2021
February 27
Webinar

34th World Neuroscience and Neurology Conference;

13th International Conference on Tissue Science and Regenerative Medicine &

38th Global Psychiatry and Mental Health Conference

Sensory modulation in peripheral and central lesions of somoto sensory system

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VS Ramachandran and AV Srinivasan et al (1998) described allesthesia and extinction of referred sensations in brachial plus lesions. K. Sathian et al (2000) suggested that intermanual referral of sensations can occur after central lesions of the Somato sensory system. This communication considers eight patients with five central lesions and three peripheral lesions of the Somato sensory system. Ecological model of sensory modulations has external and internal dimensions. This refers both physiological and behavioural responses. Disfunction in sensory integration is the ability, modulate, discriminate, co-ordinate or organise sensation adaptively. Eight patients aged between 19-51 with brachial plexus lesion one, amputation 2 patients, hemiparesis with hemisensory deficit five patients were analysed. Patients vibration and kinesthesis were tested. Stimuli were applied manually. All patientshad Magnetic Resonance Imaging of the Brain and ENMG studies. Central lesions: All the five stroke patients showed intermanual referral of sensations between 3rd and 4th month after developing hemisensory deficit. Three had thalamic stroke and two patients has temparoparietal infarct. Intermanual referred sensations to the affected hand in these patients were not referred to the affected leg. Intermanual referred sensations were poorly localised and the facial sensations were referred with increased intensity in the thalamic patients. When applied pressure on the normal hand resulted in the extinction of pain sensation side and pain recurred within one minute of the pressure being relieved. Peripheral Lesions: Amputation of Limbs: Both the patients (below elbow and knee amputation) showed intermanual referral sensation within ten days. The referred sensation of touch and vibration lacked spatial organisation and poor localization with a relatively high threshold. Brachial Plexus Lesion: Patient had sensation intermanually referred in a topographically organised manner in the phantom limb. Intriguingly, simultaneous stimulation of the specific region (e.g. thumb) of the face and the corresponding finger of the right hand resulted in a mutual cancellation or extinction of the referred sensations in the phantom limb. 1.0 Intermanual referral of the sensations in the phantom limb occurred immediately in Brachial plexus lesion and amputation, whereas it occurred after a delay of three month is in hemiparesis with hemisensory deficit. This can be utilised for extinction of pain in the paretic side and in the Phantom limb. 2.0 Sensations were referred intermanually in a topographically organized manner in Brachial Plexus lesions, whereas in amputations and hemiparesis with hemisensory deficit, lacked spatial and poor localization. Disclosure: Dr. Venkatesan has nothing to disclose. 1. Willis WD Jr.: The sensory system with emphasis on structures important for pain. Brain Res Rev 2007; 55:297-313. 2. Miller LJ, Reisman JE. An ecological model of sensory modulation, Chapter 4,292-324. 3.DOBKIN BH. The clinical science of neurological rehabilitation. Second edn, Chapter 4, 292-324. 4. Srinivasan AV, Ramachandran VS, Ramachandran R,et al, 127th Annual Meeting of the AMRICAN Neurological Association; Oct 12 to 16,2002. Marriot Marquis Hotel, New York. 5. Srinivasan AV, Velmurugendran CU, Roger-Ramachandran, et al,. Anaesthesia and extinction of referred sensations after brachial plexus avulsion 5th American Academyof Neurology Meeting: Minneapolis.1998. 6. RamachandranVS, Hirstein W. The perception of phantom limbs: the DO.Hebb lecture. Brain. Sensory modulation disorder (SMD) affects sensory processing across single or multiple sensory systems. The sensory over-responsivity (SOR) subtype of SMD is manifested clinically as a condition in which non-painful

stimuli are perceived as abnormally irritating, unpleasant, or even painful. Moreover, SOR interferes with participation in daily routines and activities co-occurs with daily pain hyper-sensitivity, and reduces quality of life due to bodily pain. Laboratory behavioral studies have confirmed abnormal pain perception, as demonstrated by hyperalgesia and an enhanced lingering painful sensation, in children and adults with SMD. Advanced quantitative sensory testing (QST) has revealed the mechanisms of altered pain processing in SOR whereby despite the existence of normal peripheral sensory processing, there is enhanced facilitation of pain-transmitting pathways along with preserved but delayed inhibitory pain modulation. These findings point to central nervous system (CNS) involvement as the underlying mechanism of pain hypersensitivity in SOR. Based on the mutual central processing of both non-painful and painful sensory stimuli, we suggest shared mechanisms such as cortical hyper-excitation, an excitatory-inhibitory neuronal imbalance, and sensory modulation alterations. This is supported by novel findings indicating that SOR is a risk factor and comorbidity of chronic non-neuropathic pain disorders. This is the first review to summarize current empirical knowledge investigating SMD and pain, a sensory modality not yet part of the official SMD realm. We propose a neurophysiological mechanism-based model for the interrelation between pain and SMD. Embracing the pain domain could significantly contribute to the understanding of this condition's pathogenesis and how it manifests in daily life, as well as suggesting the basis for future potential mechanism-based therapies. Tactile over-responsiveness was characterized some decades ago as consisting of defensive-protective behaviors which are accompanied by stress responses to nociceptive qualities of sensory stimuli. Specifically, non-painful sensory stimuli are often experienced by individuals with this disorder as aversive, bothersome and lingering. Despite these reports, the pain sensory system has been neglected in both the Sensory modulation disorder (SMD) clinical and research domains. Interestingly, allodynia, a clinical term not implying a mechanism, refers to pain due to a stimulus that does not normally provoke pain. Consequently, allodynia represents a condition where the response mode differs from the stimulus mode, the latter of which may be induced by various non-painful stimuli such as light touch, cool or warm stimuli. Therefore, we suggest allodynia to mirror sensory over-responsivity (SOR), a subtype of SMD, by perceiving non-painful sensations as irritating, unpleasant or painful. According to the International Association for the Study of Pain, pain is "an unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage." This definition of pain has led our research efforts for the past decade, where we have endeavored to further our understanding of the SOR phenomenon, by studying its phenotype as well as its underlying mechanisms.