Neuroplasticity after a spinal cord injury

V. Dietz
Spinal Cord Injury Center, Balgrist University Hospital, 8008 Zurich, Switzerland

After a spinal cord injury (SCI) of the cat or rat, neuronal centers below the level of lesion exhibit plasticity than can be exploited by specific training paradigms. In individuals with complete or incomplete SCI, human spinal locomotor centers can be activated and modulated by locomotor training (facilitating stepping movements of the legs using body weight support on a treadmill to provide appropriate sensory cues) (for review see Dietz 2002,2003). Individuals with incomplete SCI benefit from locomotor training such that they improve their ability to walk over ground. Load- or hip joint-related afferent input seems to be of crucial importance for both the generation of a locomotor pattern and the effectiveness of the training (Dietz et al. 2002). However, it may be a critical combination of afferent signals that is needed to generate a locomotor pattern after severe SCI. Mobility of individuals after a SCI can be improved by taking advantage of the plasticity of the central nervous system and can be maintained with persistent locomotor activity. In the future, if regeneration approaches can successfully be applied in human SCI, even individuals with complete SCI may recover walking ability with locomotor training (Curt et al. 2004). During the past few years, several approaches to spinal cord repair have been successfully established in animal models. For their use in clinical trials of SCI in human beings, specific difficulties that affect the success of clinical trials have to be recognised (Dietz and Curt 2006). First, transection of the spinal cord is commonly applied in animal models, whereas contusion, which generally leads to injury in two to three segments, represents the typical injury mechanism in human beings. Second, the quadrupedal organisation of locomotion in animals and the more complex autonomic functions in human beings, challenge translation of animal behaviour into recovery from SCI in people. Third, the extensive damage of motor neurons and roots associated with spinal cord contusion is little addressed in current translation studies. Fourth, there is increasing evidence for a degradation of neuronal function below the level of the lesion in chronic complete SCI (Dietz and Müller 2004; Dietz 2010). This degradation might have a considerable impact in chronic SCI subjects for a regeneration-inducing treatment. Therefore, it's relevance needs to be investigated.

References:

Curt A, Schwab ME, Dietz V (2004) Providing the clinical basis for new interventional therapies: Refined diagnosis and assessment of recovery after spinal cord injury. Spinal Cord 42:1-6.

Dietz V (2002) Proprioception and locomotor disorders. Nature Reviews Neuroscience 3:781-790.

Dietz V (2003) Spinal pattern generators for locomotion. Clinical Neurophysiology (Review) 114:1379-1389.

Dietz V (2010) Behavior of spinal neurons deprived of supraspinal input. Nature Reviews Neurology 6: 167-174.

Dietz V, Curt A (2006) Neurological aspects if spinal cord repair: promises and challenges. Lancet Neurology (Review) 5: 688-694.

Dietz V, Müller R (2004) Degradation of neuronal function following a spinal cord injury: mechanisms and countermeasures. Brain 127: 2221-2231

Dietz V, Müller R, Colombo G (2002) Locomotor activity in spinal man: Significance of afferent input from joint and load receptors. Brain 125:2626-2634.