Bioscene

Bioscene
Volume- 21 Number- 03
ISSN: 1539-2422 (P) 2055-1583 (O)
www.explorebioscene.com

Concept of Neuroplasticity and its Application in Orofacial Region

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The neurological term phantom limb alludes to the complex of rich and vivid perception that are referred to an amputed body part. These perceptions are most commonly reported following limb amputation. These phantom limb sensations can be painful or non-painful. Neuroplasticity is the ability of brain to change or rewire throughout a person's life. Our body is inverted anatomically on to the cerebral cortex. Sensitive body parts with many nerve endings are represented by larger cortical areas in somatosensory cortex while the body parts with higher muscular activity are represented by larger cortical areas in the primary motor cortex too. Neuroplasticity plays a crucial role in the head and neck regions also.

Introduction

History behind phantom limb

The term ''phantom limb'' was coined by Ambrose Pare, a French surgeon who documented cases of amputees experiencing sensations in their missing limbs. One of the most famous historical cases of phantom limb sensations is that of James Thurber, a civil war soldier who lost his arm in a battle. Despite the loss of his arm, Thurber reported feeling intense pain and sensations in his phantom limb.

In the 19th and early 20th centuries, researchers such as Silas Weir Mitchell and Charles Sherrington began studying phantom limb sensations in more detail. They proposed theories to explain why amputees could still feel sensations in limbs that were no longer there. The discovery of neuroplasticity shed light on how the brain adapt to changes such as limb loss.

The history behind phantom limb experiences a fascinating journey of discovery and understanding the complex interplay between the brain nerves and perception.[1]

Phantom limb pain

Phantom limb pain is a phenomenon where individual who have had a limb amputed continue to feel pain or other sensations in the missing limb. This can be puzzling and distressing experience for those who suffer from it. The pain can vary in intensity and may be described as burning, tingling, stabbing or throbbing. It can also be triggered or worsened by various factors such as changes in weather, stress or even touching other parts of the body.[2]

Neurology behind phantom limb

Neuroplasticity is the brains remarkable ability to reorganize and adapt itself by forming new neural connections in response to experience, learning and injury. Neuroplasticity plays a crucial role in understanding why amputees can still feel sensations in a limb that is no longer there. When a limb is amputed, the brains sensory and motor regions that used to correspond that limb reorganize. Despite the physical absence of the limb, the neural networks associated with it remain active. This can lead to the sensation of phantom limb, where the individual feels as though the missing limb is still present and may even experience sensations such as pain, itching or movement in the non-existing limb. The areas representing the missing limb become hyperactive or cross wired with neighbouring regions. This rewiring can create a miss match between the brains perception of body and its actual physical state, leading to the sensation of phantom limb.[3]

Additionally, neuroplasticity can also contribute to the treatment of phantom limb pain. Techniques such as mirror therapy, virtual reality have been developed to harness the brains ability to reorganize and alleviate phantom limb sensations. By engaging the brain in activities that retrain its perception of the missing limb, these therapies can help to reduce phantom limb pain and improve the individual's quality of life.[4]

Application of Neuroplasticity in Orofacial Region

The diagnosis and management of pain is one of the most demanding and fulfilling facets of general practice. Orofacial pain is estimated to affect 22% of the general population within a 6-month period. Moreover, the head and neck region experience a higher prevalence of chronic and persistent pain than any other section of the body. It is common for orofacial pain to be misdiagnosed. The localization and interpretation of pain symptoms are complicated by the convergence of sensory neurons to higher centres.

Neuroplasticity, also known as neural plasticity or brain plasticity, is a process that involves adaptive structural and functional changes to the brain. This concept could be applied as most possible reason behind some conditions affecting orofacial region which includes, Phantom odontogenic pain or atypical

odontalgia, Phantom bite syndrome, Burning mouth syndrome and Frey's syndrome or gustatory sweating.[5]

Atypical odontalgia

McElin and Horton published the first description of atypical odontalgia (sometimes referred to as idiopathic or phantom tooth pain) in 1947. Despite being widely verified, this clinical condition is infrequently described. Persistent toothache after pulp extractions, apicoectomies, or tooth extractions is typically its defining feature.Atypicalodontalgia has also been linked to inferior alveolar nerve block and facial trauma. After receiving endodontic therapy, 3% to 6% of individuals experience atypical odontalgia, according to epidemiologic data. Atypical odontalgia typically manifests as protracted episodes of excruciating or throbbing pain in the alveolar process or teeth, and This is in the lack of any discernible odontogenicethology seen on radiographs or in clinical observations. The patient has chronic pain, but sleep is uninterrupted, and there may be a brief period of time when they are symptom-free.[6]

It's common for patients to have trouble locating their discomfort. and The initial trauma location is usually where it hurts the most, although it can also extend unilaterally or bilaterally to nearby regions. All age groups are impacted, with the exception of children; women in their mid-40s are the most afflicted. The maxilla's molars and premolars are most frequently impacted. Uncertain outcomes are obtained via local anaesthetic[7] block, and analgesics—including opioids—rarely provide pain relief for patients. AndRegrettably, atypical odontalgia is sometimes misdiagnosed as a regular post-treatment or post-trauma issue. Psychological comorbidity has not been shown in atypical odontalgia, despite the allure of thinking so. There is a noticeable degree of demoralization, similar to several chronic pain syndromes. Nevertheless, it's unclear if this is the condition's cause or effect.

For atypical odontalgia, numerous classification and diagnostic criteria have been put forth. Once every other head and neck ailment has been ruled out, it is still an exclusion diagnosis. Many times, after undergoing several endodontic or surgical procedures, patients experience neither improvement nor worsening of their problems.Recognizing neurologic indications involving adjacent structures and other teeth that are serviced by the same nerve is essential for an accurate diagnosis. It's yet unknown what causes atypical odontalgia. Marbach proposed in 1978 that phantom limb pain and atypical odontalgia had a similar origin.[8]

Research on deafferentation has shown that changes can occur in the organization and activity of both central and peripheral nerves following injury which accounts for neuroplastic process and chronicity of pain and other associated symptoms (dysesthesia, paresthesia) may arise from this. For instance, such discomfort is

believed to be caused by neuromas consequent to nerve damage.and Sensitization of pain fibers, sprouting of neighboring afferent fibers, sympathetic stimulation of afferents, cross-activation of afferents, loss of inhibitory mechanisms, and phenotypic flipping of afferent neurons are other mechanisms implicated in the pathophysiology of pain which mainly attributes to concept of neuroplasticity[9]

Phantom bite syndrome

Oral sensorimotor functions may change as a result of dental occlusion modifications. Orofacial movements are generated and controlled by the face primary motor cortex Changes in motor function or sensory inputs can cause face-M1 neuroplasticity.[10]

Phantom bite syndrome, or PBS, is typified by pain during occlusion that isn't associated with any obvious abnormalities. These patients develop a preoccupation with dental care because they are frequently obsessed with their occlusion or the alignment of their teeth. The majority of them have had bad dental experiences in the past and attribute the failure to the dentists' "unskilled" work. Undiagnosed medical conditions including body tilt and persistent back pain are frequently linked to "wrong bites."[11]

PBS was once thought to be a psychogenic or psychiatric condition, similar to dysmorphophobia, monosymptomatichypochondriacal psychosis, severe personality disorder, and paranoia1 However, based in part on how PBS patients respond to psychopharmacologic therapy and research on other phantom feelings, new investigations have speculated that central nervous system dysfunction occurs in these patients. PBS was reinterpreted by Marbach as "altered central processing." In view of psychopharmacological results, mental comorbidities, and the distribution of the side experiencing occlusal discomfort, we maintained this theory. [12]

The thalamus and primary sensory region of the ipsilateral side receive partial projections of the physiological oral sensation, but the opposite thalamus and primary sensory cortex receive the majority of them. Additionally, there exists a positive and negative correlation between masticatory muscle action and cortical activation, encompassing the frontal lobe

patients repeatedly try to verify that their occlusion is in place because they find it painful, even though they don't seem to be moving or acting strangely. The most straightforward explanation for these clinical symptoms is that, although it's unclear if they're primary or secondary, the opposite thalamus may be more active as the principal conduction channel as a result of focusing on one side of the oral cavity's senses.[13]

Burning mouth syndrome

According to International Headache Society burning mouth syndrome is defined as intraoral burning or dysaesthetic sensation, recurring daily for more than 2 hours per day over more than 3 months, without clinically evident causative lesions. Although etiology of BMS is considered to be multifactorial, recent studies explored various underlyingpsychopathological and neuropathic disorders.[14]

According to previous studies[15]BMS shares a range of characteristics with other forms of chronic pain. This characteristics may results from alterations in CNS function caused by hyper stimulation of the N-methyl-D-aspartic acid (NMDA) receptors ofthe dorsal trunk leading to neuroplasticity phenomena.As a result of this entity common harmless stimuli (like irritation due to dentures) are reinterpreted by the central nervous system as being located above the pain threshold and thus interpreted as harmful.[16]

Some recent studies highlights the behaviour of BMS as a form of oral phantom pain. Few hypothesis were proposed in this sense. It implies that a conventional axonal reflex would activate a set of nociceptors which release neuropeptides at the terminal branch of trigeminal nerve that mediates neuronal inflammation. A second hypothesis proposes the possibility of an indirect reflex that would arise in the afferent nerves of oral cavity which process at a central level in the trigeminal nerve, and then trigger a reaction at the level of the autonomic nervous system. The third theory relates to the dual efferent and sensory functions of capsaicin receptors.These receptors, which are present in C-polymodalnociceptors, are thought to play a role in controlling the processes leading up to neurogenic inflammation.[14]

Frey's syndrome or Gustatory sweating.

It is also known as Gustatory Hyper-Hydrosis, Auriculo-Temporal Syndrome or Baillarger Syndrome.Frey's syndrome is a rare condition first described by Polish Neurologist Łucja Frey in 1923, Frey's syndrome (FS), characterized by unilateral sweating and flushing of the facial skin that occurs during meals, usually in the area of the parotid gland. Frey's syndrome is a frequent and unpleasant complication of parotidectomy. It has also been reported following radical neck dissections, carotid endarterectomy, and submandibular gland excision, and mandibular condylar fracture, surgical approaches to the mandible, autonomic neuropathy in diabetes mellitus, herpes zoster infections and obstetrics forceps trauma. Neuroplasticity changes can be induced by trauma (i.e., transection) of peripheral sensory or motor nerves or manipulations of sensory inputs or motor functions.FS is related to aberrant regeneration of nerve fibres after trauma.[17]The origin of parasympathetic secretory fibers to parotid

gland is located in the medulla, in the inferior salivary nucleus. After synapsing in the otic ganglion, the postsynaptic parasympathetic fibers join the auriculotemporal nerve (a branch of the mandibular nerve V3) to supply secretomotor fibers to the parotid and surrounding mucus glands and vasodilator fibers to the vasculature. Postganglionic sympathetic fibers originating in the superior cervical ganglion join the internal and external carotid artery nerve plexus to supply vasculature and sweat glands. Although most postganglionic sympathetic nerve fibers release (nor) epinephrine, those innervating the sweat glands are cholinergic.[18]Cutting the auriculotemporal nerve during surgery of parotid gland, interrupts postganglionic parasympathetic fibers that supply the parotid gland, and sympathetic fibers for local sweat glands.[19]The disrupted parasympathetic secretomotorfibers normally destined for the parotid are misdirected during regeneration. Instead, these fibers aberrantly reinnervate sweat glands and cutaneous vessels in the territories of the auriculotemporal and large auricular nerve distributions. Hence, sweating or dermal flush occurs during salivary stimulation (ie eating or thinking of eating / food) may give rise to sweating, flushing, and heating in the regions anterior to the ear, over the angle of the mandible, and over the site of the parotid and can be easily diagnosed using Minor's iodine–starch test or infrared thermography. While physical symptoms are generally considered mild, FS can be a major source of psychosocial morbidity for patients. Aberrant regeneration of parasympathetic fibers cut between otic ganglion and the salivary gland tissue, leads to aberrant innervation of sweat glands and subcutaneous vessels.[20]This pathophysiologic mechanism explains neuroplasticity and related behavioural changes may not only reflect adaptive modifications that are beneficial but could instead represent maladaptive modifications that result in impaired function.

Phantogeusia

Phantogeusiais a phenomenon characterized by the perception of tastes in the absence of external stimuli, resulting from alterations in the brain's taste processing systems. In individuals with phantogeusia, the brain's neural connections have adapted and reorganized in a way that generates the perception of tastes without external stimuli. This adaptation is thought to be a result of neuroplasticity, which enables the brain to compensate for damage, disease, or experience-related changes by reorganizing its neural connections.[21] This adaptability is thought to be mediated by changes in the strength and number of synaptic connections between neurons, as well as the formation of new neurons and neural pathways .[22]Damage to the brain's taste processing systems, such as the insula or anterior cingulate cortex, can lead to reorganization and adaptation of neural connections.[23] These changes can result from damage to the brain, such as stroke or traumatic injury[22] where the brain attempts to compensate for lost or altered taste function.[24] Neuroplasticity can also be driven by experience and learning, leading to changes in the brain's taste processing systems.[21]For

example, repeated exposure to certain tastes or smells can lead to changes in the brain's neural connections and the development of phantogeusia.[22]Additionally, neuroplasticity can be influenced by cognitive and emotional factors, such as attention, expectation, and emotional state.For example, individuals with high levels of anxiety or stress may be more likely to develop phantogeusia, as their brains are more prone to reorganization and adaptation in response to perceived threats. Understanding the relationship between phantogeusia and neuroplasticity is crucial for the development of novel treatments aimed at alleviating phantogeusia by harnessing the brain's neuroplastic potential.

Conclusion

Neuroplasticity underscores the brain's remarkable ability to adapt and reorganize, offering hope for improved outcomes in rehabilitation, pain management, and functional recovery within the orofacial region. Understanding these principles is crucial for advancing treatment modalities and enhancing quality of life for patients with orofacial conditions.

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