

# Clinical Treatments I

## HYPOTONUS, DISUSE AND TRUE ATROPHY

### **Definitions:**

Hypotonus: abnormally decreased or deficient muscle tone

Atonic: without normal tension or tone

Muscle Atrophy: a decrease in the size of muscle cells and therefore size of the muscle affected. Atrophy causes a severe degree of hypotonicity and wasting of muscle tissue. There are two types of muscle atrophy, disuse and true

Disuse Atrophy: atrophy secondary to any primary lesion causing immobility, or due to failure to exercise part or all of the body.

True Atrophy: loss of function due to de-ervation of muscle, through pathology or trauma, or degeneration of muscle tissue itself, through disease process (ex. Muscular Dystrophy)

### **Cause:**

- Loss of function and/or immobility due to any pathology or trauma
- Secondary to central or peripheral nervous system disorder with reduced or absent neural input to the musculature
- Muscle wasting usually becomes obvious after 4-6 weeks and progresses to its maximum by 12 weeks (Magee).
- Circulatory changes due to neural injury take approximately 3 weeks to become significant (Magee)

### **Physiological and Pathological Changes:**

#### Hypotonus:

- Innervation intact
- Cell number, no change
- Cell size, reduced
- Muscular tone decreased
- Fibrosis unlikely but may be present
- Contractures unlikely but may be present
- AROM normal
- PROM may be normal, or reduced in the presence of contractures, joint dysfunction, or other pathology
- RROM may be reduced or weak.

# Clinical Treatments I

## Disuse Atrophy:

- Innervation intact
- Cell number and size: no change or reduced (time factor)
- Muscular tone decreased
- Contractures less common, but may be present
- AROM may be reduced, rarely absent
- PROM as per hypotonus
- RROM usually reduced, but not absent

## True Atrophy:

- Innervation disrupted
- Cell number and size reduced
- Muscular tone decreased
- Fibrosis may be present
- Contractures may be present
- AROM absent
- PROM as per hypotonus
- RROM absent

## **Signs and Symptoms:**

- Muscle weakness or paralysis, usually specific and localized
- Some degree of muscle wasting, decreased girth
- Possible presence of adhesions, fibrous or myofibrositic nodules
- Possible presence of contracture
- Tissue may feel cool or boggy

## **Assessment:**

### History:

- Systemic dysfunctions, pathologies seriously affecting the neuromuscular system
- Type/position of immobilization, and/or immobilization device
- Length of time immobilized

### Observation:

- Posture, compensations, tissue colour, scarring

### Palpation:

- Note presence/absence and location of fibrosis, contracture, adhesions

### Movement:

- AROM/PROM/RROM
- MMT for specific muscular involvement

# Clinical Treatments I

## Neurological:

- Essential to include dermatome, myotome testing in true atrophy, as well as sensation testing where the atrophy is secondary to a central nervous system disorder. Reflex testing may also be of assistance.

## Pain Referral:

- Pain may be secondary to nerve regeneration processes
- Pain may be of muscular origin (connective tissue resistance, muscular loss of flexibility or fatigue)

## Special Tests:

- Girth measurement
- As per specific primary or related dysfunction(s) indicated

## **HYPERTONUS**

Hypertonicity can result in pain and tenderness, secondary to ischemia and/or the formation of active trigger points and muscle spasm. Abnormalities of posture, position and movement result and over time can lead to abnormal lengthening or shortening of structures (fibrosis and contracture).

### **Definitions:**

Tonus: normal or non-pathological tone

Hypertonus: abnormally increased or excessive muscle tone often with increased resistance to stretch, but possibly with a normal resting length (H&K)

Hypertrophy: enlargement of existing muscle fibers (T&D)

Hyperplasia: increase in number of muscle fibers

### **Treatment of Hypertonus**

#### Postural /Positional:

Trigger Point Release  
Stretching  
Point pressure/C-bowing/S-bowing  
X-Fiber Frictions/ mm stripping  
Origin-insertion approximation (reset mm. Spindle)  
Pin and stretch

#### Hydro:

Heat or neutral warmth

#### Remex:

Active movement/stretching  
Postural correction

# Clinical Treatments I

## Acute vs. Sub Acute Muscle Spasm

Acute	Sub Acute
Treatment Goal:	Treatment Goal:
Remove the cause	Remove the cause (Posture/Activities/Etc.)
Address metabolic function	Correct Joint function
Warm tissue/Cool Tissue	Decrease muscle guarding
Reset muscle/pt education	Restore muscle balance

<b>Acute</b>	Sub Acute
Treatment Techniques:	Treatment Techniques:
Rehydrate if post event	Deep Breathing/ Visualization
Deep Breathing/ Neural Stroke	Tx. Proximal and Bilateral Structures
Agonist Contract Stretch/AROM/PROM	Muscle Stripping/ Deep Circulatory work
Effleurage/Flushing/Petrissage	Flushing/ Petrissage
GTO/MTAR Release	Hold Relax Stretching
Hold Relax/ Ag. Contract	Joint Mobs
Tissue approximation	MFTPs/MTAR/ Fascial Release
Joint Play (GR 1&2 Oscillation-H&K316)	
<b>RESET MUSCLES</b>	<b>RESET MUSCLE</b>

# Clinical Treatments I