Force of Muscle Contraction
• The force of contraction is affected by:
  • Number of muscle fibers stimulated (recruitment)
  • Relative size of the fibers—hypertrophy of cells increases strength

Force of Muscle Contraction
• The force of contraction is affected by:
  • Frequency of stimulation—frequency allows time for more effective transfer of tension to noncontractile components
  • Length-tension relationship—muscles contract most strongly when muscle fibers are 80–120% of their normal resting length

Velocity and Duration of Contraction
Influenced by:
1. Muscle fiber type
2. Load
3. Recruitment

Muscle Fiber Type
Classified according to two characteristics:
1. Speed of contraction: slow or fast, according to:
   • Speed at which myosin ATPases split ATP
   • Pattern of electrical activity of the motor neurons

Muscle Fiber Type
2. Metabolic pathways for ATP synthesis:
   • Oxidative fibers—use aerobic pathways
   • Glycolytic fibers—use anaerobic glycolysis

Muscle Fiber Type
Three types:
• Slow oxidative fibers
• Fast oxidative fibers
• Fast glycolytic fibers
Influence of Load
\(\uparrow\) load \(\rightarrow\) \(\uparrow\) latent period, \(\downarrow\) contraction, and \(\downarrow\) duration of contraction

Influence of Recruitment
Recruitment \(\rightarrow\) faster contraction and \(\uparrow\) duration of contraction

Effects of Exercise
Aerobic (endurance) exercise:
• Leads to increased:
  • Muscle capillaries
  • Number of mitochondria
  • Myoglobin synthesis
• Results in greater endurance, strength, and resistance to fatigue
• May convert fast glycolytic fibers into fast oxidative fibers

Effects of Resistance Exercise
• Resistance exercise (typically anaerobic) results in:
  • Muscle hypertrophy (due to increase in fiber size)
  • Increased mitochondria, myofilaments, glycogen stores, and connective tissue

The Overload Principle
• Forcing a muscle to work hard promotes increased muscle strength and endurance
• Muscles adapt to increased demands
• Muscles must be overloaded to produce further gains

Smooth Muscle
• Found in walls of most hollow organs (except heart)
• Usually in two layers (longitudinal and circular)

Peristalsis
• Alternating contractions and relaxations of smooth muscle layers that mix and squeeze substances through the lumen of hollow organs
  • Longitudinal layer contracts; organ dilates and shortens
  • Circular layer contracts; organ constricts and elongates

Microscopic Structure
Spindle-shaped fibers: thin and short compared with skeletal muscle fibers
Connective tissue: endomysium only
SR: less developed than in skeletal muscle
Pouchlike infoldings (caveolae) of sarcolemma sequester Ca$^{2+}$
No sarcomeres, myofibrils, or T tubules

**Innervation of Smooth Muscle**
- Autonomic nerve fibers innervate smooth muscle at diffuse junctions
- Varicosities (bulbous swellings) of nerve fibers store and release neurotransmitters

**Myofilaments in Smooth Muscle**
- Ratio of thick to thin filaments (1:13) is much lower than in skeletal muscle (1:2)
- Thick filaments have heads along their entire length
- No troponin complex; protein calmodulin binds Ca$^{2+}$

**Myofilaments in Smooth Muscle**
- Myofilaments are spirally arranged, causing smooth muscle to contract in a corkscrew manner
- Dense bodies: proteins that anchor noncontractile intermediate filaments to sarcolemma at regular intervals

**Contraction of Smooth Muscle**
- Slow, synchronized contractions
- Cells are electrically coupled by gap junctions
- Some cells are self-excitational (depolarize without external stimuli); act as pacemakers for sheets of muscle
- Rate and intensity of contraction may be modified by neural and chemical stimuli

**Contraction of Smooth Muscle**
- Sliding filament mechanism
- Final trigger is ↑ intracellular Ca$^{2+}$
- Ca$^{2+}$ is obtained from the SR and extracellular space

**Role of Calcium Ions**
• \( \text{Ca}^{2+} \) binds to and activates calmodulin
• Activated calmodulin activates myosin (light chain) kinase
• Activated kinase phosphorylates and activates myosin
• Cross bridges interact with actin

**Contraction of Smooth Muscle**
• Very energy efficient (slow ATPases)
• Myofilaments may maintain a latch state for prolonged contractions

Relaxation requires:
• \( \text{Ca}^{2+} \) detachment from calmodulin
• Active transport of \( \text{Ca}^{2+} \) into SR and ECF
• Dephosphorylation of myosin to reduce myosin ATPase activity

**Regulation of Contraction**

*Neural regulation:*
• Neurotransmitter binding \( \rightarrow \) \( [\text{Ca}^{2+}] \) in sarcoplasm; either graded (local) potential or action potential
• Response depends on neurotransmitter released and type of receptor molecules

*Hormones and local chemicals:*
• May bind to G protein–linked receptors
• May either enhance or inhibit \( \text{Ca}^{2+} \) entry

**Special Features of Smooth Muscle Contraction**

*Stress-relaxation response:*
• Responds to stretch only briefly, then adapts to new length
• Retains ability to contract on demand
• Enables organs such as the stomach and bladder to temporarily store contents

*Length and tension changes:*
• Can contract when between half and twice its resting length

**Hyperplasia:**
• Smooth muscle cells can divide and increase their numbers
• Example:
• estrogen effects on uterus at puberty and during pregnancy

**Types of Smooth Muscle**

Single-unit (visceral) smooth muscle:
- Sheets contract rhythmically as a unit (gap junctions)
- Often exhibit spontaneous action potentials
- Arranged in opposing sheets and exhibit stress-relaxation response

**Types of Smooth Muscle: Multiunit**

Multiunit smooth muscle:
- Located in large airways, large arteries, arrector pili muscles, and iris of eye
- Gap junctions are rare
- Arranged in motor units
- Graded contractions occur in response to neural stimuli

**Developmental Aspects**

- All muscle tissues develop from embryonic myoblasts
- Multinucleated skeletal muscle cells form by fusion
- Growth factor agrin stimulates clustering of ACh receptors at neuromuscular junctions
- Cardiac and smooth muscle myoblasts develop gap junctions

**Developmental Aspects**

- Cardiac and skeletal muscle become amitotic, but can lengthen and thicken
- Myoblast-like skeletal muscle satellite cells have limited regenerative ability
- Injured heart muscle is mostly replaced by connective tissue
- Smooth muscle regenerates throughout life

**Developmental Aspects**

- Muscular development reflects neuromuscular coordination
- Development occurs head to toe, and proximal to distal
- Peak natural neural control occurs by midadolescence
- Athletics and training can improve neuromuscular control

**Developmental Aspects**

- Female skeletal muscle makes up 36% of body mass
• Male skeletal muscle makes up 42% of body mass, primarily due to testosterone
• Body strength per unit muscle mass is the same in both sexes

Developmental Aspects
• With age, connective tissue increases and muscle fibers decrease
• By age 30, loss of muscle mass (sarcopenia) begins
• Regular exercise reverses sarcopenia
• Atherosclerosis may block distal arteries, leading to intermittent claudication and severe pain in leg muscles

Muscular Dystrophy
• Group of inherited muscle-destroying diseases
• Muscles enlarge due to fat and connective tissue deposits
• Muscle fibers atrophy

Muscular Dystrophy
Duchenne muscular dystrophy (DMD):
• Most common and severe type
• Inherited, sex-linked, carried by females and expressed in males (1/3500) as lack of dystrophin
• Victims become clumsy and fall frequently; usually die of respiratory failure in their 20s
• No cure, but viral gene therapy or infusion of stem cells with correct dystrophin genes show promise