Summary

Remarkable progress in therapy and diagnostics of neurological diseases, including brain and spinal cord injuries, has been achieved during last years. However, the nervous system with its complicated feedback circuits systems reaching from the periphery to the brain remains still unclear to our knowledge. Regeneration following serious injuries of the peripheral or central nervous system is often incomplete, leading to its poor functional outcome. Thus for the patients it means to become reconciled with a permanent disability. It is caused by limited recovery of the nervous system. Physiological circuits and neuronal nets are altered by an injury. In our study we try to elucidate some kind of an adaptation process of the peripheral and spinal nervous system using some of the neurophysiological methods. We studied the spinal inhibitory circuits and its afferent and efferent pathways and its role in acute and chronic lesions of the nervous system. We tried to reveal its potential capability in nervous system recovery. Our research includes several topics which have not been published yet.

We focused on a spinal inhibitory reflex namely a cutaneous silent period in several conditions. In physiological conditions, we studied the influence of temperature on the cutaneous silent period; in pathological conditions we tried to utilize our findings in brachial plexus injuries. Foremost, clinical research leads to attempt an implementation of our findings to the clinical practice.

Influence of temperature on large-diameter fiber conduction is well known, as cooling significantly reduces a conduction velocity. There is lack of knowledge about the influence of temperature on small-diameter fibers, especially on A-delta fibers. Thus we analyzed the influence of temperature on both large-diameter and A-delta fibers by testing the spinal inhibitory reflex (cutaneous silent period) which is formed by activation of these small-diameter fibers. We confirmed that the velocity conduction is getting affected by changing temperature

in large-diameter fibers. We proved also its influence on A-delta fibers. We conclude that limb temperature should be taken into account when testing the cutaneous silent period in clinical setting, as different limb temperature affects latencies of the cutaneous silent period as well as function of the large-diameter fiber conduction.

We studied this spinal inhibitory reflex in severe brachial plexopathies and tried to establish its role as a new diagnostic tool in these patients. We hypothesized that the cutaneous silent period is absent in severe traumatic lesion, especially in a case of damaged afferent part of the appropriate cervical root. Surprisingly, the cutaneous silent period was still present in most of brachial plexus lesions. Thus we focused on traumatic root avulsions. We tried to use this neurophysiological method to confirm a particular root avulsion of C5, C6, C7, and C8, respectively. Preserved cutaneous silent period was present in most of these cases. Root avulsion seems not to be overall associated with absence of the cutaneous silent period in an appropriate dermatome thus partial presence of the afferent fibers regardless particular dermatome has to be present. Only severe multi-segmental root avulsion leads to loss of the cutaneous silent period. Thus this method cannot be recommended as a reliable test for diagnosis of a single root avulsion. Our results are complementary with other studies where absence of the cutaneous silent period in cervical myelopathy was present. Preservation of this reflex in brachial plexus palsies indirectly confirms well preserved functional spinal cord integrity. The clinicians may use this simple spinal inhibitory reflex as a physiologic aid in brachial plexus injuries to exclude spinal cord lesions.