

Denervation hypersensitivity

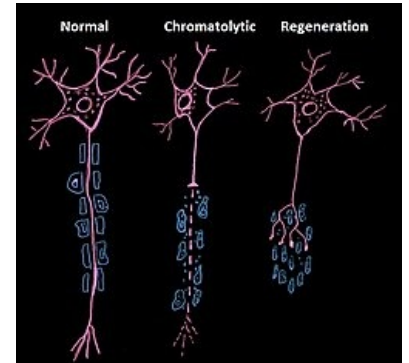
Principal

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

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 denervated 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)*.



Neuron damage and regeneration

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.