Seventy-Seventh General Session of the International Association for Dental Research

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The 1999 General Session of the IADR was held in Vancouver, British Columbia, where the transition from winter to spring this year was accompanied by a great deal of rain and wind. This was extra incentive to follow closely the organized symposia and the vast numbers of oral and poster presentations during the meeting. The meeting took place at the imposing Vancouver Trade and Convention Centre at the waterfront. The organization was excellent, and there were 3,600 abstracts and more than 6,000 participants.

This year's annual session was preceded by a day-long satellite symposium that brought together experts in the fields of orofacial pain and dysfunction, each of whom was working on a chapter of a book. This book, which will provide state-of-the-art knowledge on these topics for the dental curriculum, is intended for use by dental students at the undergraduate level. The symposium consisted of the authors' presentations of their chapter overviews, which were then critiqued and discussed. Dr Hargreaves' editorial in this issue of the journal also refers to this event.

During a coinciding session on neuroscience, the application of irritating substances to tooth pulp (mustard oil by Nomura et al, bacterial lipopolysaccharide by Chaisin et al, J Dent Res 1999;78:117) was reported to induce C-fos expression in the brain stem, especially in the peri-obex region and in subnucleus caudalis/interpolaris, implicating these regions in the transmission of dental nociception. When the inflammatory agent mustard oil was injected into the temporomandibular joint (TMJ) of cats, fos-like immunoreactivity was observed in the ipsilateral paratrigeminal nuclei and the dorsal and ventral part of the subnucleus interpolaris. The absence of labeling in the rostral part of this nucleus, in the subnucleus oralis, or in the chief sensory nucleus, suggests that the TMJ afferents that project directly to the dorsal rostral nuclei probably involve larger-diameter fibers that are not involved in nociception (Ellis and Capra, J Dent Res 1999;78:117).

Another symposium presented research on neuronal modulators of wound healing. P. T. Marucha discussed the ways in which neuronal factors could influence the process: they are involved in the recruitment of inflammatory cells, in the regulation of blood flow via epinephrines and steroids, and in the regulation of immunologic processes, through cytokines. Experiments in rats and humans illustrated that stress may affect chemotaxis (IL-8), neutrophil function, and wound healing also in the clinical situation, thus increasing the possibility of infection. It remained unclear whether sleep disturbances could cause similar delays in wound healing, either by themselves or via the stress that accompanies them. K. M. Hargreaves underlined the necessity of the integrity of the peripheral neuronal system in providing appropriate regulatory mechanisms for neurogenic inflammation and healing, both of which involve such neuropeptides as substance P and CGRP. If the peripheral tissue is damaged (eg, by capsaicin application or desensitization) a decrease of up to 80% in healing capacity can be observed; this is expressed as decreased angiogenesis. In his lecture, Dr Hargreaves also illustrated the effect of cannabinoids and anandamine, whose peripheral receptors are functionally active in peripheral pulp tissue (bovine and human). Anandamine appears to inhibit CGRP release in peripheral tissue, and it also inhibits plasma extravasation in vitro. As such, these products and their receptors might be

important in mediating (decreasing) peripheral nociception during hyperalgesia, in addition to their central effects.

M. R. Byers lectured on the differing responses of pulp tissue according to the degree of damage, from reversible changes to necrosis. Injury and inflammation lead to enlarged receptive fields, and while mild injuries can be repaired via nerve sprouting and healing, deeper irritation will lead to vasodilatation and the formation of reparative dentin. A continued irritation will cause neutrophil attraction and continuous inflammation, which in the chronic state can also incite periapical nerve effects. Injury especially is expressed as an increase in nerve growth factor (NGF), which regulates the synthesis of substance P, CGRP, and other mediators. It appears that NGF is carried by fluid produced by fibroblasts into the dentin and odontoblasts, influencing dentinal repair. Also at the level of the trigeminal ganglion the receptors for NGF change, and c-fos expression in the brain stem may alter, depending on which tissue is stimulated.

In the concluding lecture of this symposium, J. F. Sheridan elucidated the relationship between the nervous system, the endocrine system, and the immune system. When stress activates the central nervous system, a neurocrine modulation of gene expression during inflammation is observed, which decreases wound healing. Stress, on the other hand, is an individualistic response, which leads to variable increases in plasma cortisone levels. The adrenocorticotrophic hormone (ACTH) reaction to the creation of a wound is greatly increased when stress is increased, for example, in rats that are restrained; this necessitates longer healing times. A normalization of healing time is observed when the ACTH receptor is blocked. Corticosterone inhibits the "trafficking" of inflammatory cells through its influence on proinflammatory cytokines and chemokines. To what extent these new insights into the role of stress in healing and neurogenic inflammation are already transferable to the clinical situation is unclear.

Some presentations focused on clinical studies, discussing, for example, the positive effect of continuous positive airway pressure (Morrison et al, J Dent Res 1999;78:213) and a mandibular protrusion device (Fransson et al, J Dent Res 1999; 78:213) in sleep apnea patients. A controlled study showed that sleep bruxers are neither more vigilant nor more prone than controls to react to a motor command during the daytime (Rompré et al, J Dent Res 1999;78:213). Fricton et al (J Dent Res 1999;78:272) reported their preliminary findings on long-term clinical outcomes, mandibular dysfunction, pain, and imaging changes in groups of patients who had undergone nonsurgical, surgical, or implant-surgical treatment for disc displacement 7 years prior. Clinical and radiologic changes were more apparent in the surgically treated patients, and surgery that involved implants was associated with more severe changes, as observed on magnetic resonance images. Sommers et al (J Dent Res 1999;78:271) associated tinnitus with tenderness to palpation of the lateral pterygoid area, which led them to speculate that a prolonged protrusion of the mandible could be involved, although an even more important finding of their study was that persons reporting ear symptoms may have a general tendency to report more physical symptoms. A Swedish study (Wolf et al, J Dent Res 1999;78:272) reported pain relief in 75% of patients with long-lasting pain, 4 to 9 years after the first examination. Truelove et al reported that in a randomized clinical trial on the use of nonsplint conventional treatment, soft-splint treatment, and hard-splint treatment, no significant differences regarding clinical signs and symptoms could be observed after 12 months; the splint therapy (soft or hard) was in no way superior to the nonsplint conventional treatment (Truelove et al, J Dent Res 1999;78:292).

With respect to TMJ disc displacement with reduction, a comparison was made between treatment using a mandibular flat plane hard splint, a maxillary anteriorly positioned splint worn during the night, and a sham palatal splint, in addition to behavioral treatment and exercises. No differences were observed between the 3 groups after 3, 6, or 9 months (Anderson et al, J Dent Res 1999;78:292). Several studies focused on the Axis I and II of the Research Diagnostic Criteria, confirming both that the Axis I factors were largely independent of Axis II factors, and that, upon testing, the Axis I factors showed good reliability and predictive validity for potentially significant psychologic disturbance (Dworkin et al and Ohrbach et al, J Dent Res 1999;78:292).

In a study evaluating risk factors for the development for TMD in general and disc displacement in particular, an apparent discrimination between clenching and grinding as parafunctions was reported, in which grinding was considered less "dangerous" than clenching the teeth (Velly et al, J Dent Res 1999;78:491). In addition, in a group of 211 patients, an increased occurrence of disc perforation with age was observed in 28% of the subjects, regardless of disc position. The absence of a positive correlation between clinical symptoms, except for joint sounds, and the existence of disc

perforation was confirmed (Tateishi et al, J Dent Res 1999;78:510).

In a series of studies on biologic markers in TMJ synovial fluid, it was concluded that tumor necrosis factor α was present in patients with connective tissue disease and that its presence was related to pain on mandibular movement and allodynia. High levels of plasma serotonin were observed in relation to serum serotonin in patients with rheumatoid arthritis (Nordahl et al, J Dent Res 1999;78:539–540).

Pain pressure thresholds (PPT) were evaluated and found to be reliable in the short- and long-term (Cimino et al, J Dent Res 1999;78:357), especially when measured relative to a non-trigeminal reference site (Fredriksson et al, J Dent Res 1999;78:357). Pain pressure thresholds appeared to fluctuate with the menstrual cycle (Isselee et al, J Dent Res 1999;78:555). They also appeared to be unchanged after prolonged gum-chewing, which, by contrast, caused significant increases on VAS scales for perceived pain and masticatory fatigue (Michelotti et al, J Dent Res 1999;78:358). Estrogen appeared to decrease proteoglycan content and the number of hypertrophic chondroblasts in rat condylar cartilage (Harper et al, J Dent Res 1999;78:438–439).

An experimental pain model in humans confirmed previous findings in animals: neuronal plasticity occurred relatively quickly and was expressed as initial inhibition and subsequent excitation of the R2 component of the trigeminofacial reflex, which was more prominent in women (Moopen and Widmer, J Dent Res 1999;78:555).

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