Physiotherapy in the Spastic Syndrome

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The spastic syndrome after lesion of the CNS is not a primary impairment. Nowadays it is seen as an adaptive process, which can hinder recovery of function after a paresis as well as create secondary complications. The restructuring of the CNS after a lesion is regulated by lesion-induced plasticity but the activity or non-activity of neuronal pathways seems to contribute to this process. Therefore, to influence the recovery process, therapy in upper motor neuron syndrome should be impairment orientated. The development of so-called "positive symptoms" of the upper motor neuron syndrome needs to be delayed by avoiding stimuli which increase the tonic inputs to the motor neurons. This includes adequate handling by the nursing and therapeutic team, and use of peripheral inhibitory stimuli, in particular from joint receptors to achieve the most functional distribution of tonus between muscle chains and their antagonists. Additionally, secondary changes in muscle and tissue have to be avoided by (active) passive stretching of muscles. At the same time the negative signs of the upper motor neuron syndrome have to be treated. Activity in descending projections for voluntary movements has to be increased to regain the capability to excite motoneurons below threshold and to inhibit motoneurons of the antagonists. For this an analysis of the patient’s motor performance is especially important. Many patients experience their paresis like an external load (a feeling of abnormal heaviness of the plegic limbs). Therefore, they increasingly plan motion which is used for movements against resistance. Such motor plans include an extraordinary amount of static activity. Mainly cocontraction activity and only little reciprocal inhibition are executed to maintain stability of the body against the putative load. The efficiency of static control pathways can be increased by this, whereas there is little increase in efficiency in the inhibitory pathways. Such a behavior supports the development of muscle hypertonus, while the ability to precisely inhibit this tonus again is not facilitated. Physiotherapy for recovery of central paresis therefore has to include a behavioral therapy. With a systematic repetitive BASIS training, pathways for voluntary movements with a high proportion of reciprocal inhibition can be activated. When a patient can shorten and lengthen his/her muscles again, meaning that sufficient descending control of reflex activity has been regained, only then should postural activities be trained.