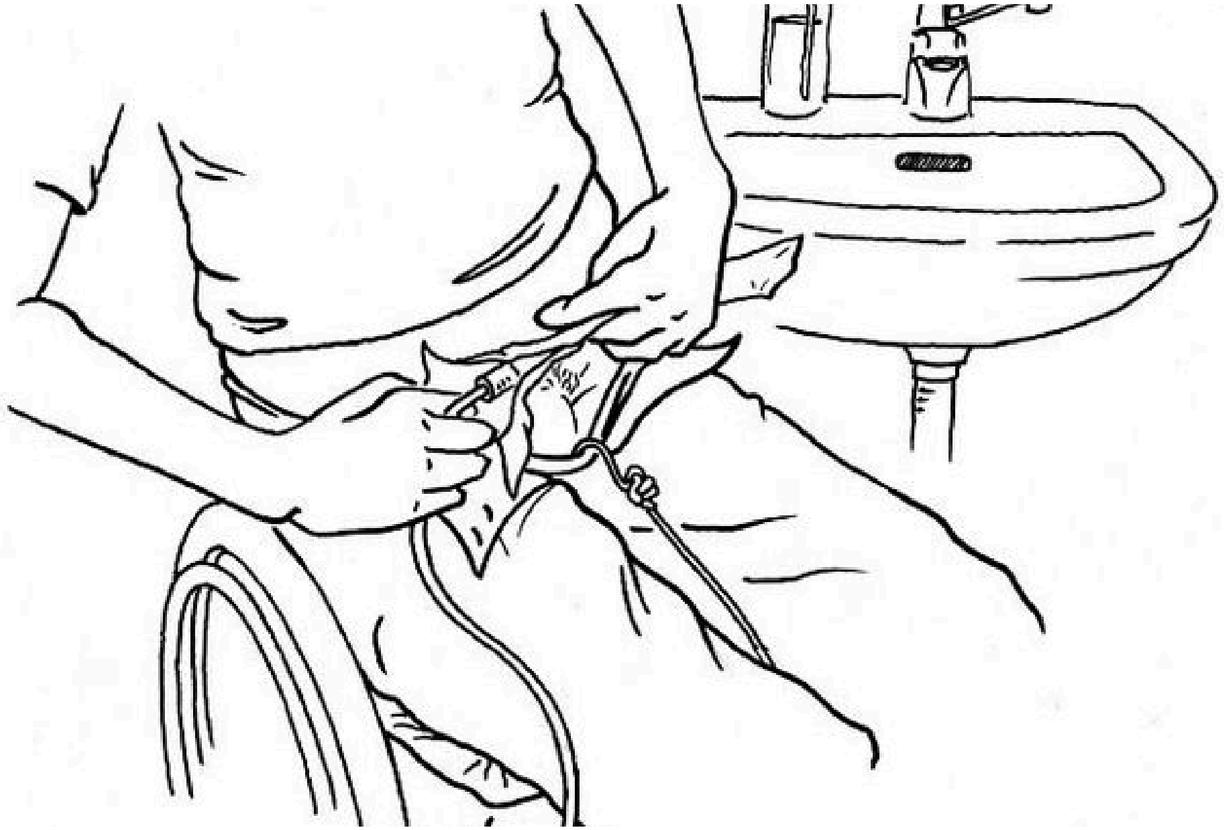
A- Clam augmentation cystoplasty .
B- Detrusor myectomy .
C- Denervation procedures .
D- Urinary diversion

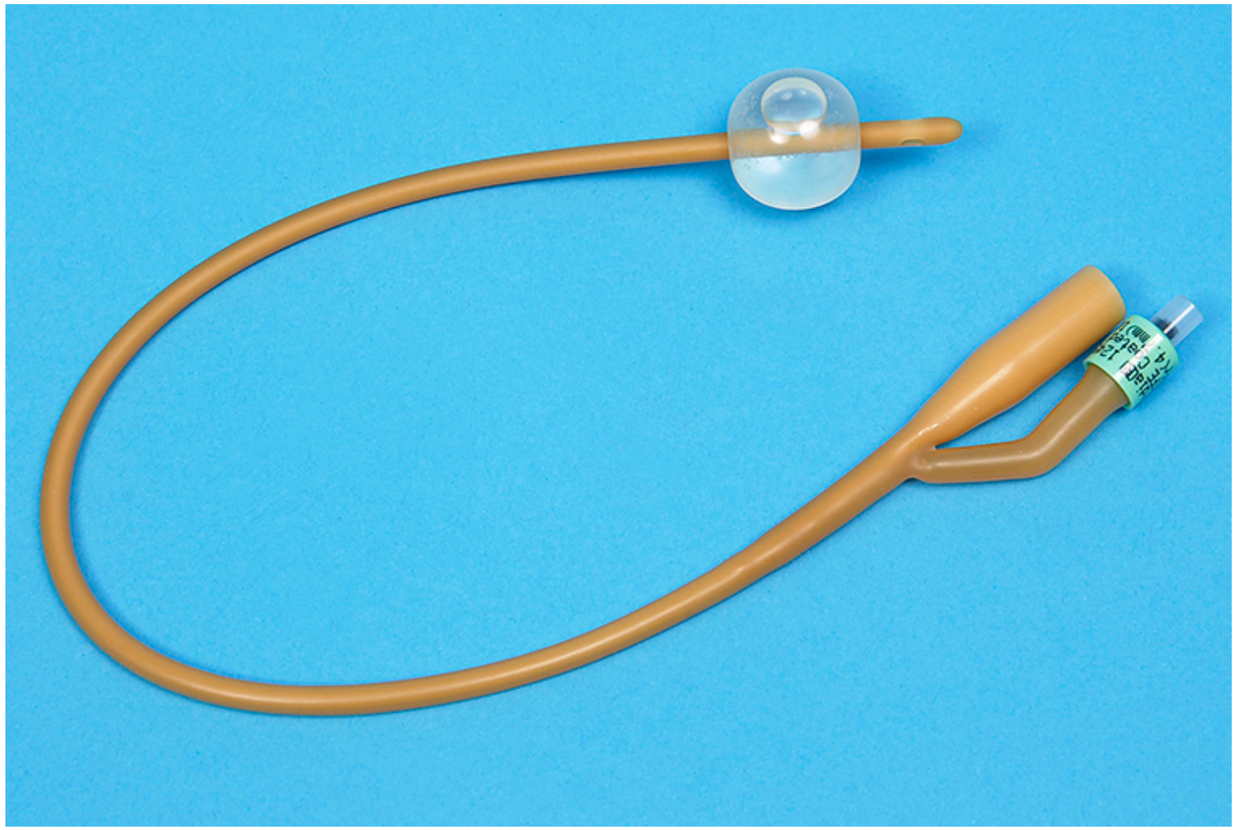






2-management of voiding dysfunction
-Medications
-Catheterization





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