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SMOOTH MUSCLE CELLS:

These muscles have their characteristics, which may differ from those of skeletal muscles. In addition to that, smooth muscle cells may also differ from organ to organ in their organization, physical dimension responses to stimuli, and innervation. Generally, they are divided into multi-unit smooth muscle and single unit smooth muscle.

In multi-unit: each muscle fiber operates independently of all other fibers. In single unit muscles, their function needs cooperation of many muscle fibers to perform a function. Muscle fibers, in this type, are connected to each other by gap junctions to synchronize their contraction (functional syncytium).

Organization of contractile proteins in smooth muscle and mechanism of contraction:

The organization of contractile proteins is different from that in skeletal muscle. The actin filaments are attached to a dense structure inside the muscle known as **dense bodies**. These actin filaments radiate between dense bodies. In the midway between dense bodies, few myosin filaments are found where they overlap with actin filaments. The mechanism of contraction in smooth muscle cells also involves actin myosin interaction, but with different mechanism than that found in skeletal muscle. When smooth muscle is stimulated, it takes longer time than striated muscle to induce contraction (long latent period), the total contraction time is about 30 times more than that in skeletal muscle. These appear because of the slow attachment and detachment of contractile proteins, which results in slow cycling of cross bridges.

The mechanism of contraction in smooth muscle also involves an increase in Ca++ concentration, but the source could be different than in skeletal muscle. The source in skeletal muscle is only from the endoplasmic reticulum, which has high representation in skeletal muscle, while in smooth muscle the main source is extracellular and some contraction can be induced also by the release of Ca++ from intracellular stores (sarcoplasmic reticulum), which is moderately developed in smooth muscle (not well as in skeletal muscle).

The release of Ca++ into the cytosol induces activation of a protein known as **calmodulin** by forming calmodulin-Ca++ complex (4 Ca++ bind to one calmodulin). The activated calmodulin-Ca++ complex will induce activation of an enzyme called **myosin kinase**. This enzyme will **phosphorylate** the regulatory chain on **myosin head**. The phosphrylated myosin can interact with actin to induce contraction.

The relaxation of smooth muscle cells also involves a decrease in Ca++ concentration by increased activity of Ca++ pumps located at the plasma membrane and sarcoplasmic reticulum. In addition to that, the mechanism of relaxation also involves dephosphorylation of myosin heads by an enzyme called **myosin phosphatase**.

In some instances, the smooth muscle contracts and their contraction is sustained. This is known as **latch phenomenon**. This is due to much decrease in cycling frequency of cross bridges. Which is probably due to a decrease in myosin phosphatase activity, that results in a decreased dephosphorylation of myosin head (remain longer time activated). Little ATP molecules are consumed during this phenomenon.

Membrane potential and action potential in smooth muscle cells:

The resting membrane potential in smooth muscle is less negative than in skeletal muscle. It is about -60 to –50mV in smooth muscle. The characteristics of action potentials are also different in smooth muscle. Many types of action potential are found on smooth muscle fiber:

1. Spike potentials (have short duration): these can be elicited by external stimulus.

2. Action potential in with plateau: similar to the action potential that found in cardiac muscle. The onset is rapid as in spike potential, but repolarization takes longer time.

This type of action potential has importance in organs where longer contraction period is needed, such as in uterus. The longer action potential and the plateau in this type are due to activation of Ca++ channels. These channels are activated slowly, and their opening is maintained for longer time than Na+ channels.

3. Slow wave potentials: some smooth muscle cells are self-excitatory. This property is due to rhythmic variations in membrane potential that appear at muscle membrane. These rhythmic variations are known as **slow waves**. These waves are probably caused by changes in Na+ pump activity, or changes in conductance of ion channels. Slow wave are not action potentials and they cannot induce contraction in smooth muscle. When the peak of these slow waves rises above threshold, they can generate spike potentials, which result in contraction of smooth muscle.

Neural and hormonal control of smooth muscle contraction:

Muscle cells are innervated by autonomic fibers. The terminals of these fibers are ending diffusely between cells and not forming organized synapses, such as the motor end plate in skeletal muscle. The transmitter in autonomic fibers is found in varicosities of the fine terminals of nerve fibers. The released transmitters from these varicosities act on their receptors to induce activation or inhibition of the contraction in smooth muscle.

In addition to neural control, some smooth muscle membrane has receptors for hormones, neuropeptides, or other factors. These also control the activity of smooth muscle cells when their receptors on smooth muscle are activated.

The mechanism by which smooth muscle cells are activated may include activation of Ca++ channels at the sarcolemma or activation of phospholipase C. The later results in the formation of IP3 (inositol trisphosphate) and release of Ca++ from the sarcoplasmic reticulum. The mechanism of inhibition may include formation of cAMP or cGMP, which induces phosphorylation of some proteins that activate K+ channels or proteins involved in relaxation, or inhibition of proteins involved in contraction.