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Clinical Commentary

Coordination training in individuals with incomplete spinal cord injury: consideration of motor hierarchical structures

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Study design: Clinical commentary.
Objective: To discuss the method of coordination training to enhance motor skills in persons after spinal cord injury (SCI).
Method: From the literature and clinical experience, we learn that basic motor skills like walking are refined and maintained through the millions of repetitions that take place as part of normal development. These coordinated patterns function effectively as a form of training to the system because of the presence of neural pathways that mediate commands between higher and lower levels of the central nervous system (CNS). When these pathways are disrupted as a result of a lesion, the question that arises is whether retraining can be effective.
Results/Discussion: The question is directed at the common practice among rehabilitation professionals to prescribe and carry out tireless repetitions of coordinated motor activities in people with SCI lesions. We discuss this fundamental question from the perspective of understanding differences in maturation and function of higher motor centres and lower motor centres.
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Introduction

Coordinated movement demands considerable practice or training.[1–4] A child takes about three million steps before the pattern of walking is mature at about 6 years of age.[1] Similarly, in sports, it is estimated that a professional basketball player shoots as many as one million baskets before the movement is perfected.[1] The importance of coordination training is described in textbooks on motor learning[3] and motor development.[4] The process of coordinated learning, according to Kottke,[2] involves learning to perform a movement with minimal activation of agonist muscles while inhibiting muscles that do not effectively contribute to the desired activity. In the healthy human motor system, where all the prerequisites for movement are present, training of coordinated activity, such as walking and running, is effectively achieved through the many repetitions that occur as a normal part of human motor development and exploration of the environment.[4] In this article, we would like to focus on the question of whether the training of basic motor skills of the lower extremities is an effective strategy for motor relearning in those subjects who have an incomplete spinal cord injury (SCI).

Organization of the neuromotor system

Our motor system is organized hierarchically, with the uppermost level mediated through the primary motor cortex (localized in the *gyrus precentralis*) and the lowermost levels mediated through the spinal motor neurons. In this hierarchical structure, the uppermost components are generally responsible for the initiation of the movement, whereas the lower level components are responsible for the organization and execution of the movement (eg how to make a movement). At the lowermost level, all the motor commands converge on the motor neuronal pool in the spine.[5] The motor neuronal pool is a prerequisite for all muscle contractions; only after a depolarization of these motor neurons can a muscle contraction take place. It is for this reason that Sherrington[6] described this path from the motor

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neuronal pool in the spine to the muscle as 'the final common pathway'.

Maturation

There is evidence that the *corticospinal* tract in man matures predominantly after birth according to a fixed program. For a review on this topic, we refer the reader to the article by Forssberg[7] on the ontogeny of human locomotor control. In this work it was shown that innate pattern generators in the spine produce stepping at birth and that during the first years of life it is controlled in a piecewise fashion by higher organization centres so that infant stepping is transformed to normal *plantigrade* stepping. During normal development, no additional coordination training is needed to stimulate the outgrowth of the *corticospinal* tract. In a classic investigation on the onset of walking in the Hopi Indian culture by Dennis it was shown that a severe restriction of movement did not influence the onset of walking compared to unrestricted movement in early infancy.[8] This experiment shows us that the development of walking behaviour is innate. When this basic motor skill has developed, the child refines this motor skill to the ever-changing environment with endless repetitions as also we learn from Kottke *et al*[1] and Gallahue.[4]

CNS lesion

In the case of a lesion to the CNS, the resulting functional deficits are determined by which groups of nerve cells are severed. The site of lesion, either in the spinal cord or the brain, determines whether it may be possible to compensate for the damage, or in the worst case, lose all capability to move voluntarily. Lesions of the spinal cord affecting the upper motor neuron (UMN) can clearly be distinguished from lesions of the lower motor neuron (LMN). Generally speaking, after a lesion to the UMN, muscle contractions are still possible although the highest cortical control may be missing. In contrast, lesions on the level of the LMN typically result in flaccid paresis. Furthermore, after a lesion of the UMN, it may be possible for the person to relearn some movements or to compensate for them in such a way that they may be able to return to independent functioning.

For those that suffer from severe lesions of the CNS, much research has been done on the central pattern generator (CPG), the foundation level of motor control in the spine. The question arises: to what extent can we activate the CPG in case of a spinal cord lesion? Originally, experiments on CPGs were conducted on spinalized cats by Grillner[9] and he demonstrated that it was possible for these animals to treadmill walk, even on a split belt operating at different speeds. Similar CPG studies are being conducted for treadmill walking in spinal man.[10–14] Although it is possible to synchronize and improve the EMG signals after treadmill training in humans with a complete SCI, the ability of individuals with SCI to improve their overground walking after treadmill training is limited.[12,15,16] One reason for this lack of transfer is that the proprioceptive feedback of the moving feet on a stationary surface is lacking.[17,18] Moreover, it was also shown that people with incomplete paraplegia profited more from treadmill training than individuals with complete lesions.[12,15,16] With this in mind, it becomes very important to establish an exact diagnosis of the SCI immediately after the lesion because time-consuming therapies appear to be more appropriate for the incomplete injury.[12,15] Furthermore, maintaining neuronal circuitries 'trained' in incomplete SCI should make these circuitries more easily accessible for eventual regeneration or compensatory plastic processes.[17,19–21]

Most lesions of the CNS are incomplete.[22] In animals, walking ability may be regained with less than 50% of the spinal cord left intact.[23] Recent investigations on incomplete SCI rats showed that even the *rubrospinal* tract is able to reorganize itself and make new sprouts and connections after a partial denervation.[24] This provides a basis for hope among individuals with incomplete SCI.

Discussion

Critical remarks concerning coordination training for the incomplete SCI lesion

It is our recommendation that we cautiously bear in mind the following when treating the individual with SCI. Instead of focusing on the *corticospinal* tract and trying to train the *phylogenetically* youngest part of the motor system first, it may be beneficial to adopt an approach where we try to influence the motor system at the lowest level initially, namely, by influencing the CPGs, the *phylogenetically* older system. From several group studies,[15,21,25] it was highlighted that circuitry in the human species is able to learn, especially when this training is combined with loadbearing. We should consider the fact that the *corticospinal* tract is not the only tract to project to the motoneurons in the spine. The *reticulo*-, *vestibulo*- and *rubrospinal* tracts also terminate on these motoneuron pools and these latter tracts may be able to compensate for a damaged *corticospinal* tract. To us, this approach seems more logical because normal motor development in the human proceeds from the lower level to the highest level, which is the *corticospinal* tract.

Also, we should bear in mind that extrapolations from animal experiments to humans should be done with great caution when arguing that treatments that influence the *corticospinal* tract may lead to a breakthrough in SCI recovery. Anatomically and functionally, the *corticospinal* tract in cat and rat differs from those in higher primates and humans. In quadrupeds the front legs have a different function than the arms in bipeds.[26] A one-to-one transfer from experiments on mammals that use four limbs for locomotion to the higher primates that ambulate *bipedally* is not possible. Furthermore, one of the reasons why so much research

has been done on this tract in rat and mice is that the *corticospinal* tract can be anatomically located very easily. There is a resulting overemphasis on the role of this tract in *quadrupedal* locomotion. In humans it is known that the other *supraspinal* tract systems as well as the CPGs are of greater importance in walking.[17] There may also be more practical reasons than scientific reasons for the preponderance of research on the *corticospinal* tract. Monetary and ethical considerations have limited the practicality of performing basic research experiments on primates, although higher primates using two legs for locomotion will sustain a much more suitable animal model for individuals with SCI. Nevertheless, even in the higher primates the *corticospinal* projections to lower limb motoneurons are different from that in humans, probably caused by the different use of the limb.[27] Last, but not the least, most studies performed in rodents, cats or opossums use juvenile to young adult animals. Since the development of the *corticospinal* tract is not accomplished in young animals, this may provide another example of why the animal model system does not necessarily simulate the conditions in human adults with SCI.[28,29]

Therefore, when considering the delayed maturation of the *corticospinal* tract, the incompatibility of standard animal models with the situation in humans, and the minor successes in regaining little, if any, normal function in human species after complete SCI, we come to the conclusion that regeneration of the *corticospinal* tract is barely possible, if not impossible. In the case of incomplete lesions, it should be the task of the physiotherapist or other rehabilitation professional to find out whether it is possible to activate the CPGs in the spine in whatever way possible. This may help regulate the excitability of the motoneuronal pool by providing it with an abundance of sensory information.[30,31] It may also optimize the CPGs that are in contact with *supraspinal* tracts so that the spinal circuitry is maintained operational in case of possible reorganization or compensation of the *supraspinal* tracts.[20,21] The person might not attain *plantigrade* walking, but being able to walk with a crutch and *digitigrade* stepping is most likely to be preferred over using a wheelchair at all times. It may be less than the expected goal of the physiotherapist to see a person walking with a rather primitive walking behaviour but in certain individuals it may be all that is realistically achievable for that lesion. Coordination of newly gained motor behaviour will have to be paired with a lot of practice – much like in the case of the human infant that practices basic motor skills in an ever-changing environment – and hopefully an alternative way of overground movement will be achieved.

Importance of endless repetitions

The knowledge that many repetitions are necessary in an intact motor system to perfect a movement[1–4] infers that for the training of coordination of a damaged CNS, the amount of therapy, training and practice must be at least the same[25,31] or even more[2] as compared to the healthy motor system.[1,2] Time-consuming therapies, which require numerous repetitions, are bound to have a higher impact on coordination than those therapies that are only performed twice per week for half an hour.

Health care professionals and family members who are able to motivate the person to practice for countless hours on their own may in some cases be the most important 'tool' for the individual with SCI. In creating new apparatuses[32] and developing therapies[13,14] that enable the person to practice on their own presents one of the biggest challenges for all those that are involved in the treatment of the person with SCI lesions. However, therapies that claim to repair the *corticospinal* tract should be cautiously considered. It is the task of the rehabilitation professional involved in the treatment of the individual with a complete SCI lesion not to reinforce unrealistic hopes by saying that with a lot of practice function will return. This would be inappropriate for the person with a complete SCI.[12] On the other hand, it is only possible, with a lot of practice, to optimize the function of incomplete SCI.[30,31]

Conclusion

In this article, we advocate intensive physiotherapy for those individuals who have an incomplete lesion with some *supraspinal* control and who are willing to invest a lot of time for practising the basic motor skills like walking. For these individuals, a functional improvement might be achieved. The hours of practice involved are innumerable, if we take the amount of practice of the normal healthy infant into account (three million steps for a 6-year-old).[1] For complete spinal cord injuries, the functional benefit of intensive training on a treadmill for overground walking is not yet proven.

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