Movement is a sign of life. It enables voluntary locomotion, reflexive reactions for the purposes of protection (such as in the event of flight or a fall) as well as the complex functioning of our metabolism. The many different tasks that are accomplished by the muscle physiology provide a glimpse into the complexity of movement. The following article provides - after a brief review of the relevant muscle structures - an exact overview of the physiologic sequence of forms, the sliding filament theory, and the electrophysiologic and energetic processes during a contraction. Furthermore, the topic of muscle strength, with regard to bodily adjustment to exertion and relaxation is reviewed.

Muscle Types

Muscles and their cells are specialized according to their particular functions. Thus, there are 3 types of muscle:

- **Skeletal muscles**
- **Smooth muscles**
- **Cardiac muscle**
Teleokinetic and ereismatic biomotor functions in humans are facilitated by approx. 400 skeletal muscles, which make up about 40% of the body mass in an average person. This muscle type is innervated by the somatic nervous system and is, therefore, normally voluntarily contractible. Another important feature is that the skeletal muscles can rapidly exert high force but are easily fatigued.

The hierarchical structure of skeletal muscles is illustrated as:

- Total muscle → muscle fascicle (muscle fiber bundle) → muscle fiber (muscle cell) → myofibrils in sarcoplasm (cytoplasm of the muscle cell) → myofilaments (actin and myosin = proteins, which affect the contraction of a muscle cell)

Upon closer inspection, a regular structure can be detected inside a myofibril. It consists of many lined-up sarcomeres, which describe the section between 2 z-discs.


Sarcoplasmic reticulum surrounds myofibrils. These tubules are aligned longitudinal to
the myofibrils, hence the name longitudinal tubuli, longitudinal system, or L-system. **Terminal cisternae**, which transverse muscle fibers, serve as $\text{Ca}^{2+}$ stores.

**Classification of muscle fibers:**

The type of muscle fiber determines how much power a muscle can exert and how enduring it is. Muscle fibers can be classified by functional criteria or by the content of myoglobin.

Considering the myoglobin content, muscles can be classified as:

<table>
<thead>
<tr>
<th>White or pale muscles</th>
<th>Red muscles</th>
</tr>
</thead>
<tbody>
<tr>
<td>Contain little myoglobin</td>
<td>Contain much myoglobin</td>
</tr>
<tr>
<td>Predominantly anaerobic glycolysis</td>
<td>Predominantly aerobic glycolysis</td>
</tr>
<tr>
<td>Fast and forceful contraction</td>
<td>Slow and less forceful shortening of the muscle</td>
</tr>
<tr>
<td>Rapidly tiring</td>
<td>High endurance</td>
</tr>
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</table>

Based on functional criteria, the classification of muscle fibers is as follows:

- **S fibers** (‘slow’), also called Group I fibers (similar to red muscle fibers)
- **FR fibers** (‘fast, fatigue-resistant’) also called Group IIA fibers (classified between red and white muscles, i.e., medium-power exertion, aerobic, and anaerobic metabolism, etc.)
- **FF fibers** (‘fast, fast fatigable’) or Group IIB fibers (corresponds to white muscles)

**Skeletal muscles can also be classified into:**

**Type I fibers:**

These are the slow-twitch fibers or slowly contracting fibers. They are also known as red and slow fibers.

**Type II fibers:**

These are fast contracting fibers that comprise 2 forms with different resistances to fatigue.

- Type IIa

They are red fast oxidative fibers with medium resistance to fatigue.
Type IIₐ

These are the white fibers or fast glycolytic fibers. They have the least resistance to fatigue.

Smooth muscles

Smooth muscles, which are located in all inner organs, are innervated through the autonomic nervous system and are, therefore, not voluntarily contractible. They regulate blood pressure inside the vessels, cyclic changes from the contraction and relaxation of the gastrointestinal tract, and other internal processes.

Smooth muscle cells are much smaller than skeletal muscle cells. The contractile elements are actin and myosin, which are organized in a net-like structure, forming a smooth surface as can be observed through an optical microscope.

The dense bodies and dense areas correspond to the z-plates of skeletal muscles and contain the actin filaments connected to the sarcolemma (cell membrane of the muscle cell).

The power of the myosin filaments is transferred to the sarcolemma by virtue of the intermediate filaments (for example, desmin, vimentin, and filamin) being connected with the dense bodies and dense areas. The endoplasmic reticulum serves as a Ca²⁺ store, as in the skeletal musculature. However, in this case, the Ca²⁺-binding protein is calmodulin instead of troponin C.

Cardiac muscles

Cardiac musculature contains elements of both smooth and skeletal muscles but is differentiated by its special muscle tissue. Cardiac muscle tissue must be extremely enduring – an entire lifetime – at low-power exertion.
The following are important histological features of cardiac musculature:

- Cardiac muscle cells are arranged in a parallel fashion
- **Intercalated discs** (also called glossy stripes) are arranged in a stair-step fashion and connect the muscle cells with one another
- Transmission of cardiac muscle stimuli occurs via **gap junctions**
- Mechanical stability is facilitated by **maculae adherens** and **fasciae adherens**
- Specialized cardiac muscles facilitate the myogenic, autonomous stimulus regulation
- Myofibrillar structure of sarcomeres, similar to skeletal muscles

**Musculature Contraction – Basics and Process**

Due to the existence of different types of musculature, there is a variety of different contraction processes, which are more or less similar, depending on the type. This is why the following section individually examines these processes.

**Skeletal Muscle Contraction – Sliding Filament Theory**

In order to understand the contraction of striated musculature - the **sliding filament theory** - one must be familiar with the following terminology:

- **Motor unit**: Contains muscle fibers of a common branch, which are innervated by a single α-motor neuron (source: anterior horn of the spinal cord). The action potential of a single α-motor neuron is responsible for the simultaneous contraction of all muscle cells of a motor unit.
- **Electromechanical coupling**: Describes the process of transforming an electrical impulse into a mechanical muscle contraction.

**Sliding filament theory**: Describes the interaction of the actin filaments with the myosin filaments, which leads to mechanical muscle contraction. In this mechanism, there is no change in the length of myofilaments during a contraction but only a shortening of the sarcomeres through a sliding-by of the myofilaments. This causes the shortening of the muscle as a whole.
The sliding process is based on the so-called **cross-bridge cycle**, which functions as follows:

1. Due to the ATPase activity of the **myosin head**, it is capable of independently splitting ATP. Thus, the myosin head is activated, and the energy gained from the ATP split can be ‘stored’ there.
2. The activated myosin head accumulates perpendicularly at the connection point of the actin filament, which builds a **cross-bridge**. This short-term chemical bond between the actin and myosin molecules can exist since the protein troponin C is bound to the calcium ions released by the intracellular stores (sarcoplasmic reticulum).
3. The myosin head tilts by 45°, which pulls the bound actin filament towards the **middle of the sarcomere**. The sarcomere shortens.
4. Following the positional change of the myosin head, its conformation will be changed so that a new ATP molecule can be bound. When this happens, the myosin head disconnects from the actin filament, and a new cross-bridge cycle can begin. This cycle is also called the **oar paddle**.

Muscle contraction ends by lowering the intracellular Ca$^{2+}$ level due to the **cessation of the action potential**.
Electromechanical observation of the contraction process of skeletal muscles

1. Transmission of the action potential from the central nervous system via motor neurons to the motor endplate (= transmission from the motor neuron to the muscle cell).
2. Dispersion of the transmitter acetylcholine, which occupies the postsynaptic n-cholinoreceptors.
3. Na⁺-influx via the opening of non-selective ion channels → depolarization of the sarcolemma (= endplate potential).
4. Triggering of an action potential through several large endplate potentials → diffusion to the entire sarcolemma as well as along the transverse tubules.
5. Activation of tension and dihydropyridine-sensitive Ca²⁺ channel proteins → marked stimulation and opening of ryanodine-sensitive Ca²⁺ channels.
6. Markedly increasing the release of Ca²⁺ ions → suddenly increasing Ca²⁺
concentration around the myofibrils, from $10^{-7}$ to $10^{-5}$.

7. Contraction trigger.

**Note:** A voluntarily triggered contraction stems from the cerebral cortex whose neurite transmits the electrical impulse to the anterior horn of the spinal cord.

**Energetic observation of the bodily work and effort during skeletal muscle contraction**

Depending on which kind of physical strain (intensity or duration) a body is exposed to, it will utilize different sources of energy to meet the demand:

- **Short, intensive effort (e.g., sprinting):** ATP synthesis from existing creatine phosphate and anaerobic glycolysis of glucose from muscle glycogen with lactate release → pH level falls for a short time from 7.4 to 7.2.
- **Somewhat longer lasting effort (minutes):** Glucose breakdown from muscle glycogen through aerobic glycolysis and ATP recovery through oxidative phosphorylation.
- **Bodily exertion for hours:** Glucose breakdown from muscle and liver glycogen and triacylglycerol (lipolysis and β-oxidation).

**Contraction forms of the skeletal musculature**

- **Isotonic muscle contraction:** muscle tension remains the same
- **Isometric muscle contraction:** muscle length remains the same
- **Auxotonic muscle contraction:** muscle tension and length change in parallel
- **Support muscle contraction:** isometric muscle contraction immediately followed by isotonic contraction
- **Impact muscle contraction:** isotonic muscle contraction followed by isometric muscle contraction

**Development and regulation of muscle power during skeletal muscle contraction**

The amount of force a muscle can exert at the end, and the detailed gradations and its application, depends on the following:

- **Amount of mobilized motor units:** The higher the amount of recruited motor units, the higher the deployed force.
- **Frequency of action potentials:** The more action potentials arrive within a short period of time at the muscle fiber, the more intensive the power exertion and the shortening of the muscle. In superposition (many action potentials within a short period of time), maximal contraction – a so-called *tetanic contraction* – will occur.
- **Shortening velocity:** The faster a muscle shortens, the higher its deployment of force (Hill’s tension velocity relation).
- **Pre-tension:** The initial tension influences the deployed force in isometric or isotonic contractions.
Muscle work and bodily adjustment to exertion during contraction of skeletal musculature

Work in the physical sense is accomplished when a distance is crossed. Muscle work can be computed by considering the product of muscle shortening and load. This means that mechanically speaking, no work is performed during an isometric contraction. The invested energy is simply transformed into heat.

The capability of the human body to adapt to physical exertion or inexertion is extraordinary. For instance, in a phase of inactivity (perhaps due to immobilization) atrophic changes can be observed within a short period of time. Equally, the body can adapt muscicularly to higher exertion. The mechanisms of muscle performance increase depending on its type are as follows:

- **Red muscle fibers**: Increase of the myoglobin content, the number of mitochondria, and capillary formation.
- **White muscle fibers**: Increase in the number of myofibrils and glycogen storage → Increase of muscle diameter (**muscle hypertrophy**).

Length-tension curve for skeletal muscle contraction

Muscle elasticity is facilitated by the collagen layer of the sarcolemma and connective tissue between the muscle fibers. When a resting muscle is engaged, its tension will rise. That means, the longer a muscle is in a state of strain, the more force has to be exerted. This interaction is called the **length-tension curve**.

**Clinical feature**: In the case of excessive strain, muscle fibers are destroyed, which is colloquially known as a muscle tear.

Smooth Muscle Contraction

**Differentiation of the working muscle cell types during smooth muscle contraction**

Different contraction behaviors determine the classification into **single-** and **multi-unit smooth muscles**. The former is a muscle unit, which is coupled by **gap junctions** and, therefore, works as one coherent functional unit, i.e., the muscles contract together. This muscle type is found predominantly along organ walls and blood vessels.

For multi-unit smooth muscles, the muscle cells are capable of contracting independently of one another due to the predominantly autonomic innervation. Since there are relatively few gap junctions, the electric coupling occurs through a basal membrane-like layer. Furthermore, the neurotransmitters are distributed by so-called **varicosities**. This type of smooth muscle can be found, among others, in the ciliary muscle of the **iris** and the **arrector pili muscle**.

**Special features of smooth muscle contraction**

- **The capability of spontaneous, autonomous contraction**: Due to the innervation through the autonomic nervous system (involuntary processes), existing automatic processes can be adjusted according to the situation.
- The predominant share of the incoming Ca$^{2+}$ ions stems from the extracellular...
Smooth muscle contraction step-by-step:

1. Ca\(^{2+}\) binds to calmodulin
2. Activation of myosin-light-chain-kinase (MLCK) through the Ca\(^{2+}\)-calmodulin complex
3. Accretion of the complex to caldesmon → activation of a certain enzyme to facilitate phosphorylation
4. Phosphorylation of the light chain of the myosin head through MLCK, using ATP
5. Contraction via cross-bridge formation
6. The secession of the remaining phosphate from the light chain of the myosin head through myosin-light-chain phosphatase (MLCP) → dissolution of the actin-myosin-bond

Cardiac Muscle Contraction

Cardiac muscle contraction is facilitated by specialized cardiac muscle cells of the electrical impulse formation system of the heart. The electrical impulses are transmitted via gap junctions to cardiac muscle cells and, later on, to the working myocardium. The specialized cardiac muscle cells can be divided into the electrical impulse formation system and conduction system. The system has the following components:

- Sinus and AV nodes
- Bundle of His
- Bundle branches also called tawara branches
- Purkinje fibers
- **Working myocardium:** These are cells, which are connected via gap junctions and form a syncytium, where the conduction of electrical impulses can occur rapidly. Both syncytia are isolated from each other through the annulus fibrosus (valve connective tissue). The working myocardium ‘takes care’ of mechanical work.

From stimulation to cardiac muscle contraction

The stimulation stemming from the sinus node, i.e., the electrical impulse formation system, is transmitted from the syncytium to the AV node. This serves simultaneously as a ‘backup electrical impulse formation system’. From there, the impulse is passed on from the bundle of His to the bundle branches. Their function is to rapidly spread the impulse to the apex of the heart and papillary muscles of the heart valves.

**Note:** Stimulation of both atria → conduction to papillary muscles → impulse transmission
to the Purkinje fibers (final branch of the impulse conduction system); in this phase, the atria are already relaxed.

Electrophysiological observation of cardiac muscle contraction

Since the actual process of contraction occurs in the working myocardium, we will discuss the process of development and cessation of an action potential in this section. First, it is important to note that localized processes of the action potential are similar to others as they pass through partially similar stages.

The action potential comprises 3 phases:

1. **Notch phase:** Caused by the activation of the Na\(^+\) channels with a membrane potential of about -60 mV. A Na\(^+\) influx is triggered, which leads to the depolarization of the sarcolemma and increases membrane potential to about +30 mV (overshoot). This, in turn, leads to the deactivation of the Na\(^+\) channels.

2. **Plateau phase:** The depolarization leads to a temporarily off-set activation of the Ca\(^{2+}\) channels, which causes a decreased membrane potential of about -30 mV. This leads to a Ca\(^{2+}\) influx from the extracellular space, which enables the electromechanical coupling. In this phase, the action potential of the chamber myocardium is different from that of the nerve and muscle cells since there is a temporary equilibrium between the depolarizing and repolarizing currents.

3. **Rapid repolarization phase:** Through the activation of tension-dependent K\(^+\) channels, the conductivity of Na\(^+\) and Ca\(^{2+}\) decreases more rapidly. Furthermore, the K\(^+\) channels serve to stabilize the membrane potential at rest.

The action potential of the myocardium lasts about 300–450 ms, depending on the exact location of measurement and heart frequency.

References


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