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Pain Control

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Preface

Current pain treatment is successful in many patients, but nevertheless numerous problems have to be solved because still about 20 % of the people in the population suffer from chronic pain. A major aim of pain research is, therefore, to clarify the neuronal mechanisms which are involved in the generation and maintenance of different pain states, and to identify the mechanisms which can be targeted for pain treatment. This volume on pain control addresses neuronal pain mechanisms at the peripheral, spinal, and supraspinal level which are thought to significantly contribute to pain and which may be the basis for the development of new treatment principles. Chapters on nociceptive mechanisms in the peripheral nociceptive system address the concept of hyperalgesic priming, the role of voltage-gated sodium channels in different inflammatory and neuropathic pain states, the hyperalgesic effects of NGF in different tissues and in inflammatory and neuropathic pain states, and the contribution of proteinase-activated receptors (PAR) to the development of pain in several chronic pain conditions. Chapters on nociceptive mechanisms in the spinal cord address the particular role of NO and of glial cell activation in the generation and maintenance of inflammatory and neuropathic pain, and they discuss the potential role of local inhibitory interneurons, of the endogenous endocannabinoid system, and the importance of non-neuronal immune mechanisms in opioid signaling in the control of pain. Furthermore, it is presented how spinal mechanisms contribute to the expression of peripheral inflammation. At the supraspinal level, the role of the amygdala and their connections to the medial prefrontal cortex in pain states are addressed. A particular chapter discusses the experimental methods to test central sensitization of the nociceptive system in humans. Finally, differences and similarities of the neuronal systems of pain and itch are reported. Altogether, the chapters demonstrate that both the concentration on single key molecules of nociception and the interference with disease-related mediators may provide novel approaches of pain treatment.

Jena, Germany — Hans-Georg Schaible

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