Spinal Locomotion in Cats

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1. Introduction

Locomotion is based on the synchronization between the flexion and extension of the limbs and reflex circuits of the spinal cord. However, the spinal cord alone is not sufficient to enable walking, and the input of the brain centers is also necessary [1]. The alternation between the extension and flexion of the pelvic limbs involves the central pattern generators (CPGs). The CPGs are spinal locomotor circuits with pacemaker proprieties, located in the thoracolumbar spinal cord, which generate bilateral rhythmic and repetitive contractions and relaxations of the flexors and extensor muscles in the absence of descending motor tracts and supraspinal inputs [1][2]. The supraspinal control of the CPGs originates in the nuclei, located in the midbrain (“mesencephalic locomotor region”). These nuclei initiate locomotion by activating the reticulospinal neurons in the brain stem. In cats, there are the following two major descending motor tracts: the medial longitudinal fascicle, with cells originating in the medial pontomedullary reticular formation, and the lateral vestibulospinal tract [3].

Locomotor training after spinal cord injury is based on the principle that sensory stimuli reactivate and reorganize the spinal locomotor circuit [4]. Over the last decade, the expression of hind limb locomotion after Spinal Cord Injury (SCI) at T13 in kittens [5][6] and adult cats has been investigated [7][8]. After 2–3 weeks of land treadmill training, both were able to walk and recover full-weight-bearing. To modulate the flexor–extensor pattern, some researchers suggest the association of pharmacologic management [9][10]. Thus, the adult cat spinal cord, in vivo, was proposed as a classical model in studies of mammalian hindlimb CPGs [11][12][13], allowing the translation of these findings to a clinical setting.

2. Spinal Locomotion in Cats Following Spinal Cord Injury

After spinal cord injury, both humans and cats can experience spontaneous recovery that is observed even without functional rehabilitation, and can be explained by the same cellular mechanisms that can occur in spontaneous remodeling [14][15][16]. This makes it difficult to separate, concerning the clinical signs that are indicative of neurological recovery over time, the evolution that is mainly due to the action of rehabilitation training from that of spontaneous recovery [17].

Early and important studies in neuroscience were performed in acute and chronic adult spinal cats and were based on a complete section of the spinal cord in a laboratory setting, which resulted in the paralysis of the pelvic limbs (grade 0 in MFS) [4][7][8][18][19]. The breakthrough of intensive rehabilitation in SCI was based on the studies mentioned above [17]. Furthermore, training procedures that showed small stepping movements on the land treadmill led to a marked improvement in the locomotor performance [20], although it appears that the type of locomotor training was the most important factor in the recovery of locomotion [21].

The main difference between those studies, when compared to ours, is the velocity applied on the treadmill. In our study, locomotor training was performed, in some cases, bipedal training that progressed to quadrupedal training, which allowed the spinal cats to adapt to the treadmill and made it possible to reach higher treadmill speeds. Lovely et al., 1990 [21] achieved stepping on the land treadmill with velocities of 0.05–0.15 m/s^{-1}, which was a slow speed when compared to our study (0.22 m/s increasing to 0.5 m/s). The maximum land treadmill speed at which spinal locomotion could be obtained was reported from 0.5 to 0.6 m/s, only after three weeks of training [22]. Our study accords with these training procedures.

Lovely et al., 1990 [21] also performed daily locomotor training, similar to our protocol, but with an early training approach in the acute phase, justifying their results. The same study showed that a period of four to six months was needed to achieve ambulation, while Barbeau and Rossignol (1987) proved that acute spinalizated adult cats (T13) were capable of demonstrating a gait with the weight support of the hindlimbs without showing knuckling, from three weeks up to one year [7]. On the other hand, Edgerton et al., 1991 [23] performed 30 min of daily training for six months, reaching a performance plateau at tow to four months. Regarding time, this training was similar to the one implemented in our protocol, although 33% (*n* = 3) reached a plateau performance in one month.

Barbeau and Rossignol, 1990 [24] introduced pharmacological management in cats between one and three months after spinalization. The cats reached a treadmill velocity of 1.0 m/s before and after an injection of a serotonergic monoamine drug (5-HT). They concluded that the injections of 5-HT increased the cycle duration by about 80%, and that noradrenergic, dopaminergic, and GABAergic drugs could improve the initiation of locomotion (in an early stage), and modulate the well-established locomotor pattern (in a chronic stage). In addition, studies were performed on the association between pharmacologic management and FES [25]. In this regard, some results showed that electrical stimulation could initiate or modify locomotor activity in a similar way to pharmacological neural modulation [20]. These findings show that the use of electrostimulation can be a neurorehabilitation modality, used to enhance the restoration of full-weight-bearing locomotor function [26].

Smith et al., 1982 [27] demonstrated that spinal cats could exhibit excellent weight support during locomotion, applying treadmill speeds of ≤0.8 m/s, which suggested velocity as a major factor in the recovery of ambulation. In addition, the results have shown that, in the first 7–10 days post-section, cats only executed small hindlimb movements, with little to no weight support, which could be enhanced with pharmacological treatment, stimulating the central plasticity that could be reflected by the evolution of the locomotor pattern over time [10].

When compared to our study, on both modalities of the locomotor training (BLT and QLT), combined with FES, ambulation was obtained in 56% of cats (*n* = 5). Thus, only 10% of the white matter of the spinal cord is required to initiate and maintain the locomotor pattern, in a voluntary way, on the land treadmill [28], which can probably be justified by the residual descending motor tracts post-injury [29]. In our study, one cat recovered their DPP, obtaining medical discharge after two weeks of INRP. As for the other three cats exhibiting SS, two had medical discharge one month after INRP and the other one did two months afterwards. It is suggested that severe injury contains sub-functional connections that are capable of transmitting a supraspinal influence on the neural circuits of CPG, below the injury [30][31]. Following the researches of Dimitrijevic et al. 1987, Militskova et al. refers to this type of lesion as “discomplete SCI”, which agrees with Gerasimenko et al., 2017 [32], who suggest that locomotor training can significantly improve sensorimotor and autonomic function after SCI [33].

In the neurophysiological field, it has been proposed that the use of an α2-noradrenergic agonist, such as clonidine, could initiate treadmill locomotion faster in cats with acute and chronic spinal injury [34], and modulate the spinal locomotor pattern [10], allowing fictive locomotion, which may be recovered spontaneously in several weeks. Though in chronic spinal cats, the activation of the receptors does not express the spontaneous spinal locomotion [35], excitatory amino acid receptor agonist NMDA, injected intrathecally, could result in a dramatic improvement in the locomotor pattern.

In some studies, fictive locomotion has been achieved. This fictive locomotion could be defined as a rhythmic pattern that occurs in the absence of any movement [2][36], and in chronic spinal cats, it can occur spontaneously, which indicates functional changes in the interneurons and CPGs [4][37].

Our strict INRP suggested that BLT could activate proprioceptive afferents (groups Ia, Ib, and II) [37][38] throughout stretch bicycle movements, performed by the therapist, in alternation with the stimulation of the perineal region [39] and the base of the tail [14], which regulated, in some part, the duration of various sub-phases of the step cycle (frequency and speed). Moreover, the hip joint influences the locomotor rhythmic generation and has a potent effect on the intrinsic neural circuits, because, when the hip is flexed, the spinal cat stops the stepping movement [40]. Furthermore, the QLT may activate the CPG that regulates the fore- and hindlimb locomotion, and this can be blocked when the propriospinal pathways that connect the cervical and lumbar enlargements are interrupted [41][42]. Thus, the QLT stimulates the coordinating propriospinal system, providing forelimb–hindlimb coordination [43]. In our study, the cat that showed recovery of DPP could perform QLT in the first instance.

The QLT presumably increases the synaptic activity by interneuron stimulation, which is responsible for locomotor patterns and intrinsic electrophysiological proprieties [44][45] that are vital to neural control [46]. Locomotor training can have an impact on spinal cord autonomy, influence sensory inputs, induce neuromodulation and learning abilities of the spinal

network, promote depolarization of the descending motor pathways [46], and upregulate the neurotrophins, particularly the neurotrophic factor derived from the brain (BDNF) that plays an important role in central nervous system (CNS) neuroplasticity, contributing to the restoration of function [20]. The intense repetitive training may have driven neuroplasticity, which eventually results in the ability of voluntary movement, by reorganizing its structure, function, and connections [47][48][49][50][51].

The QLT promotes stimulation of the afferent pathways by receptors, located in the muscles (intrafusal fibers), joints (nerve endings), and skin. This type of stimulation allows a dynamic interaction with the CPG circuit [3][52][53], and cutaneous neural stimuli in the digit region that stimulates the expression of reflex locomotion, allowing the observation of neural reorganization after four weeks of locomotor training on the treadmill [7][14][54]. During the exercises, several interconnected components are activated, which are necessary to obtain correct locomotion [4].

The vestibulospinal and reticulospinal tracts are responsible for posture, meaning that they have a greater positive effect on the extensor muscles [55], so the introduction of the slope during QLT will stimulate these muscles and, therefore, the neuroplasticity of the tracts mentioned above. Thus, a 10° slope is suggested by Maier et al., 2009 [56], and 25° is suggested by Tillakaratne et al., 2014 [57].

Locomotor training that allows neural plasticity should be performed for 30–60 min [52][15], which is in agreement with the INRP presented in this study. The INRP is associated with kinesiotherapy exercises that play a role in synaptic neuroplasticity, in the sense of neuroremodeling [58][59]. To achieve a balance between excitation and inhibition, in the passage of an obstacle, there is a stimulation of receptors located in the distal dorsal region of the limb that will allow excitation of the flexor muscle group of the ipsilateral limb, when performing the protraction phase, and that, with the same stimulus, can excite the extensor muscles that are necessary to obtain the postural phase of the step cycle [60]. An example of the above is active-assisted and/or active kinesiotherapy exercises, which are included in locomotor training [61]. Included in the INRP are the cavalettis rails for stimulating the passage of obstacles.

The cat that had recovered DPP showed fast progression when performing these exercises, supporting the notion that DPP is considered a favorable prognostic factor [29][62][63][64]. Therefore, the ability to execute early locomotor training, reaching the mentioned performance guidelines, may suggest a higher probability of recovery ambulation in a shorter amount of time.

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