A close relationship between painful and non-painful (somatosensory) percepction was noticed already in the past centuries and led into development of many analgesic methods. Only basic neuroanatomical and neurophysiological research using animal models of nociception was able to shed more light on their proper mechanisms until the era of modern non-invasive imaging methods.

The main aim of this thesis was to analyze non-invasively, in human volunteers and patients with chronic pain, spatiotemporal relations between brain evoked responses to painful (or aversive) and non-painful stimuli. In next step, to discuss the roles of different engaged mechanisms in found interactions and suggest recommendations for further research of pain.

4 experimental studies (3 in healthy volunteers and 1 in patients with failed back surgery syndrome) are presented. Using high-resolution EEG, phasic electrical stimulation of median, tibial or sural nerve(s), and source analysis of recorded data, modulations of all repersentative components of somatosensory evoked potentials (SEPs) by several interfering conditions were analyzed. In healthy volunteers, effects of heterotopic repetitive heat pain administered to the right side of the body (ipsilateral to electrically stimulated nerve) were tested in Experiment 1 (compared to the same-site non-painful thermal stimulation) and Experiment 2 (compared to heat pain administered to the left side of the body). In Experiment 3 we tried to isolate endogenous anticipatory and/or attentional modulations by using homotopic target (aversive or non-aversive) stimuli administered in predictable (similar to Expeirments 1 and 2) or unpredictable manner. In Experiment 4 the modulatory effects of spinal cord stimulation (SCS) on painful (n. suralis) and non-painful (n. tibialis) SEPs were analyzed.

In the same extremity administration, both phasic and tonic inhibitory effects of pain on short-latency (exogenous) cortical SEPs were found. Heat pain administered contralaterally to the tibial nerve electrical stimulation led to phasic reduction of short-latency SEPs only. Mid- and long-latency (endogenous) components were modulated in more complicated way – both augmenting and attenuating effects of painful and non-painful heat were found. Anticipation of target stimuli led to reduction of short-latency and facilitation of mid- and long-latency SEPs. Augmenting effects of anticipation on mid- and long-latency SEPs were enhanced in unpredictable (compared to predictable) blocks of Experiment 3. During SCS, robust reduction of most cortical SEPs was seen. However, the response of midcingulate cortex in painful sural nerve stimulation was enhanced by SCS.

Results suggest parallel engagement of both bottom-up and top-down influences in observed modulations. We emphasize the hypotheses of interference between nociceptive and non-nociceptive processing on the cortical level. In anticipation, similar attenuating effects compared to pain probably result from endogenous activation of descending inhibitory systems. On the other hand, many reported augmenting effects point to the key role of increased attention in processing of aversive stimuli. In SCS, enhanced cingulate SEP points to the active involvement of cerebral cortex in its analgesic mechanisms. Implications for further pain research are discussed.