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REVIEW ARTICLE

PATHOPHYSIOLOGY OF OROFACIAL PAIN: A REVIEW

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ABSTRACT

The International Association for the Study of Pain (IASP) defines pain as "an unpleasant sensory and emotional experience which we primarily associate with tissue damage or describe in terms of such damage, or both." This definition recognizes that pain is a perception. The nervous system mechanism for detection of stimuli that have the potential to cause tissue damage is very important for triggering behavioural process that protect against current or further tissue damage. This is done by reflex reaction and also by preemptive actions against stimuli that can lead to tissue damage such as strong mechanical forces, temperature extremes, oxygen deprivation, and exposure to certain chemicals.

The clinician who attempts to understand and manage pain needs to have a thorough appreciation for different types of pain that can be encountered which are classified as acute pain, chronic pain, primary, secondary, neuropathic and inflammatory.

Dental pains may have their origin in the dental pulps or in the periodontal structures.
Pulpal pains may be classified as acute, chronic, recurrent or mixed with periodontal elements. Periodontal pain is deep somatic pain of the musculoskeletal type. As such, it is more localized than is pulpal pain.

Nondontogenic pain can be classified as muscular, cardiac, sinus, TMJ pain, neurovascular pain and musculoskeleton pain. Pain gives a warning of tissue damage and activation of defensive mechanisms, with the aim of prevention of further damage. Knowing the pathways and mechanisms of pain, possible causes and different characters of orofacial pain, clinical examination will eventually lead to a proper diagnosis.

INTRODUCTION

The International Association for the Study of Pain (IASP) defines pain as "an unpleasant sensory and emotional experience which we primarily associate with tissue damage or describe in terms of such damage, or both." [1]

This definition recognizes that pain is a perception. The nervous system mechanism for detection of stimuli that have the potential to cause tissue damage is very important for triggering behavioural process that protect against current or further tissue damage. This is done by reflex reaction and also by preemptive actions against stimuli that can lead to tissue damage such as strong mechanical forces, temperature extremes, oxygen deprivation, and exposure to certain chemicals .The term nociception refers to the sensory process that is triggered and pain refers to the perception of a feeling or sensation which the person calls pain and describes variably as irritating, sore, stinging, aching, throbbing or unbearable. [2]

The alerting function of pain evokes protective responses (reflex motor withdrawal and behavioural responses), and is intended to keep tissue damage to a minimum. The capacity to experience pain has a protective role. If tissue damage (cellular breakdown with liberation of biochemical substances) is unavoidable, a cascade of changes occurs in the peripheral and central nervous system responsible for the perception of pain. [3]

Various presentations of pain

The clinician who attempts to understand and manage pain needs to have a thorough appreciation for different types of pain that can be encountered.

Acute pain - Acute pain is defined as 'pain of recent onset and probable limited duration. It usually has an identifiable temporal and causal relationship to injury or disease'.

Chronic pain- Chronic pain was originally defined as pain that has lasted 6 months or longer. More recently it has been defined as pain that persists longer than the temporal course of natural healing, associated with a particular type of injury or disease process. Chronic pain 'commonly persists beyond the time of healing of an injury and frequently there may not be any clearly identifiable cause'. [4]

Primary versus Secondary pain - The site where pain occur may or may not identify the location of the source of pain. If pain does in fact emanate from the structures that hurt, it constitutes a primary nociceptive input. If however the true source of pain is located elsewhere, the area of discomfort represents secondary pain. Secondary pain is also called as heterotopic pain.

Somatic versus Neuropathic pain - Pain emanating from a particular area may result from noxious stimulation of the somatic structures .When this occurs, the nociceptive impulses are being received and transmitted by normal components of the sensory nervous system. Such pain is referred to as somatic pain. Quite a different type of pain, however, may emanate not from abnormality in the somatic structures but from abnormality in the neural components that innervate the area. Such pain is termed neuropathic.

Superficial versus Deep somatic pain-Pain emanating from the cutaneous and mucogingival tissues present clinical characteristics that are similar to other exteroceptive sensations .They are precisely localized by the patient and relate faithfully to the provocation in timing, location and intensity.In contrast pains resulting from stimulation of deeper musculoskeletal and visceral structures resemble other proprioceptive and interoceptive sensations. As such they are more diffusely felt respond less faithfully to provocation and frequently initiate secondary effects such as referred pain.[5]

Inflammatory pain - Tissue injury initiates an inflammatory reaction that characteristically induces pain. Inflammatory pain is due to the action of prostaglandins and bradykinin substances released by inflammatory process .Prostaglandins are important mediators of inflammation, fever and pain. They are synthesized by the constitutive enzyme, cyclo-oxygenase-1 (COX-1), and its isoform enzyme COX-2, which is induced in peripheral tissues by cytokines, growth factors and other inflammatory stimuli. Although in some situations prostaglandins contribute to pain by directly activating nociceptors, they are generally considered to be sensitizing agents. Prostaglandins increase levels of cyclic AMP and may enhance nociceptor sensitization by reducing the activation threshold for TTX-R sodium channels via a protein kinaseA pathway.[6]

Pain of dental origin

Dental pains may have their origin in the dental pulps or in the periodontal structures. These two categories will be discussed separately, since their clinical presentationare different.

Dental pain of pulpal origin

The pain is of threshold type so no response occurs until the threshold level is reached. Pulpal pain responds to noxious stimulation, it responds to impact shock, thermal and chemical irritants, the pain is often very difficult for patient to localize.

Pulpal pains may be classified as acute, chronic, recurrent or mixed with periodontal elements. A basic clinical feature of pulpal pain is that it does not remain the same indefinitely. Generally it resolves become chronic or proceeds to involve the periodontal structures.

Identification of Pulpal Pain

History and examination - When investigating acute dental pain, the history should focuson:

• location
• type
• frequency and duration
• onset
• exacerbation and remission (for example the response to heat or cold)
• severity

Associated pathology and referred pain should also be considered.[7]

Which tests can assist in diagnosis?

There are several simple tests that may assist in diagnosis of dental pain.

Pulp sensitivity test -Dry ice, or an ordinary ice stick (made in a plastic or glasstube), is placed on the cervical third (neck region) of the tooth crown. A response to the stimulus indicates that the pulpal tissue is capable of transmitting nerve impulses. No response may indicate pulp necrosis.

Percussion test - Using an instrument handle, the tooth is tapped along the longitudinal axis. A painful response suggests possible periapical inflammation.

Probing -Placing a fine, blunt probe gently into the gingival sulcus surrounding the tooth enables the health of the gingival tissues to be assessed. Bleeding and/or sulcus depths greater than 3–4 mm indicate gum disease.

Mobility test - Holding a tooth firmly on the buccal (cheek) and lingual sides between the fingers enables mobility to be assessed. All teeth have a small amount of mobility (<0.5 mm), but visible movement suggests loss of bone support around the root of the tooth.

Palpation -Careful palpation around the area of concern may reveal tenderness and the type and extent of swelling.[8]

Pain of periodontal origin

Periodontal pain is deep somatic pain of the musculoskeletal type. As such, it is more localized than is pulpal pain. It responds to provocation proportionately and in graduated increments, rather than as a threshold response like pulpal pain.

The receptors of the periodontal ligament are capable of precise localization of the stimulus. Therefore, periodontal pains of all types rarely present any real diagnostic problem, because the proper offending tooth is readily identified. In the PDL are proprioceptors which allow a precise localization of the pressure stimuli, so a periapical pain is easily diagnosed.

The causes of periodontal pain are many and varied . It may occur as a primary periodontal inflammatory condition arising from a local cause such as trauma, occusal forces. Pain may occur as a result of dental prophylaxis, endodontic treatment, orthodontic treatment, inadequate opposing occlusal contact, overcontoured or undercontoured proximal contact points . [5]

Referred Pain

The term referred or reflected pain, denotes the pain felt in the body part which is remote from the place of stimulation or tissue damage. A reflected pain originates in one place (eg. the lower first molar), and is felt in the other (eg. ear). Contrary to that, odontalgia is the pain caused by pathological changes in other places and reflected on the teeth.

Referred pain is felt in anare innervated by a different nerve from the one that mediates the primary pain. Both teeth and non-odontogenic sources (such as muscles, sinuses, pathology, and so forth) can refer pain to other teeth or to other anatomic regions of the head and neck.[9]

Non odontogenic pain

Muscular toothache - Muscle or myofascial pain is a condition characterized by dull, aching muscles with localized tender areas. These tender areas can refer pain to other structures in consistent patterns. Jaw muscles can refer pain to the teeth, and this can be perceived as dental or intraoral pain.

Cardiac toothache - The clinical description of ischemic heart disease is characterized by substernal pain, which spreads to the shoulders, arms and neck. In some cases, the pain may spread to the jaws and teeth. The cause of cardiac pain referred to the orofacial region can be explained by convergent mechanisms in the trigeminal complex. Spreading of the pain to the orofacial area is less frequently observed and would be the consequence of converging, at the spinalthalamictractus, of afferent cardiac fibers with second order trigeminal neurons responsible for innerving the dental sensitivity. However, the most frequent symptom is the pain in the jaw area innerved by the upper cervical roots C2 and C3.

Sinus or nasal mucosal toothache - Problems in the maxillary sinuses and/or paranasal mucosa can refer pain to the upper teeth. The pain is usually felt in several teeth as dull, aching or throbbing. Sometimes it is associated with pressure below the eyes and it can increase with lowering the head, putting pressure over the sinuses, coughing or sneezing. Tests performed on your teeth, such as cold, chewing and percussion, can increase the pain from sinus origin. A history of an upper respiratory infection, nasal congestion or sinus problem should lead to suspicion of a "sinus toothache."

Neurovascular toothache- Neurovascular pains or headache is a common complaint. Typically headache is pain localized to the cranium. However it may also present as a variant involving the orofacial region hence mimicking toothache.[10]

Temporomandibular joint pain - Signs and symptoms of temporomandibular disorders (TMDs) may include pain, impaired jaw function, malocclusion, deviation or deflection, limited range of motion, joint noise, and locking. Because of many etiologic factors, the diagnosis and treatment of patients with temporomandibular joint disorders is complex. Temporomandibular joint disorders can be subdivided into muscular and articular categories.

Myogenic disorders include myalgia, myospasm, fibrosis etc. Articular disorders include synovitis, joint effusion, fracture, internal disc derangement and arthritis. Temporomandibular joint disorders defined by the American Association of Orofacial Pain (AAOP) as:

'A collective term embracing a number of clinical problems that involve the masticatory musculature, the Temporomandibular joint and associated structures, or both '.
Temporomandibular joint disorders are common in adults. In epidemiologic studies, up to 75% of adults show at least one sign of joint dysfunction .On examination as many as one-third have at least one symptom, which include jaw or neck pain, headache, and clicking within the joint. [11]

Musculoskeletal pain

Myofascial Pain Dysfunction Syndrome (MPDS) -

Myofascial pain is a common form of pain arising from hyperirritable foci in muscle, usually referred to as myofascial trigger points.

Active trigger point: An active trigger point causes spontaneous pain at rest, with an increase in pain on contraction or stretching of the muscle involved.

Latent trigger point: A latent trigger point is a focal area of tenderness and tightness in a muscle that does not result in spontaneous pain.[12]

Pathophysiology: [26]

Precipitating factors of MFP may cause the facilitated release of acetylcholine at motor end plates, sustained muscle fibre contractions and local ischaemia with release of vascular and neuroactive substances, and muscle pain. More acetylcholine may then be released, thus perpetuating the muscle pain and spasm.

Clinical Features

The diagnosis of MFP is based on the history and clinical examination of the patient. MFP is characterized primarily by pain and tenderness of muscles of mastication, the pain being regional and unilateral. MFP is characterized by regional, unilateral pain. Patients typically localize the pain toareas around the ear, the angle, body of the mandible and the temporal region. Referral patterns include intraoral, auriculotemporal, supraorbital and maxillary areas depending on the muscles involved and the intensity of the pain.

Physical examination usually reveals limited mouth opening, presence of limited mouth opening in patient may also indicate TMJ pathology. Tenderness to palpation usually present in ipsilateral masticatory muscles is a distinguishing feature. The masseter is the muscle most commonly involved (> 60%), the medial pterygoid and temporal is muscles are involved in about 40–50% of cases.

Areas of referred pain may includeperioral and intraoral (teeth) structures and depend on the muscles involved and the intensity of pain .Referral to the teeth may be prominent and may often cause misdiagnosis as dental pathology. Referral of pain from trigger points in the deep part of the masseter muscle includes the TMJ and ear, causing possible misdiagnosis with intra-articular or ear disorders.

Differential Diagnosis

MFP needs to be differentiated from other conditions that may affect the masticatory muscles. Inflammation of a muscle or myositis secondary to infection or trauma is commonly seen in dental practice. Myositis is usually associated with a pertinent history or significant clinical findings such as muscle or regional swelling, redness and dental or periodontal infection. The affected musclesare tender, located in the vicinity of the inflammation and accompanied by limitation of mouth opening. Myositis may precede or be associated with a painful contraction or myospasm in the regional muscles that is of acuteonset. The differentiation from painful TMJ disorders may also be complex due to overlapping symptoms. Regional pain, pain-referral patterns and pain evoked by mandibular movement are common to both MFP and TMJ disorders.

The occurrence of regional primary or metastatic tumours may induce TMD-like symptoms and should be excluded. Particularly, patients with a history of previous malignancy are at risk and should be referred for relevant imaging studies.[13]

Principles of Pain Diagnosis

Diagnosis of pain complaint consists essentially of three major steps:

- Accurate identification of the location of the structure from which the pain emanates.
- Establishment of correct pain category that is represented in the condition under investigation. Establishment of the proper pain category is dependent upon a good understanding of genesis and mechanism of pain.
- Choice of the particular pain disorder that correctly accounts for the incidence and behaviour of the patients pain problem.

Initial Pain Assessment

Pain management depends on a comprehensive assessment. This is especially true for the patient with persistent pain. Pain assessment should be ongoing (occurring at regular intervals), individualized, and documented so that all involved in the patient's care have a clear understanding of the pain problem.

As a result of the pain assessment, the clinician should understand the nature of the pain in terms of its etiology, pathophysiology and syndrome; its impact on many domains of life; and relevant premorbid conditions and comorbidities that will influence treatment decisions. This understanding requires detailed questions about the pain characteristics, an assessment of the impact of the pain in multiple domains, and an evaluation of related concerns.

Based on this information, the findings on a physical examination and review of records and existing laboratory and imaging data, a working diagnosis can be developed that includes an understanding of the pain's etiology, pathophysiology and syndrome. From this formulation, a plan of care can be developed that may include the need for additional evaluation and an initial set of therapies to address the pain and other concerns.

This process of assessment can be straightforward and brief in the setting of acute pain related to trauma or surgery. It increases in complexity and the time required as the pain becomes persistent, fails to respond to conventional therapy, or is observed to be occurring in a biomedical or psychosocial context that complicates the understanding of the pain or poses challenges in management.

Initial pain assessment guidelines

- Obtain a detailed history, including an assessment of pain characteristics
- Conduct a physical examination, emphasizing the neurological and musculoskeletal examination.
- Obtain and review past medical records.

Diagnosis of orofacial pain

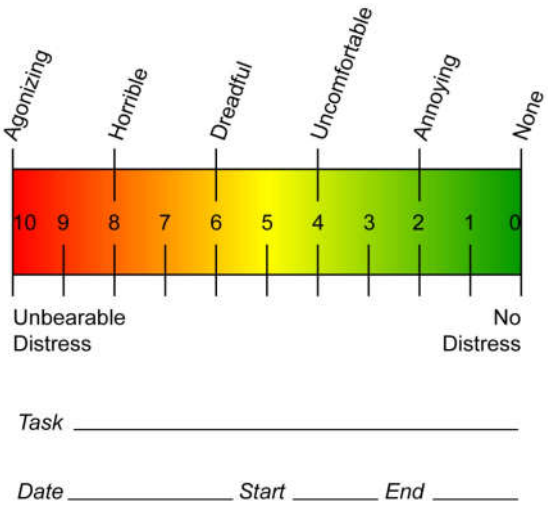
The history is the most important means of diagnosing orofacial pain. In order to differentiate the widely distributed causes, it is essential to determine key points about the pain, especially:

Location

Valuable information can be obtained by asking if the pain is localised or diffuse, and watching the patient's reaction. For example, patients frequently point with one finger when describing pain of dental causes or trigeminal neuralgia, but atypical facial pain is much more diffuse, and may radiate across the midline.

Character

Patients should be asked about the severity and character of the pain i.e. whether the pain is 'sharp', 'dull', 'aching', 'throbbing' or 'shooting'. However, one should bear in mind that patients often have difficulty finding adequate descriptors. Ask the patient to rate the pain severity on a scale of zero (no pain) to 10 (most severe pain that the patient has experienced), or ask them to mark this on a line divided into10 equal sections (visual analogue scale) or use an assessment instrument such as the McGill Pain Questionnaire. These 'tools' help assess the severity of pain, accepting always that it is subjective, and they may also be useful in monitoring the response to treatment. Disturbance of the normal sleep pattern by pain is also useful in assessing the severity.



Duration

The average duration of each episode may help diagnosis. For example, pain from exposed dentine is fairly transient (lasting only for seconds) while the pain from pulpitislasts for a much longer period. Trigeminal neuralgia is a brief lancinating pain lasting up to about five seconds, although some patients report a persistent background less severe pain-more of a dull ache; migrainous neuralgia typically lasts 30 to 45 minutes, while atypical facial pain is typically persistent.

Frequency and periodicity

Determine whether the pain occurs at specific times or related to specific events. A 'pain diary' can help. For example, the pain of temporomandibular pain dysfunction syndrome may be more severe on waking if this is associated with nocturnal para-functional activity such as clenching or tooth grinding. The pain of sinusitis is often aggravated by lying down. Periodic migrainous neuralgia frequently disturbs the patient's sleep at a specific time each night, around 2am.

Precipitating, aggravating and relieving factors

It may be necessary to resort to leading questions to ask if temperature, biting, posture, analgesics, alcohol etc affect the pain. For example, heat often aggravates dental pain; touching

a trigger zone may precipitate trigeminal neuralgia attacks, stress may worsen atypical facial pain, and alcohol may induce episodes of migrainous neuralgia.

Associated features

Some types of pain maybe associated with other features which are helpful diagnostically, such as the swollen face in dental abscess, nausea and vomiting in migraine, or nasal stuffiness or lacrimation in migrainous neuralgia. The cause of most or ofacial pain is established mainly from the history, and examination findings are also helpful, not least in excluding local pathology.

However, it is important to consider the usefulness of additional investigations, particularly imaging of the head and neck, using CT or MRI. It is crucial not to misdiagnose and thus mislabeling the patient as having psychogenic pain, and not to miss an underlying brain tumour underlying a patient with supposed 'idiopathic' trigeminal neuralgia.[5]

CONCLUSION

Pain gives a warning of tissue damage and activation of defensive mechanisms, with the aim of prevention of further damage. The stimulus which damages or threatens to damage a tissue activates the nociceptors which in turn carry the information by a system of neurons to cortex, where it is processed to recognize and properly diagnose the cause of pain. This is not always easy, due to numerous variations within the clinical findings, and the latent possibility that pain has been referred from odontogenic structure to non odontogenic ones and vice versa.

Knowing the pathways and mechanisms of pain, possible causes and different characters of orofacial pain, clinical examination will eventually lead to a proper diagnosis.

An odontogenic source of pain is well defined and has an apparent cause. Any deviation from standard clinical status should be taken with caution. Once odontogenic cause of pain has been excluded, other potential causes of orofacial pain should be taken into consideration, in order to establish a valid diagnosis.

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