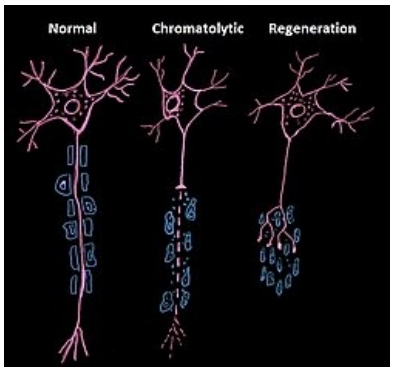
Denervation hypersensitivity

Principal

In massive **damage to a motor nerve** that innervates a skeletal muscle, such as when the axon is cut or crushed, results in complete **degeneration of the distal part of the axon**. Structures innervated directly by destroyed neurons subsequently develop extreme sensitivity to *acetylcholine*. There is no change for neighboring structures.

Mechanism of formation



Neuron damage and regeneration

This hypersensitivity is created in the area of postsynaptic structures as a **consequence of the absence of the transmitter *(acetylcholine)'*** *that was carried by the neuron before it was damaged.* These structures respond to the absence of signals by activating more receptors and synthesizing new ones. The area of the muscle membrane reacting to the presence of *acetylcholine* increases and extends outside the neuromuscular plate. Under physiological conditions, adult-type nicotinic receptors containing the ε subunit are present on this membrane. However in dennervated muscle, receptors with the fetal δ subunit begin to appear. After the regeneration of the neuron, these receptors disappear again and the sensitivity returns to normal.

We observe similar hypersensitivity *even in denervated smooth muscles and exocrine glands (except sweat glands)*.

Examples

Postganglionic sympathetic fibers leading to one pupil are cut in the experimental animal. After a certain time, noradrenaline is administered intravenously to the animal. This causes a wide dilation of the pupil on the denervated side, while a shorter and less pronounced response is observed on the side with retained innervation.

Links

Literature used

- GANONG, William F. *Přehled lékařské fyziologie.* 1. edition. Jinočany : H & H, 1995. ISBN 80-85787-36-9.