Neuroplasticity of Motor Pathways

Author: Andrew C. Smith, PT, DPT, PhD

FACT SHEET

ACADEMY OF NEUROLOGIC PHYSICAL THERAPY
SPINAL CORD INJURY SIG

Academy of Neurologic Physical Therapy

American Physical Therapy Association

5841 Cedar Lake Road S. Ste 204
Minneapolis, MN 55416
952-543-5348

Fax: 952-545-6073

info@neuropt.org
www.neuropt.org

Motor Disruption After SCI

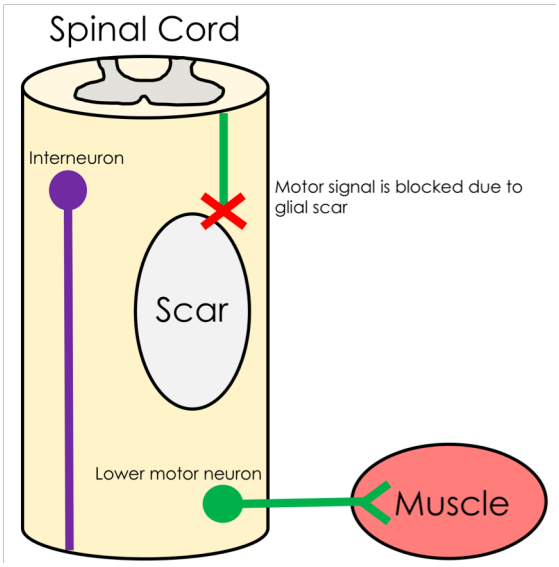

Figure 1: Motor signal is blocked due to descending motor tract injury and glial scar

Following a spinal cord injury (SCI), there is a disruption to the signal between the brain and the lower motor neuron that drives muscle activation and movement.[1] Both the initial injury to the spinal cord and the scar that forms afterward contribute to this disruption (see Figure 1).[1]

Rewiring May Be Possible

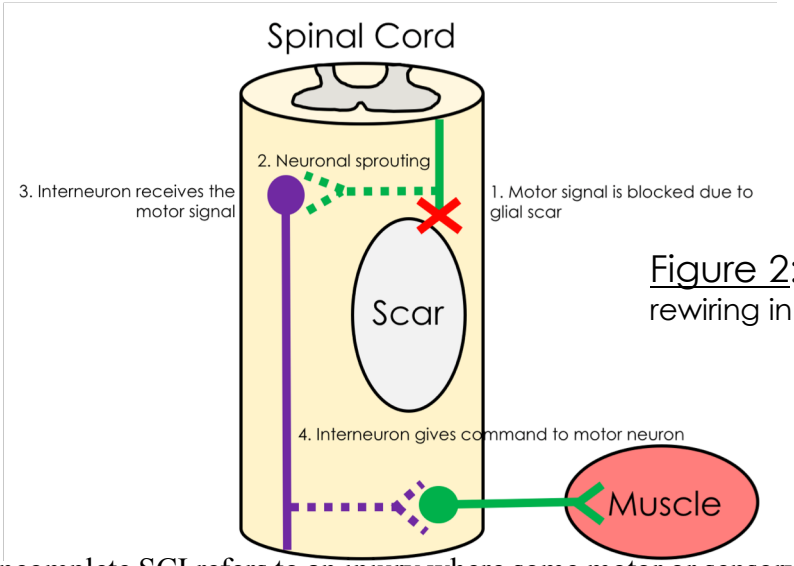

Figure 2: Mechanism of rewiring in the spinal cord

Incomplete SCI refers to an injury where some motor or sensory function is still present below the level of the injury.[2] In the case of an incomplete SCI, the re-wiring of nerves plays an important role in recovery.[2] Studies in animals have helped us understand how this re-wiring works.[3,4] In these animal studies, as little as 25% of remaining nerves allowed for recovery of voluntary walking ability.[3,4] These studies suggest that nerves are able to sprout outwards to

communicate with spinal interneurons, which then relay the motor command to the muscle via the motor neuron (see Figure 2).[5-10] This motor pathway-interneuron connection can be enhanced with medication and with activity-based interventions such as treadmill training.[11,12]

Research in humans using brain stimulation and nerve stimulation suggests that this pathway is preserved in humans.[13-19] While more research is needed, the available evidence provides clinicians with an understanding of what is likely taking place in the nervous system of our patients with SCI. With active physical therapy interventions such as locomotor training on a treadmill[2] and repetitive practice of task-oriented activities,[20] we are likely encouraging and promoting this re-wiring. This ability of the spinal cord to re-wire means that neurologic recovery is possible… which is exciting and hopeful news!

References:

1. Ahuja CS, Nori S, Tetreault L, et al. Traumatic spinal cord injury – repair and regeneration. *Neurosurgery*. 2017; 80(3S):S9-S22.
2. Smith AC, Knikou M. A review on locomotor training after spinal cord injury: reorganization of spinal neuronal circuits and recovery of motor function. *Neural Plast*. 2016;doi:10.1155/2016/1216258.
3. Windle WF, Smart JO, Beers JJ. Residual function after subtotal spinal cord transection in adult cats. *Neurology*. 1958;8(7):518-521.
4. Eidelberg E, Walden JG, Nguyen LH. Locomotor control in macaque monkeys. *Brain*. 1981;104(Pt 4):647-663.
5. Bareyre FM, Kerschensteiner M, Raineteau O, Mettenleiter TC, Weinmann O, Schwab ME. The injured spinal cord spontaneously forms a new intraspinal circuit in adult rats. *Nat Neurosci*. 2004;7(3):269-277.
6. Courtine G, Song B, Roy RR, et al. Recovery of supraspinal control of stepping via indirect propriospinal relay connections after spinal cord injury. *Nat Med*. 2008;14(1):69-74.
7. van den Brand R, Heutschi J, Barraud Q, et al. Restoring voluntary control of locomotion after paralyzing spinal cord injury. *Science*. 2012;336(6085):1182-1185.
8. Rosenzweig ES, Courtine G, Jindrich DL, et al. Extensive spontaneous plasticity of corticospinal projections after primate spinal cord injury. *Nat Neurosci*. 2010;13(12):1505-1510.
9. Flynn JR, Graham BA, Galea MP, Callister RJ. The role of propriospinal interneurons in recovery from spinal cord injury. *Neuropharmacology*. 2011;60(5):809-822.
10. Filli L, Schwab ME. Structural and functional reorganization of propriospinal connections promotes functional recovery after spinal cord injury. *Neural Regen Res*. 2015;10(4):509-513.
11. Vavrek R, Girgis J, Tetzlaff W, Hiebert GW, Fouad K. BDNF promotes connections of corticospinal neurons onto spared descending interneurons in spinal cord injured rats. *Brain*. 2006;129:1534-1545.
12. Rank MM, Flynn JR, Battistuzzo CR, Galea MP, Callister R, Callister RJ. Functional changes in deep dorsal horn interneurons following spinal cord injury are enhanced with different durations of exercise training. *J Physiol*. 2015;593(1):331-345.
13. Nathan PW, Smith MC. Fasciculi proprii of the spinal cord in man. *Brain*. 1959;82:610-668.
14. Burke D, Gracies JM, Mazevet D, Meunier S, Pierrot-Deseilligny E. Non-monosynaptic transmission of the cortical command for voluntary movement in man. *J Physiol*. 1994;480:191-202.
15. Pierrot-Deseilligny E. Propriospinal transmission of part of the corticospinal excitation in humans. *Muscle Nerve*. 2002;26(2):155-172.
16. Gracies JM, Meunier S, Pierrot-Deseilligny E. Evidence for corticospinal excitation of presumed propriospinal neurones in man. *J Physiol*. 1994;475(3):509-518.
17. Pierrot-Deseilligny E. Transmission of the cortical command for human voluntary movement through cervical propriospinal premotoneurons. *Prog Neurobiol*. 48(4-5):489-517.
18. Iglesias C, Nielsen JB, Marchand-Pauvert V. Corticospinal inhibition of transmission in propriospinal-like neurones during human walking. *Eur J Neurosci*. 2008;28(7):1351-1361.
19. Petersen NT, Pyndt HS, Nielsen JB. Investigating human motor control by transcranial magnetic stimulation. *Exp Brain Res*. 2003;152(1):1-16.
20. Beekhuizen KS, Field-Fote EC. Sensory stimulation augments the effects of massed practice training in persons with tetraplegia. *Arch Phys Med Rehabil.* 2008;89:602-608.