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JA SYMPOSIUM

Recent advances in pain medicine: from bench to bed; August 17, 2009, Kobe, Japan

Opening remarks

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In late 2000, the US Congress passed into law a provision, which the President signed, that declared a 10-year period as the Decade of Pain Control and Research which began from January 1, 2001. The American Pain Society has actively supported the Decade of Pain Control and Research, and it has been a focal point for the development of numerous programs to heighten awareness and treatment of pain and research funding. Cutaneous sensation produced by noxious and innocuous stimuli is transmitted to the dorsal horn of the spinal cord via primary afferent fibers and recognized as pain and touch in the somatosensory cortex. While acute pain has a fundamental role as a protective system against the occurrence or threat of injuries and diseases, chronic pain associated with diseases such as postherpetic neuralgia, and rheumatoid arthritis often outlasts its biological usefulness and in itself causes functional disturbance. Neuropathic pain is a significant and largely unresolved medical problem characterized by spontaneous pain, hyperalgesia, and tactile pain (allodynia). Over several decades, non-steroidal anti-inflammatory drugs (NSAIDs) and opioids have been used for clinical management of pain. Neuropathic pain is persistent and can be particularly problematic because they are often poorly managed by NSAIDs and conventional opioid analgesics. Kohno demonstrated that although excitatory transmission remained intact, presynaptic GABA release was reduced in lamina II neurons of an isolated adult rat spinal cord slice preparation in neuropathic pain models. He also reviewed the role of neuron–microglia interactions in the spinal cord in neuropathic pain [1].

Pain associated with cancer, particularly when tumors metastasize to bone, is often severe and debilitating. Better understanding of the neurobiological mechanisms underlying cancer pain are likely to lead to the development of more effective treatments. Kawamata et al. demonstrated that transient receptor potential vanilloid subfamily 1 (TRPV1) was up-regulated, and the μ opioid receptor was down-regulated in peripheral neurons in bone cancer. Activation of spinal cannabinoid receptor type 1 (CB1) has been shown to inhibit glutamatergic excitatory postsynaptic currents in the spinal cord slices of naïve rats and C-fiber-evoked neuronal response of the dorsal horn. He found that CB1 was located in the axon terminals of excitatory spinal interneuron, and its expression is preserved in bone cancer. Furthermore, he reported that spinal CB1 activation by an exogenously administered CB1 agonist reduced bone cancer-related pain. Therefore, spinal

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CB1 activation might be effective, and the reduced analgesic effect of morphine could be rescued by its combination with a TRPV1 antagonist for treatment of bone cancer pain [2].

A common cause of persistent pain and hyperalgesia in humans is post-operative pain. Tissue damage associated with surgery initiates a local change in the sensitivity of nociceptors and also triggers central sensitization, an increase in the excitability of neurons in the spinal cord, which may contribute to the severity of post-operative pain. Inoue discussed the benefits and risks of epidural analgesia (EA) and intravenous patient-controlled analgesia (PCA), the clinical implication of EA, and the future direction of multimodal post-operative analgesia. He demonstrated that intravenous NSAID plus the combination of local anesthetic and opioid in epidural bolus before continuous epidural infusion (CEI) and the addition of PCA to CEI was a better choice for epidural post-operative analgesia with concomitant reduction of side effects [3].

Deafferentation pain is clinically defined as pathological pain that is associated with a partial or complete loss of sensory input from a portion of the body following lesions in somatosensory pathways (e.g. phantom limb pain and nerve injury-associated pain). Patients with deafferentation pain complain of a complex quality of pain, and its treatment can be difficult. Understanding on how the brain changes during chronic pain or responds to pharmacological or other therapeutic interventions has been significantly changed by developments in neuroimaging of the central nervous system. Functional magnetic resonance imaging (fMRI) has been used to map cerebral activations related to nociceptive stimuli in rodents. Sumitani et al. demonstrated that mirror visual feedback (MVF) treatment could improve deafferentation pain and visually induced motor imagery by MVF was more effective for reducing deep pain rather than superficial pain. They also discussed the relationship between MVF treatment and spinal cord stimulation therapy using fMRI [4].

We hope that today's discussions will be a step towards developing strategies for the prevention and treatment of neuropathic pain.

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