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Spinal cord injury: Neurosurgical Rewiring of Neuromuscular and Spinal cord circuitry restores Locomotor Functions

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Opinion

At this point in time (2015) there are only limited numbers of treatment options available for spinal cord injury (SCI) paralysis and they (e.g. stem cells; implanted stimulation devices) are all in clinical trials. The treatment procedure presented here is founded on over 40 years of neurophysiological studies in locomotor development and computational cognitive systems. The treatment is meant to be cost-effective, alternative, as well as complementary to the existing options. This synaptic competitive-learning (SCL) therapy involves controlled neurapraxia (nerve crush) surgery of limbs main nerve trunks. I have experimentally proved the concepts in spinal cord complete injury (SCIc) animal model. What is synaptic competitive-learning (SCL)? In rats a SCIc from new born up to 20 days age does not result in paralysis. Body weight-support, standing, stepping all remain intact into adulthood. Beyond 20 days, however a SCIc always results in incapacitating complete paralysis [1]. I found in 1991 that the function preservation seen in the young rats is due to restorative SCL rewiring within the isolated cord and the limb muscles. At older ages this rewiring capability is lost. Now the question is: can such rewiring be done in SCI in adult?

Briefly, SCL is the naturally-endowed, post-birth synaptic connectivity phenomenon in the intact spinal cord and in the limb muscles neuromuscular junctions during locomotor learning (crawl, sit, stand, step, and walk) and maturation. At birth, each muscle fibre is innervated by more than three, four motor terminals derived from different motoneurons. Then, as locomotor learning continues motor activity-dependent, competition takes place between the motor terminals to gain control of that muscle fibre. Functionally fitting terminal is retained and stabilized while the others are pruned. Eventually, each muscle fibre is controlled by a single motor terminal. At this stage stretch and reciprocal inhibitory reflexes mature and reach their adult activity pattern. And by late infancy these SCL wirings in the limb muscles and spinal cord circuitry get completed and locomotor maturation (in rats 20 days age) is reached. This post-birth (epigenetic), activity-dependent, competition-based synaptic connectivity phenomenon [2] gives the brain-spinal cord circuitry unlimited motor learning and memory-storing capability throughout life as opposed to the pre-birth, genetically pre-determined, rather restricted connectivity processes.

I designed a simple neurosurgical procedure (Krishnan, 1991; 2001) in the adult SCIc amphibian animal model that would simulate and reinstall each of the stages of the SCL mechanisms in the injured cord circuitry and the limb muscles. This involves controlled neurapraxia (nerve crush) of the paralyzed limbs motor nerve trunks (sciatic, femoral, and obturator). The procedure interrupts impulse conduction in about 20-30 percent of the sensory-motor axons. All the limb muscles become temporarily partially denervated. Then, spontaneous sprouting of intact motor axons follows within these muscles and reinnervates the denervated fibres. When the interrupted motor axons regenerate and arrive (in the animal model six to eight weeks post-neurapraxia) into their muscles synapse competition takes place between the sprouted and the regenerated motor terminals to gain control of the muscle fibres. This competition is motor activity-dependent. Functionally appropriate connections are selected and stabilized while others are pruned. In the meantime, the motoneurons soma sizes become plastic and are resized. A similar synapse competition-selection takes place on their soma-dendritic surfaces. The excitatory-inhibitory (E-I) synaptic connections weights impinging on the motoneurons undergo corrective repositioning and the E-I balance between synergists-antagonists muscles is restored. 6-10 weeks following the SCL therapy swimming and stepping commenced in these

animals that was sustained and improved for several months of post-operative observation (see videos).

https://www.youtube.com/results?search_query=krishnanspinal

How do we know for certain that it is indeed the SCL therapy that brought the motor restoration, and not due to regeneration across the injury site? First, none of the untreated control SCIc animals showed recovery. They remained complete paralyzed and indeed developed extensor-adductors spasticity and "scissoring" of the limbs. Second, in all the neurapraxia treated animals spasticity was absent. Instead, they regained the normal flexion position of the limbs. Third, the treated animals recovered swimming and ground progression that persisted for several months of post-operative survival. How does neurapraxia treatment prevent spasticity? Selective neurectomy is a well-known neurosurgical procedure for relief of intractable spasticity. This consists of permanent, partial surgical denervation (hyponeurotization) of spastic muscle/s. In controlled neurapraxia procedure there is no permanent partial denervation. The transient denervation and the ensuing synapse competition and the resizing of motoneurons soma reduces/abolishes spasticity. Computational modelling shows that the synapse competition and the resizing of soma sizes enable the motoneurons to selectively eliminate topographically incorrect synapses and self-correct errors in their firing properties.

What is Neurorehabilitation? Present rehabilitation programs e.g. Body weight-supported treadmill walking is incorrectly called neurorehabilitation. Indeed they are rather desperate, arduous retraining of the injured cord circuitry that is stubbornly learning-resistant. Whereas SCL therapy rewires neuromuscular and cord circuitry. Spasticity is abolished and neuro relearning commences. In this intrinsically generated locomotor performance all muscles of the limbs participate. When this relearning is combined with retraining then it becomes true neurorehabilitation. Our research [3,2] (2009; 2013) shows that in human SCI paralysis, Botulinum toxin (BoTx) when administered in small doses to select muscles in serial sessions can reinstall SCL mechanisms precisely similar to the neurapraxia procedure and bring about long lasting motor recovery. Indeed, BoTx procedure will be non-invasive compared to neurapraxia. In human SCI over 70 percent of them are incomplete injuries. Neurosurgical and neuropathological studies have shown that even if a small percentage (20-30) of neural tissue has survived the injury then that could contribute to substantial recovery. The SCIc animal model presented here shows that significant functional motor recovery can be achieved even in severest injury [4]. This means in incomplete injuries SCL therapy would bring even far greater recovery. Researchers might question why I chose an amphibian model. I did these experiments at my retirement over ten years ago in the total absence of funding and technical assistance. I invite neurosurgeons and neurologists to study the two SCL treatment procedures: experiment, debate and translate into clinical trials in the nearest future.

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