Neurology Update: The Sequelae of Spinal Cord Injury

CPD Program for Primary Care 2017-2018 March 2/18

Dr. Karen Ethans, MD, FRCPC (PM&R), Associate Professor
University of Manitoba

- Disclosures: In the last 2 years:
- Allergan -Received honorarium for invited talk given
- Ipsen and Merz- Received honorarium for attendance on advisory board
- Ipsen – Received research funds for involvement in clinical trial

Objectives

At the conclusion of this program, the attendee will:

- Be familiar with the American Spinal Injury Association classification scale, what is meant by "complete spinal cord injury"
- Recognize the signs and symptoms of Autonomic Dysreflexia as a medical emergency and formulate an initial treatment plan
- Be familiar with, and initiate workup and treatment of, some of the chronic medical sequelae of SCI including heterotopic ossification, osteoporosis, orthostasis, syringomyelia, neuropathic pain and spasticity
- Troubleshoot with a patient on initial management of changes in neurogenic bladder and bowel routines

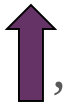
Spinal Cord Injury Classification American Spinal Injury Association

- Last normal neurologic level =level, NOT radiologic
  - To and including triceps intact, sensation to middle finger=C7
- Tetraplegia – upper limbs affected ie C8 and above.
- Paraplegia – upper limbs not affected (T1 and below)
- A-E
  - A= "complete" – no sensory or motor to and include anal region (S4-5)
  - B= "sensory incomplete" – sensory to include (S4-5)
  - C="'"motor incomplete" – motor function present but most weaker than grade 3 (so non-functional)
  - D= "motor incomplete" – motor function with at least grade 3 or more in most muscles below injury
  - E= recovery motor and sensory function

Spinal Cord Syndromes

- Anterior spinal cord syndrome
  - With disruption of blood supply from anterior spinal artery
  - Anterior 2/3 of cord infarct – only dorsal tracts preserved (LT, prop, vibration). Severe paralysis, poor outcome
- Central Cord Syndrome
  - Arms more affected than legs. Corticospinal tracts to arms run in humunculus more medial than legs. Self care very difficult
- Brown Sequard syndrome
  - Hemisection of cord, loss of motor function/proprioception one side, loss of temperature opposite. Neuropathic pain common. Motor recovery reasonable
- Cauda equina syndrome
  - Injuries/disc herniations L1 or distal
  - Areflexic and asensate bladder and bowel, erectile dysfunction +/- LE weakness

Autonomic dysreflexia

- A medical emergency!
- Occurs in those with T6 and above
- Noxious stimulus
- Activates spinal sympathetics
- No descending inhibition
- Spinal sympathethics reflex to efferent sympathetics
- BP  , risk of stroke, intracranial hemorrhage, seizure

AD Etiology

- Three commonest:
- Bladder
- Bowel
- Cutaneous
- Other: Musculoskeletal, abdominal

Clinical Presentation

- Symptoms: **Pounding headache, hyperhydrosis, flushing/rash above,** nasal congestion, blurred vision, palpitations, increased spasticity
- Signs: Paroxysmal **hypertension***, bradycardia/tachycardia, arrhythmias, miosis, piloerection
- *Remember resting blood pressure often 90/50 in tetraplegia

Management

- **Prevention**
  - avoid noxious stimuli
  - proper, regular bowel and bladder care
  - skin care, pressure relief, nail care
- **Education**
  - *recognition*
  - treatment
  - need to educate own health care givers, carry wallet card, Medic alert

Acute Treatment

- 1) If suspect AD, check BP and HR/-monitor q 5 mins
- 2) Sit patient up
- 3) Loosen constrictive clothing/leg bags
- 4) Check urinary drainage
  - Foley - ?kinks, gentle irrigation, replace if needed
  - No Foley - cath

PVA.org – Consortium for SCI Medicine. Clinical practice guidelines

Acute Treatment con’t

- 5) If AD still present, sys BP>150, consider Rx (normal BP for tetraplegic is often 90-100 systolic)
- 6) Check for fecal impaction
- 7) If still not successful treatment need workup eg abdominal path, UTI, HO, skin and nail pathology, fractures, DVT

Autonomic dysreflexia treatment

- ***Acute AD Drug Treatment: First Line Therapy***
  - Ca2+ channel blockers –
    - Nifidepine - 10 mg s/l (bite/swallow or pierce with pin) q 30 mins to abort attack
  - ACE inhibitor
    - Captopril – 12.5-25 mg s/l or po (25mg equiv Nifedipine 10 mg)
    - onset 5-10 mins s/l, 15-30 mins po
    - Direct arterial dilators –
      - Nitrates –s/l 0.3 mg or transdermal, remove if hypotensive, s/e headache
      - IV in severe AD
- ***Acute AD Drug Treatment: Second Line Therapy***
  - Hydralazine 10-20 mg IV
  - Nitroprusside 0.5-1.5 ug/kg/min
  - **α-1 blockers** - only adrenergic discharge blocked
    - *phentolamine* 2-10 mg - in acute, often not effective

AD Prophylaxis

- Topical anaesthetic before catheterizing or bowel care (Lidocaine gel)
- Local anaesthesia prior to diagnostic procedures eg: cystoscopy, gastroscopy
- Premedicate before sexual activity (NOT IF USING PDE5 INHIBITORS!!!), electroejaculation, or other unavoidable triggers
- *Nifedipine 10 mg po 30 minutes prior*

Recurrent/frequent AD

- Alpha-1 antagonist - terazosin/ prazosin, etc - used with minor symptoms
- Alpha-2 agonist (clonidine) - prevents recurrent episodes - but lose effectiveness over time due to down regulation alpha-2 receptors

Orthostatic hypotension

- Most severe in post acute period
- Lesions T5 and above especially affected
- Chronically BP low - 90/50 typical in tetraplegia
- ?treatment

Treatment of hypotension

- ***None if asymptomatic***
- Pressure stockings (at least 20-30 mm Hg) NOT TEDS
- Up from lying position slowly
- Abdominal binder
- Fludrocortisone (Florinef) 0.1 mg OD-qid (not for renal failure) or midodrine 5-10 mg bid-tid

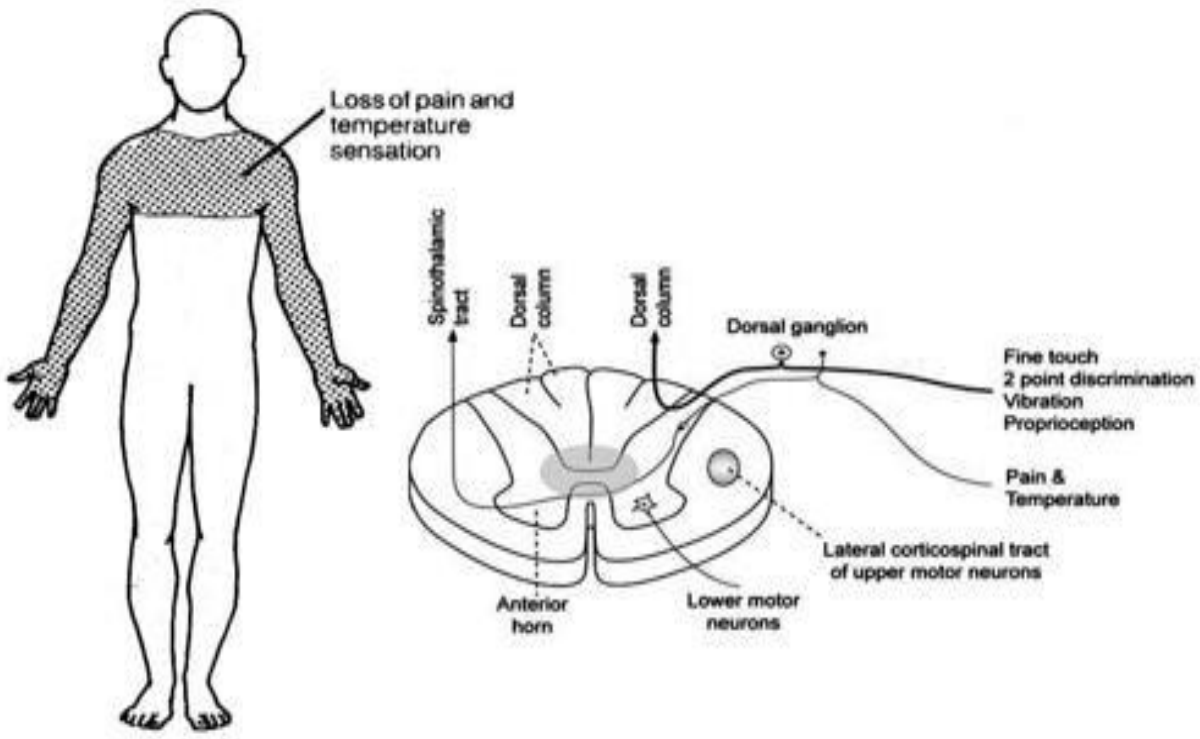
Dependent edema

- Decreased sympathetic outflow thus decreased vascular tone
- Loss of active muscle contractions thus venous pooling and less venous return to heart
- Rule out cellulitis, DVT, fracture
- Treatment
  - Elevate legs (watch out increased U/O)
  - Compression stockings (NOT anti-embolism stockings)
  - UNLESS there is other indication of total body fluid overload, CHF etc, do NOT recommend diuretics

Syringomyelia - “Syrinx”

- “Cyst” in spinal cord - expansion of central canal, cause unknown
- rising lesion, impairment level rising, function decreasing
- increased loss power, pain and temp sensation hands – why??
- Dx - MRI, clinical
- Tx - Avoid valsalva,

drain neurosurgical shunt



Heterotopic Ossification

- Laying down of bony matrix in soft tissues around a joint
- unknown cause
- forms mature lamellar bone, calcifies, causes ***progressive loss of ROM***
- below injury, Hips> knees- affects seating, transferring, dressing
- incidence from 10% – 53%, develop between 1 – 6 months post SCI (peak 2 months)
- Diagnosis
  - clinical - heat, swelling, erythema, eventual decrease ROM
  - x-rays*, bone scan, alkaline phosphatase* +/- CT/MRI

Treatment HO

- Tx:
  - ROM - not aggressive
  - bisphosphonates - non cyclic etidronate
  - NSAIDS Probably only in acute phase?
  - Radiation – either in inflammatory phase or peri-operative to prevent recurrence after surgery
  - Surgery

Cardiovascular disease

- Mortality rate from CV disease is 228% HIGHER in SCI
- 46% of SCI patients 30 years of age and older die of cardiovascular disease
- There is a 60-90% increased risk of heart attack in sedentary SCI patients when compared to sedentary able bodied controls
- In active men and women with SCI the % body fat ranges from 16-24 and 24-32 respectively, compared with 15 and 23% in able bodied controls

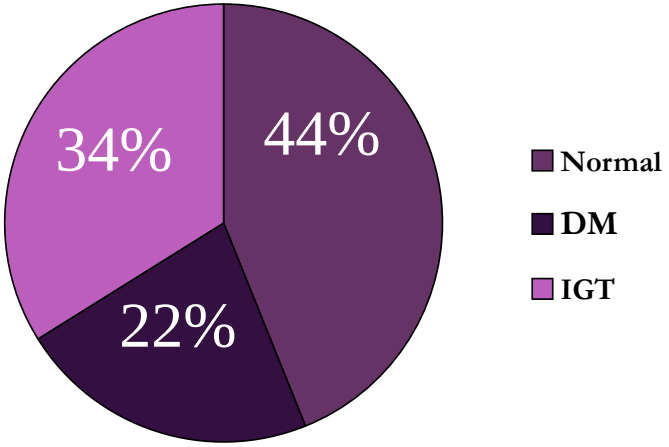
Cardiovascular Risk Factors

- Hypertension – not really issue….
- Diabetes
- Hypercholesterolemia (or dyslipidemia)
- Obesity
- Inactivity
- Smoking

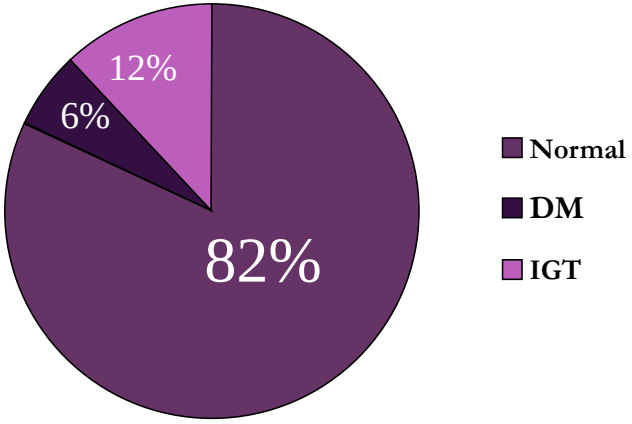
Diabetes

- 22% of paraplegics have Diabetes, compared to 11% of the general population, Imai et al. 1994

- Below, data from Bauman and Spungen, 1994



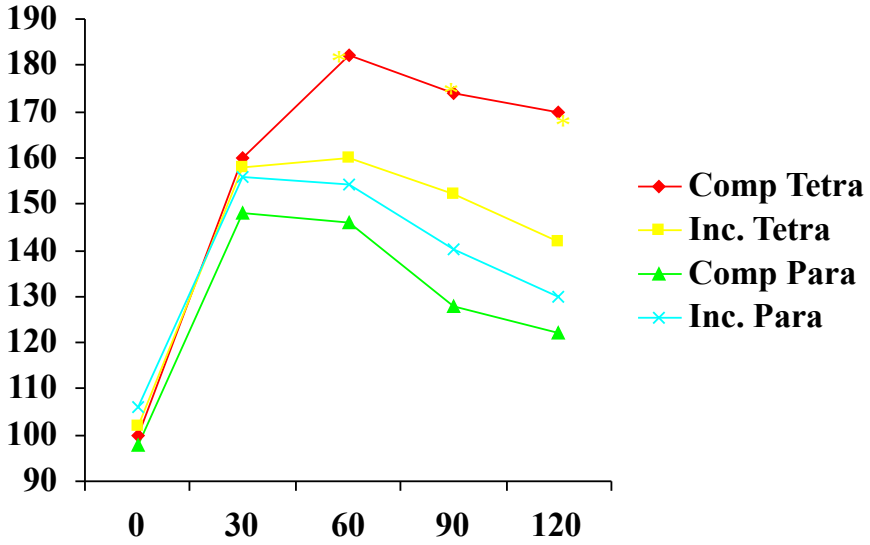
SCI population



Control

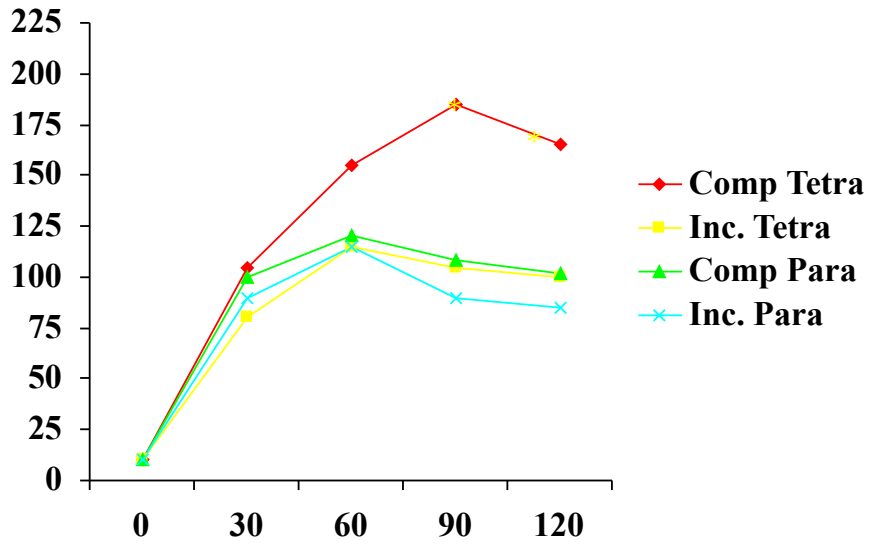
Diabetes – Glucose Intolerance

Bauman and Spungen, 1994



Plasma Glucose (mg/ml) vs Time (min)

*P<0.05



Plasma insulin (mcU/ml) vs Time (min)

*P<0.05

Lipids/dyslipidemia

- HDL-cholesterol is cardioprotective
- 10% of Americans have an HDL < 35 mg/dL compared to 24-40% of SCI patients
- Lower levels of HDL are found in tetraplegics vs paraplegics
- Complete injuries have lower HDL compared to incompletes at similar levels
- Aerobic exercise may not be possible
- *Exercise and cardiopulmonary fitness* increases HDL in both the able-bodied and SCI population

Immobility

- Unable to get aerobic exercise, or if just using upper limbs often overuse
- Need to find mode of aerobic exercise to perform without overuse upper extremities

Osteoporosis

- "a systemic disease characterized by low bone mass, and microarchitectural deterioration of bone tissue with *resultant increase in fragility and risk of fracture*"

Pathophysiology

- Osteoporosis is a well known complication in SCI, and occurs to a severe degree in these patients- neurological damage due to SCI is culprit, and not immobilization alone

- Seen on x-ray as early as 6 weeks after SCI
- Lumbar preserved (100-112%)
- Hips 28%, distal femur 37-43%, prox tibia 36-50% Normal
- Upper limb often normal or better, don't accept radius imaging only!
- *Manitoba issue –BMD testing inaccessible, no lift

Neuropathic pain – At or below level

- Narcotics often not helpful except ? tramadol?
- NSAIDS of little use
- antiepileptics (e.g. gabapentin* 900-4600 mg/day, pregabalin** 75-150mg bid, lamotrigine* in incomplete)
- TCA's (e.g. amitryptiline* 10-50 mg qhs)
- THC/Cannabinoids
- relaxation therapy
- TENS, acupuncture,
- Treat noxious stimulus

- *off label
- **Has indication but NOT COVERED by pharmacare!!!

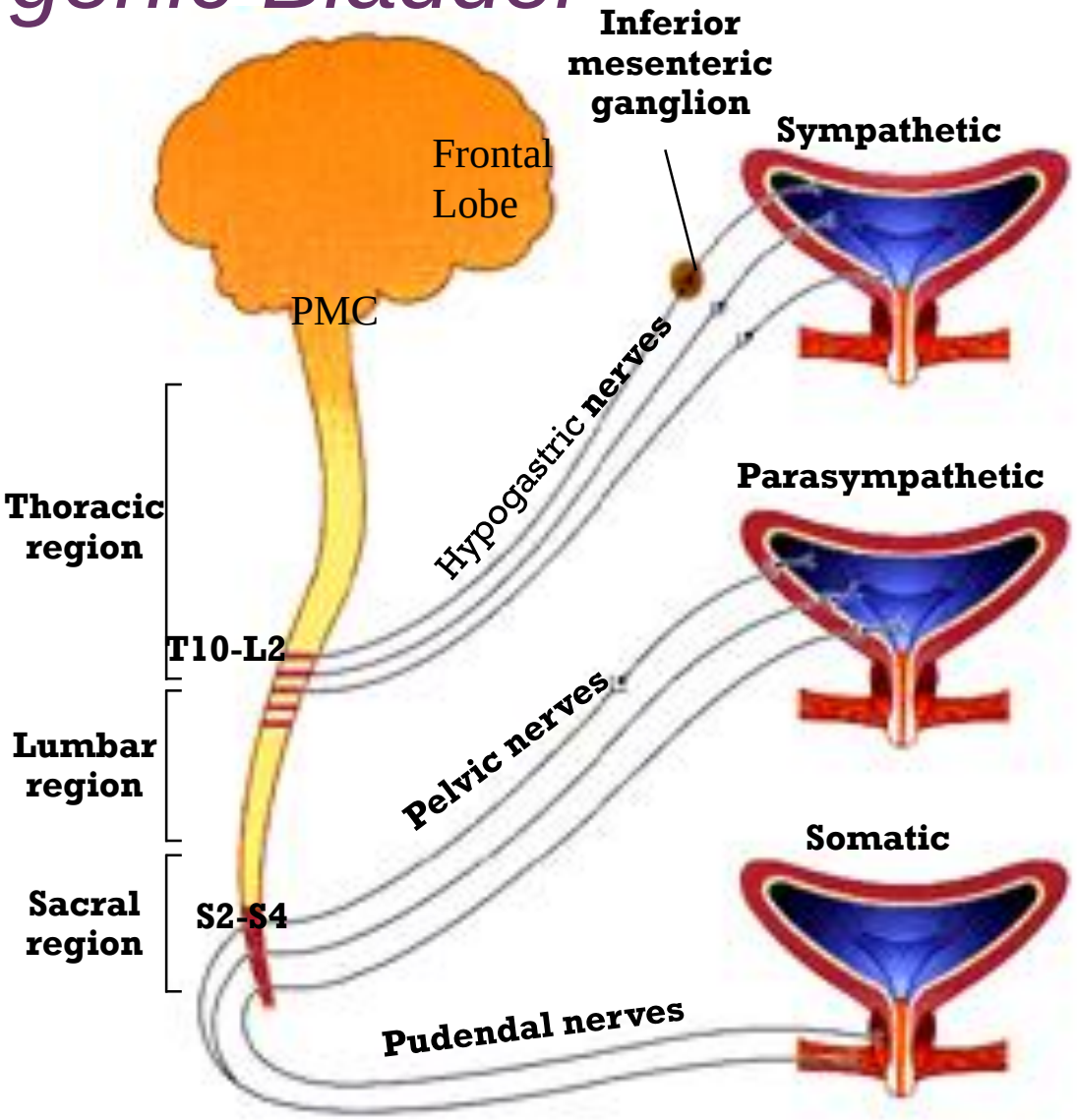
Spasticity

- Part of the UMN syndrome
- Velocity dependent increase in tone
- Shaking (clonus), cramping, tight, violent spasms
- Treatment
  - Nothing
  - Stretches
  - Systemic – (baclofen, tizanidine, benzodiazepines, cannabinoids)
  - Focal – if focal area a problem –(eg adductors hips) – botulinum toxin
- If sudden increase spasticity, look for something else (UTI, fracture, fecal impaction….

Musculoskeletal pain

- Overuse injuries upper extremities exceedingly common
  - Shoulders – wheeling, weightbearing
  - Extensors of forearm (tennis elbow)
  - Carpal tunnel – wheeling/transferring
- Pain of upper back/neck muscles
  - Poor posture
  - Over development shoulder protractors, over stretch and weak retractors
  - Contributes to shoulder problems
- Treatment: Improve biomechanics, conserve shoulders/transfers, wheeling gloves, treat myofascial pain

Neurogenic Bladder

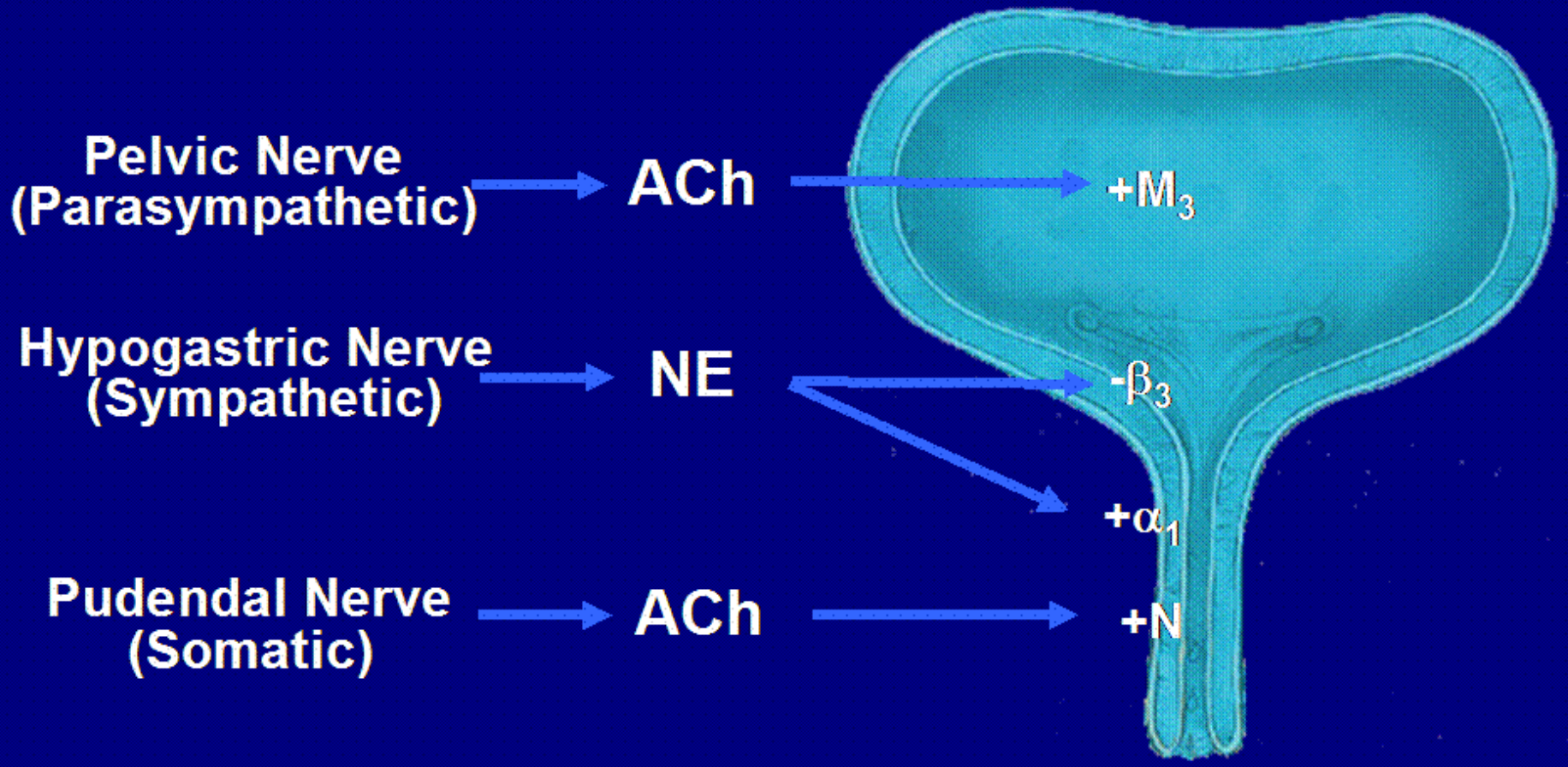


DESD

- GOALS

1) Prevent kidney damage- maintain low pressures and regular bladder emptying
2) Prevent incontinence
3) Prevent recurrent UTI's/stones with regular bladder emptying

LUT Peripheral Motor Innervation



DESD Management

- 1) Enhance bladder volumes
  - R/O UTI, stones
    - Relax hyperreflexic/spastic detrusor
      - Anticholinergics – oxybutinin, tolterodine, solifenacin
      - B3 agonists - mirabegron
      - Botulinum toxin
      - Augmentation cystoplasty
    - intermittent catheterization needed to empty regularly
- 2) Enhance emptying
      - Intermittent Cath
      - Condom drainage plus sphincterotomy (higher quads who cannot cath)
      - Alpha blockers (for incomplete voiders)/avoid anticholinergics
      - Indwelling foley – needs regular changing –AVOID clogging – AD!!

Antimuscarinic Side Effects

- Mad as a hatter: CNS, delirium

- Red as a beet: Direct vasodilation
- Blind as a bat: Cycloplegia

- Hot as a hare: Thermoregulation
- Dry as a bone: ↓ Sweat & secretions

Neurogenic Bowel

- Upper Motor Neuron
  - Lesion above conus medularis
  - Reflexic propulsion intact, spastic
  - Transit Times higher
    - mean = **80.7 +/- 11 hrs**
      - [controls: 39.1 +/- 5 hrs]
      - Slowest distally.
  - Inability to initiate BM as the external sphincter not under voluntary control, spastic
  - Often sphincter-rectal co-contraction

Neurogenic Bowel

- Lower Motor Neuron
  - injury to: cell bodies in the conus medullaris, their axons in the cauda equina, or the pelvic nerve.
  - NO spinal cord mediated peristaltic activity (only slow activity by mesenteric plexus - fluid absorption continues, thus hard stools)
  - Transit time ***very*** slow (?6 days?)
  - External anal sphincter denervated, thus incontinence common, especially smearing, leaking

Neurogenic Bowel Routine

- **Goals of a bowel care program:**
  - Minimize unplanned BM (“accidents”)
  - Stool evacuation at a regular, predictable time (longer it is there, harder it gets to evacuate).
  - Duration < 60 min.
  - Minimize GI problems
  - ** often takes months to establish.

Neurogenic Bowel Routines

- Upper motor neuron
  - Q1-2 days
  - Aim – soft, formed stool that's easily evacuated
  - High fiber, high fluid diet
  - Oral laxative night before routine (PEG, lactulose)
  - Warm beverage or food 30 minutes prior
  - bisacodyl suppository into rectum (clear out rectum first if stool present)
  - Assume upright or side laying position
  - Digital rectal stim q5-10 minutes as needed
  - +/- forward lean/ mild valsalva

Neurogenic Bowel Routines

- Lower Motor Neuron
  - Daily **or several times** per day
  - Aim- firm dry stool that can be retained between routines, yet not too hard to evacuate. Generally avoid stool softeners
  - Upright or side laying position
  - Valsalva and manual disimpaction until rectum clear of stool.
  - If too soft/liquid, consider fiber supplement

Summary

- NUMEROUS systems affected by SCI
- Many of these issues will present over the lifetime of person with SCI, thus family MD needs to be aware of these issues and look out for them, initiate diagnosis and treatment plan