First Course "Basics in Motion analysis"

TRAMA Project

September 10 -12 th 2007

Helga Hirschfeld
Karolinska Institutet
Dept. of Neurobiology, Care Sciences and Society
Motor Control and Physical Therapy Research Laboratory

SPASTICITY

- Definition
- Signs and Symptoms
- Assessments
- Treatments

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Condition resulting from brain trauma (incl. CP), spinal cord injury, systemic degenerative processes (MS)

- Spastic spasms are vigorous and painful
- Impaired voluntary movement and posture control

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Definition in terms of signs and symptoms rather than in terms of neurophysiological mechanisms.

Motor Disorder characterized by velocity dependent increase in the tonic stretch reflex (contributes to muscle tone) and exaggerated tendon jerks resulting from hyper-excitability of the stretch reflex *(Lance 1980)*

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TONIC STRETCH REFLEX = tonic autogenic recruitment of motoneurons in response to a slow increase in muscle length.

Combined action of different peripheral receptors via spinal reflex loops.

Tonic stretch reflex is polysynaptic.

Feldman 1966 model - Equilibrium Point Hypothesis

= treshold of tonic stretch reflex

Spasticity ➡ decreased treshold

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MUSCLE TONE is the force with which a muscle resists being lengthened.

Provides important feature of muscles:

1) Spring-like behavior (intrinsic stiffness)
2) and neural component (tonic stretch reflex)

Slow stretch: muscle resists stretching due to passive elastic properties, then at certain length, recruitment begins (threshold of the tonic stretch reflex) first component peripheral independent of reflexes, second component has a reflex nature (velocity dependent)

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PHASIC STRETCH REFLEX

Emerges in response to a change in the level of the receptor stimuli. Represent a short burst leading to a twitchy movement. Monosynaptic reflexes are phasic reflexes

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Signs of spasticity

1. increased resistance to passive movement (uni-directional)
2. velocity dependent
3. hyperactive tendon jerk

Signs of rigidity

1) resistance bi-directional
2) independent to velocity
3) no hyperactive tendon jerks

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POSSIBLE MECHANISMS RESPONSIBLE FOR SPASTICITY

It is assumed that spasticity is associated with a deficit in spinal inhibitory mechanisms (pre- and postsynaptic) and a disruption of the normal functioning of certain descending systems.

However these systems are still not well defined in humans and their role in voluntary motor control is unclear.

In following slides examples of possible mechanisms

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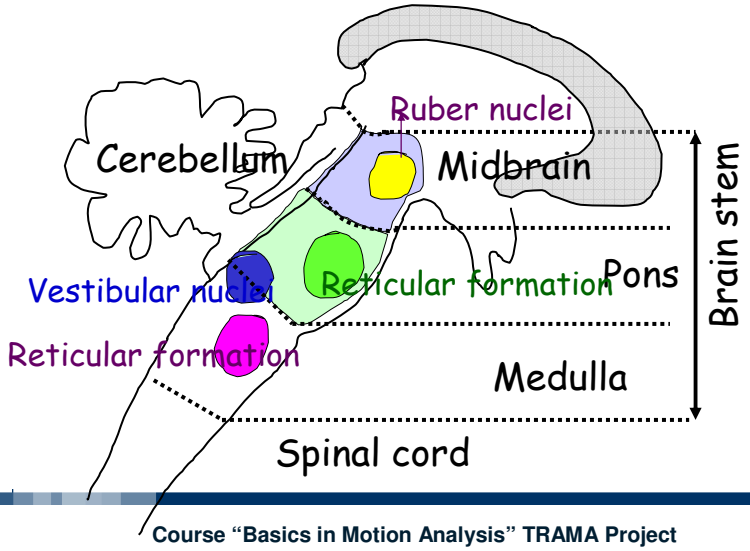

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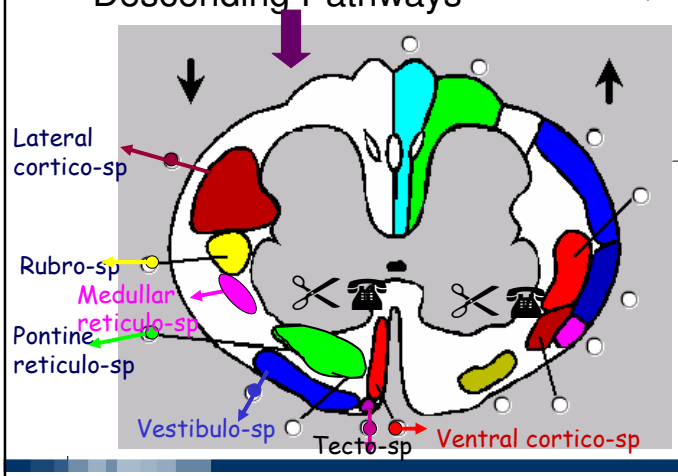
Descending Pathways

(from Neurolab, 2004)



Ventromedial tracts

\+ Ext.

\- Flex.

Dorsolateral tracts

\+ Flex.

\- Ext.

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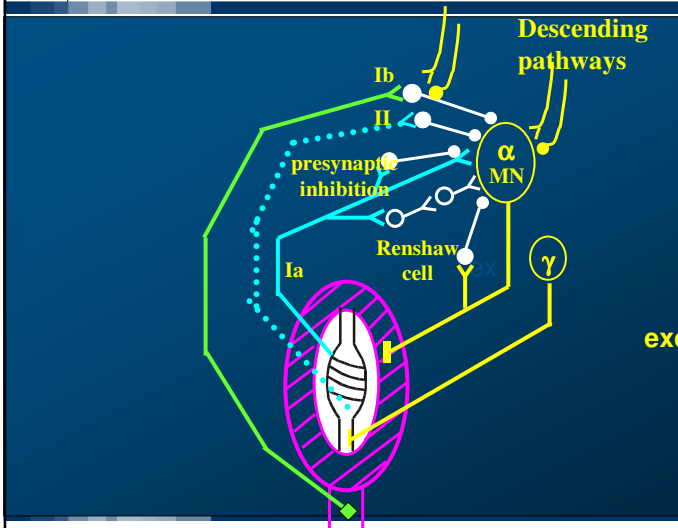

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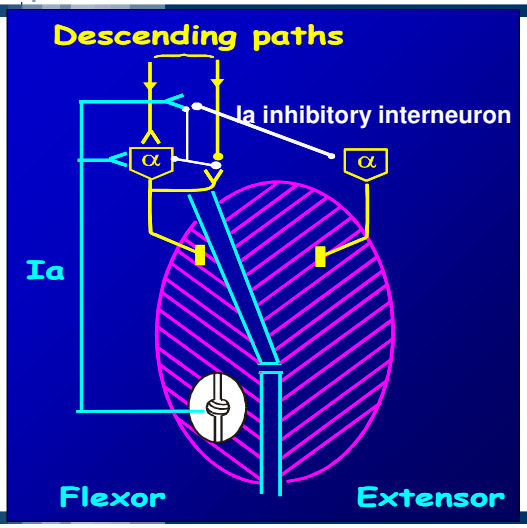

Reciprocal inhibition

Agonist / antagonist activation in voluntary movements

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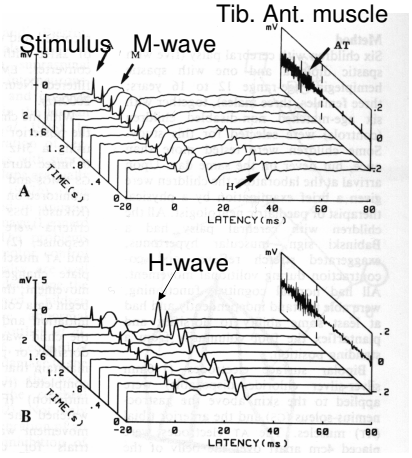

Lack of reciprocal inhibition

H-REFLEX (SOLEUS MUSCLE)

(Leonard et al 1991)

CONTROL (14 Y)

SPAST. DIPLEGIA (14 Y)

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TONIC VIBRATION REFLEX

High frequency, (100 Hz) low amplitude (1mm) muscle vibration may induce action potentials in response to every cycle of vibration.

TVR is accompanied by suppression of monosynaptic reflexes of same muscle (e.g. of H-reflex)
Suppression of presynaptic origin.

Vibration increases excitation of Ia, Ib and group II endings of vibrated muscle. Different frequency different effects - kinesthetic illusions.

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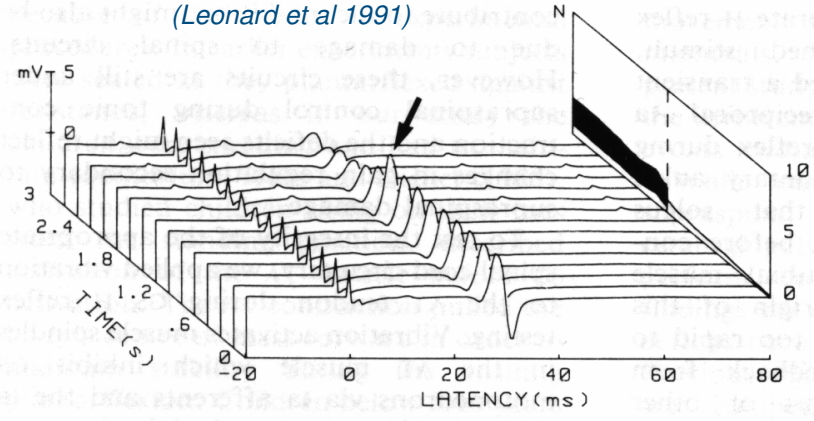
H-reflex and Vibration on triceps surae muscle
(Leonard et al 1991)



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Babinski's sign
indicator of CNS damage
Includes ext. of great toe and abduction of toes in response to stroking of the sole of the foot

Clonus
auto-oscillation in hyper-excitable monosynaptic stretch reflex loops, generating series of alternating bursts in flexor och extensor muscles of a joint (6-8 Hz), in response to quick passive or active movement
Clonus lasts for seconds, minutes - has to be stopped mechanically!

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CLINICAL ASSESSMENT OF SPASTICITY

ASHWORTH SCALE / MAS (0-4)

Description of muscle tone scores 1-5
1- no increase in tone
5- affected part is rigid in flexion or extension

SPASM SCALE (Penn 1989)

Frequency of spasm scores 0-4
0 - no spasm
4 - spasms occurring more frequently than ten times per hour

Scales are subjective, reflect experimenters general impression

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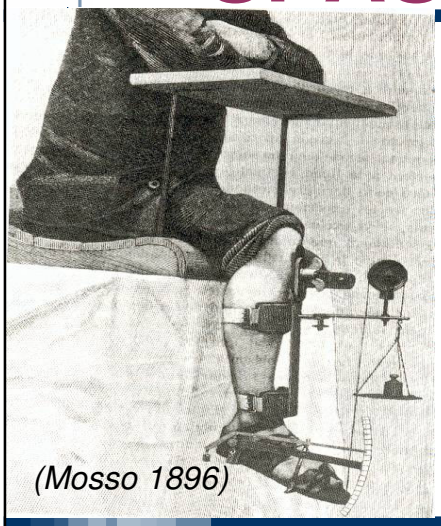

Instrumental assesment

Measure of muscle tone

Myotonograph
measured deflection of the foot
due to gravity

Pendulum Test of Spasticity (Drop Test)

Isokinetic dynamometer acts as an electronic goniometer

(not applicable for most of the muscle groups)

MYOTONOMETER (under evaluation in our lab) *www.neurogenic.com*

Quantitative measure of muscle compliance,
can be used in passive or active state of muscle

Muscle "compliance" is an intrinsic property of the muscle in which tension within the muscle increases during lengthening without a change in the neural drive to the muscle.

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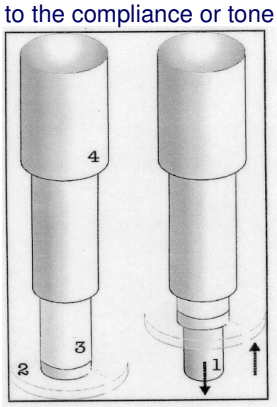
The myotonometer assesses the amount of resistance or force a muscle exerts against the probe –(probe gently pushed perpendicular to muscle fibers).Is directly proportional to the compliance or tone of a muscle.



This figure depicts the general operation of the probe. The probe is the mechanical part of the Myotonometer and is capable of sending information pertaining to force and tissue displacement to the computer. (1) Inner probe (2) Plexiglass collar (3) Inner shaft (4) Handle. The user grasps the handle and applies downward pressure perpendicular to the muscle. As pressure is applied, the inner probe pushes into the muscle whereas the plexiglass collar remains relatively motionless on the skin surface. Specialized transducers monitor ongoing pressure changes with the accompanying changes is displacement between the inner probe (1) and outer collar (2).

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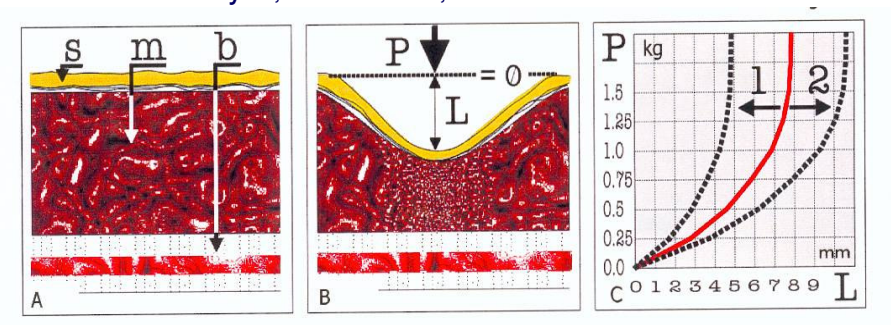
s - skin layer; m -muscle; b - bone



By applying downward pressure on the Myotonometer (P) on the skin overlying the muscle, the muscle deforms and offer counter pressure.

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Myotonometer is a tissue compliance meter.
Transducers within the probe measure the amount of underlying tissue displacement per unit of force applied to the muscle by the probe.

Length-tension curves generated from recordings show the amount of stretch to the muscle per unit of applied force.

Tissue compliance meters are valid and reliable measures of muscle tone and compliance.

Can be used to differentiate levels of severity of spastic condition

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TREATMENT

•**DRUG THERAPY** - most promising

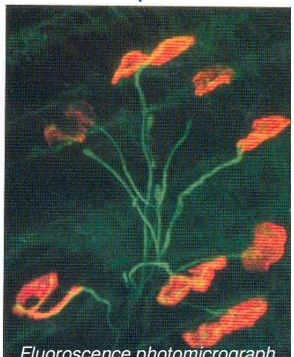
Injections of BUTOLIN-TOXIN A affects the pre-synaptic release of ACTH at the motor end plate

Baclofen pump

•**Neurosurgical procedures (rhizotomi)**

•**Physical therapy,** (in combination with drug therapy)

Motor end plate



Fluoroscence photomicrograph (Purves et al 2001)

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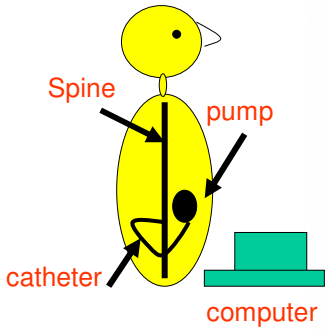
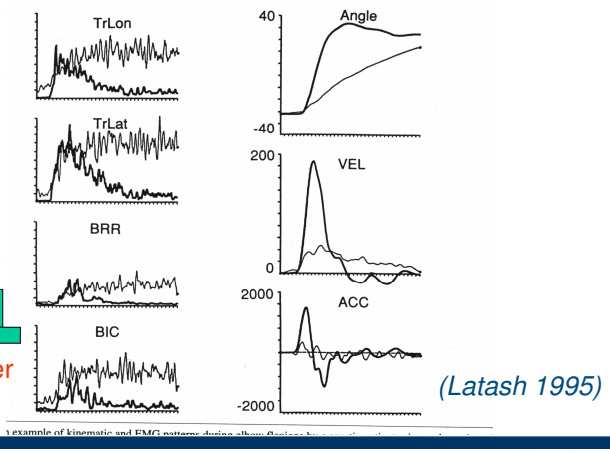
Elbow flexion before and after administration of intrathecal baclofen





(Latash 1995)

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Physical Therapy

- **Treatment focus on functional limitations.**

Active movement - patient has to overcome the spasticity influencing motor behavior by himself.

- **Strength training, Endurance training**

Treadmill training / weight support

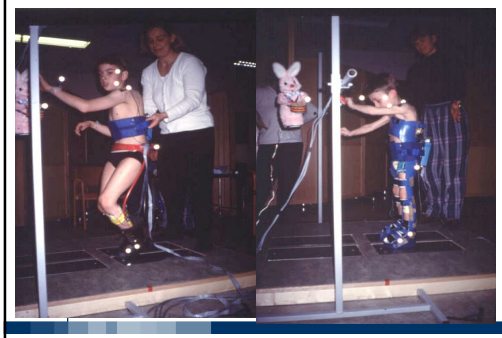
- **Muscle extensibility and joint mobility**
- **Skeletal alignment/ standing / sitting**

prerequisite for coordination of posture and voluntary movement. (Reference posture)

- **Relaxing techniques**

Importance: active movement - patient has by himself to overcome the spasticity influencing motor behavior.

Compensation of lack in alignement (standing shell),reduces background EMG and allows coordination of posture and voluntary movement.



Working Hypothesis

Compensating body posture , simulating postural orientation in standing will improve arm movement control.

Arm push while standing