CHRONIC OROFACIAL PAIN: DIAGNOSTIC AND THERAPEUTIC CHALLENGE

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Summary

Chronic orofacial pain is reviewed, especially from the aspect of common diagnostic and therapeutic dilemmas. Chronic orofacial pain is a more frequent phenomenon than usually regarded, as time rich with emotional tension greatly favours the specific release of endogenous conflicts, which may also be manifested by chronic orofacial pain, especially psychogenic orofacial pain.

In this article, characteristic modalities of neurogenic, somatic and psychogenic pain in the orofacial region are described, pointing to the main diagnostic criteria and therapeutic possibilities. The rationale for setting up and organizing pain clinics is stressed, as treatment by several specialists at the same place is more convenient, both to the patient and the practitioner, and gives a better chance for success than referral to other specialists, especially to psychiatric clinics.

Key words: Pain, facial, chronic; Pain, neurogenic; Pain, somatic; Pain psychogenic

Introduction

Orofacial pain is the most frequent reason patients seek dental attention. However, the precise diagnosis of orofacial pain is not always simple. In that respect, the greatest diagnostic (and therapeutic) problem presents chronic pain, especially when there is no obvious organic cause (for example, malignant disease).

Pain is an unpleasant emotional experience resulting from either physical or psychological trauma. Painful experience includes not only a perception of sensations evoked by noxious stimuli, but also the reaction or response to such stimuli. Unless the patient is supposed to be a conscious malingering or layer, the painful experience is always unpleasant and always real. The psychological component of this experience has always been accepted and psychological factors, such as previous experience, prejudice, emotional status etc, may significantly influence and intensify it or cause the persistence of pain even when the original tissue damage has disappeared.

Pain of more than 6 months' duration is considered to be chronic. In contrast to acute pain, which serves a biologic purpose as either a protective mechanism or a warning signal, chronic pain does not serve any biologic purpose and appears to be permanent and intensified by psychological factors. In fact, chronic pain may be considered as a product of complex interaction of biologic, i.e. organic and psychological factors, and of an acquired "illness
behaviour” as well as some behavioural factors (bruxism habit, chronic muscle contraction) which significantly contribute to arising pain.

Chronic orofacial pain may have many different clinical presentations. However, all these conditions (except cancer pain, which may have chronic presentation but does not have diagnostic or therapeutic dilemmas) can be grouped into three main pain categories: neurogenic pain (caused by structural abnormalities within the neural components), somatic pain (caused by abnormalities in normal structures, but sometimes having referred pain characteristics) and psychogenic pain (caused primarily by disturbances in the psychogenic sphere, but also showing sometimes changes within the neural component).

**Neurogenic Pain**

Neurogenic pain is estimated to be caused by a functional abnormality within the nervous system. However, the exact etiopathogenetic mechanism of the most prominent representative of this chronic orofacial category - a paroxysmal (idiopathic) trigeminal neuralgia is still not precisely determined.

**Paroxysmal Trigeminal Neuralgia**

Paroxysmal trigeminal neuralgia (PTN) is a precisely defined clinical entity, with a clinical picture that distinguishes it from other, more or less similar orofacial pain syndromes. Although known for centuries, PTN still produces controversies, beginning with terminological (idiopathic or primary trigeminal neuralgia, tic douloureux, etc) closing with therapeutic (a plethora of treatment modalities, no one guaranteeing permanent pain relief).

**Clinical presentation.** PTN is characterized by lancinating pain, the most intensive pain ever experienced by humans. Pain is most frequently described as a sudden, sharp, severe stabbing or lancinating, lasting relatively briefly (from a few seconds to a minute). It is of paroxysmal nature, repeating throughout the day (rarely at night). Pain is characteristically provoked by stimulation of one, more or less constant sensory region of the skin of the face or oral mucosa, so-called trigger-zone, which is one of the patognomonic symptoms. Trigger-mechanism is usually provoked by light touch, talking, chewing or swallowing. After provoking a trigger-mechanism, pain is elicited within a single division of the trigeminal nerve (most often maxillary or mandibular). Immediately after the attack, there is a short “refractory period” when it is impossible to provoke pain. The patient is usually pain-free between attacks, but sometimes a dull, constant pain may persist between attacks of sharp pain.
Periods of spontaneous remission of pain attacks are characteristic for the PTN. They can be brief (few months) or more protracted (till a year) - an especially confusing feature because coincidental treatment may be given the (undeserved) credit for bringing on what is in fact a spontaneous subsidence of pain. However, the spontaneous remissions gradually get shorter, especially in untreated cases. Spontaneous recovery has not been reported.

**Diagnostic criteria.** As pain, a most prominent symptom of the PTN, demonstrates individual experience which is difficult to present objectively, the diagnosis of PTN is not always simple. It is primarily based on the detailed description of pain characteristics, as well as on the elimination of other possible causes of pain.

Classic criteria for making the diagnosis of PTN are: (1) the intensive, brief pain of paroxysmal character, with complete remissions occurring between attacks; (2) the existence of a trigger-zone provokable by even a light sensory stimulation; (3) unilateral pain confined to the distribution of the affected nerve-division; (4) normal neurological findings at examination, without objective sensory or motor disturbances.

There are no laboratory tests or radiographic findings pointing to the PTN. Nevertheless, each patient with presumable diagnosis of PTN should be submitted to detailed clinical and radiographic examination, including the routine laboratory tests, in order to exclude other syndromes and diseases with similar clinical presentation. In diagnosing PTN, a positive response to local anaesthetic injection or the undertaken medication reinforces the clinical impression and confirms the diagnosis. Specific changes of somatosensory evoked potentials in PTN patients are also observed, but it is not clear whether these results may be used with certainty in differential diagnosing of the PTN.

The PTN may be confused with three main types of syndromes: (1) paroxysmal (idiopathic) neuralgia involving other nerves; (2) symptomatic neuralgia; and (3) facial pain other than paroxysmal neuralgia.

Idiopathic neuralgia may affect, although only occasionally, other nerves than trigeminal - the glossopharyngeal and the superior laryngeal nerves. Many pain characteristics are similar to the PTN, but pain appears in tonsils and the root of the tongue, referring to the ear and the throat. It is usually provoked by swallowing or speaking. The diagnosis of glossopharyngeal neuralgia may be established by simple local anaesthetic spraying of the throat: a relief of pain indicate the glossopharyngeal neuralgia.

Symptomatic (secondary) trigeminal neuralgias are caused by both peripheral and central lesions of the trigeminal nerve. Intracranial causes of secondary neuralgias may be the posterior and the middle cranial fossa lesions, and extracranial causes comprise compression or entrapment of peripheral trigeminal branches. In the very beginning of the disease, symptoms may imitate PTN, but gradually pain becomes more constant, usually accompanied
by signs of affection of other nerves (neurological, sensory or motor disturbances). Detailed oral, otorhinolaryngological, neurological and radiographic examinations, as a rule, reveal the cause of pain (in contrast to the PTN).

Chronic orofacial pain other than idiopathic neuralgia may sometimes mimic true PTN pain. This is especially the case with vascular and psychogenic pains. However, pain in these conditions is more constant, localized on the wider region and, especially with psychogenic pain, not confined precisely to the certain nerve division. Nevertheless, sometimes a great clinical experience is needed to differentiate these syndromes from PTN solely on the basis of clinical characteristics of pain.

**Treatment.** Due to unknown aetiology and pathogenesis of the PTN, there is no uniform mode of treatment. Several drugs and procedures were proposed in due course, but only some of them maintained judgement of time.

Generally speaking, treatment of PTN can be medical or surgical. Numerous surgical procedures, which can be didactically grouped into intracranial and extracranial ones, are reserved for patients who do not respond to medication, or cannot use it for other reasons (for example, allergy).

Medical treatment in many treatment protocols takes the first line owing to the discovery of Blom that the antiepileptic drug carbamazepine (Tegretol, Mazepine), can provide pain relief in PTN patients. Although there is other anticonvulsant medication useful in the treatment of PTN, carbamazepine is still the sovereign remedy for that purpose, effective in 60-90% of the treated patients. Moreover, this drug is so specific in abolishing PTN pain that can be used even for diagnostic purpose. The therapeutic dose of the drug gradually rises from 200-400 mg daily to the maximal 1200 mg daily dose owing to the attained effect. Unfortunately, in 25-50% of the patients, after several years of successful treatment, carbamazepine becomes ineffective.

An analysis of results of the carbamazepine treatment of PTN, performed at the Clinic of Oral Surgery, Faculty of Stomatology in Belgrade, however, showed slightly poorer effect of this medication than is usually presented (Tab. 1). The positive effect of the treatment, in this series, was noticed in only 11 of 19 patients (58%), with pain relief of 12 months maximum. At the same time, side-effects were noticed in 7 patients, although not of a serious character (dizziness, drowsiness and sluggishness).

Peripheral nerve blocks (blockades) are, usually, the next step in the PTN treatment. This term comprises the injection of some neurolytic substances in the vicinity of the affected peripheral nerve for the purpose of relieving pain. The injection technique is the same as for the anaesthetic blocks performed routinely for achieving local anaesthesia, only with the substances other than local anaesthetics.
### Table 1. Clinical Data of Patients with PTN and Results of Carbamazepine Treatment

<table>
<thead>
<tr>
<th>Patient</th>
<th>The Affected Nerve*</th>
<th>Duration of PTN</th>
<th>Initial Dose**</th>
<th>Painless Period</th>
<th>Side-effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>V3</td>
<td>3 months</td>
<td>0.6</td>
<td>3 months</td>
<td>yes</td>
</tr>
<tr>
<td>2.</td>
<td>V3</td>
<td>4 weeks</td>
<td>0.6</td>
<td>4 months</td>
<td>#</td>
</tr>
<tr>
<td>3.</td>
<td>V2/3</td>
<td>5 years</td>
<td>0.6</td>
<td>5 days</td>
<td></td>
</tr>
<tr>
<td>4.</td>
<td>V3</td>
<td>1 month</td>
<td>0.6</td>
<td>12 months</td>
<td></td>
</tr>
<tr>
<td>5.</td>
<td>V2</td>
<td>2 years</td>
<td>0.6</td>
<td>0</td>
<td>#</td>
</tr>
<tr>
<td>6.</td>
<td>V3</td>
<td>7 years</td>
<td>0.6</td>
<td>0</td>
<td>yes</td>
</tr>
<tr>
<td>7.</td>
<td>V2</td>
<td>1 month</td>
<td>0.6</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>8.</td>
<td>V2</td>
<td>8 years</td>
<td>1.2</td>
<td>6 weeks</td>
<td></td>
</tr>
<tr>
<td>9.</td>
<td>V3</td>
<td>1 month</td>
<td>0.6</td>
<td>0</td>
<td>yes</td>
</tr>
<tr>
<td>10.</td>
<td>V3</td>
<td>4 years</td>
<td>0.6</td>
<td>1 month</td>
<td></td>
</tr>
<tr>
<td>11.</td>
<td>V3</td>
<td>4 years</td>
<td>1.2</td>
<td>1 month</td>
<td></td>
</tr>
<tr>
<td>12.</td>
<td>V3</td>
<td>3 months</td>
<td>0.6</td>
<td>0</td>
<td>yes</td>
</tr>
<tr>
<td>13.</td>
<td>V3</td>
<td>2 years</td>
<td>0.6</td>
<td>0</td>
<td>#</td>
</tr>
<tr>
<td>14.</td>
<td>V2</td>
<td>2 years</td>
<td>0.6</td>
<td>6 months/still</td>
<td></td>
</tr>
<tr>
<td>15.</td>
<td>V2/3</td>
<td>3 years</td>
<td>1.2</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>16.</td>
<td>V2/3</td>
<td>3 months</td>
<td>1.2</td>
<td>0</td>
<td>yes</td>
</tr>
<tr>
<td>17.</td>
<td>V3</td>
<td>1 month</td>
<td>0.6</td>
<td>3 months</td>
<td>yes/#</td>
</tr>
<tr>
<td>18.</td>
<td>V3</td>
<td>2 years</td>
<td>0.6</td>
<td>9 months/still</td>
<td></td>
</tr>
<tr>
<td>19.</td>
<td>V3</td>
<td>1 week</td>
<td>0.6</td>
<td>3 months/still</td>
<td></td>
</tr>
</tbody>
</table>

* - V2 – Maxillary Nerve; V3 – Mandibular Nerve  
** - g/daily  
# - refuses therapy

The use of solely local anaesthetics for the same purpose (peripheral nerve block) is least effective, producing pain relief in PTN only sporadically and in the beginning of the condition⁸,¹⁵. Controlled, double blind study of the effect of peripheral nerve blocks with local anaesthetic (lidocaine) and the solution of streptomycin in lidocaine in the treatment of PTN, performed in two separate institutions (Yugoslavia and UK)²³, however, showed that local anaesthetic may be efficient in some patients, with a pain relief period of more than 30 months (Tab. 2). Nevertheless, local anaesthetics are used primarily for diagnosing PTN, as test-blocks performed at the first visit. If the pain relief is achieved even after the relapse of local anaesthesia, our policy is not to undertake any other procedure till the recurrence of pain. So, in these cases, peripheral nerve blocks with local anaesthetics may be regarded as a therapeutic procedure.

Ethyl-alcohol (95%) is most frequently used for peripheral nerve blocks. Alcohol causes neurolysis when contacting peripheral nerve⁸, producing anaesthesia of the region innervated by the treated nerve division, which is an unpleasant consequence of the alcohol blockade.
Table 2. Treatment Results of Peripheral Streptomycin/Lidocaine versus Lidocaine Alone Injections in the Treatment of PTN

<table>
<thead>
<tr>
<th>Patient</th>
<th>Blockade*</th>
<th>Treated Nerve**</th>
<th>Painless Period</th>
<th>Recurrence</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>L</td>
<td>V3</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>2.</td>
<td>S/L</td>
<td>V2</td>
<td>5 months</td>
<td>yes</td>
</tr>
<tr>
<td>3.</td>
<td>L</td>
<td>V3</td>
<td>17 months</td>
<td>no</td>
</tr>
<tr>
<td>4.</td>
<td>S/L</td>
<td>V2</td>
<td>3 weeks</td>
<td>yes</td>
</tr>
<tr>
<td>5.</td>
<td>L</td>
<td>V2</td>
<td>26 months</td>
<td>no</td>
</tr>
<tr>
<td>6.</td>
<td>L</td>
<td>V2</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>7.</td>
<td>S/L</td>
<td>V2</td>
<td>2 weeks</td>
<td>yes</td>
</tr>
<tr>
<td>8.</td>
<td>S/L</td>
<td>V3</td>
<td>30 months</td>
<td>no</td>
</tr>
<tr>
<td>9.</td>
<td>L</td>
<td>V3</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>10.</td>
<td>S/L</td>
<td>V3</td>
<td>3 months</td>
<td>yes</td>
</tr>
<tr>
<td>11.</td>
<td>S/L</td>
<td>V2</td>
<td>6 months</td>
<td>yes</td>
</tr>
<tr>
<td>12.</td>
<td>S/L</td>
<td>V3</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>13.</td>
<td>S/L</td>
<td>V3</td>
<td>14 months</td>
<td>no</td>
</tr>
<tr>
<td>14.</td>
<td>L</td>
<td>V2</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>15.</td>
<td>L</td>
<td>V2</td>
<td>8 months</td>
<td>yes</td>
</tr>
<tr>
<td>16.</td>
<td>S/L</td>
<td>V3</td>
<td>6 months</td>
<td>no</td>
</tr>
<tr>
<td>17.</td>
<td>L</td>
<td>V2</td>
<td>0</td>
<td></td>
</tr>
</tbody>
</table>

* - L - Lidocaine Alone;  
S/L - Solution of Streptomycin in Lidocaine  
** - V2 – Maxillary Nerve; V3 – Mandibular Nerve

In addition, pain recurs after 6 to 24 months8, and subsequent blocks are more difficult to perform and less effective due to the fibrous tissue formed after previous injection8. This is the reason some authors do not recommend peripheral alcohol blocks as a treatment modality for PTN patients10.

It seems that anhydrous organic alcohol glycerol is a better alternative to ethyl-alcohol for peripheral nerve blocks in PTN. Due to its hygroscopic properties, it diffuses through perineurium, absorbing water and subsequently damaging nerve axons24. It is shown that glycerol has a satisfactory effect on pain relief in PTN when applied peripherally in the vicinity of the affected nerve25. A preliminary study, conducted at the Clinic of Oral Surgery in Belgrade22, showed pain relief from 6 to 24 months in 70% of patients with PTN treated with peripheral glycerol nerve blocks (Tab. 2). It is suggested that glycerol should be injected 20-30 minutes after local anaesthetic, as not to be unnecessarily diluted22.

An attempt to use the solution of streptomycin in lidocaine for peripheral nerve block in PTN patients deserves attention, preliminary results of the procedure being firstly reported in 198626. The procedure comprised 5 peripheral nerve blocks, the time between each injection varying from 3-7 days.
according to the intensity and frequency of pain attacks. Even this preliminary study showed very encouraging results, the double blind, controlled one demonstrated a slightly less favourable, but still beneficial effect23.

Open surgery on peripheral nerves, so-called peripheral neurectomy, has long been advocated for the treatment of PTN8. By surgical avulsion of a certain segment of the affected peripheral nerve, a relatively long period of pain relief is achieved. The major disadvantage of peripheral neurectomy is that total anaesthesia of the corresponding region results, while pain relief is only temporary. Repeating the procedure is much more difficult because of the presence of fibrous scar tissue, and should be performed at the other site. These are the reasons peripheral neurectomy is not widely used10.

Cryosurgical techniques, based on the freezing effect of liquid nitrogen probes at the surgically exposed nerve, have been used for the treatment of PTN for approximately two decades27,28. Cryoanalgesia can be achieved even without surgical exposure of the affected nerves. The advantages of cryotherapy are relatively fast recovery of sensation (approximately after 3 months), relatively long remissions, the possibility of performing on an outpatient basis and the absence of major complications.

Intracranial percutaneous or open surgery is usually left for resistant cases of PTN, when other procedures fail. It comprises blockade of the gasserian ganglion, percutaneous retrogasserian coagulation or major surgery on the sensory root of the trigeminal nerve. The injection of glycerol into the trigeminal cistern30 has increasingly gained followers10. Pain relief is comparatively long (even more than 6 years) and recurrence is quite low (12-31%)30-32. Percutaneous retrogasserian radio frequent rhizotomy selectively destroys preganglionic trigeminal nerve fibres, through small electrode, by thermo-coagulation of trigeminal rootlets33, giving favourable effect but comparatively frequent recurrence. Major intracranial surgery is out of the scope of oral and maxillofacial surgery.

In conclusion, it should be stressed that an ideal procedure in the PTN treatment does not exist. The treatment plan is, therefore, individual, adapted to every single patient, as well as to the possibilities and experience of the therapist. Even if there are some differences in treatment protocols between institutions treating PTN patients, general trend is directed to less invasive procedures, primarily medical therapy, and open surgery being left for resistant cases and in late stages of the condition. At the Orofacial Pain Centre at the Faculty of Stomatology in Belgrade, priority is given to less invasive procedures in the treatment of PTN, such as medical treatment and peripheral nerve blockades with glycerol or the solution of streptomycin in lidocaine. The results of these types of treatment are quite satisfactory23,25,26, which justifies the recommendation of using such procedures as a first step in the treatment of PTN patients.
Somatic Pain

Somatic pain may be divided into superficial and deep somatic pain. Superficial somatic pain has a bright quality of stimulating nature and is involved in acute pain; deep somatic pain, on the other hand, has a dull, deep, depressing quality, and is often involved in chronic orofacial pain.

Musculoskeletal pain falls into the category of deep somatic pain; in the orofacial region, this pain category refers to temporomandibular joint and masticatory muscle problems, usually coexisting and overlapping with signs and symptoms. This is the reason why the term arthromyalgia, proposed by Harris, seems to be the most appropriate for the chronic pain syndrome otherwise variably called the temporomandibular joint dysfunction syndrome, myofascial pain dysfunction syndrome or, at the beginning, Costen’s syndrome. However, in quite a number of patients, temporomandibular joint dysfunction can be distinguished from myofascial pain dysfunction. In the rest of unclear cases it is crucial to recognize and identify the basic and primary cause of discomfort as the treatment may significantly vary, from solely to combined medical, dental and surgical.

Temporomandibular Joint Pain

Temporomandibular joint pain arises due to the “internal derangement”, defined as an abnormal relationship of the articular disc (meniscus) to the mandibular condyle, glenoid fossa and articular eminence. The first sign of the internal derangement is a painless joint sound (click), which occurs on movement of the joint, indicating a subluxation of the posterior band of the meniscus. The click remains painless as long as the main insertions of the meniscus into the mandibular condyle poles are not threatened. A painful click arises when the main insertions begin to fail and the whole meniscus begins to luxate. This is possible only if there is a stretching or tearing of the insertions of the meniscus into the condylar poles, and this is accompanied by pain and tenderness to palpation over the lateral pole - a usual clinical finding.

Arthralgia is characterized by continuous, localized joint pain that increases with jaw function. The maximum jaw opening motion is sometimes limited or deviated to the affected side. The joint is usually tender on palpation. The condition is termed also as capsulitis and synovitis. Localized arthralgia/arthritis can be caused by external trauma to the joint or by repetitive malfunction of the jaw (fingernail biting, bruxism, and clenching teeth). Due to subluxation, meniscus no longer separates the condyle from its fossa to the same degree as in its correct anatomical position and a malocclusion may result. On the other hand, occlusion may affect the joint indirectly, by altering muscle activity, and directly. However, many studies suggest there is no demonstrable relationship between the occlusion and
temporomandibular joint pain\textsuperscript{39}. Finally, as the direct relationship between emotional factors, inducing the increased muscle activity, and temporomandibular joint dysfunction has been demonstrated\textsuperscript{40}, a hypothesis is developed, connecting emotional status, muscle activity and joint dysfunction, although correlations between these factors have not been clearly established\textsuperscript{39}.

Due to different possible etiological factors, it is apparent that, consequently, the treatment also can be directed to various presumable sources. There are several treatment modalities, from simple reassurance and occlusal correction (necessary to allow bilateral mastication), to joint surgery comprising arthrocentesis, meniscoplasty or meniscectomy\textsuperscript{41,42}. The usual policy is a conservative approach - to try with the least invasive procedures (occlusal correction, making bite-guard appliances, physiotherapy, psychotropic medication), leaving surgery for those cases where conservative treatments have failed\textsuperscript{42}.

\textbf{Muscular (Myofascial) Pain}

Masticatory muscle myalgia can occur as a result of direct muscle tissue trauma, forceful jaw-closing habits (bruxism) or prolonged protective muscle activity secondary to regional pain\textsuperscript{43}. Typical characteristics of myofascial pain are dull, aching, continuous pain increasing with function, with local palpable tender bands in the muscle and trigger points within these bands\textsuperscript{44}. Trigger points are tender to palpation and exogenous factors such as tension, trauma or weather changes, which may increase pain from mild to agonizing\textsuperscript{4}. In early stages, the pain may be more localized, but with chronicity, the trigger point can refer pain to more distant sites\textsuperscript{45}.

The mechanism for these chronic muscle pain phenomena is not clear, but speculations include theories involving localized hypoxia, central sensitization and neurogenically induced pain-peptide secretion at the tender sites\textsuperscript{3}.

Specific myofascial trigger point therapies include stretch and (cold) spray, post-isometric relaxation, injection of trigger points, massage applied to the taut bands and trigger points and physiotherapy (ultrasound, electrical stimulation)\textsuperscript{4}. However, the success of the treatment depends on the precise differential diagnosis to the temporomandibular joint pain and psychogenic pain syndromes.

\textbf{Psychogenic Pain}

Chronic orofacial pain of psychogenic nature is a much more frequent phenomenon than is usually regarded\textsuperscript{47}. The time rich with emotional tension greatly favours the specific release of endogenous conflicts, which may be manifested by chronic orofacial pain as well. According to some reports\textsuperscript{48}, 2-5\%
of patients with chronic orofacial pain have no demonstrable organic disease, or have an exaggerated response to minimal pathology.

Psychogenic pain, like any psychosomatic disturbance, can arise in three ways, each manifesting differing degrees of severity and chronicity: (1) as a manifestation of stress in an otherwise fit and stable person; (2) as a symptom of psychiatric illness; or (3) as a feature of an unchanging hypochondriacally personality trait. It is important to recognize that this pain is real, arisen initially in dilated blood vessels. It is supposed that there is a disturbance of the central descending pain inhibitory system that normally filters chronic peripheral discomfort in these patients.

Psychogenic pain is characteristically constant and nagging, of aching and burning nature, located deep into tissues and difficult to describe. It is not well localized and crosses over normal neurological boundaries. The typical patient is, according to Miller, a middle aged female, edentulous, haggard and importunate. As there is no oral or dental abnormality which can be associated to the apparent symptoms, the term “atypical facial pain” is frequently used, although it is not officially recommended. However, the term “atypical” seems to be more convenient than the term “psychogenic” in routine communication, especially with a patient, because it does not suggest that the pain is imagined - a fact which could cause the patient to become upset.

Psychogenic orofacial pain can be manifested differently, the most interesting (and characteristic) presentation being atypical odontalgia. This condition is characterized by continuous or throbbing pain localized in a single tooth (or several teeth in the region), otherwise indistinguishable from that of an acute pulpitis or periodontitis. Affected teeth are extremely sensitive to pressure or thermal stimuli. Some cases appear to have been precipitated by a dental procedure, such as the fitting of a bridge or an extraction. The main feature of the condition is the lack of any detectable sign of dental pathology, although the detailed anamnesis can reveal previous attempts to “treat” the condition by unnecessary dental procedures (pulpectomies, root fillings, apicoectomies, extractions, etc.). Although symptoms may vary, a carefully taken history usually reveals some emotional problems or recurrent migrainous attacks. This is the reason the condition is assumed to be a state of hyperalgesia of pulpal/periodontal pain receptors, suggesting a vascular disturbance.

To arrive at an accurate diagnosis of psychogenic pain, the first and most important step is to take a complete history and to obtain relevant information, such as: chief complaint(s), location of pain and its characteristics (quality, intensity, temporal behaviour, provoking, aggravating or relieving factors), chronology of complaints and personal and past medical history. Objective signs of any kind of oral pathology usually lack. However, investigations of tooth pulp evoked potentials (TPEP) have revealed some indicative, although discrete, changes in patients with psychogenic pain. These changes are seen in a shorter latency of the first negative wave on the side with pain compared to the opposite side or to control patients. Also, the
second negative wave has a prolonged latency on the side with pain, with a plateau instead of an amplitude peak (Fig. 1). Changes of TPEP in psychogenic pain patients indicate the existence of slower impulse transmission and the reduction of cognitive inhibitory pain control at both synaptic junctions (at pons and thalamus).

![Fig. 1. Typical TPEP in:](image)
(a) Healthy individual: N1 - the first negative wave showing the first synaptic junction; P1 -the first positive wave showing impulse transmission in lemniscus medialis; N2/P2 - the second negative and the second positive waves showing impulse transmission at the thalamo-cortical level;
(b) A patient with atypical facial pain: on the side with pain the changed N2 wave, with plateau phenomenon

Treatment of psychogenic orofacial pain is not simple, especially if one persistently tries to find out an organic cause of present pain. The patient usually undergoes unnecessary dental or surgical treatment without any improvement (it even gets worse), and pain moves from one tooth or place to another. The patient should be subjected to essential dental care only, with additional explanation that the actual pain is a migraine-like pain in the teeth and surrounding tissues due to dilated blood vessels or hyperactive muscles, provoked by emotional pressure and stress. Finally, it was shown that tricyclic antidepressants improve symptoms in patients with psychogenic pain, as well as with facial arthromyalgia. It seems that they are successful even in the absence of depression. The explanation of antidepressants’ efficacy probably lies in the fact that they increase the serotonin, noradrenaline and dopamine concentrations in synapses, preventing their re-uptake. Tricyclic antidepressants are effective in the vast majority of psychogenic pain patients; however, the improvement can not be demonstrated immediately, but a few months after the beginning of the therapy.
**Possible Conclusions**

Many patients experiencing or anticipating chronic orofacial pain seek oral and dental treatment. In addition, an orofacial region is one of the most complex of all regional areas in terms of identification of pain\textsuperscript{48}, having an extremely complex cranial and cervical innervation and an intricate vascular supply. There are many other pain modalities and types apart from those mentioned in this review (for example, different vascular pains, post-traumatic and secondary neuralgias, etc.). It is obvious that precise diagnosing of all types of chronic orofacial pain requires a lot of experience and undertaking of the most promising procedures only after careful evaluation of all available data.

Whenever a patient presents for evaluation chronic orofacial pain, it is essential: (1) to take a detailed and complete history; (2) to perform a detailed clinical examination of the orofacial region; (3) not to have prejudice that the patient necessarily has an organic condition provoking pain; (4) to have thorough insight into all possible causes of orofacial pain, i.e. organic, functional and psychogenic; and (5) to have no hesitation in referring a patient for further evaluation if there is no obvious cause of pain found instead of performing unnecessary (usually irreversible) dental procedures.

Having in mind the fact that chronic orofacial pain presents a diagnostic and therapeutic challenge to health practitioners, it should be dealt with by clinicians of different specialities, and it seems that the multidisciplinary concept in treating orofacial pain patients is the best approach. The basic rationale of setting up and organizing specialized pain clinics is the necessity of treating pain patients by several specialists at the same time and place. Only joint management of chronic orofacial pain patients provides an overall insight into pain cause itself and the necessary diagnostic and treatment procedures.
References