MUSCLE PHYSIOLOGY

PART 4: MOVEMENT AND MUSCLE PROBLEMS

The previous three articles in this series explored the form and function of muscle and, in particular, skeletal muscle. This article outlines how the contraction of muscles (outlined in part three of this series) causes limb movements, and addresses some common muscle problems.

Muscle contraction would produce very little movement in the body were it not for the fact that muscles are attached to bones by tendons and can therefore pull on the bones and cause movement at the many joints of the body.

TENDONS

Tendons, which attach muscle to bone, are extremely strong because they are formed from the collagen fibres of connective tissue that surround the different muscle layers (see part one): the endomysium, perimysium and epimysium. Tendons ‘grow out of’ the muscle and gradually become more calcified as they approach bone. The collagen fibres lie longitudinally in the tendon and this, in addition to their poor arterial blood supply, makes them difficult to repair when severed. Tendons have great tensile strength and some elasticity but they are not compressible (they simply bend). Their main function is to transfer the force of tension from contracting muscles to bone but they also limit movement and provide support if the tendon crosses a joint (for example the patellar tendon).

LEVERS

The force, speed and range of movement are determined by where and how the muscle is connected to bone. A lever is a bar (in this case a bone) that rotates about a fulcrum (or joint) (McLaren, 2005). This means the direction and strength of the force produced by muscle can be altered. In this way, for example, contracting the biceps in the upper arm causes flexion at the elbow joint resulting in movement of the forearm (Fig 1), because the biceps tendon is attached to the bones of the forearm (Fig 2). The result is that a relatively small movement in the biceps produces a large movement of the forearm. However, joints and bones are served by more than one muscle and an integrated action is needed in all these muscles to produce controlled, fluid movements.

TYPES OF SKELETAL MUSCLE

Muscles can be classified as:
- Prime movers (or agonists), which initiate and maintain the desired movement, for example the biceps muscle, which produces elbow flexion (Fig 1);
- Antagonists, which produce the opposite movement and resist the actions of the prime movers, including ‘braking’ at the end of a movement range. The triceps muscle acts as the agonist to biceps, producing the opposite movement and limiting its scope of movement (Fig 1);
- Synergists, which help prime movers to produce the desired effect or cancel out unwanted movements (for example if a muscle crosses more than one joint or can produce more than one movement). An example of a synergist muscle is the supraspinatus, which assists the deltoid muscle at the beginning of abduction of the arm at the shoulder joint.

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Sometimes prime movers and antagonist muscles contract together to stabilise a joint. In the standing position, for example, both the quadriceps and hamstring muscles are contracted to stabilise the knee.

**AGE**

Muscle is the biggest protein mass in the body and, like all other protein, is lost with age while connective tissue and fat deposits increase. From our middle years to about 80 we lose about 30% of our muscle mass. As a result, muscle strength is reduced by about 50% by the age of 80 (Marieb, 2006).

Regular exercise and eating well, particularly taking supplements to increase intake of amino acids, can help to minimise muscle deterioration, but hormonal changes and changes in protein turnover also increase muscle loss and are less easily rectified.

As muscle mass is lost there is a reduction in the ability to do physical work and falls and muscle injuries become more likely.

**MUSCLE-RELATED PROBLEMS**

McLaren (2005) and Marieb (2006) provide more detailed discussions of the following muscle disorders.

**Muscle cramp**

This is a sustained involuntary spasm of a muscle that makes it tight and painful. Spasms usually occur following exercise or at night and may result from low blood sugar levels, ion imbalance, dehydration or spinal cord irritability (Marieb, 2006).

**Muscle strain**

Also called a ‘pulled muscle’, this is due to excessive stretching and possible tearing of a muscle. The muscle itself becomes painful (myalgia) and inflamed, and joint movement is also painful.

**Muscle fatigue**

This is a physiological inability of muscle to contract, which occurs when there is insufficient adenosine triphosphate (ATP) available. This is a nucleotide that serves as an energy source for many metabolic processes. Build-up of lactic acid after exercise also contributes to muscle fatigue.

**Myopathies**

Disorders that impair the function of skeletal muscle are known as myopathies. They can be caused by defects in the muscle itself or because of vascular, endocrine or metabolic disorders that affect muscles. For many muscle disorders there is no effective medical treatment to prevent muscle degeneration.

**Atrophy**

This refers to a decrease in muscle mass and can follow damage to the nerve supply to a muscle or result from long periods of inactivity (for example prolonged bedrest or following a cerebrovascular accident). Atrophy is a common feature of a number of diseases including diabetes mellitus and hypothyroidism. Muscle fibres shorten and their diameter decreases so that they are unable to function optimally.

**Hypertrophy**

Most muscle hypertrophy takes place in healthy people who undertake strength training that increases muscle mass, such as body builders. It can also occur, however, in some other conditions such as Duchenne dystrophy (where the muscle growth is due to excessive development of fat and fibrous tissue while the muscle cells themselves atrophy) and congenital myotonia (McLaren, 2005).

**Necrosis or muscle death**

Death of the muscle fibres is common in a number of muscle disorders such as muscular dystrophies and can also occur as a result of trauma, ischaemia, inflammation and infection of the muscle (McLaren, 2005).

**Rigor mortis**

The ‘stiffness of death’ occurs when stimulation of the muscle cells ceases. Some muscle fibres will have been in mid contraction at the moment of death and so the myosin heads are still attached to the actin filaments (see part three). Since there is no more ATP, the cross-bridges cannot release and remain stuck in the contracted position. This stiffness wears off some time after death as muscle proteins break down.

**CONCLUSION**

This series has explored the function of muscles, in particular skeletal muscle, which allows movement ranging from the blinking of the eyes to the coordinated movements of a trained athlete (Thibodeau and Patton, 2005). Our muscles function throughout our lives and without them, life would be impossible.

**REFERENCES**


form is Duchenne muscular dystrophy, which is caused by a missing gene on the X chromosome.

**Myaesthenia gravis**

This disease is characterised by muscle weakness, particularly in the face and throat (Marieb, 2006), which can progress to all four limbs and cause respiratory arrest due to involvement of the respiratory muscles. It occurs when the body’s immune system attacks muscle cells at the neuromuscular junction so that neurons are unable to stimulate the muscle cells adequately.