neuropathic component. Evidence supporting the performance of these tools in this context will be reviewed, and the strengths and limitations of this approach will be discussed.

Until consensus is agreed on a diagnostic approach to neuropathic pain, screening tools will serve to identify potential patients with neuropathic pain, particularly by non-specialists and this is probably their chief clinical strength. Their ease of use make these tools attractive because they provide immediately available information. Screening tools fail to identify about 10–20% of patients with clinician diagnosed neuropathic pain indicating that they may offer guidance for further diagnostic evaluation and pain management but clearly, they do not replace clinical judgment.

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QUANTITATIVE SENSORY TESTING: ASSESSMENT OF THE NEUROPATHIC COMPONENT IN LOW BACK PAIN

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Background and aims. In order to assess an impaired sensory function in neuropathic back pain the QST protocol of the German Research Network on Neuropathic Pain (DFNS) was used.

Methods. The QST protocol of the DFNS consists of seven tests measuring 13 parameters: thermal detection thresholds for the perception of cold, warm and paradoxical heat sensations, thermal pain thresholds for cold and hot stimuli, mechanical detection thresholds for touch and vibration, mechanical pain sensitivity including thresholds for pinprick and blunt pressure, a stimulus/response-function for pinprick sensitivity and dynamic mechanical allodynia, and pain summation to repetitive pinprick stimuli.

Results. Patients with different back pain syndromes were assessed: Facet joint arthropathy, and other radicular or pseudoradicular back pains. Back pain patients presented different sensory plus signs, i.e. pinprick-hyperalgesia, hyperalgesia to cold or blunt pressure, dynamic mechanical allodynia. Many patients showed sensory minus signs as well, i.e. hypoesthesia or hypoalgesia to thermal and mechanical stimuli. In the case of a radicular lesion A-fiber function was gradually reduced following the rule "A-beta > A-delta", while C-fiber function was almost preserved.

Conclusions. QST data from back pain patients show similar somatosensory phenotypes. Sensory plus signs indicate that peripheral or central sensitization of nociceptive pathways contributes to the back pain. Sensory minus signs most likely reflect a nerve damage pointing to the presence of a neuropathic pain component. The mixture of both sensory plus and minus signs is consistent with the mixed pain concept of nociceptive and neuropathic components in many back pain syndromes.

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TREATMENT OF NEUROPATHIC PAIN: INTERVENTIONAL TREATMENT OPTIONS

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Treatment of chronic, neuropathic low back pain continues to pose a burden for patients, although significant scientific advances in delineating pathophysiologic mechanisms have facilitated the development of targeted pharmacological and interventional treatments. 7% of low back pain patients experience associated neuropathic pain and although the exact numbers are unknown, it is believed that an estimated 5–10% of patients will be refractory to the majority of modalities used to treat chronic low back pain. The treatment algorithm for interventional therapies for neuropathic pain should be; failure of refractory treatments and combination therapy.

Interventional therapies considered for neuropathic low back pain are:

Neuroablative procedures
Nerve blocks.
Percutaneous RF techniques.

Neuromodulation
Spinal infusion.
Stimulation of the CNS or PNS.

Pulsed radiofrequency

Local anesthetic peripheral and sympathetic blocks provide useful diagnostic information but tend to afford only temporary therapeutic benefits in patients with peripheral neuropathy.

Despite the wide use of sympathetic nerve blocks in different pain syndromes, no substantive review in the literature on their role in pain treatment; a degree of multimodal therapy is inherent in most studies which may be beneficial for the patient but masks the outcome assessment measures.

Percutaneous radiofrequency techniques like dorsal root ganglion rhizotomy are also used for the palliation of pain in several neuropathic pain syndromes. A systematic review within the framework of the Cochrane

Collaboration Back review group shows that there is limited evidence that RF-DRG is more effective than placebo, conflicting evidence for RF of lumbar facet pain, limited evidence suggesting that intradiscal RF may not be effective in relieving discogenic pain.

Recently a new mode of radiofrequency, pulsed radiofrequency have been appearing in the literature. PRF was conceived as a novel, potentially safer mode of administration of RF energy. In order to further elucidate the mode of action of PRF and to define its true value in the management of chronic pain, more research on this promising technique is justified.

When these techniques do not sustain adequate pain relief, spinal cord stimulation (SCS) or intrathecal therapy represent reasonable options. The level of evidence supporting this modality remains moderate. Pooled results of a recent systematic review of the SCS literature (one RCT, one cohort, 72 case studies) suggest significant benefit, with 50% or greater improvement in pain relief, in roughly 62.5% of patients.

A final option is intrathecal therapy. An important challenge posed by intrathecal delivery of medications is an inability, thus far, to address the correct target in the spinal cord. The only drug approved for use in intrathecal therapy is morphine and ziconitide (although hydromorphone and fentanyl, and the alpha-2 agonist clonidine are routinely used in clinical practice). While there is evidence for long-term analgesic efficacy, patient selection is critical and should be based on objective evidence of nonreversible pathology, coupled with a failure to achieve adequate results from oral upload therapy and/or an inability to tolerate the side-effects of oral opioids. In a recent review of 297 articles related to intrathecal drug therapy, investigators concluded that the scientific evidence for efficacy is incomplete.

In this lecture all efficacy of all techniques mentioned will be presented on the basis of evidence based medicine.

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Workshop – Basic Sciences 3: TRANSIENT RECEPTOR POTENTIAL CHANNELS – AN UPDATE

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Workshop Summary: TRANSIENT RECEPTOR POTENTIAL CHANNELS – AN UPDATE

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The transient receptor potential (TRP) channels constitute a large and diverse family of channel proteins that are expressed in many tissues and cell types in both vertebrates and invertebrates. Recently, TRP channels have collected much attention as molecular gateways in sensory systems, an interface between the environment and the nervous system. Several TRP channels transduce thermal, chemical, and mechanical stimuli into inward currents, an essential first step for eliciting thermal and pain sensations. Precise regulation of the expression, localization, and function of the TRP channels is crucial for their sensory role in nociceptor terminals, particularly after tissue damage or inflammation. In this workshop, three speakers will talk about recent findings about TRP channels. Dr. Tominaga will have a short review of TRP channels and also his new findings of warm sensitive TRP channels. Dr. Reeh will talk about his findings about inflammation-mediated changes in single-fiber activity using TRPV1 knockout mice. Dr. Koltzenburg will talk about cold sensitivity of sensory neurons, especially other cation channels in addition to TRPM8.

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THERMOSENSITIVE TRP CHANNELS AND NOCICEPTION

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TRP (transient receptor potential) channels were first described in Drosophila in 1989, and in mammals, TRP channels comprise six related protein families (TRPC, TRPV, TRPM, TRPA, TRPML, TRPP). TRP channels are best recognized for their contributions to sensory transduction, responding to temperature, nociceptive stimuli, touch, osmolarity, pheromones and other stimuli from both within and outside the cell. Among the huge TRP super family of ion channels, some have been proven to be involved in thermosensation detecting ambient temperatures from cold to hot. There are now nice thermosensitive TRP channels (TRPV1, TRPV2, TRPV3, TRPV4, TRPM2, TRPM4, TRPM5, TRPM8 and TRPA1) with distinct temperature thresholds for their activation. Interestingly, some of the thermosensitive TRP channels are expressed specifically in sensory neurons and involved in nociception. Involvement of TRPV1, TRPV3, TRPV4 and TRPA1 in nociception has been confirmed at an animal level using mice lacking the TRP channels. I will summaries the recent progress in the research of thermosensitive TRP channels and nociception especially by focusing on TRPV1 and TRPA1. In addition, I will discuss about the physiological significance of warmth sensitive TRP channels (TRPV3, TRPV4, TRPM2, TRPM4 and TRPM5).