Spinal Cord Injury 3

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Resident Teaching

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Outline

1. Autonomic dysreflexia
2. Neurogenic bowel/bladder
3. Control of spasticity
4. Community integration/rehabilitation (Dr. McGowan)

1. Autonomic Dysreflexia

- An acute syndrome of excessive, uncontrolled sympathetic output that can occur in patients who have had an injury to the spinal cord (generally at or above T6)
- Due to spinal reflex mechanisms remaining intact despite the injury
- Very manageable syndrome, but if not recognized promptly, can lead to death

Pathophysiology

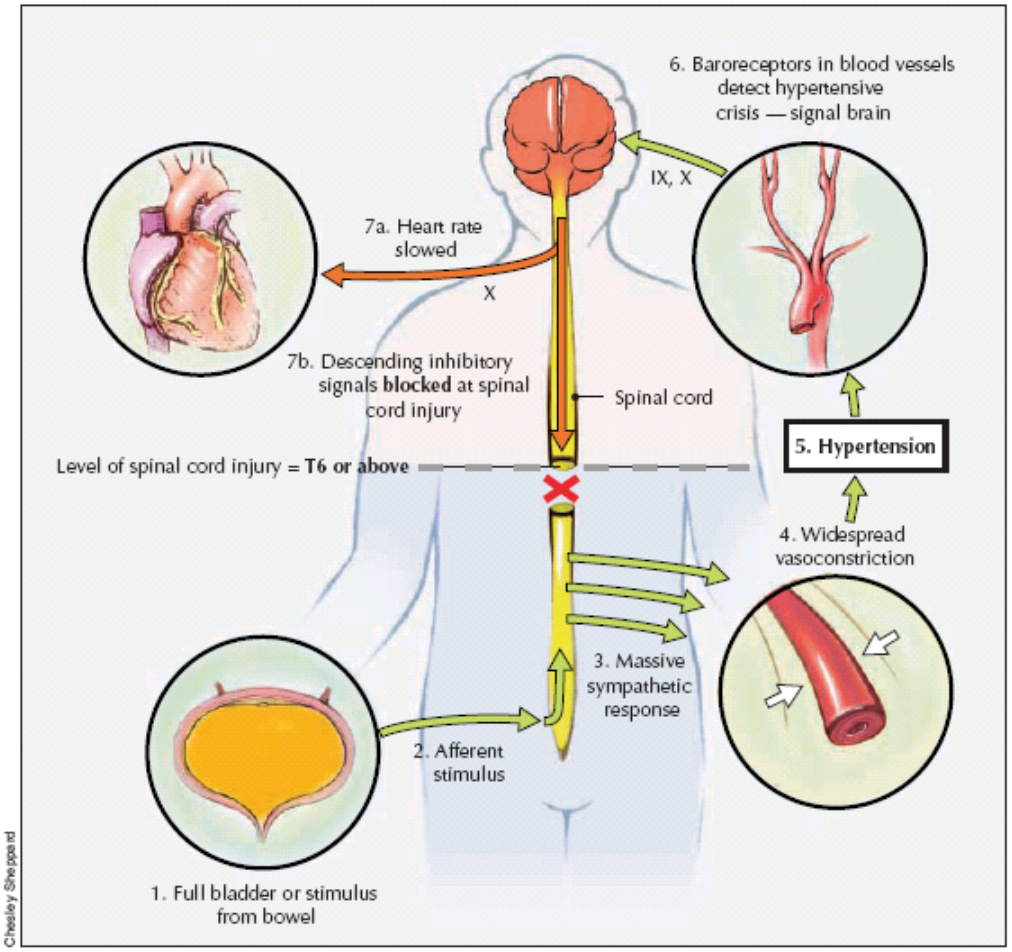


Fig 1: Diagram illustrating how autonomic dysreflexia occurs in a person with spinal cord injury. The afferent stimulus, in this case a distended bladder, triggers a peripheral sympathetic response, which results in vasoconstriction and hypertension. Descending inhibitory signals, which would normally counteract the rise in blood pressure, are blocked at the level of the spinal cord injury. The roman numerals (IX, X) refer to cranial nerves.

Clinical Features

- Symptoms:
  - Bilateral pounding headache
  - Sweating above level of SCI, nasal congestion, malaise, nausea, blurred vision
- Signs:
  - Hypertension (remember normal for SCI ~90/60)
  - Reflex bradycardia (sometimes tachycardia)
  - Flushed, sweating above SCI level
  - Pale, cool skin below SCI level

DDx

- Migraine, cluster headache
- Essential hypertension
- Posterior fossa tumours
- Pheochromocytoma
- Toxemia of pregnancy

Table 1: Precipitants of autonomic dysreflexia

Category	Noxious stimulus*
Bladder	Infection
	Distension
Urinary tract	Urethral distension
	Instrumentation
	Calculus
Gastrointestinal	Distension
	Instrumentation
	Infection or inflammation
	Ulceration
	Reflux
Anorectal	Distension
	Instrumentation
	Hemorrhoids
	Anal fissure
Dermatologic	Pressure sore
	Ingrown toenail
Skeletal	Heterotopic ossification
	Fracture
	Joint dislocation
Reproductive	Labour and delivery
	Menstruation
	Testicular torsion
	Ejaculation
	Intercourse
Hematologic	Deep vein thrombosis
	Pulmonary embolism
Central nervous system	Syringomyelia
Medications	Nasal decongestants
	Sympathomimetics
	Misoprostol

*Any stimulus that might be expected to cause pain or discomfort in a person without spinal cord injury.

Table 2: Treatment of autonomic dysreflexia

Intervention	Rationale
1. Sit the patient upright	May cause an orthostatic decrease in blood pressure
2. Loosen any tight clothing or constrictive devices	Decreases noxious stimuli
3. Monitor the blood pressure every 2 to 5 minutes during the episode	Allows for rapid pharmacologic intervention where indicated
4. If no indwelling catheter is present, perform an intermittent catheterization	Bladder distension is the most common precipitant of autonomic dysreflexia
5. If an indwelling catheter is present, check it for obstructions and irrigate the catheter	Bladder distension is the most common precipitant of autonomic dysreflexia
6. If symptoms are still present and systolic blood pressure is 150 mm Hg or greater, treat the blood pressure pharmacologically	Risk of adverse sequelae increases when the systolic blood pressure exceeds 150 mm Hg
7. If symptoms are still present and systolic blood pressure is less than 150 mm Hg, manually disimpact the bowel	Bowel problems are the second most common precipitant of autonomic dysreflexia
8. If symptoms persist, search for other precipitants (see Table 1)	Other precipitants, such as a pressure sore, ingrown toenail or fracture, must be found and treated appropriately to prevent further episodes
9. Consider admission or referral if symptoms persist or no precipitant is found	Patient is at risk of further episodes if no precipitant is found and should be monitored until the symptoms resolve and the blood pressure returns to normal

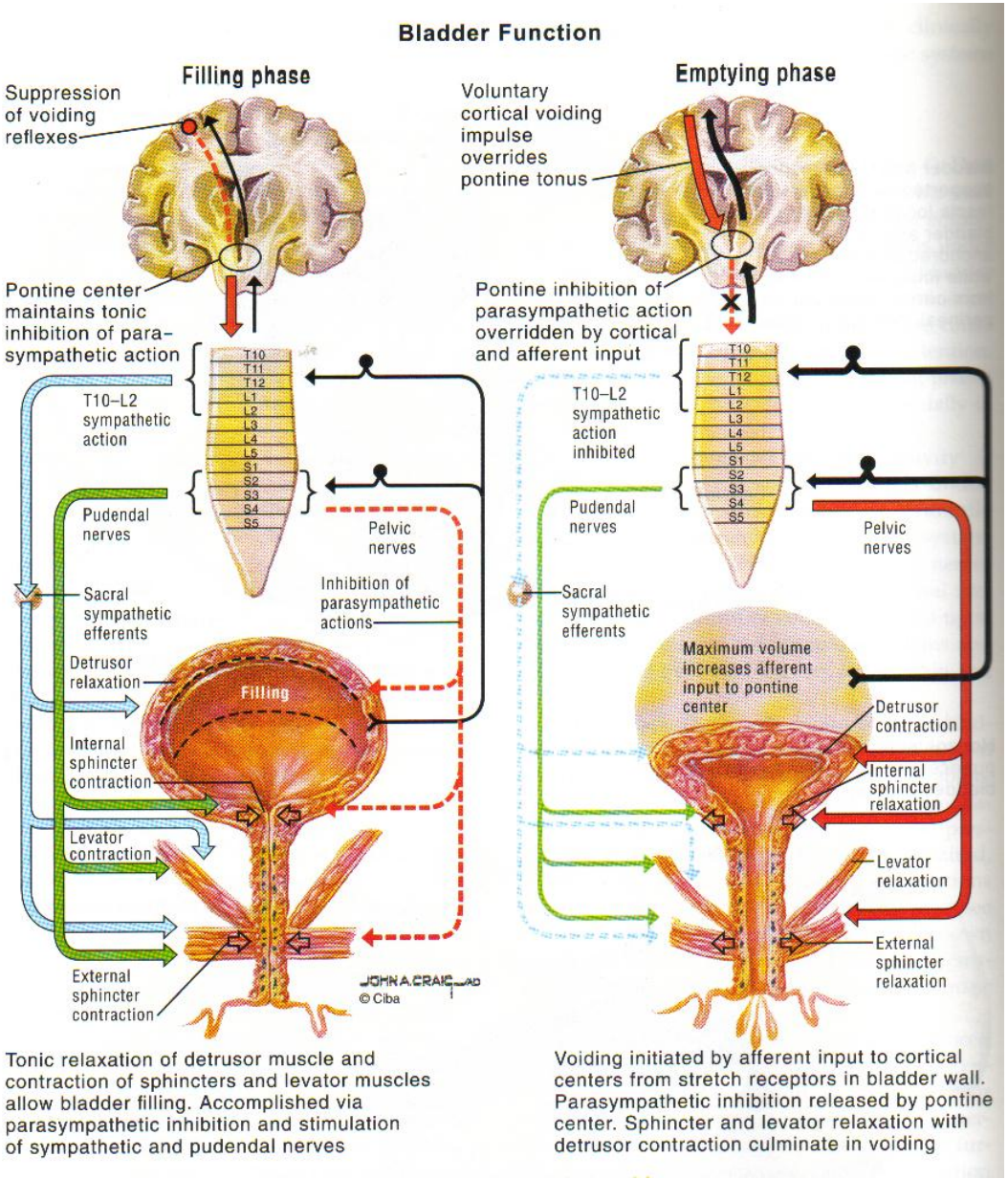
Pharmacologic Options

- Sublingual nifedipine 10mg
- IV phentolamine (anticholinergic)
- Nitroprusside
- Can use prophylaxis for recurrent episodes (eg during bladder cath):
  - Beta blocker
  - Pyridium (urinary anesthetic)
  - Phenoxybenzamine (alpha blocker)

2. Neurogenic Bladder

Normal Urination

Sympathetic



Parasympath.

Urination in SCI

- Initial approx 3 months is spinal shock
  - Decreased autonomic and somatic activity, detrusor areflexia, urinary retention and constipation
  - Internal and external urethral sphincters are normal
- When spinal shock wears off, bladder function returns but detrusor becomes overactive (detrusor hyperreflexia)

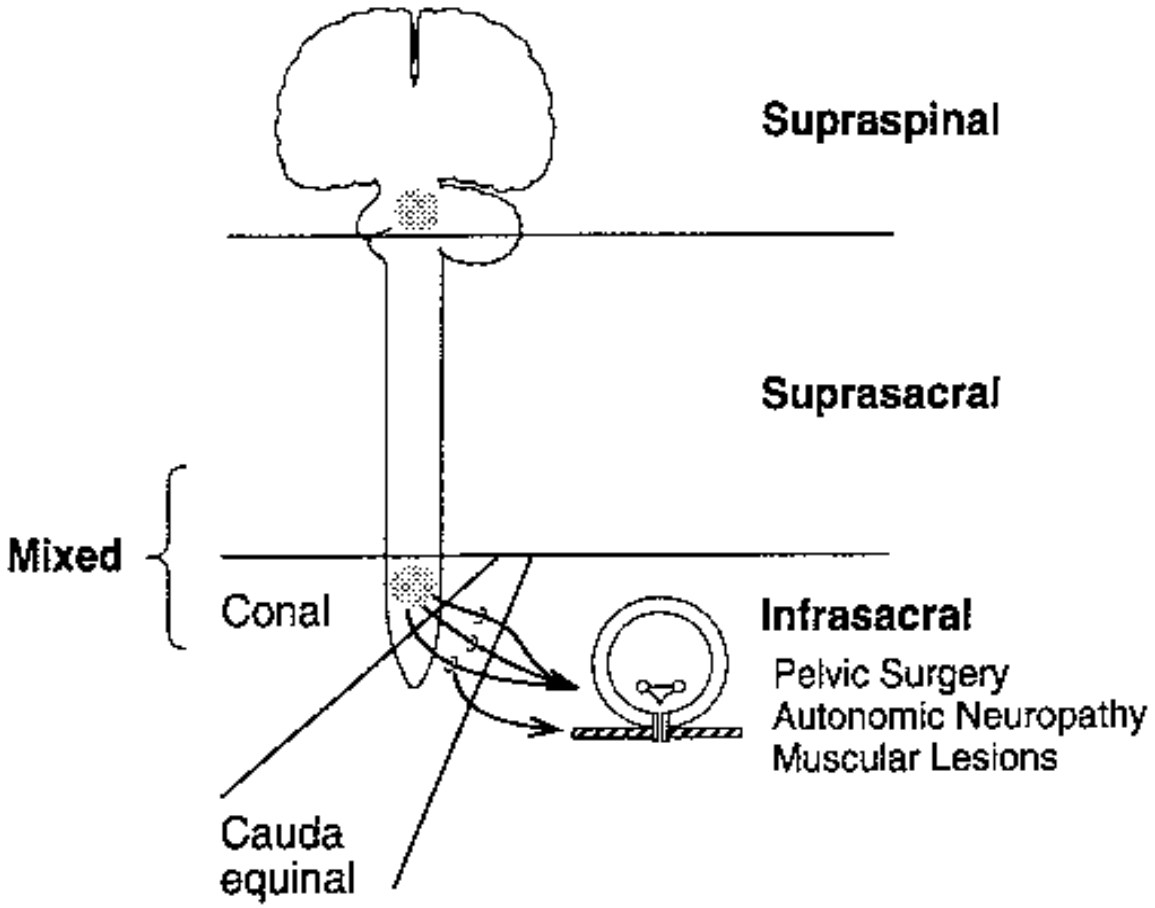


FIGURE 27–4. Anatomical classification of the neurogenic bladder.

Level of Injury

- Supraspinal:
  - Brain injury (CVA, tumour)
  - Loss of centrally mediated inhibition of pontine voiding reflex
  - Involuntary bladder contractions with sphincter synergy, usually with preserved sensation
  - Preserved voluntary sphincter function
  - Normal bladder pressures maintained
  - Voluntary bladder emptying maintained
  - Treat with timed voiding and anticholinergics

Level of Injury (2)

- Suprasacral:
  - Lesions above S2
  - Detrusor hyperreflexia (involuntary bladder contractions without sensations – autonomic bladder)
  - Sphincter dyssynergy causes high bladder pressures
  - Bladder fills and empties spontaneously (or with cutaneous stimulation
  - Treat with intermittent cath and anticholinergic

Level of Injury (3)

- Intrasacral:
  - Lesion below S2, includes conus injury, cauda equina, and peripheral nerve injury
  - Detrusor areflexia
  - Urinary retention leads to overflow incontinence
  - Associated with loss of bulbocavernosus and anal wink reflex and perineal sensory loss

Evaluation of Bladder Function

- Urodynamics
  - Measures intravesicular pressures during retrograde bladder filling
  - Presence or absence of detrusor reflex detected
  - If present, ask pt to suppress urge to void (inability to suppress is detrusor hyperreflexia)
- Sphincter Electromyography (EMG)
  - Voluntary sphincter contraction tests intactness of supraspinal innervation

Treatment

- Goal is to preserve renal function (prevent UTI, stones, ureteral reflux) and optimize urinary continence
- If inadequate emptying or increased bladder pressures, use intermittent cath + anticholiner
- If voluntary bladder emptying maintained with frequency/urge incontinence, anticholinergics and behavioural therapy used

Anticholinergics

- Oxybutynin Cl (Ditropan ®) 2.5 mg po bid to tid (Ditropan XL ®) 10 – 30 mg po od (Uromax ®) 15 mg po od (Oxytrol ® - Oxybutynin patch) 2 patch/week

- Tolterodine L-tartrate (Detrol ®) 4 mg bid, (Detrol LA ® 4-8 mg po od)

Neurogenic Bowel

- Normal bowel function:
- Vagus nerve innervates upper segments of GI tract
- Pelvic splanchnic nerves – parasymp fibers from S2-4 – to descending colon and rectum
- Hypogastric nerve – symp fibers from L1-3 – to lower colon and rectum
- Pudendal nerve – somatic S2-4 – innervates external anal sphincter

Neurogenic Bowel

- Lesion above conus (UMN) causes underactive propulsive peristalsis and overactive segmental peristalsis, leads to colonic slowing, constipation, fecal incontinence
- Treat with scheduled bowel emptying, using enema or suppository

3. Spasticity

Pathophysiology

- Due to UMN lesion causing absence of inhibitory influence on alpha and gamma motor neurons
- Causes uninhibited reflex arc between aMN and Ia afferents from muscle spindles resulting in a hypertonic state of muscles with clonus

Clinical Features

- Increased resistance to passive movement
- Increased reflexes
- May be painful, can affect ability to sit in wheelchair or lay in bed
- Often exacerbated by same triggers for autonomic dysreflexia
- After period of spinal shock, spasticity starts with flexors over 3-6mos, then extensors which ultimately predominate

Benefits of spasticity

- Maintains muscle tone and therefore bulk, therefore helps prevent decubitus ulcers
- Muscle contractions may help prevent DVTs
- May be useful in bracing

Grading Spasticity – Ashworth Scale

1. Normal
2. A “catch” when affected part flexed/extended
3. Passive movements easy
4. Passive movements difficult
5. Affected part rigid in flexion/extension

Treating Spasticity

- Decrease triggers – ie prevent
- Prolonged stretching
- Oral meds
  - Potentiate GABA, increase pre-synaptic inhibition of aMN
    - Valium (start 2mg TID, up to 20mg TID)
    - Baclofen (start 5mg TID, up to 20mg QID)
  - Ca channel blocker, decrease muscle depolarization
    - Dantrolene (start 25mg daily, up to 100mg QID)

Treating Spasticity (2)

- Botox
- Surgery
  - Intrathecal baclofen to ablative procedures to cordectomy

References

1. Greenberg, M.S. Handbook of Neurosurgery, 6th Ed. 2006
2. Blackmer, J. Rehabilitation Medicine: 1. Autonomic Dysreflexia. CMAJ • OCT. 28, 2003; 169 (9)
3. Lansang, R. Neurogenic Bowel. eMedicine May 7, 2008