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Headache and olfactory sensor

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Introduction: Migraine is a neurovascular disorder characterized by recurrent unilateral headaches accompanied by nausea, vomiting, photophobia and phonophobia. Migraine headache is associated with trigeminal nerve activation and calcitonin gene-related peptide (CGRP) release from the trigeminovascular system. Various factors have been identified as being migraine triggers, including foods, stress, hormones, sensory stimuli, and so on. Individuals with migraine are often aware of the things that serve as triggers for them, since part of migraine management is avoidance of triggers. One of factors on the onset of migraine is olfactory sensory. This is review article to examine the relationship between headaches and smell disturbance. And the psychological effects of odors are in this group. Most common causes of smell disturbance are nasal and sinus disease, upper respiratory infection and head trauma. Smell or taste dysfunction can have a significant impact on quality of life. In general, the olfactory system regenerates poorly after a head injury. Most patients who recover smell function subsequent to head trauma do so within 12 weeks of injury. Olfactory dysfunction in some people as loss of smell sense, and in some one the psychological impact of an increased sense of smell, which affect their quality of life.

Anatomy and Physiology: The human sense of smell depends on the functioning of not only cranial nerve I (olfactory nerve) but also portions of cranial nerve V (trigeminal nerve). Smell receptors are located within the olfactory neuroepithelium, a region of tissue found over the cribriform plate, the superior septum and a segment of the superior turbinate. The free nerve endings of cranial nerve V are located diffusely throughout the nasal respiratory epithelium. The axons (C- and A -delta fibers) project to the trigeminal sensory nucleus and to the spinal, principal and mesencephalic trigeminal nuclei. Nociceptive afferents descend in the trigeminal tract and terminate in the spinal nucleus. Trigeminal information is relayed to the amygdala from the trigeminal sensory nuclei via the lateral parabrachial complex. It must be noted that electrophysiological data indicate that an area of increased trigeminal chemo sensitivity might be located at the anterior third of the septum. Indeed, most odorant molecules have the propensity to simultaneously stimulate olfactory and trigeminal systems in the nasal cavity. The trigeminal system provides an important pain-transmitting link from the cranial vasculature to the CNS. In humans, unilateral stimulation of the trigeminal ganglion results in increased bilateral cortical blood flow, slightly more on the stimulated than on the contralateral site. This may provide the anatomical link between cerebral neurons and the trigeminovascular system which is the central communication for the afferent pain to the brainstem and the central aspects of the migraine symptoms.

Several studies indicate that olfactory receptor responses to chemical stimuli can be modified by activation of the trigeminal nerve. Also in some studies, the results have showed a significant difference in the olfactory threshold at the immediate post-operative period. Therefore, the injured trigeminal fibers are probably associated with the increase in the olfactory threshold after the surgery; it has been shown in normosmic subjects that trigeminal stimuli are perceived as more intense when they were accompanied by olfactory stimulation. On the other hand, in a small sample of women with congenital anosmia, nausea and vomiting of pregnancy occurred in only 1 pregnancy, suggesting that olfaction is a highly selected trigger for nausea and vomiting of pregnancy. The shared nausea and vomiting experience of hyperemesis gravidarum and migraine headache among women suggests there is a common mechanism.

Conclusions: Thus, previous trigeminal activation induced an increase in olfactory sensitivity and it must be argued that both time and intensity of stimulations probably play a role in modulating interactions

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