

muscle physiology lecture 15

molecular mechanisms of nociception

even if a patient is not currently in pain, it does not necessarily mean that their muscle function has returned to normal pre-pain levels even if their total force output may return to somewhat normal values. this is because there can be long term consequences of pain, especially if it is persistent. long periods of nociceptor activity, or pain sensation, can hypersensitize the nervous system so that even regular movements transmit messages of pain.

nociceptors - pain alerts you to possible threats and triggers a protective response. sometimes, pain outlives its role as a threat alarm and continues to trigger the protective response. pain to a certain extent is good.

dendrites - receiving station, incoming information goes here

cell body - houses the nucleus inside of it

axon - can be really long in the spine

nerve ending - place to send messages

ATP gets released from damaged tissue from the epithelial cells and activates the nociceptors. ATP can depolarize nociceptors. through repeated injury or excessive inflammatory conditions, chemical receptors can become more sensitive and increase in number. if there are more receptors, then there can be more binding, and more binding means the nerve will be triggered more. This means that the nerve will be more responsive to the same concentration of chemicals. calcium is able to affect gene expression and is also linked to long term changes. The influx of calcium and sodium is permitted by the NMDA receptor. consistent firing causes genes to turn on and create more receptors, which allows for firing to be even easier. neuronal sensitization is a sign of an increased excitability of afferent neurons, which results in pain signals being amplified since the nociceptors will fire with ease in response to innocuous stimuli.

allodynia - the nervous system's sensitization to pain, messages of pain are often transmitted in response to non-painful stimuli.