**Liste-152**

**NEURONAL MECHANISMS OF SPASTICITY**

Dimitrijevic MR, Pinter M, Ludwig Boltzmann Institute for Restorative Neurology and Neuromodulation, Hofzeile 18-20, 1190 Wien, Austria, and Department of Physical Medicine and Rehabilitation, Baylor College of Medicine, Houston, Texas, USA

Muscle hypertonia characterized by a velocity-dependent increase of tonic stretch reflex is a well recognized clinical finding in movement disorders. Sherrington's discovery that the stretch reflex generates muscle contractions, which underlie posture, gave rise to numerous experimental studies aiming at defining the underlying mechanisms of muscle hypertonia or spasticity. Magoun and Rhines proposed that spasticity is of supraspinal origin and the result of an impaired balance between facilitatory and inhibitory descending influences through the spinal cord. Moreover, there is clear evidence in monkeys and humans that spasticity can be observed after lesions of the primary motor cortex or the premotor cortex. The development of the concept of "premotor center" of the spinal cord and the demonstration of convergency of different descending tracts to the "premotor center" led to the understanding that we should not place any emphasis on various neuronal descending systems since these are parallel channels, which converge upon the premotor center, including the propriospinal and segmental interneuronal pathways.

In order to demonstrate how the propriospinal and segmental interneuronal systems are involved in generating muscle hypertonia and hypotonia, under simulated "supraspinal control", we shall present the results of our studies on externally controlled afferent input to the spinal cord in subjects with complete and incomplete chronic spinal cord injury.

Epidural spinal cord stimulation can generate afferent input to the segmental interneuronal and propriospinal spinal cord systems. By delivering a train of stimuli of different frequencies and amplitudes to the posterior structures of the spinal cord, we have been able to demonstrate alteration in muscle tone and induce reflex responses and locomotor-like movement. Thus, we shall provide evidence that in order to generate spasticity it is essential to have additional supraspinal input.