CHRONIC ELECTRICAL STIMULATION OF AXOTOMIZED NEURONS IN THE RABBIT. R. Orozco*, T. Gordon, L.A. Davis* and G. Goldsand*. Department of Pharmacology and Surgery, University of Alberta, Edmonton, Alberta, Canada, T6H 2H7.

When a mammalian peripheral nerve is severed, there is an initial decline, then stabilization in the diameter of the fibers which remain in continuity with the cell bodies (Davis et al. J. Physiol. 285: 543, 1978). This atrophy can be reversed if functional connections are remade with denervated end-organs, as a result of either re-establishing neural activity or by allowing the flow of trophic factors. Two observations, namely that the most rapid atrophy coincided with a fall in neuronal activity in the injured neurons, and that the silenced sensory axons atrophied relatively more than active motor axons (Hoffer et al. Brain Research, 178, 347-361, 1979) suggested to us that superimposing neural activity might reverse atrophy in axotomized neurones.

Extracellular cuff electrodes were bilaterally implanted around the sciatic nerve and the common peroneal or tibial nerve in 12 rabbit hindlimbs for chronic stimulation and monitoring of the compound action potential (CAP). Evoked CAP amplitude and latency were measured and used as a reference for nerve diameter. In a second operation common peroneal or tibial nerves were cut bilaterally and ligated distal to their cuff electrodes. Electrical stimulation of the proximal segment of the nerve was carried out at a frequency of 10 Hz, 8 hours a day for up to 200 days in one limb while the other served as control. CAP and latency were again recorded at 5 days intervals to monitor changes in axon diameter. Evoked CAP amplitude declined to about 25% of preoperative values with a similar time course irrespective of whether the axotomized neurons were chronically stimulated or not. An increase in CAP latency was also similar in stimulated and nonstimulated nerves.

The present study shows that chronic electrical stimulation neither prevents atrophy in axotomized neurons nor stimulates recovery of atrophic neurons. Thus, the most likely explanation for the recovery of axotomized neurons after reinnervation is the reinstitution of trophic factors.

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