Neuropathic Pain

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This congress was organized by the French chapter of International Association for the Study of Pain (IASP). It was focused on a topic, neuropathic pain, which is changing rapidly at the moment. It was held in French but only English translations will be given in this summary, which reports the 3 lectures from a plenary session that referred to taxonomy, epidemiology, and physiopathology. Although these 3 lectures did not consider orofacial pain per se, they are very relevant to it.

There are many chronic orofacial conditions that are progressively better understood and whose neuropathic nature is established, suspected, or discussed. A neuropathic mechanism is established for trigeminal neuralgia and neuralgic pain after a traumatic injury of large nerve trunks (ie, posttraumatic neuralgia of the lingual or inferior alveolar nerve). Although they do not display the typical neurologic symptoms (anesthesia, allodynia, and hyperesthesia in a nerve trunk territory), atypical odontalgia and atypical facial pain also express some neurologic symptoms. However, stomatodynia (burning mouth syndrome) is different, since the pain is bilateral and does not follow any nerve trajectory, but its does display some neuropathic characteristics that are increasingly recognized. At the other extreme of the continuum, myofascial pain and fibromyalgia are characterized by diffuse symptoms, with no clear nerve lesion but with a clear abnormal functioning of the nervous system. This raises a question—what is neuropathic and what is not? This question applies to pain in the trigeminal region as well as in the rest of the body and was at the basis of the 3 lectures.

Nadine Attal addressed the taxonomic context of neuropathic pain. She first referred to the present definition provided by the IASP which defined neuropathic pain as "pain initiated or caused by a primary lesion or dysfunction in the nervous system." She pointed out that this definition has been useful in distinguishing some characteristics of neuropathic and nociceptive types of pain but is now considered too loose. For example, this definition does not help distinguish normal activation (eg, by central sensitization) from true neuropathic dysfunction resulting from physiologic neuroplasticity. A more precise definition has been recently proposed by a group of experts: "neuropathic pain arises as a direct consequence of a lesion or disease affecting the somatosensory system."[1] In this new form, the terms "dysfunction" and "nervous system" would be replaced by "disease" and "somatosensory system," respectively. In the orofacial field, this would mean that symptoms of muscle pain resulting from spastic or dystonic dysfunctions would not be considered as being due to a neuropathic condition. Stomatodynia would be considered neuropathic if the lesion or disease could be established. Then Dr Attal pointed to the lack of validated diagnostic criteria to establish a diagnostic tool for neuropathic pain but emphasized existing new tools based on several European and North American questionnaires that are important for helping first-order care providers in treatment choice and for clinical research. The recent French neuropathic pain diagnostic questionnaire (DN4)[2] has shown that 5% of the general population suffers from moderate to severe neuropathic pain. Finally, she noted that both characteristics of the lesion, as shown by these tools, and location of the symptoms are useful for clinical diagnosis.

Jean Bruxelle presented the results of an epidemiologic study sponsored by the French chapter of IASP. This study was not based on questionnaires but the data were obtained through direct observation of 1,397 successive patients seen at their first visit in 19 pain centers, 29 pain units, and 40 other consultation sites. About 400

patients (30%) were found to have neuropathic pain with equal proportions of patients with pure neuropathic pain and neuropathic mixed with nociceptive pain. These neuropathic pain patients had their pain for 2 years on average. The neuropathic pain was either central (10%) or peripheral (90%). The central neuropathic pain was mostly due to stroke, multiple sclerosis, or syringomyela, and the peripheral neuropathic pain was directly related to surgery in 25% of the cases, to nonoperated radiculopathy in 16%, and to operated radiculopathy in 14%. Altogether, surgery preceded or was related to the neuropathic pain in 47% of the cases. Classical trigeminal neuralgia and post-zoster pain accounted for only 3% each. Therefore, this study emphasized the role of surgery in the occurrence of neuropathic pain. Although orofacial pain was not isolated in the study from the other neuropathic pain conditions, there are other studies that have shown that the impact of oral surgery on the incidence of neuropathic pain after nerve injury has been underestimated.[3] In addition, there is a strong possibility that chronic orofacial pain with a neuropathic component of varying importance may occur after common dental procedures such as pulpectomy and tooth extraction.

Didier Bouhassira discussed the clinical relevance of animal models for neuropathic pain. He underlined the gap between neuropathic pain in humans and what can be observed in animal models for neuropathic pain. He began by reminding the audience that effective control of neuropathic pain is poor, since only 40% of the patients receive satisfactory pain control.[4,5] This is in spite of the huge number of both pharmacologic and physiologic studies published during the 2 last decades. That animal models have been only partly useful from a pharmacologic standpoint is shown by the small number of new effective drugs that have been introduced as a result of research. In addition, most drugs, such as gabapentine, pregabalin, or lidocaine patch, belong to an old class of pharmacologic agents. One reason for the relative failure of animal models of neuropathic pain may be found in the weak reproduction of human symptoms in animal models. Provoked pain is only partially reproduced, and spontaneous pain is usually not present, although it is a dominant symptom in human neuropathic pain. Nonetheless, these models have been useful for the study of pain mechanisms, although most of the mechanisms have not been confirmed in humans. There is one noticeable exception: ectopic discharges have been recorded in humans by microneurography. The meaning of this exception is not clear, however, since ectopic discharges are present in diabetic neuropathies not only in patients with pain but also in those without pain. These discharges could therefore be due to the nerve lesions rather than to the pain itself.[6] The differences between humans and animals in the outcomes of pharmacologic trials are also intriguing. Many substances, including some nonsteroidal anti-inflammatory drugs, have been shown to be very effective in animals and totally inefficient in humans; this suggests that animal models are characterized by a very high sensitivity and a very low specificity. Didier Bouhassira concluded by noting the increasing gap in communication between basic scientists and clinicians and by expressing his view that more substantial interactions and willingness to cooperate represent a most important challenge in the near future.

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