Wissenschaftlicher Verein Muskel und Schmerz
The Meeting of the Scientific Association for Muscle and Nerve
Bernreid, Germany
May 2012

The scientific meeting of the "Wissenschaftlicher Verein Muskel und Schmerz" (Scientific Association for Muscle and Nerve) took place at the beginning of May in the Schloss Höhenried, a lovely location on the Starnberg Lake near Munich, Germany. This Association brings together German and Swiss scientists interested in the study of the pathophysiology and therapy of muscle pain. The program comprised four topical reviews and two scientific presentations by members and invited speakers. The topical reviews addressed the involvement of the autonomous system in pain (W. Jänig [Kiel] and W. Magerl [Mannheim]), the activity of microglia in spinal cord lesions (E.D. Schomburg [Göttingen]), the relationship between psychological profiles and low back pain (M. Hasenbring [Bochum]), while the two scientific presentations discussed new data on quantitative sensory testing (QST) in temporomandibular disorder (TMD) patients (D. Pfau [Mannheim]) and the role of muscle fasciae in the etiology of myofascial pain (U. Hoheisel [Mannheim]).

There is a close relationship between the nociceptive and the autonomic systems at all levels of the nervous system, and the pain experience is accompanied by a powerful stereotyped modulation of diverse motor functions involving the autonomic and neuroendocrine systems through circuits that include the spinal cord, brainstem, hypothalamus, amygdala, and other brain centers. For instance, subdiaphragmatic vagal afferent activity modulates mechanical nociceptive threshold and inflammatory mediator-induced hyperalgesia via the hypothalamo-pituitary-adrenal axis. Indeed, ongoing activity in vagal afferents inhibits the release of epinephrine from the adrenal medulla. Thus, removal of this tonic activity results in disinhibition of inputs to the adrenal medulla, leading to an increase of plasma epinephrine, a decrease of the mechanical nociceptive threshold, and an enhancement of bradykinin-induced hyperalgesia. Furthermore, vagal activity may represent the link between acute stress, sympathetic activation, and analgesia via a baroreflex mechanism that physiologically counteracts sympathetic activation. A decrease in baroreflexes parallels a decrease in vagal activity.

It is well known that a noxious stimulus activates a large neural brain network. Many of these areas also participate in the regulation of the function of the autonomic nervous system. New data indicate that the autonomic responses produced by the anticipation of pain and the experience of pain share common central neural networks similar to those known to be active in pain experience and pain anticipation. It was also pointed out that sympathetic and motoric reactions are spinally coupled, that the nociceptive muscle vasoconstriction leads to hypercapnia, and that pain is not a stress caused by hypercapnia; it does not lead to a significant cardial and sudomotor reflex activity but to a pronounced tonic vasoconstriction without habituation to repeated nociceptive stimuli.

Microglia and astrocytes play central roles in inflammatory processes in the central nervous system and rapidly respond to neural lesions. Within a few minutes from an experimentally produced spinal cord lesion, microglial cells begin sending processes toward the lesion forming a "shielding" ring around the lesion leading to microgliosis. This microglial migration is triggered by both the nitric oxide and adenosintriphosphate gradient in the lesion vicinity. Microglial cells and astrocytes behave differently: the microglia cells become active first and remain active for up to 28 days postlesion, while the astrocytes are initially inactive and become activated 3 months postlesion. Dr Schomburg hypothesized that axonal regeneration in the spinal cord may be prevented by the microgliosis, raising the question of whether an administration of methylene blue (that inhibits the synthesis of nitric oxide) could hinder microgliosis and thereby favor axonal repair.

The avoidance-endurance model of pain (AEM) differentiates between fear-avoidance patients and pain-endurance patients and postulates different psychological and behavioral responses to pain that may influence the transition from acute to chronic pain. A 6-month prospective study supported this hypothesis by showing that the baseline psychological characteristics of patients with unspecific back pain could predict the development of chronic pain. At baseline, the patients were divided

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into three groups: the fear-avoidance, the distress (negative stress)-endurance, and the eustress (positive stress)-endurance patients. The fear-avoidance patients scored higher in pain catastrophizing, the distress-endurance patients displayed elevated anxiety/depression and helplessness/hopelessness, while the eustress-endurance patients had the highest humor/distraction scores, pain persistence, and positive mood despite pain. At the 6-month follow-up, all three maladaptive groups revealed a higher pain intensity than the adaptive patients, while disability was elevated only in the fear-avoidance and distress-endurance patients. As the psychosocial and behavioral responses associated with chronic pain are common to diverse samples of pain patients despite differences in somatic diagnosis, it is likely that the AEM model of pain can be applied also for orofacial pain patients in order not only to tailor their management but also to diagnose early the patients who are at risk to develop chronic pain.

QST is used to diagnose and assess the severity of nerve damage, allowing the diagnosis of many different types of neuropathies, including peripheral neuropathies. It can also be used to try inferring the pain mechanisms involved in different TMD patients. The TMD patients were first classified as sensitive or insensitive based on their tender point score (cutoff of 10 tender points). The two patient groups did not differ as far as psychological parameters and pain duration and all patients did not fulfill the diagnostic criteria for fibromyalgia (FM). The sensitive patients were more sensitive compared to healthy controls and to insensitive TMD patients as far as their QST profile for face, hand, and back, similar to additionally tested FM patients. However, sensitive TMD patients had a short pain duration, which argues against a transition from TMD to FM over time. Data rather suggested an overlap in pathophysiology between the FM and sensitive TMD patients, eg, a disturbance of central pain processing in the sensitive TMD patients.

A question that has been raised in recent years is whether muscle fasciae are a potential source of myofascial pain. The rat and human thoracolumbar fascia (TLF) is densely innervated with substance P (SP)- and calcitonin gene-related peptide (CGRP)-positive free nerve endings, most of which are located in the outer layer of the fascia and the subcutaneous tissue. In naive rats, about 10% of dorsal horn neurons in the spinal segments Th13-L2 receive input from the TLF, most of which receive additional input from deep tissues (muscles) as well as from the skin. The number of neurons with a receptive field in the TLF rises significantly after an experimentally induced inflammation of a low back muscle (multifidus). Moreover, neurons in spinal segment L3, that do not receive input from the fascia in normal animals respond to fascia in animals with inflamed muscle. A similar behavior occurs after nociceptor sensitization without inflammation, eg, after nerve growth factor (NGF) application to the multifidus muscle. Indeed, a second NGF injection performed 5 days after the first one leads to an increase of dorsal horn neurons with input from deep tissues and from the TLF. These data suggest that the nociceptive input from the thoracolumbar fascia may contribute to the pain in patients with low back pain.

In accordance to the tradition of the Association, ample time was left for formal and informal discussions so that the participants had enough time to interact and discuss the topics in a relaxed atmosphere. A very pleasant and enjoyable concert completed the scientific meeting.

Sandro Palla
Associate Editor

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