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Review
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ODONTOGENIC PAIN

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Summary

Pain has the function of a warning to 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 and recognized as pain. Most somatosensory information from the area of orofacial system is transported via n. trigeminus. In order to remove pain, it is necessary to recognize and properly diagnose the caus of pain. This is not always easy, due to numerous variations within the clinical findings, and the latent possibility that pain has referred from odontogenis structure onto the nonodontogenis ones, and vice versa.

Knowing the pathways and mechanisms of pain, possible causes and different characters of orofacial pain, as well as a thorough anamnesis, clinical examination and testing will eventually lead to a proper diagnosis. An odontogenic source of pain is well defined and has an apparent cause, and therefore the provocation tests lead to the symptoms contained in the anamnesis. 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.

***Key words*:** odontogenic pain; non-odontogenic pain; referred pain; differential diagnostics.

INTRODUCTION

Pain is an unpleasant, subjective, sensational and emotional experience associated with actual or potential tissue damage [1]. It belongs to the sensations that bring information about the state of the organism and its relation with the environment directly to the brain [2].

Pain is not a disease and it is always subjective [3], it is manifested, in addition to pain, as the activity of sympathicus, producing fear, anxiety, pupilla dila-

tion, tears, tachycardia, hypertension, nausea, vomiting, sound effects, and facial expressions. The level of perception of pain is not constant: the threshold of pain and responses to pain vary under different conditions [4,5]. Awareness of pain occurs at the thalamocortical level where many complex interactions shape the overall experience and response. Postcentral girus determine awareness of the localization of stimuli, temporal lobus, with the help of memory, identifies the nature of the stimuli, frontal lobus and limbic system provide emotional reactions and the hypothalamus and pituitary gland control of autonomic and endocrine response [6,7].

It is exactly pain which is the most common reason for patients to come to the dental clinic; this pain usually originates in the tooth itself or its supporting structures.

In order to establish a proper diagnosis, it is absolutely important to take anamnesis, ie, a detailed subjective description of the painful condition of the patient, including the quality, volume, duration, frequency and periodicity of pain.

Anamnesis is complemented by the clinical finding of the therapist, which consists of inspection, palpation, thermal testing, testing the pulp vitality, inspection of periodontium, percussion and taking of an X-ray [8,9,10].

Pain as an unpleasant emotional experience is typically associated with actual or potential tissue damage. It has a warning function of a tissue damage and it activates the defense mechanisms in order to prevent further damage [11-14].

The pain-process involves a number of chemical pain mediators. Thus, it is known that the teeth are innerved by sympathetic nerve fibers, which release norepinephrine as a mediator, and the sensory fibers, which release acetylcholine and substance P [15]. Of other mediators, there are also vasoactive peptides and calcitonin, which participate in the increase of the dentine sensitivity [16]. Nerve fibers that connect teeth with the central nervous system belong to the fifth brain nerves (N. Trigeminus) and autonomic nervous system (sympathetic nervous system) [17,18].

Sensory nerve fibers in the pulp consist of myelinized A-fibers, which prevail, and non-myelinized C-fibers. Of the former, these are mainly A-delta fibers, which conduct the impulses faster, while, speaking of the latter, C-fibers, which are thinner and slower conducting [19].

A-delta fibers are responsible for strong, immediate, sharp, well localized pain [20], and C-fibers for dull, continuous, and irradiating pain [21]. Today, the most accepted theory of transmission of pain stimuli through the dentin to the

pulp, is hydrodinamic theory, proposed by Gysi [22], and later developed by Brannstrom et al. According to this theory, pain provoked by stimuli (thermal, chemical and mechanical) is a consequence of fluidal flow in the dentinal tubuli, at the speed of 2-4 mm / sec. Such circulation stimulates the mechanoreceptors and leads to the initiation of neural impulses in subodontoblastic plexus Raschkov and interodontoblastic plexus Bradlow in the pulp, resulting in the emergence of pain.

The effect of a short heat or cold stimulus is explained by the hydrodinamic theory in the following way: the application of hot stimuli on the exposed dentin leads to the expansion of dentinal fluid, whereas the application of cold stimuli causes its contraction. Both types of stimuli cause fluid flow, thus the activation of mechanoreceptors of the sensory nerves.

Chemical stimuli applied to the exposed dentin (sweet and salty foods) also lead to a faster flow of dentinal fluid to the surface of teeth. This is a consequence of low concentration of the dentinal fluid, which - because of its lower osmolarity – tends to flow towards a higher concentration of liquids and it is this current that re-stimulates the mechanoreceptors. However, if an isotonic liquid is applied onto the exposed dentin, painful sensations are not reported [23].

CLASSIFICATION AND DIFFERENTIAL DIAGNOSTICS OF PAIN

Odontogenic pain

Odontogenic pain has its source in the pulpodentinal complex and/or periapical tissue [24,25]. These two structures are functionally and embryonicly different and, consequently, the pain originating in these areas is perceived differently.

Dentinal and pulpal pain

According to clinical classification, we can distinguish several conditions of the pulp. These are: a healthy pulp, inflammatory-altered pulp, with the possibility of recovery (reversible pulpitis), inflammatory-altered pulp, without the possibility of recovery (irreversible pulpitis), and pulp necrosis.

In a healthy pulp, the cold and warm stimuli produce pain which lasts 1-2 seconds. Dentin-hypersensitivity is a result of exposed dentin, as dentin reacts to thermal, chemical, osmotic and tactile stimuli and pain is sharp, strong and short-lasting. However, not every exposed sensitive dentin shall necessarily provoke pain; open dentinal tubuli are usually more sensitive than occluded ones. A diagnosis of dentin hypersensitivity is set by the finding of exposed dentine

and by means of a provocation test (dragging the probe over the exposed dentin will cause a short, sharp pain).

Reversible pulpitis is most commonly found after the restorative treatment, as a result of inadeqate preparation procedures of the teeth and / or toxic components of the materials used, or the ditching around the filling. It is characterized by a short-term pain on the cold, which quickly disappears after the removal of pathological stimuli. To diagnose reversible pulpitis, the clinician needs anamnesis, clinical status, X-ray findings, and then he can identify its causes as caries, fracture, worn-out filling, ditching, recent restorations of the teeth, etc.

In irreversible pulpitis, pain is initially transferred by A-delta and C-fibers, and as the inflammatory process progresses, C-fiber-transmission prevails, which is then reflected by the change of the character of pain.

The symptoms include:

- intense, persisiting pain on warm stimuli, and after the removal of the stimulus, the pain changes to dull and pulsating (A-delta fibers have already transferred the information expediently, whereas the C-fibers are still transmitting);
- pain subsides on cold stimuli because of vasoconstriction and consequent reduction of intrapulpal pressure. This phenomenon points to the advanced necrosis of the pulp;
- spontaneous pain;
- when pain is dominated by C-fibers transmission it becomes diffused, and the cause is more difficult to identify;
- intensive and prolonged pain can lead to referred pain (in the ear, temporal region, cheek).

Irreversible pulpitis is difficult to diagnose until the stage when periradicular tissue is affected. However, as the inflammation spreads to periapical tissue, the tooth becomes sensitive to percussion and so the affected tooth is easily identified.

In pulp necrosis the pulpal sensory neurons are damaged, for which reason it does not respond to thermal and electrical stimuli, but in the case of partial necrosis a reaction may be present, because the C-fibers are more resistent to hypoxia than A-delta fibers. This is usually the case with multirooted teeth, and when pulp is extirpated it may still be sensitive [10,11].

Periadicular pain

Periradicular pain is usually caused by spreading of the infection from the pulp into periapical tissue. In the PDL are proprioceptors which allow a precise localization of the pressure-stimuli, so a periapical process is easily diagnosed.

If the periapical process has imminently followed the irreversible pulpitis, the clinical finding will contain symptoms of both irreversible pulpitis and periapical process (sensitivity to bite, with a dull, persisting, pulsating pain). With the progression of the inflammatory process through the alveolar bone, symptomatology is amplified, and the patient may report fever, malaise, swelling and rash. We distinguish between a localized process (abscess) and a process with unlimited progression into the tissue (cellulitis). The process shall expand in the direction of least resistance, and the most painful phase of the penetration is when it reaches the periosteum. After this, the process shall penetrate the submucosal tissue or produce a fistula, or spread into the soft-tissue spaces. By creating a fistula, the pain is soothed, and the process turns into a chronic [10,11,16].

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.

The key location of the phenomenon of the reflected pain is the spinal core of the trigeminal nerve. It is located caudal-most in the complex of the cores of nervus trigeminus toward the spinal cord, where it connects directly to supstantia gelatinosa and the spongiosis zone. The spinal core is divided into subnucleus oralis, subnucleus interpolaris and subnucleus caudalis.

The mechanism of the referred pain is explained by the theory of convergence. Aferrent nociceptive nerve fibers which conduct the stimuli from different parts of the head and neck converge in the area of the second neuron of the sensory pathway of pain in subnucleus caudalis of the spinal core of nervi trigemini. Aferrent fibers which also converge here belong to cranial nerves V, VII, IX and X, and the upper cervical nerves C2 and C3 respectively.

This means that painful signals from the facial area, teeth, temporomandibular joint, ear, pharynx, larynx, skull and other adjacent structures converge in the joint nociceptive area of the neurons located in subnucleus caudalis of the spinal core of n. trigeminus. It is the very proximity of these converging nerves, coming from different parts of head and neck, which causes complex synaptic communication in the subnucleus caudalis.

When the system for pain transfer gets activated, and nerve impulses travel to the brain centers, the higher centers are not able to identify the cause of the painful stimuli. Postcentral girus which determines the awareness of localiza-

tion of the painful stimuli, tends to contribute the painful sensations to e.g. recently restored teeth from which it has, until very recently, been receiving the actual painful signals. Toothache can reflect not only to the head and neck area, but to other teeth as well [10,11]. Differential diagnostics of pain of odontogenic origin is shown in Table 1 [10].

Table 1. Differential diagnostics of pain of odontogenic origin (adopted from ref. 10)

Pulpal	Symptoms	Radiographic	Pulp tests	Periapical tests
Normal	None of significance	No periapical changes	Responds	Not sensitive
Reversible	May or may not have slight symptoms to thermal_ stimulus	No periapical changes	Responds	Not sensitive
Irreversible	Similar to reversible; also may have spontaneous or severe pain to thermal stimuli	No periapical radiolucent changes; one exception: occasional condensing osteitis	Responds (possibly with extreme pain on thermal stimulus)	May or may not have pain on percussion or palpation
Necrotic	None to thermal stimulus Other symptoms: see under Periapical	See under Periapical	No response	Depends on periapical status
Periapical				
Normal	None of significance	No significant change	Response	Not sensitive
Acute apical periodontitis	Significant pain on mastication or pressure	No significant change	Response or no re- sponse (depends on pulp status)	Pain on percussion or palpation
Chronic apical periodontitis and apical cyst	None to mild	Apical racliolucency	No response	None to mild on percussion or palpation
Acute apical abscess	Swelling and/or significant pain	Usually a radiolucent lesion	No response	Pain on percussion or palpation
Suppurative apical periodontitis (chronic apical abscess)	Draining sinus or parulis	Usually a radiolucent lesion	No response	Not sensitive
Condensing osteitis	Varies (depends on pulp or periapical status)	Increased trabecular bone density	Response or no re- sponse (depends on pulp status)	May or may not have pain on percussion or palpation

NONODONTOGENIC PAIN

When the clinical examination has excluded odontogenic causes, extracranial causes of orofacial pain should be taken into consideration: salivary glands,

sinuses, nose, throat, thyroid gland, eye, ear, heart, esofageal cardiac sphincter, and lungs. Painful syndromes of the jaw which emulate toothache are divided into acute (neuralgia n. trigemini, "cluster" headaches, acute otitis media, acute maxillary sinusitis, cardiogenic jaw-pain, sialolithiasis) and chronic (temporomandibular joint disorders and cheek-muscle pain, atypical facial pain, allergic sinusitis, causalgia, posterherpetic neuralgia, facial pain as a result of malignant neoplasms) [26-33]. Overview of the symptoms is listed in Tables 2 and 3 [11].

Table 2. Differentiating acute nonodontogenic pains (adopted from ref. 11)

Condition	Nature	Triggers	Duration
Odontalgia	Stabbing, throbbing, noncpisodic	Hot, cold, tooth percussion	Hours-days
Trigeminal neuralgia	Lancinating, electrical, episodic	1-2 mm locus on skin/ mucosa, light touch triggers pain	Seconds
Cluster headache	Severe ache, retroorbital component, episodic	REM sleep, alcohol	30-45 min
Acute otitis media	Severe ache, throbbing, deep to ear, noncpisodic	Lowering head, barometric pressure	Hours-days
Bacterial sinusitis	Severe ache, throbbing in multiple posterior maxillary teeth, nonepisodic	Lowering head, tooth percussion	Hours-days
Cardiogenic	Short-lived ache in left posterior mandible, episodic	Exertion	Minutes
Sialolithiasis	Sharp, drawing, salivary swelling, episodic	Eating, induced salivation	Constant low-level ache, sharp brief episodes when triggered

In a study by Oklješa et al. [34], conducted within the framework of the scientific research by Faculty of Dental Medicine in Zagreb, it has been investigated how often patients come to the dental clinic because of toothache or generally any pain in the mouth, and what the respective percentages of acute or chronic pain were. The research was conducted on the sample of 2735 respondents over a period of 1 year. Pain was present in the 16.49 % of patients, and the remaining 83.51 % of the patients were without pain. With regard to the duration of pain, acute pain has been significantly higher (about 84 %) than chronic pain (about 16 %). The structure of acute (*Fig 1*) and chronic (*Fig 2*) pain odontalgia was present in 89 % of all cases of acute pain in 90 % of all chronic pain, which was significantly higher than the remaini ng types of pain combined. The representation of acute odontalgia with respect to the total number of patients was 12.36 % of patients, and the structure of is shown in *Figure 3*; chronic odontalgia was represented in 2.38 % of patients, and the structure is shown in *Figure 4*.

Table 3. Differentiating chronic aching and burning pains (adopted from ref. 11)

Condition	Nature	Triggers	
Odontalgia	Dull ache	Hot, cold, tooth percussion	Days-weeks
TMJ internal derangemeuls	Dull ache, sharp episodes	Opening, chewing	Weeks-years
Myalgia	Dull ache, degree varies	Stress, clenching	Weeks-years
Atypical facial pain	Dull ache with severe episodes	Spontaneous	Weeks-years
Phantom tooth pain	Dull ache with severe episodes	Spontaneous	Weeks-years
Neuralgia-inducing cavitational osteonecrosis	Dull ache with severe episodes	Spontaneous	Weeks-years
Allergic sinusitis	Dull ache,	Lowering head	Weeks-months
	Malar area, multiple posterior maxillary teeth		Seasonal
Causalgia	Burning	Posttrauma, postsurgical	Weeks-years
Post-herpetic neuralgia	Deep boring ache with burning	Spontaneous after facial shingles	Weeks-years
Cancer-associated facial pain	Variable, motor deficit, paresthesia	Spontaneous	Days-months

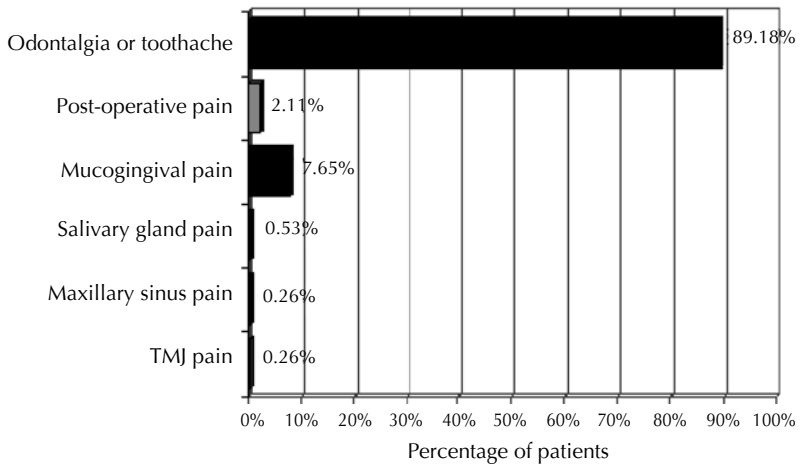

Fig. 1. Structure of acute pain: percentage of all patients with acute pain (adopted from ref. 34).

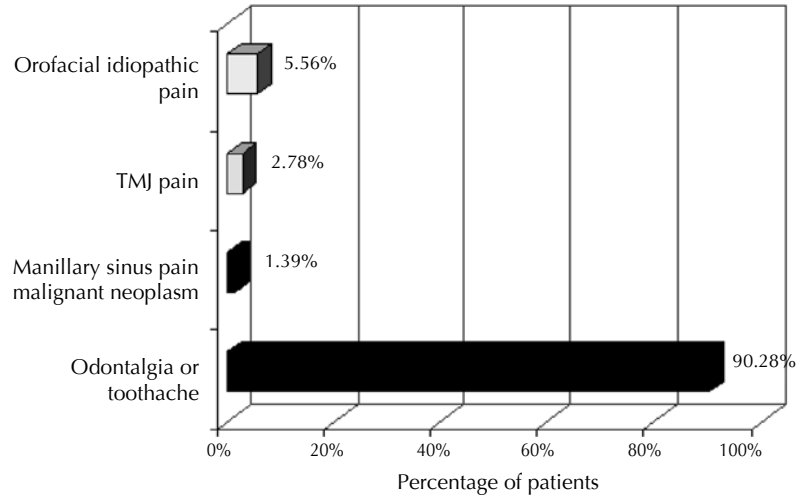

Fig. 2. Structure of chronic pain: percentage of all patients with chronic pain (adopted from ref. 34).

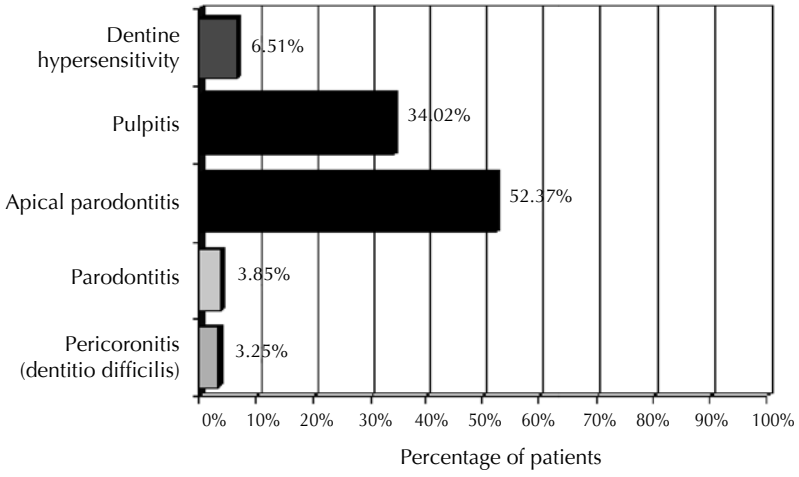

Fig. 3. Structure of acute odontalgia: percentage of all with acute odontagia (adopted from ref. 34)

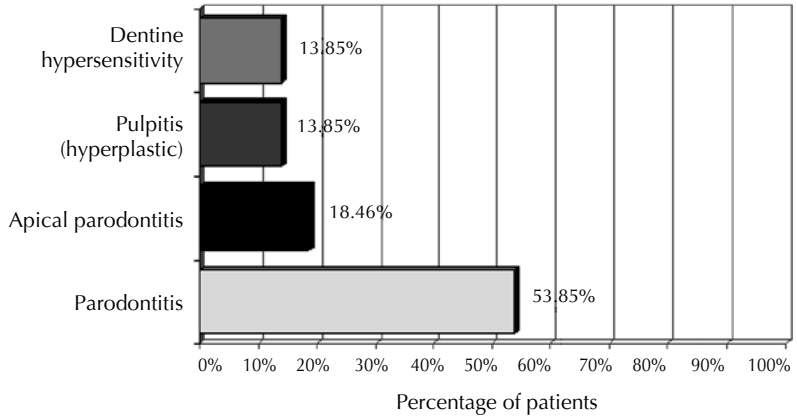

Fig 4. Structure of chronic odontalgia: percentage of all with chronic odontalgia (adopted from ref. 34). This research showed that only one in six patients come to the office of doctor of dental medicine because of pain, which points to an increased awareness of the patient regading care for their oral health. However, in order to reduce the incidence of pain in dental patients, especially when it comes to odontogenic pain, more attention should be paid to educate patients in the implementation of oral hygiene and regular check-up visits, for the purpose of an early diagnosis of possible pathological changes and preventive action [35].

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Sažetak

Odontogena bol

Bol ima funkciju upozoravanja na oštećenje tkiva te funkciju aktivacije obrambenih refleksa radi prevencije daljnjeg oštećenja. Podražaj koji oštećuje tkivo ili prijeti oštećenjem tkiva aktivira nociceptore koji prenose informaciju sustavom neurona do kore velikog mozga, gdje se obrađuje i interpretira kao bol. Većinu somatosenzornih informacija iz područja orofacijalnog sustava prenosi n. trigeminus. Da bismo uklonili bol, potrebno je prepoznati i dijagnosticirati uzrok boli. To nije uvijek jednostavno zbog mnogih varijacija kliničke slike te mogućnosti boli da se referira iz odontogenih struktura u neodontogene i obratno.

Poznavanje puta i mehanizma boli te mogućih uzroka i karaktera orofacijalne boli, odnosno detaljna anamneza, klinički pregled i testiranja, dovest će do prave dijagnoze. Odontogeni izvor boli obično ima tipičan karakter, evidentan uzrok, te se provokacijskim testovima dobivaju simptomi navedeni u anamnezi. Svakom odstupanju od klasične slike treba pristupiti s oprezom. Isključivanjem odontogenog uzroka boli treba ispitati ostale potencijalne uzroke orofacijalne boli kako bi se postavila pouzdana dijagnoza.

***Ključne riječi*:** odontogena bol; neodontogena bol; odražena bol; diferencijalna dijagnostika.

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