Characteristics of Whole Muscle Contraction

Dr.Viral I. Champaneri, MD
Assistant Professor
Department of Physiology

Learning objectives

- **Series elastic component**
- **Fast fibers v/s Slow fibers**
- **Motor unit**
- **Muscle tone**
- **Muscle fatigue**

Learning objectives

- **Muscle rigor & Rigor mortis**
- **Muscle hypertrophy & atrophy**
- **Hyperplasia of muscle**
- **Macromotor unit**
- **Fibers type**

Series – Elastic Component of Muscle Contraction

- When muscle fibers ➔
- Contracts against a Load
- Portion of the muscle fibers
- Do not contract

Series – Elastic Component of Muscle Contraction

- **Portion of the muscle fibers ➔ Do not contract**
- **Stretch slightly**
- **As tension ➔ Increases**

Series – Elastic Component of Muscle Contraction

1. Tendons

2. Sarcolemmal ends of the muscle fibers

- Where muscle fibers attach to the tendons

3. Hinged arms of the cross bridges

Series – Elastic Component of Muscle Contraction

- Stretch of these elements….
- Contractile part of the muscle of the muscle
- Must shorten ➔ Extra 3 % to 5 %

Fast Fibers V/s Slow Fibers

Fast Fibers

- Present in : Muscle react
- Rapidly
- Large fibers for great strength of contraction

Slow Fibers

- Present in: Muscle responds
- Slowly but with prolonged contraction
- Smaller fibers

Fast Fibers V/s Slow Fibers

Fast Fibers	Slow Fibers
• Extensive Sarcoplasmic reticulum (SR) • For rapid release of Ca^{2+} ions to initiate contraction	• Extensive Blood vessels system and capillary • To supply extra amount of O_2

Fast Fibers V/s Slow Fibers

Fast Fibers	Slow Fibers
• Large amount of Glycolytic enzymes • For rapid release of the energy by Glycolysis	• Increased number of Mitochondria • To support high levels of Oxidative metabolism

Fast Fibers V/s Slow Fibers

Fast Fibers

- Less Extensive Blood supply
- Oxidative metabolism is of secondary importance
- Fewer Mitochondria

Fast Fibers V/s Slow Fibers

Slow Fibers

- Contains large amount of Myoglobin
- Combines with Oxygen and
- Stores oxygen until needed
- Speeds Oxygen transport to the Mitochondria

Fast Fibers V/s Slow Fibers

Slow Fibers → RedMuscle

- Contains large amount of Myoglobin
- Myoglobin → Reddish appearance
- Red Muscle

Fast Fibers V/s Slow Fibers

Fast Fibers ➔White Muscle

- Deficiency of Red Myoglobin
- In Fast Muscle ➔
- White Muscle

Fast Fibers V/s Slow Fibers

Fast Fibers ➔ White muscle	Slow Fibers ➔ Red muscle
• Jumping • Short-Distance Powerful Running	• Prolonged • Continued muscle activity

Motorneuron

- **Each Motor neuron**
- **Leaves the Spinal cord ➔**
- **Anterior horn of the spinal cord**

Motorneuron innervates ➔Muscle fibers

- **Motorneuron ➔ Innervates**
- **Multiple different muscle fibers**
- **Number depending on the type of the muscle**

Motor Unit

- **All *Muscle fibers* innervated**
- ***By single motor nerve fiber***
- **Called ➔ Motor Units**

Number of Muscle fibers in Each Motor Unit

- **Small muscles ➔ Laryngeal Muscles**
- **React rapidly**
- **Whose control must be exact (Fine)**
- ***2 to 3 Muscle fibers ➔ Each Motorunit***

Number of Muscle fibers in Each Motor Unit

- **Large muscles ➔ *Soleus Muscle***
- ***Do not require ➔ Fine Control***
- **Several hundred Muscle fibers ➔ Each Motorunit**

Number of Muscle fibers in Each Motor Unit

- *Average*
- **80 to 100 muscle fibers**
- **To each motor unit in body**

Motor Unit

- **Not bundle together**
- **In muscle**

Motor Unit

- *Overlap*
- **Other motor units in *Microbundles of***
- ***3 to15 fibers***

Interdigitation of Motor Unit

- *Allows separate Motor units to contracts*
- **In *support of one another***
- **Rather than ➔ As individual segments**

Skeletal Muscle Tone

- *Certain amount of tautness*
- **Remains ➔ *In skeletal muscles***
- ***At Rest***
- **Called ➔ Muscle Tone**

Cause of Skeletal Muscle Tone

- **Skeletal muscle ➔ *Do not contract***
- ***Without an action potential***
- ***To stimulate the fibers***
- ***Except in certain pathological conditions***

Cause of Skeletal Muscle Tone

- **Results entirely from**
- **A low rate of nerve impulses**
- **Coming from ➔ Spinal Cord**

Spinal Cord Impulses

- Controlled partly by
- Impulses transmitted from ➔Brain
- To appropriate Anterior motoneurons

Spinal Cord Impulses

- Controlled partly by
- Impulses originate in ➔Muscle Spindle
- Located in ➔Muscle tendon

Muscle Fatigue

- **Prolonged**
- **Strong contraction of a muscle**
- **Leads to → Muscle Fatigue**

Causes of Muscle Fatigue

1. Depletion of the muscle glycogen
2. Diminishes the number of Acetylcholine vesicles
3. Interruption of blood flow through contracting Muscle

Study in Athletes ➔ Muscle Fatigue

- **Increases**
- ***Direct proportional* to the**
- ***Rate of depletion of the Muscle Glycogen***

Complete Muscle Fatigue

- **Interruption of the blood flow**
- **Through a contracting muscle**
- **Leads to almost complete muscle fatigue**

Interruption of blood flow ➔ Muscle Fatigue

- **Within 1 or 2 minutes because of the**
- **Loss of *nutrient* supply**
- **Loss of *Oxygen* supply**

Most of the Muscle Fatigue

- Inability of the contractile and metabolic
- Processes of the muscle fibers
- To continue the same work output

Muscle Rigor ➔ Irreversible

- **When muscle fibers are completely**
- ***Depleted of ATP & Phosphorylcreatinine***
- **Develop stage of Rigidity ➔Muscle Rigor**

Muscle Rigor ➔ Irreversible

- **In rigor ➔ Almost all myosin heads**
- **Attach to Actin but in**
- ***Abnormal***
- ***Fixed and***
- ***Resistant way***

Muscle Rigor ➔ Irreversible

- **Muscles effectively**
- **Are locked into place**
- **And become quite stiff to the touch**

Rigor Mortis ➔ Reversible

- **Several hours after death…..**
- **All the muscles of the body**
- **Go in to a state of *contracture*…**
- **Called *“Rigor mortis”***

Rigor Mortis ➔ Reversible

- The muscles ➔ Contraction
- Become ➔ Rigid
- Even without ➔ Action potentials

Cause Rigor Mortis

- **Loss of all the ATP**
- ***Required to cause separation of the cross-bridges***
- **Of Myosin filaments from Actin filaments**
- **During relaxation process**

Muscle remains in Rigor Mortis

- **Until the muscle proteins ➔**
- **Are destroyed**

Muscle Proteins ➔ Destroyed

- From the Autolysis
- Caused by enzymes released from Lysosomes
- Some 15 to 25 hours later
- Events occurs more rapidly at higher temperature

Muscle Hypertrophy

- Total mass
- of the
- Muscle enlarges

Muscle Atrophy

- Total mass
- of the
- Muscle decrease

Muscle Hypertrophy ➔ Results

- Increase in number of
- Actin (Thin) & Myosin (Thick) Filaments
- In each muscle fiber

Muscle Hypertrophy → Results

- Enlargement of the individual muscle fiber
- Called →
- Fiber Hypertrophy

Muscle Hypertrophy ➔ Occurs

- Contraction of the muscle at the
- Maximal or
- Almost Maximal force

Muscle Hypertrophy ➔ Occurs

- **Much greater extent**
- **When Muscle simultaneously loaded**
- **During Contraction e.g. Jimming**

Muscle Hypertrophy → Occurs

- Few strong contractions → Each day
- Hypertrophy →
- Within 6 to 10 weeks

During Developing Muscle Hypertrophy

- The rate of synthesis of muscle contractile protein
- Far greater
- Rate of decay

During Developing Muscle Hypertrophy

- **Greater numbers of Myosin and Actin filaments**
- **In the Myofibris**
- **Often increasing ➔ 50%**

During Developing Muscle Hypertrophy

- Some of the myofibirils
- Splits within each muscle fiber
- To form new myofibrils

During Developing Muscle Hypertrophy

- **Increasing size of the myofibrils**
- **The enzyme systems**
- **Provide energy also ➔ Increase**

During Developing Muscle Hypertrophy

- **Enzymes for Glycolysis**
- **Rapid supply of energy**
- **During short-term forceful contraction**

Muscle Unused ➔ For long Time

- Muscle Atrophy ➔
- Rate of decay of the contractile proteins
- Occurs more rapidly
- Than the rate of synthesis of the replacement

Hyperplasia of muscle fibers

- Under Rare conditions
- Of extreme muscle force generation
- Actual numbers of muscle fibers
- Observed to increase

Hyperplasia of muscle fibers

- **But by only a few percentage**
- **In addition to the fiber hypertrophy process**

Hyperplasia of muscle fibers

- **The increase in the fiber numbers**
- ***Fiber hyperplasia***
- ***Mechanism*** ➔
- ***Is linear splitting of previously enlarged fibers***

Effects of Muscle Denervation

- When muscle ➔ Loses its nerve supply
- No longer receives
- The contractile signals
- Required to maintain normal muscle size

Effects of Muscle Denervation

- **Atrophy begins**
- **Almost immediately**

Effects of Muscle Denervation

- **After 2 months**
- **Degenerative changes ➔ Also begin to appear**
- **In muscle fibers themselves**

Effects of Muscle Denervation

- If nerve supply ➔ Grows back rapidly to muscle
- Full return of the
- Function usually ➔ In 3 months

Effects of Muscle Denervation

- From 3 months onwards
- Capability of the functional return
- Becomes less and less
- With no return of function after 1 to 2 years

Final stage of Denervation Atrophy

- Most of muscle fibers
- Destroyed
- Replaced by ➔ Fibrous
- Fatty tissue

Muscle fibers do Remain

- Composed of a long cell membrane
- With a line-up of muscle cell nuclei
- But with no contractile properties
- No capability of rgenerative myofibrils if nerve regrows

Contracture

- Fibrous tissue that ➔ replaces the muscle fibers
- During denervation atrophy
- Tend to continue shortening for many months

Physical therapy given ➔To Prevent

- Keep atrophying muscles
- From developing
- Debilitating and disfiguring contracture

Achieved by ➔

- **Daily stretching of the muscle**
- **Or use of the appliances**
- **That keep the muscles stretched during atrophying process**

Denervation Hypersensitivity

- **Destruction of nerve supply ➔Muscle Atrophy**
- **Abnormal excitability of the muscles**
- ***Increased sensitivity to circulating Acetylcholine***

Fibrillations ➔LMN lesion

- *Fine*
- *Irregular* **contraction**
- **Of individual fibers**
- **Fibrillations** ***not visible grossly***

Fibrillations ➔LMN lesion

- **If motor nerve regenerates**
- **Fibrillations ➔ *Disappears***

Fasciculations

- *Jerky Visible contraction* **of** *group of muscles fibers*
- **Occurs as a result of the** *pathological discharge*
- **Of** *spinal motor neurons*

Macromotor Units in Poliomyelitis

- When some
- But
- Not all nerve fibers to a muscles are destroyed
- As in poliomyelitis

Macromotor Units in Poliomyelitis

- Remaining nerve fibers ➔ Sprouts
- Forth ➔ New axons to form many new branches
- Innervate many of the paralyzed muscle fibers

Macromotor Units in Poliomyelitis

- Macromotor units ➔
- Contains ➔ Five times the normal numbers
- of the muscle fibers
- For each motorneuron in spinal cord

Macromotor Units in Poliomyelitis

- Macromotor units ➔
- Decreases ➔ The fineness of the Control
- But allows ➔ The muscle to regain the strength

Fiber Types

- **Skeletal muscles ➔ Heterogenous tissue**
- **Made up of fibers ➔ Vary in the**
- **MyosinATPase Activity**
- **Contractile Speed and properties**

Isoforms of MHCs

- **Myosin Heavy Chains ➔Ten (10)**
- **Each of 2 types of Light chain ➔ Isoforms**

Isoforms of Actin

- **Only One (1) Isoform → of G Actin**
- **Multiple Isoforms →Tropomyosin**
- **Multiple isoforms →Troponin**

Fiber Types : Muscle Contains ➔

- **Three (3) Fibers Types:**

1. **Type I (SO ➔ Slow Oxidative)**
2. **Type II A (FOG ➔Fast-Oxidative-Glycolytic)**
3. **Type II B (FG ➔ Fast Glycolytic)**

Other Names

Type I (SO)	Type II A (FOG)	Type II B (FG)
• Slow Oxidative	• Fast Oxidative Glycolytic	• Fast Glycolytic

Color

Type I (SO)	Type II A (FOG)	Type II B (FG)
• Red	• Red	• White

Myosin ATPase Activity

Type I (SO)	Type II A (FOG)	Type II B (FG)
• Slow	• Fast	• Fast

Ca^{2+} Pumping Capacity of SER

Type I (SO)	Type II A (FOG)	Type II B (FG)
• Moderate	• High	• High

Diameter

Type I (SO)	Type II A (FOG)	Type II B (FG)
• Small	• Large	• Large

Glycolytic capacity

Type I (SO)	Type II A (FOG)	Type II B (FG)
• Moderate	• High	• High

Oxidative capacity

Type I (SO)	Type II A (FOG)	Type II B (FG)
• High	• Moderate	• High

Associated Motor Unit Type

Type I (SO)	Type II A (FOG)	Type II B (FG)
• Slow (S)	• Fast Resistant to Fatigue (FR)	• Fast Fatigable (FF)

Membrane Potential

Type I (SO)	Type II A (FOG)	Type II B (FG)
• - 90mV	• -90mV	• -90mV

Adjustment of Muscle Length

- When muscles are stretched to
- Greater than normal length
- New sarcomeres added at the ends of the muscle fibers where they are attached to the tendons

Adjustment of Muscle Length

- New sarcomeres can be added
- As rapidly
- As several per minute
- Cause another type of hypertrophy

Adjustment of Muscle Length

- Muscle remains shortened
- Continually to less than its normal length
- Sarcomere at the ends of the muscle fibers
- Disappears approx equally as rapidly