MUSCLE CONTRACTION

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INTRODUCTION

Muscle contraction is the activation of tension-generating sites within muscle fibers. In physiology, muscle contraction does not necessarily mean muscle shortening because muscle tension can be produced without changes in muscle length, such as when holding a heavy book or a dumbbell at the same position.The termination of muscle contraction is followed by muscle relaxation, which is a return of the muscle fibers to their low tension-generating state.

Muscle contractions can be described based on two variables:

- Length
- Tension

A muscle contraction is described as isometric if the muscle tension changes but the muscle length remains the same. In contrast, a muscle contraction is isotonic if muscle tension remains the same throughout the contraction. If the muscle length shortens, the contraction is concentric; if the muscle length lengthens, the contraction is eccentric. In natural movements that underlie locomotor activity, muscle contractions are multifaceted as they are able to produce changes in length and tension in a time-varying manner, Therefore, neither length nor tension is likely to remain the same in muscles that contract during locomotor activity.

Muscle contraction throughout the human body can be broken down based on muscle subtype specialization to accomplish its dynamic function. In general, muscle fibers are classified into 2 large categories.

Striated muscle fibers

- Cardiac muscle tissue (involuntary)
- Skeletal muscle tissue (voluntary)

Smooth muscle fibers

- Located in the walls of the hollow, visceral organs (involuntary)

Cardiac and skeletal muscle are types of striated muscle, although the former is under involuntary controlled by the body's autonomic nervous system (ANS). In contrast, skeletal muscle is under voluntary control designed to carry out its dynamic physiologic function. Smooth muscle, which is found in blood vessels, the gastrointestinal (GI) tract, bronchioles, the uterus, and the bladder, is under involuntary control by reflexes and the body's autonomic nervous system.

ORGAN SYSTEMS INVOLVED

The musculoskeletal system is complex and requires voluntary control which manifests in the dynamic function via striated skeletal muscle. This helps people perform most of their cognitive movements, for example, walking, talking, throwing a ball, or even sitting upright in a chair. Smooth muscle is involved in many organ systems such as the blood vessels of the cardiovascular system, the GI tract, the respiratory tract, and the reproductive tract.

MECHANISM

Striated Muscle

To understand the mechanism by which striated muscle contracts, it is first important to understand its structure. The striated muscles in our body are made up of many individual muscle fibers. Inside these muscle fibers are smaller units called myofibrils which are made of parallel thin and thick filaments. These filaments are arranged longitudinally in small units known as sarcomeres. These repeating sarcomeres give the muscle a striated appearance under microscopy, which accounts for its name.

The thick filaments are made from the protein myosin, which has 2 heavy chains, and 2 pairs of light chains (be careful not to confuse the heavy and light chains of myosin with the thin and thick filaments of myofibrils). At the tail of the thick filament, the 2 heavy chains are intertwined in a helical formation. At the other end of the thick filament, each heavy chain is paired with two light chains giving rise to

2 heads. The myosin heads have an actin-binding site which helps them attach to the thin filaments.

The thin filaments are composed of actin, tropomyosin, and troponin. Actin is a globular protein which combines with other actin globules to form two intertwined strands with a positive and negative end. The double-stranded actin filaments are covered by tropomyosin, which blocks the interaction between myosin and actin when the muscle is inactive. Troponin is composed of troponins I, T, and C, and it is located along the actin filaments next to tropomyosin.

The complex process leading to muscle contraction, called excitation-contraction coupling, begins when an action potential causes depolarization in the myocyte membrane. The depolarization is spread via the transverse (T) tubules, which are invaginations of the muscle cell membrane – these help to spread depolarization signals to the entire muscle fiber. Depolarization of the T tubules causes a conformational change in the dihydropyridine receptors, which causes the opening of nearby ryanodine receptors on the sarcoplasmic reticulum (SR), the storage site for calcium within muscle cells. When calcium is released from the SR, it binds to troponin C. This causes a conformation change, which shifts tropomyosin, allowing the myosin heads to attach to the actin filaments creating what is known as a crossbridge. Then cross-bridge cycling begins. When ATP binds to an ATP binding domain on the myosin head, it causes the myosin to dissociate from the actin, breaking the cross-bridge. ATP is then hydrolyzed into ADP and P, which causes the myosin heads to change conformation and move toward the positive end of the actin, cocking the myosin head. The phosphate is released, and the ADP bound myosin binds to a new location on the actin filament. ADP is then released which causes the myosin to return to its original position, pulling on the actin filament when doing so causing the sarcomere (and therefore the muscle fiber) to contract.These cycles continue until calcium levels in the myocyte fall, causing tropomyosin to again cover the myosin binding sites of the actin filaments.

Smooth Muscle In smooth muscle, the same thin and thick filaments discussed in striated muscle are present. However, in smooth muscle, they are not organized into sarcomeres. Additionally, the mechanism of excitation-contraction coupling in smooth muscle differs as well. The first difference is in the mechanism that Ca enters the cell. There are 3 mechanisms that increase intracellular concentration.

The first is that voltage-gated Ca channels are activated by membrane depolarization, allowing Ca to enter the cell. The second mechanism is that hormones or neurotransmitters can open ligand-gated channels on the cell membrane. Last, hormones and neurotransmitters such as norepinephrine and angiotensin II, can, via the phospholipase-C pathway, cause an increase in intracellular inositol triphosphate IP3. IP3 can bind to receptors on the SR and cause Ca to be released.[3] Once Ca is released, instead of binding to troponin C as it did in striated muscle, it binds to a protein called calmodulin. Calmodulin then activates myosin light chain kinase, which, as the name suggests, phosphorylates myosin light chain. The phosphorylated myosin light chain has ATPase activity which hydrolyzes ATP, increasing its affinity to actin. The myosin can then readily bind actin. From this point, the cross-bridge cycling is the same as it was in striated muscle. As long as there is Ca2+ bound to calmodulin, and the MLCK is still phosphorylated, the smooth muscle will remain contracted. This allows for prolonged periods of vasoconstriction in blood vessels.

MUSCLE CRAMPS

Muscle cramps result in continuous, involuntary, painful, and localized contraction of an entire muscle group, individual single muscle, or select muscle fibers. Generally, the cramp can last from minutes to a few seconds for idiopathic or known causes with healthy subjects or in the presence of diseases. Palpating the muscle area of the cramp will present a knot.

Exercise-associated muscle cramps are the most frequent condition requiring medical/therapeutic intervention during sports. The specific etiology is not well understood, and possible causes depend on the physiological or pathological situation in which the cramps appear. It is important to note that a painful contraction that is limited to a specific area does not mean that the cause of the onset of the cramp is necessarily local.

In certain clinical scenarios, the underlying etiology may be related to persistent, spastic muscle contractions that significantly can impact human function. A common example of this condition is manifested in the sternocleidomastoid muscle. Clinically, this is recognized in congenital torticollis and/or spasmodic torticollis.

Other relevant conditions in this realm include, but are not limited to the following:

- Exercise-induced and heat-related muscle cramping
- Piriformis syndrome
- Thoracic outlet syndrome (scalene muscle hypertrophy/spasticity)

Palsy

On the opposite end of the spectrum, various muscle palsies exist secondary to the long-term, downstream effects of various nerve conditions and neuropathies that can result in frankly flaccid conditions (which may be permanent or temporary). These conditions include, but are not limited to the following:

- Bell's palsy
- Guyon canal syndrome
- AIN syndrome or PIN syndrome
- Klumpke's palsy