

Title : SPINAL CORD TRANSECTION AND NERVE MUSCLE TRANSMISSION
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Introduction. Spinal cord transection is usually followed by changes in muscle physiology which may lead to the observed hyperkalemic responses to succinylcholine. The purpose of this study was to document some of these methods and their time course.

Methods. Sprague Dawley rats weighing 200-250 gm were anesthetized with diethyl ether and laminectomized at T₆-T₇ levels. The spinal cord was transected at that level under direct vision. The wound was closed and the animals were permitted to recover. The completeness of the transection was evaluated in each animal prior to the study by observing failure to extend the toes or ankle when lifted off the floor. Studies of some neuromuscular functions were conducted on post-transection day 1, 3, 5, 7, 10, 30 and 90 on both extensor digitorus longus and soleus muscles. The results presented here are principally those of the extensor. The following functions were evaluated: (A) microelectrode studies, mep frequency and amplitude, sensitivity of the endplate to iontophoretically applied acetylcholine, action potential generation, membrane resistance; (B) in vitro Na⁺ and K⁺ variation to acetylcholine application; (C) effect of application of acetylcholine and succinylcholine on the development of contracture; (D) changes in serum Na⁺ and K⁺ concentration following succinylcholine administration in vivo.

Results. There was no significant change in mep frequency, amplitude, input resistance or action potential generation in all rats studied up to 90 days on either EDL or soleus muscle. Resting membrane potential, however, showed a significant decrease from control at 3 days post-transection with gradual return towards normal between the 5th and 10th days. Spread of receptor sensitivity as indicated by membrane potential change to iontophoretically applied acetylcholine could

first be demonstrated on the 3rd day post-transection and appeared to reach a maximum spread at day 7. This phenomenon of receptor spread was sustained until the 30th day and returned to normal by the 90th day. With the development of receptor spread, acetylcholine and succinylcholine contractures of the muscle in vitro could be elicited by the 3rd day post-transection. This reached maximum by the 7th day and was reproducible until the 30th day. There was no induced contracture seen in the 90-day rats. The muscles were noticeably atrophied between 7-30 days but were approximately of normal size by 90 days. K⁺ levels both in vitro and in the serum of intact animals after the administration of 1 mg/kg succinylcholine were elevated but not statistically different from controls.

Discussion. The restriction of cholinergic receptor to the endplate region has long been thought to be a result of nerve-muscle tropism. The spread of the receptor area in a denervated muscle is thought to result from the loss of this trophic influence. The observation of Thesleff¹ in 1961 of spread of receptor after cord transection would suggest other forms of tropism also exist. This study confirms this cholinergic receptor spread after cord transection and shows it in a chronological sequence. Our study does not explain the hyperkalemia that develops following succinylcholine to spinal cord transected patients, nor does it rule out cholinergic receptor spread as being the cause. Further studies are deemed necessary to prove or disprove the relationship between receptor spread and K⁺ efflux.

Reference.

1. Johns TR and Thesleff S: Effects of motor inactivation on the chemical sensitivity of skeletal muscle. Acta Physiol Scand 51:136-141, 1961