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Meeting Highlight

Report on 8th World Congress on Pain, 17–22 August 1996

J. Pereira

Regional Palliative Care Program, Grey Nuns Community Health Centre, Room 5211, 1100 Youville Drive West, Edmonton, Alberta, Canada T6L 5X8

VANCOUVER WAS the spectacular setting for this conference. The organisation, given the magnitude of the congress, with over 4500 registrants from all corners of the globe, was impressive and it catered for a variety of interests. Cancer-related pain featured in many of the plenary sessions, topical workshops, poster presentations and associated symposia.

The concept of pre-emptive treatment was alluded to by several speakers and researchers. This concept has as its goal the prevention of chronicity. This is a concept that is important for primary health care professionals since it is they who will see the patients at an early stage, often before chronicity settles in. There is evidence from basic research to believe that by treating pain early, preferably even before the pain has started, it is possible to prevent secondary mechanisms that will lead to changes in the transmission and modulation of nociception. Two models being used to demonstrate this approach are postherpetic neuralgia and postsurgery pain syndromes (e.g. postmastectomy). In both of these chronic neuropathic pain conditions, the onset of pain is well defined and an intervention can be introduced at a very early stage. The clinical evidence of pre-emptive treatment is, as yet, insufficient as few controlled studies have addressed this problem, but it appears to hold potential.

Dr Ronald Dubner, in a plenary session, gave a summary of the three major areas that have contributed to our understanding of persistent pains and their management including sensory specialisation, neuronal plasticity and descending modulation. Neuronal plasticity remains a "hot topic", as indicated by the numerous posters and sessions dedicated to this topic. One of the well-attended workshops was that on NMDA receptors and hyperalgesia by Drs Dickensen, Eisenberg, Jorum and Mayer. They described the pivotal role of the *N*-methyl-*D*-aspartate (NMDA) receptor in hyperalgesia and the role of NMDA antagonists in the treatment of neuropathic pain.

An emerging interest amongst researchers is the role of the immune system in algesia and analgesia. The immune system is involved in the facilitation of pain by way of the pro-inflammatory cytokines, interleukin-1 (IL-1), IL-6 and tumour necrosis factor. These are released peripherally and centrally following trauma and illness. However, recent studies have also revealed the role of peripheral opioid receptors and the role of peripheral opioid peptides (B-endorphin, mey-enkephalin, dynorphin). These are abundant in immune cells of peripheral inflamed tissue. These peptides are released and activate the opioid receptors on sensory nerves, resulting in analgesia. These findings have implications for the understanding of pain in immunosuppressed patients. More recently, animal studies have shown that various opioids (in particular kappa agonists) have potent and long-lasting peripheral antinociceptive effects in the model of non-inflammatory, sciatic nerve constriction pain. This is contrary to commonly held beliefs that opiates do not relieve neuropathic pain. On the topic of sensitivity of neuropathic pain to opioids, an interesting poster by Dr Bruera of Edmonton revealed that up to 58% of patients with neuropathic pain were responsive to opioids alone. It appears more and more that the role of opioids for neuropathic pain is a continuum rather than an "all or nothing" effect. On one end of the spectrum there may be a poor response and on the other, an excellent response.

New insights were shared with regards to the mechanism of action of the analgesics and adjuvant analgesics. Recent animal studies have indicated that antidepressants relieve pain, at least in part, by blocking peripheral nerve sodium channels, rather than by exclusively blocking the re-uptake of noradrenaline or serotonin. At the membrane level, NSAIDS (non-steroidal anti-inflammatory drugs), in addition to their ability to block prostaglandin synthesis, can exert effects upon G-protein, alter interactions with several receptors and inhibit the production of cellular transcription factors. In addition, they can exert actions at several sites within the central nervous system. At the spinal level, NSAIDS can inhibit cyclo-oxygenase to reduce spinal prostaglandin release and ultimately reduce glutamate receptor activation by repetitive afferent input. These multiple mechanisms and sites of action indicate that we are just beginning to understand the complex role played by this heterogeneous family of drugs.

There has also been an increase in awareness of the neuropsychiatric adverse effects of opioids. With the increase worldwide of opioid availability and use, a spectrum of neuropsychiatric toxicities is emerging which includes delirium, organic hallucinosis, myoclonus, seizures and allodynia/hyperalgesia syndromes. Dr Eduardo Bruera discussed these

toxicities as well as the role played by the opioid metabolites and aggravating factors, such as dehydration and renal impairment. He went on to discuss the role of hydration, opioid rotations, the discontinuation of aggravating drugs and the use of opioids such as methadone, an opioid which appears to have no active metabolites. The phenomenon of opioid tolerance was discussed in several sessions and the NMDA receptor featured once again. The diminished response to an opioid may not be due to a lack of efficacy, but rather to the fact that the compensatory response that counteracts the effects of the opioid results in an enhanced nociceptive message. NMDA antagonists, therefore, may have a role to play in opioid tolerance.

Studies to date on the prevalence of cancer pain show wide variation, depending upon the population studied, the stage of the disease and the methods of assessing pain. Dr Irene Higginson's plenary session described the difficulties in recording good epidemiological data for cancer pain. These are variations in study design and assessment methods, identifying acute and chronic features, failure to assess multiple pains, varied assessment of psycho-social and spiritual influences, different causes—cancer related, concurrent disorder, treatment related. She reminded the audience that the assessment of pain should include its severity and impact on the patient and that, unfortunately, in many settings, follow-up assessments are poor or unsystematic. She discussed the use of staging systems, such as the Edmonton Staging System (ESAS), to identify patients likely to suffer severe pain and identify predictive factors which combine patients' psyco-social and clinical profiles. A system of simple repeated assessment to allow the monitoring and clinical audit of pain in advanced cancer is essential. Two systems described were the STAS (Support Team Assessment Schedule) and the ESAS.

It has long been assumed that various aspects of pain could be influenced by cultural and linguistic factors, and that these could potentially influence the findings in cross-cultural studies. Using the Brief Pain Inventory, Dr Cleeland and a team of researchers investigated the relationship between the numerical ratings of pain intensity and ratings of pain's interference with such functions as activity, mood and sleep in patients of four different national cultures. The patterns of pain interference ratings were comparable across all four national samples. Boundaries were identified between mild, moderate and severe pain intensity categories in terms of relative interference with function. These boundaries were the same for each of the four national samples in the author's analysis. Factor analysis provided additional evidence for cross-cultural similarity in self-reported ratings.