Dysfunctions of the central nervous system

Applications of FMID in plegic patients

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Epidemiology of spasticity:

- "Spasticity is a motor disorder characterized 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 (1980)

- 65–78% of patients with spinal cord injury

Maynard et al. 1990

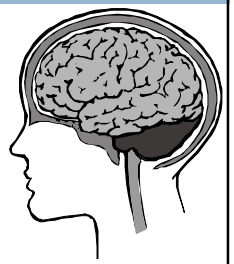
- only around 35% in stroke patients with persistent hemiplegia

Sommerfeld et al. 2004.

Stroke epidemiology

- Within three months after stroke, 19% of people will incur spasticity, and the percentage rises to 38% after 12 months.
- 65% of people who experience paralysis after stoke maintain a excessive muscle activity in the upper limbs for the 26 weeks after the event.
- Disabling spasticity occurs more commonly in young survivors (less than 55 years) or the first event of stoke.

Most common symptoms of spasticity:



- Muscle stiffness;
- Hypertonia in the upper extremities;
- Loss of fine motor control;
- Paresis;
- **"Contraction" of the soft tissue;**
- Muscle overactivity that involves reduction in the ability to relax;
- Muscle spasms;
- Postural changes of the limbs, muscle fatigue.

Compensations:

- The post-stroke spasticity is often associated with **secondary complications,** pain, and limitation in mobility.
- It is common that patients compensate certain aspects of their spasticity with certain movements and postures that may involve **changes** in the muscles and **soft tissues.**

- Lundstrom E, et al 2008; Sommerfeld DK, et al. 2004; Lamy JC,et al. 2009;

Symptoms of spasticity:

- Several studies have shown that many subjects who have been found to be spastic by clinical examination turn out **not** to have any signs of **hyperreflexia**

Sinkjœr et al. 1993, Schindler- Ivens & Shields 2004.

- The finding that **no correlation** between the degree of spasticity (assessed by the Ashworth scale) and the degree of **reduced reciprocal inhibition** has been shown

Crone et al. 1994

Spinal cord inibition?

- Changes in recurrent inhibition thus probably plays **no** major role in the pathophysiology of spasticity
  - Recurrent inhibition is mediated by Renshaw cells, which are located in the ventral horn of the spinal cord, where they receive excitatory collaterals from the motor axons and project back to the motoneurones as well as Ia inhibitory interneurones

Baldissera et al. 1981.

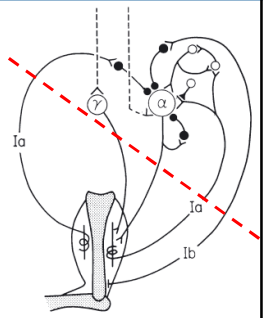


Figure 1 Diagram of various spinal pathways which may contribute to the development of spasticity.

Central or Peripheral pathology?

- "Spasticity human can not be considered as a immedita consequence of CNS injury, as it progresses during the weeks or months after the injury."
- "Normally the symptoms of post-stroke spasticity develop their greatest severity at 3 months after the event. After this period **all aggravations** of spasticity is believed to be **due to changes in the musculature.**"

- Lundstrom E et al. 2008; Sommerfeld DK et al 2004; Lamy JC et al. 2009

Structural changes in the muscles

- It has therefore been argued that spasticity may also be explained by **changes in muscle properties** and not only by hyperreflexia and alterations of the central processing of sensory input in the spinal cord

Dietz et al. 1981, Gracies 2005a,b.

- The increased muscle tone in such subjects rather seems to be caused by **structural changes** in the muscles related to contractures

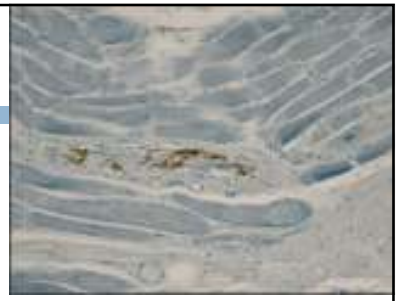
Dietz et al. 1981; Sinkjær et al. 1993, see review by Gracies 2005a,b.

Excess of connective tissue

- These changes range from:
  - a rounding of the muscle fibers
  - increase of space inter-fibers
- Up to:
  - hypertrophic fibers
  - atrophic fibers
- associated with the presence of **excessive connective tissue.**

Dietz V et al. 1981.

Muscle spindle property



- "The capsule of the muscle spindles is either attached to the perimysium, or to fascial septae, or fine connective tissue threads on in the intramuscular spaces ".

Baldissera 1981.

- "…. the extra and intra fusal muscle fibers possess complex biophysical properties that create the stiffness and / or laxity."

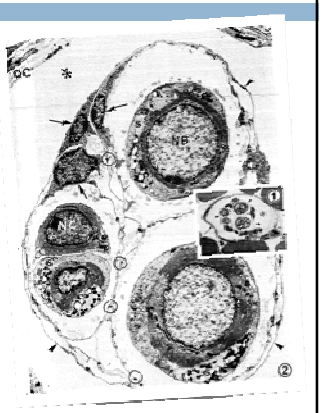
Buchthal & Kaiser, 1951; Lakie, Walsh & Wright, 1984,

Muscle spindle capsule

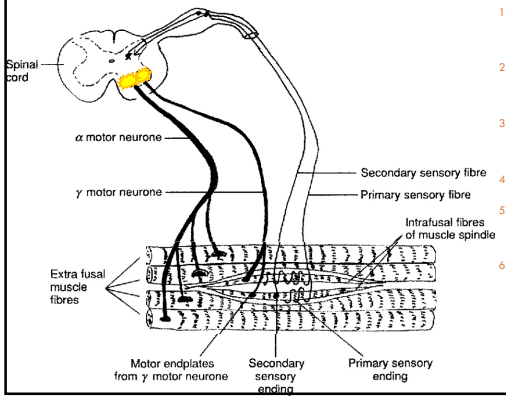
The external capsule is derived directly from the perineurium; the internal capsule is derived from endoneurium

between the two there is a space filled with a gel rich in mucopolysaccharides and **hyaluronic acid,** highly viscous

Ovalle WK, Dow PR. 1983

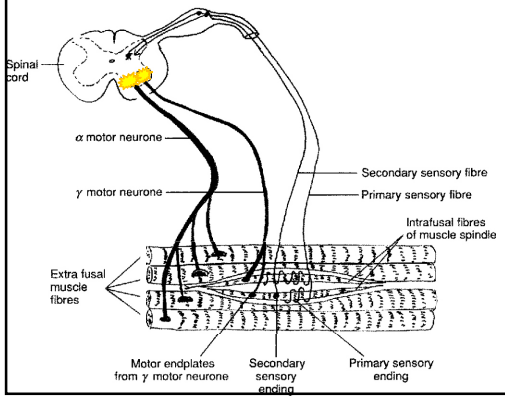


Physiology of muscle spindles



1. Gamma motoneuron become active
2. Contraction of intrafusal fiber
3. Stretching of anulospiral ring
4. Ia afferent fibre
5. Stimulation of alfa motor neuron
6. Contraction of extrafusal muscle fibres

Phatology of muscle spindle



1. Gamma motoneuron become active
2. Contraction of intrafusal fiber
3. The alteration of the capsule don't permit the stretching of the anulospiral ring

HYPERTONICITY OF EXTRAFUSAL MUSCLE FIBRES

Agonist and antagonist

- it is likely that a voluntary movement stretching a spastic muscle may produce a reflex in the antagonistic muscle that would oppose the voluntary movement.
- it has been recently reported that exaggerated stretch reflexes in the finger flexors contribute to the impairment of finger extension in stroke patients.

Kamper et al., 2003

Pharmaceutical therapy

- Reduction of the sensory feedback by the current available antispastic therapy (i.e. diazepam, baclofen, tizanidine) may thus not only reduce spasticity, but will inevitably also **influence the ability** of the patients to perform voluntary movements.
- From this point of view it is evident that antispastic therapy should be **given with care** in patients with only mild to moderate spasticity and reasonably preserved functionality.

Nielsen JB, et al 2007.

Fascia and spasticity

- "The fascial restrictions were clearly represented in a sample of children with spastic cerebral palsy."
- "we hypothesized that these factors would correlate with muscle spasticity."

Davis MF, Worden K, Clawson D, Meaney FJ, Duncan B. Confirmatory factor analysis in osteopathic medicine: fascial and spinal motion restrictions as correlates of muscle spasticity in children with cerebral palsy. J Am Osteopath Assoc. 2007 Jun;107(6):226-32. PubMed PMID: 17635903.

Thanks