PERSPECTIVES

The plasticity of the adult spinal cord continues to surprise

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Although spinal reflexes are highly modulated during normal behaviour (Zehr & Stein, 1999) and can be modified with training in adult, spinal mammals (Chen *et al.* 1999), the spinal cord has often been viewed as a relatively fixed and less interesting part of the nervous system. This view was challenged by work showing that quite normal walking patterns could be produced after treadmill training in adult animals with a complete transection at the thoracic level (reviewed by Rossignol *et al.* 2006). Without training animals did not regain the ability to walk. Thus, the spinal central pattern generator (CPG) for walking, which is normally controlled by supraspinal inputs, can modify itself after spinal cord injury (SCI) and training to function independently of descending inputs. Furthermore, studies from many centres have translated this work to the clinic, although only people with an incomplete SCI show marked benefits from treadmill training (Wernig & Muller, 1992; Barbeau *et al.* 1999). There is some evidence for a spinal CPG in humans (Calancie *et al.* 1994; Dimitrijevic *et al.* 1998), but it may be more dependent on descending inputs and therefore less able to function independently after SCI.

The paper by Frigon & Rossignol (2008) in this issue of *The Journal of Physiology* shows how precisely the normal walking pattern is reproduced, based on careful, labourious, chronic experiments. They recorded with implanted EMG electrodes from cats for 1–2 months before transecting the spinal cord and for a comparable period of time after SCI, while training the animals 3–5 times per week. Both the joint kinematics and the electrical activity of key muscle groups become remarkably similar with training to that found before SCI. How is the missing input from higher centres replaced? To study this Frigon & Rossignol also chronically implanted a cuff containing stimulating electrodes on the tibial nerve near the ankle and evoked reflexes at various times in the step cycle. Interestingly, during the stance phase short-latency reflexes in the calf muscles that were inhibitory in the intact animal became excitatory after SCI and training. The tibial nerve provides the cutaneous innervation of the paw and these receptors will be excited during the stance phase of walking. The modified reflex could provide additional force to support body weight in stance. Changes in cutaneous reflexes are well known in humans after SCI and are thought to contribute to unwanted, spastic responses. For example, innocuously brushing the skin on the leg can lead to a prolonged flexor or extensor response of the whole limb. Whether the changes Frigon & Rossignol observe occur in humans and could contribute to the effects of treadmill training remains to be investigated.

Another remarkable, recent finding from Rossignol's lab (Barriere *et al.* 2008) involves animals in which the spinal cord is initially hemisected and trained for a period of time. Then, following a complete transaction of the cord a couple of segments below the initial lesion, the animals almost immediately show a bilateral, symmetric walking pattern on a treadmill. In some sense, following the unilateral lesion and training the spinal cord 'learned' how to produce the walking pattern without descending inputs and surprisingly could 'instruct' the other side after it was injured as well. The physiological mechanisms and clinical implications of these new findings are completely unknown.

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