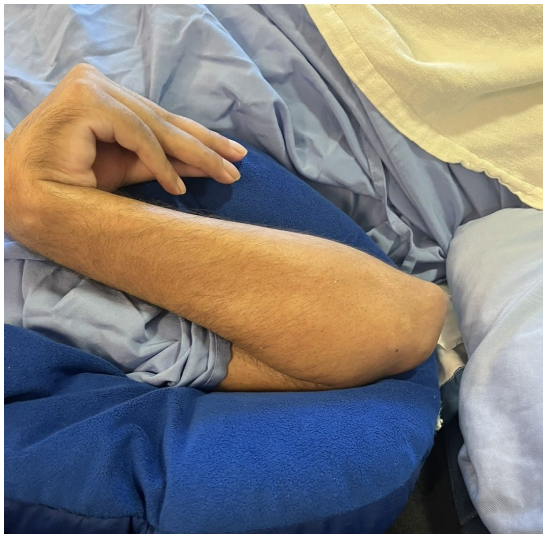
Hypertonia in Long-Term Care

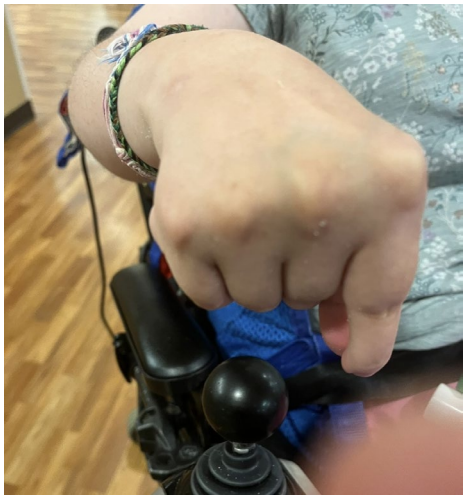
Dr. Lawrence Kei, MD, FRCPC
Physical Medicine and Rehabilitation
Clinical Instructor, UBC
September 25, 2024

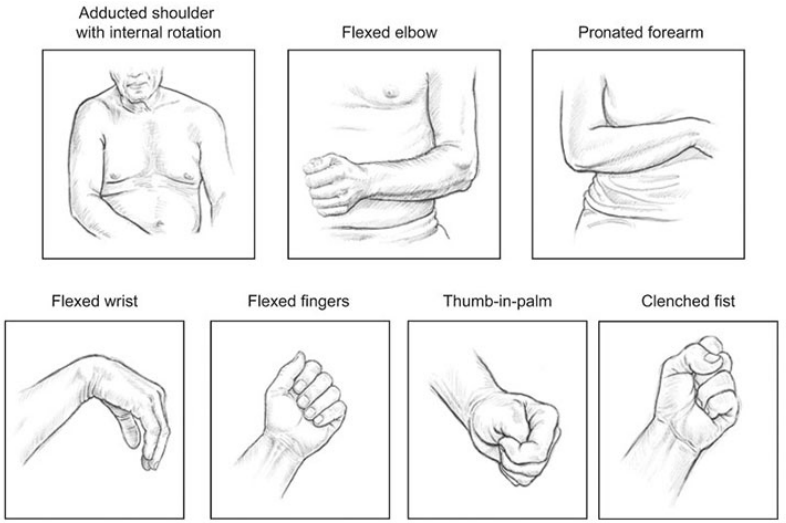
Disclosures

- Merz Therapeutics provides Nursing support at some LTC homes
- I receive no funding or other benefits from Merz Therapeutics








Adducted shoulder with internal rotation
Flexed elbow
Pronated forearm
Flexed wrist
Flexed fingers
Thumb-in-palm
Clenched fist

Objectives

1. Identify / distinguish between the different types of hypertonia present in LTC

2. Describe 3 physical management strategies for increased tone

3. Describe pharmacologic treatments for spasticity

Definitions

- Hypertonia
- Spasticity
- Paratonia
- Rigidity
- Contracture



Hypertonia

Definition

- increase in tone or resistance to movement when limb is passively moved
- Many multi-factorial

a. Non-neural component

- properties of Connective tissue/muscles crossing joint or within joint itself
- *Ex. Contractures*

b. Neural component

- Increased stretch reflex (UMN) – *spasticity*
- Extrapyramidal – *rigidity*

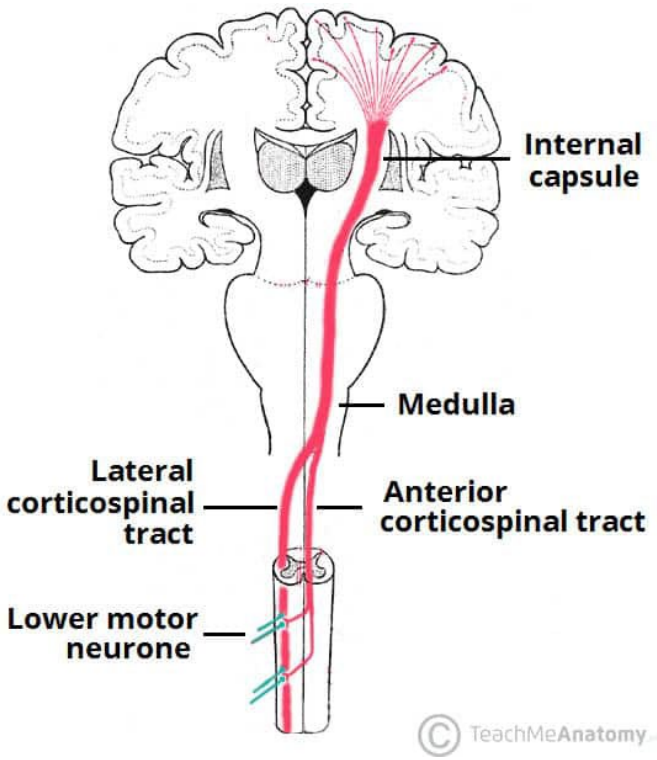
Non-neural Hypertonia = Contractures

- Final common pathway for many conditions
- Restriction of the peri-articular connective tissues
- Risk Factors: trauma, TKA, diabetes, burns, *edema*
- Immobilization*
    - Within 2 days → muscle atrophy/shortening, reduced muscle compliance (increase ratio collagen: muscle fibers)

Neural: Pyramidal vs. Extrapyramidal

Upper Motor Neuron (UMN) Syndrome

- Lesion: Lateral corticospinal spinal tract or corticobulbar tract
- "Pyramidal" (pyramids of medulla)
- Crosses
- FN: Voluntary movement at limbs
  - 10% trunk muscles (anterior corticospinal tract)



Upper Motor Neuron (UMN) Syndrome

Upper Motor Neuron (UMN) vs. Lower Motor Neuron (LMN) Syndrome

	UMN syndrome	*LMN Syndrome*
Type of Paralysis	*Spastic Paresis*	*Flaccid Paralysis*
Atrophy	No (Disuse) Atrophy	Severe *Atrophy*
Deep Tendon Reflex	*Increase*	Absent DTR
Pathological Reflex	Positive *Babinski* Sign	Absent
Superficial Reflex	Absent	Present
Fasciculation and Fibrillation	Absent	Could be *Present*



Spasticity

Definition:

- motor disorder characterised by a velocity-dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex, as one component of the upper motor neurone syndrome (LANCE)

- Pandyan et al (2005) Disordered sensorimotor control, resulting from an UMN lesion, presenting as an intermittent or sustained involuntary activations of muscles
    - Not solely a motor disorder as various afferents are involved

Stretch Reflex: Lots of supraspinal modulation

Normally stretch reflexes elicited with *high-vel stretch stimuli* (tendon hammer). - resistance is only from connective tissue (non- neural).

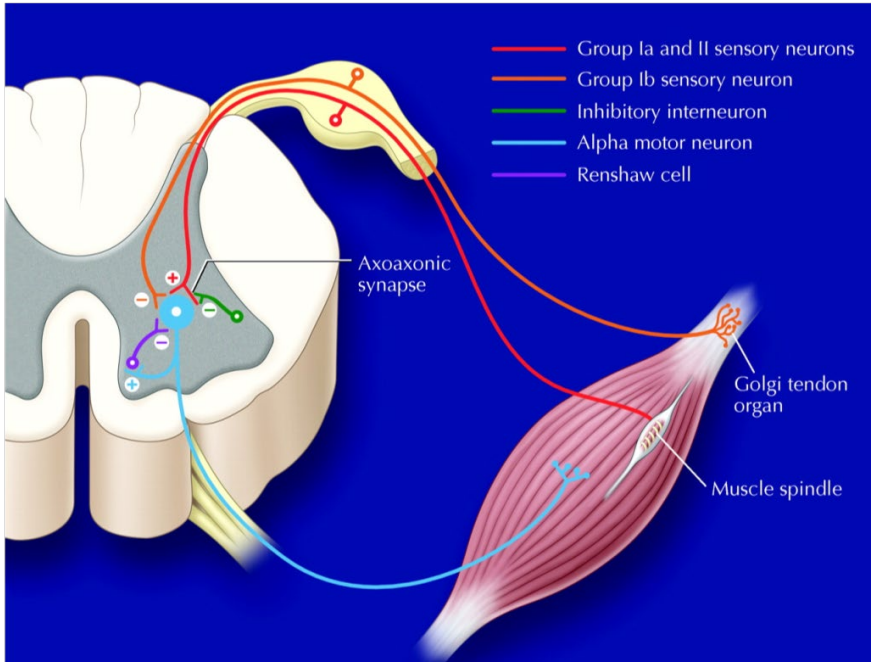
VS.

Spasticity
- stretch reflexes elicted at low velocity stretches

- Dysfunction in supraspinal inhibition
- Assessment of non-neural component requires very slow movement

Summary of these inhibitory influences:

- Reciprocal Ia inhibition (inhibition of antagonistic muscles)
- Presynaptic inhibition of Ia terminals (axoaxonic synapse)
- Nonreciprocal Ib inhibition (golgi tendon organ)
- Recurrent Renshaw inhibition
- Group II afferent inhibition



Extrapyramidal Syndrome (EPS)

- FN: modulates info coming down
    - Makes movements fluid/smooth
    - Not a discrete entity but separate parts: basal ganglia, cerebellum, vestibular nucleus, originate in brainstem
- ***Rigidity*** and bradykinesia
    - Parkinson /parkinsonian syndromes
    - *Alzheimer dementia, other dementia such as frontotemporal lobal degeneration or Lewy body disease*
- *Same resistance in all directions (flex/ext)*
- *Not velocity dependent*

VS. Spasticity

- *More resistance in one direction the other direction (flex/ext)*
- *More tone in initial part of movement – "Clasp knife spasticity"*
- *It is velocity dependent (i.e. more noticeable with fast movements)*

HYPERTONIA	
Spasticity	**Rigidity**
☐Weakness plus hyperreflexia/Babinski	☐No weakness, normal reflexes/plantar reflexes.
☐Velocity dependent	☐Not velocity dependent *(ie. Detectable with even slow movements)*
☐More resistance in one direction (affects antagonist groups asymmetric)	☐Same resistance in all directions (antagonist and agonist equal)
☐More tone in initial part of movement "Clasp knife spasticity"	☐***Cog wheel rigidity*** (Parkinson's) – Tremor superimposed on hypertonia →intermittent increase in tone during the movement - Tremor common with basal ganglia ☐***Lead pipe rigid*** (uniform tone increase)

Paratonia

- Varying definitions
- Pathology poorly understood
- 2006 expert consensus " a form of hypertonia with an involuntary variable resistance during passive movement" in any direction
- Type of paratonia may change with progression of the dementing illness
    - active assistance "facilitatory paratonia" more common early in the course of degenerative dementias, VS. active resistance (inhibitory paratonia) more common later
    - Velocity dependent (e.g., a low resistance to slow movement and a high resistance to fast movement).
    - Force dependent



ig. 1. Typical (fetal) posture of patient with advanced paratonia
ith flexion and adduction of the extremities.

Paratonia vs. Spasticity Vs. Rigidity

(oppositional) paratonia VS spasticity

1) No catch/clasp-knife response
2) resistance to movement in paratonia can be in ***any direction***

(oppositional) paratonia Vs. Parkinsonian rigidity

1) Velocity dependency of paratonia.
2) Parkinsonian rigidity = resistance is not dependent on the speed of the movement (lead pipe phenomenon)

HYPERTONIA		
Spasticity	**Rigidity**	**Paratonia**
☐Weakness plus hyperreflexia/Babinski	☐No weakness, normal reflexes/plantar reflexes.	☐No weakness
☐Velocity dependent	☐Not velocity dependent *(ie. Detectable with even slow movements)*	☐Velocity dependent
☐More resistance in one direction (affects antagonist groups asymmetric)	☐Same resistance in all directions (antagonist and agonist equal)	☐More resistance in one direction
☐More tone in initial part of movement "Clasp knife spasticity"	☐***Cog wheel rigidity*** (Parkinson's) – Tremor superimposed on hypertonia →intermittent increase in tone during the movement - Tremor common with basal ganglia ☐***Lead pipe rigid*** (uniform tone increase)	☐NO "Clasp knife spasticity"

Objectives

1. **Identify / distinguish between the different types of hypertonia present in LTC**

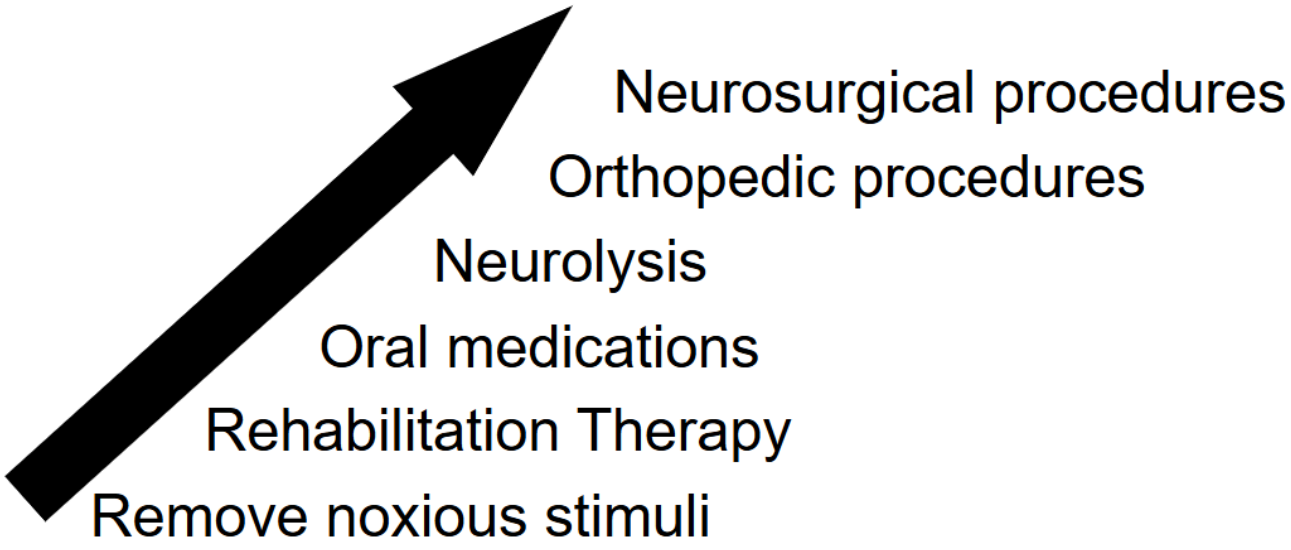
2. Describe 3 physical management strategies for increased tone

3. Describe pharmacologic treatments for spasticity

Spasticity: Goals of Treatment

- *Reduce tone/ painful spasm*
- *Improve activities of daily living (e.g., dressing, hygiene)*
- *Improve limb position (cosmesis)*
- *Improve gait / transfers*
- *Improve orthotic fit*
- *Prevent Pressure wounds (buttock, heels)*
- *Prevent Contractures**

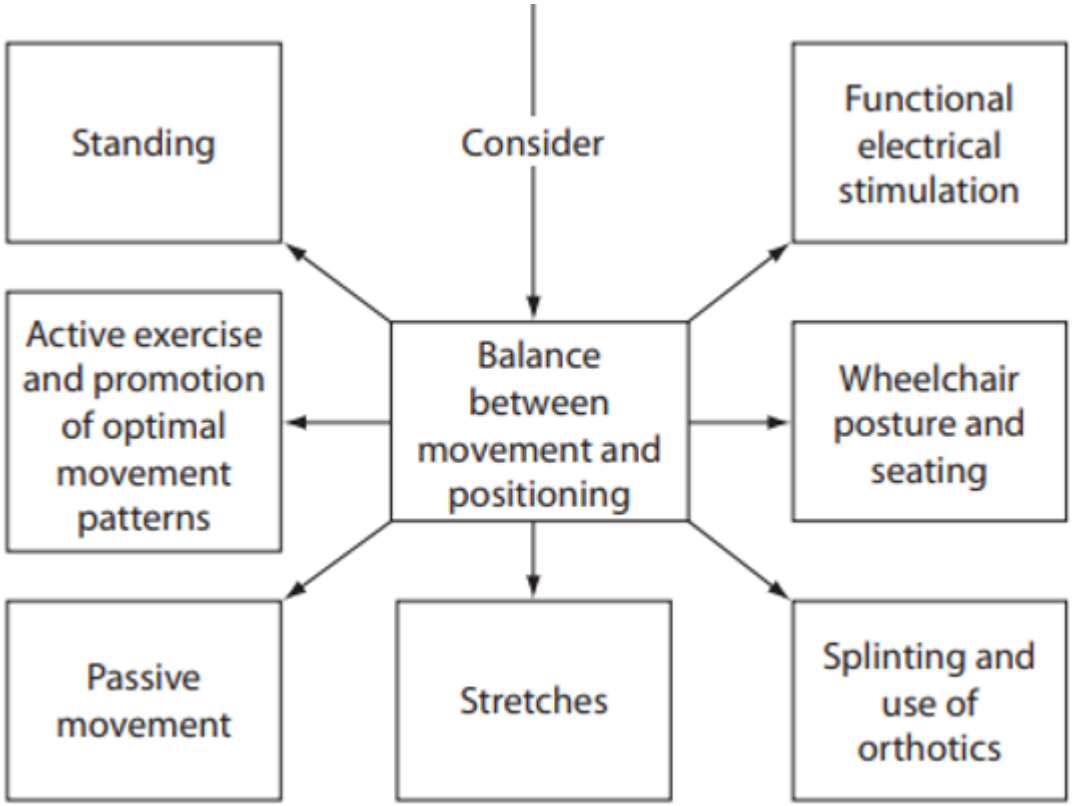
Traditional Step-Ladder Approach to Management of Spasticity



Check for Noxious stimulus!

- Bladder (UTI, stones, blocked catheter)
- Bowel (constipation, hemorrhoids)
- Ingrown toenails
- Pressure wounds
- DVT
- Heterotopic Ossification
- Poor fit in brace or wheelchair*

Physical Management Strategies



Management Strategies: Standing

- Promotion of anti-gravity muscle activity in the trunk and lower limbs
- Maintenance or improvement in soft tissue / joint flexibility → decrease risk of contracture development
- Modulation of the neural component of spasticity through *prolonged stretch and altered sensory*
- positive psychological effect

How long to stand for?

- lack of conclusive evidence.
- Duration of standing in studies varies ***20 minutes to 1.5 hours daily***,
- encourage wheelchair users *to stand daily (even few minutes)*
- *if achievable, half an hour would seem a reasonable target*.

Standing

- Fatigue → resting arms on a support (ex. kitchen table, work surface, back of an arm chair, a wall bar or a walking aid
- Slowly rising before standing to minimize spasms
- electrically powered standing frame or standing wheelchair
- Wheelchair: incorporate standing into everyday tasks
  - getting items out of high cupboards,
  - eye-level conversations





Passive Movement and Stretching

Passive movements

- recommended that all affected body parts be moved through their available ROM on a daily basis.
- Passive cycling
- Timing anti-spasticity meds to be taken 20–30 minutes before movements
- Movements should be performed at a slow speed
- *minimise triggering the stretch reflex*. Spasms may occur secondary to the sensory or stretch stimuli; if this is problematic, the movement should be paused and continued once the spasm passes.
- *avoid holding the ball of the foot, as this sensory stimulation can be a common trigger for spasms*.
- Starting positions
    - if extensor spasms in legs when lying flat, use of a positioning aid such as a T-roll, pillows or bed mechanism to bend the knees prior to starting the movements can reduce the number of spasms.

Positioning/Splinting

Theory: keep muscles in a lengthened position for as long as tolerable
→ desensitize stretch receptors by the prolonged and slow stretching

- Allow lengthening of vulnerable soft tissues in a variety of positions throughout the day/night
- Change patterns of spasticity and reduce spasms by positioning in postures ***opposite*** to the pattern of the spasticity or spasms
    - e.g. *if extensor spasms are problematic, then position in flexion*
- Key: regular changing of positions throughout the day and night.





Positioning

- Use rolled-up towel or cushion placed between the knees

- Ex. T –Roll: maintain a flexed position at the knees and hips and bring the *lumbar spine into more flexion, which in turn can reduce extensor spasticity* and *spasms*

-

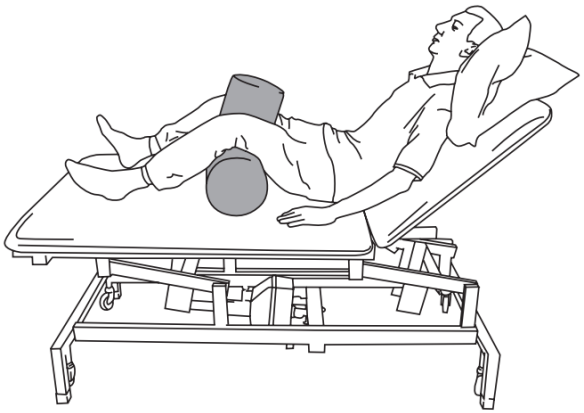
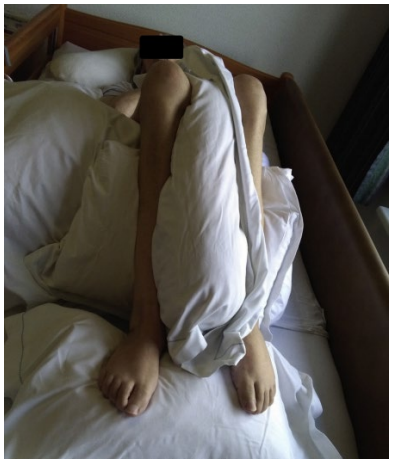


Figure 4.6 The use of a T-roll in sitting.



Figure 4.7 The use of a T-roll in side-lying.



Wheelchair and Seating

- Hypertonia may help sitting (ex. truncal weakness)
- Proper seating can reduce spasticity
    - comfortable/well supported position → minimizes tone/spasms
    - Minimalizes effects of fatigue on posture, reduces effort breath/speech

- Additional supports: headrests/tables (reduce fatigue to allow support weight of head/arms.

- Tilt in space to allow to offload

- Seat base / backrest position



Paratonia Management

Research is limited…

- Stabilizing cushions
- Massage/warm water baths/heat
- Pilot study → use of botulinum toxin.

Pharmacologic

Table 3: Dosing regimen, mechanism of action and side effects of drugs commonly used to treat spasticity

Drug	Initial dose	Daily maximum	Mechanism of action	Common side effects
Baclofen	5 mg 3 times daily	80 mg (can be higher if side effects are not a problem). Best divided into 4 doses	Centrally acting GABA analogue. Binds to $GABA_B$ receptor at the presynaptic terminal and thus inhibits the muscle stretch reflex	Daytime sedation, dizziness, weakness, fatigue, nausea; lowers seizure threshold *Withdrawal seizures and hallucinations with abrupt discontinuation*
Dantrolene	25 mg	100 mg 4 times daily	Interferes with the release of calcium from the sarcoplasmic reticulum of the muscle	Generalized muscle weakness, mild sedation, dizziness, nausea, diarrhea *Hepatotoxicity (liver enzymes should be monitored)*
Tizanidine	2–4 mg	36 mg	Imidazole derivative, with agonist action on alpha-2 adrenergic receptors in central nervous system	Dry mouth, sedation, dizziness, mild hypotension, weakness (less common than with baclofen) *Liver enzymes should be monitored*
Clonidine	0.05 mg twice daily	0.1 mg 4 times daily	Acts at multiple levels as an alpha-2 agonist in the central nervous system	Bradycardia, hypotension, depression, dry mouth, sedation, dizziness, constipation *Monitor pulse and blood pressure during treatment*
Gabapentin	100 mg 3 times daily	600–800 mg 4 times daily	GABA analogue. May have an indirect effect on GABA-ergic neurotransmission	Somnolence, dizziness, ataxia and fatigue

Note: GABA = gamma aminobutyric acid.

INTERVENTIONAL

- Diagnostic nerve blocks/Phenol injections
  - Planning for more permanent/invasive interventions (i.e. phenol, surgery)
  - Spasticity vs. contracture

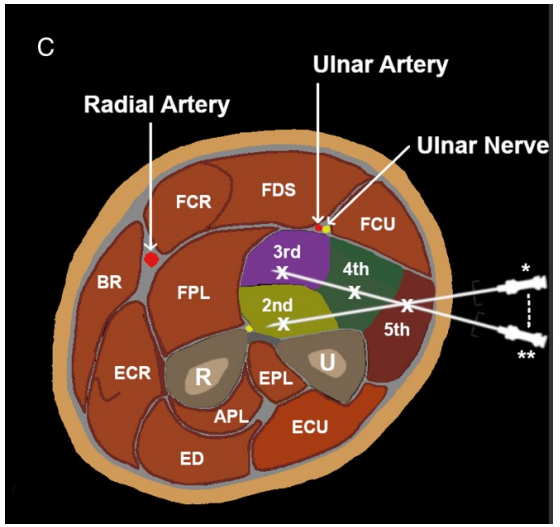
- ***Botulinum toxin injections***

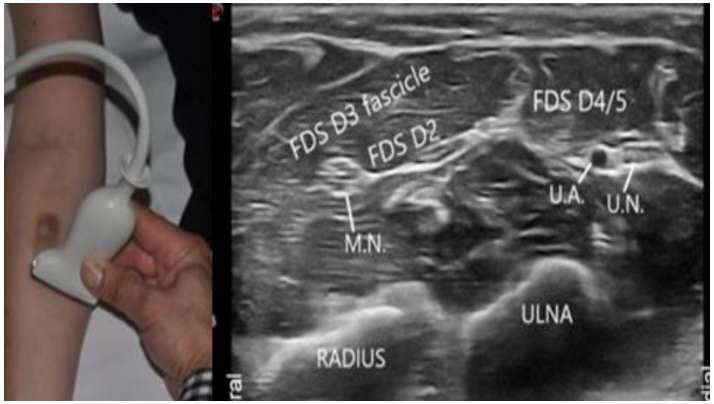
- Intrathecal baclofen Pumps

- Surgical
  - electrical stimulators:
  - Rhizotomy
  - Peripheral neurectomy
  - Tendon lengthening procedures.

Botulinum Toxin Injections

- Xeomin (upper extremity only), Botox, Dysport
- Electrical stimulation and/or US guidance
- Duration of effect ~ 3 months
- Potential Side Effects:
  - Bleeding, bruising, swelling
  - Flu-like syndrome
  - Spread of toxin effect
  - Weakness





Objectives

1. **Identify / distinguish between the different types of hypertonia present in LTC**

2. Describe 3 physical management strategies for increased tone

3. Describe pharmacologic treatments for spasticity

Thank you!

REFERENCES

1. Drenth H, Zuidema S, Bautmans I, Marinelli L, Kleiner G, Hobbelen H. Paratonia in Dementia: A Systematic Review. J Alzheimers Dis. 2020;78(4):1615-1637. doi: 10.3233/JAD-200691. PMID: 33185600; PMCID: PMC7836054
2. Stevenson, V.L., & Jarrett, L. (Eds.). (2016). Spasticity Management: A Practical Multidisciplinary Guide, Second Edition (2nd ed.). CRC Press. https://doi.org/10.1201/b21452