LITERATURE REVIEW

Pain in otorhinolaryngology

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ABSTRACT

Pain is an abnormal sensation, sometimes awkward, disturbing, which starts from a certain part of the body and is perceived by the brain. From a medical point of view, pain represents an unpleasant sensory and emotional experience, determined by actual or potential tissue damage. Considering all these characteristics, all medical or surgical specialties are involved in the knowledge of mechanisms and causes of pain, in its management and treatment. Pain must be perceived as peripheral or central. Peripheral pain originates in muscles, tendons or peripheral nerve endings. Central pain occurs as a result of spinal cord injuries or changes in the central nervous system. In specialized ambulatory practice, one can notice that about 50% of those presenting for consultation invoke pain as the primary reason. Pain in otorhinolaryngology is multifactorial, complex and frequent, its complexity deriving from the extremely rich innervation and vascularization of the cephalic extremity. In this article the authors present the clinical characteristics of pain syndromes in otorhinolaryngology and try to systemize their treatment methods.

KEYWORDS: pain syndrome, cephalalgia, neuralgias, Sluder syndrome, trigeminal neuralgia

INTRODUCTION

Pain is an abnormal sensation, sometimes awkward, disturbing, which starts from a certain part of the body and is perceived by the brain.

The word "pain" derives from the Latin word "dolor". Terms derived from "algos", which defines physical pain in Greek (neuralgia, hyperalgesia or analgesia), are also used.

The International Association for the Study of Pain (I.A.S.P.) defines pain as "an unpleasant feeling or sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage"[1].

Throughout the history, pain has been a cause for concern, interpretation or even isolation. In the Middle Ages, there were magical rituals that combined religion with medicine, thus trying to find a cure for pain. In the modern era, pain was scientifically approached based on scientific observations, experiments and evidence. Thus, the International Association for the Study of Pain (I.A.S.P.) was founded in 1974.

The pain phenomenon can be interpreted on three levels – semantic, medical and neurophysiological. Semantically and physiologically, one speaks of the antagonistic duality pain – pleasure. Disturbance in the well-being after the occurrence of pain has generated a whole philosophy and literary work from the ancients to Baudelaire and Graham Greene.

From a medical point of view, pain represents an unpleasant sensory and emotional experience, determined by actual or potential tissue damage. Considering all these characteristics, all medical or surgical specialties are involved in the knowledge of mechanisms and causes of pain, in its management and treatment.

PATHOPHYSIOLOGY OF PAIN

In the medical practice, pain must be perceived as peripheral or central. Peripheral pain originates in muscles, tendons or peripheral nerve endings. Central pain occurs as a result of spinal cord injuries or changes in the central nervous system; it may take the appearance of what we know as psychogenic pain[2].

The pain sensation has three main components: sensory-discriminative, emotional or cognitive-behavioural. The sensory-discriminative system processes the information about the intensity, duration, nature or location of pain. However, the cognitive compo-

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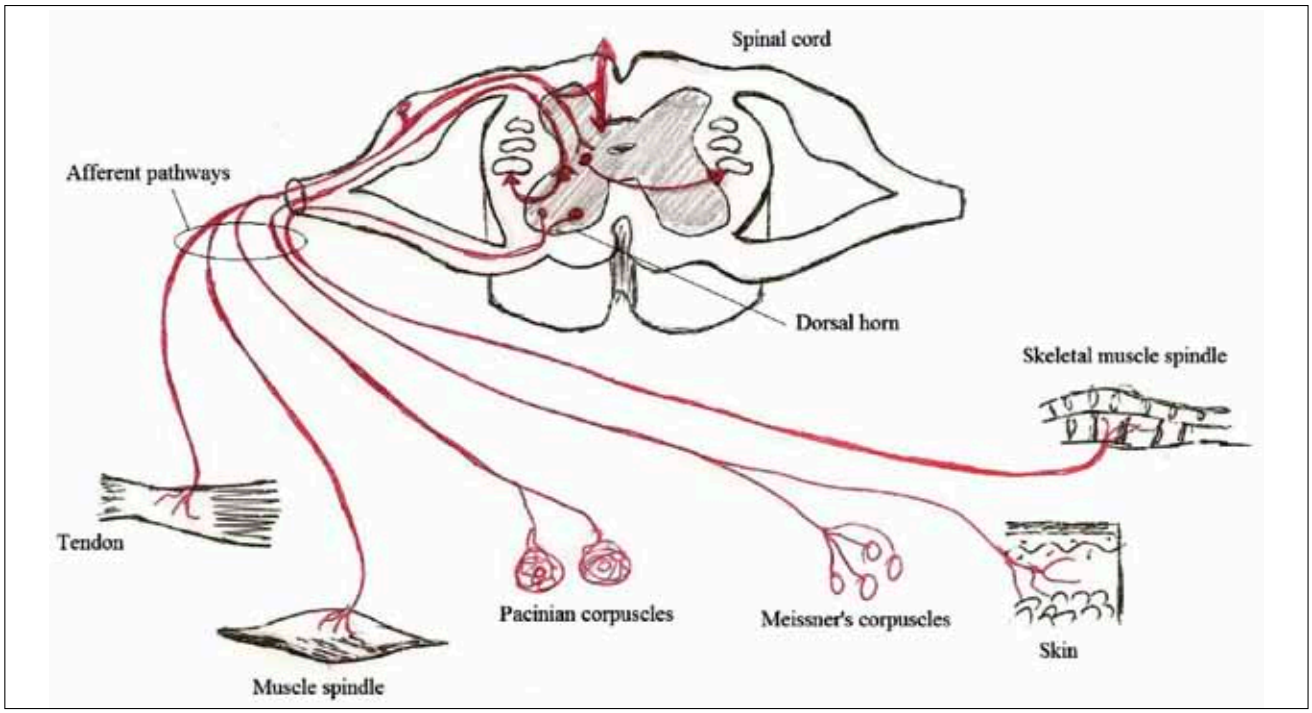


Figure 1 Pain afferent pathways

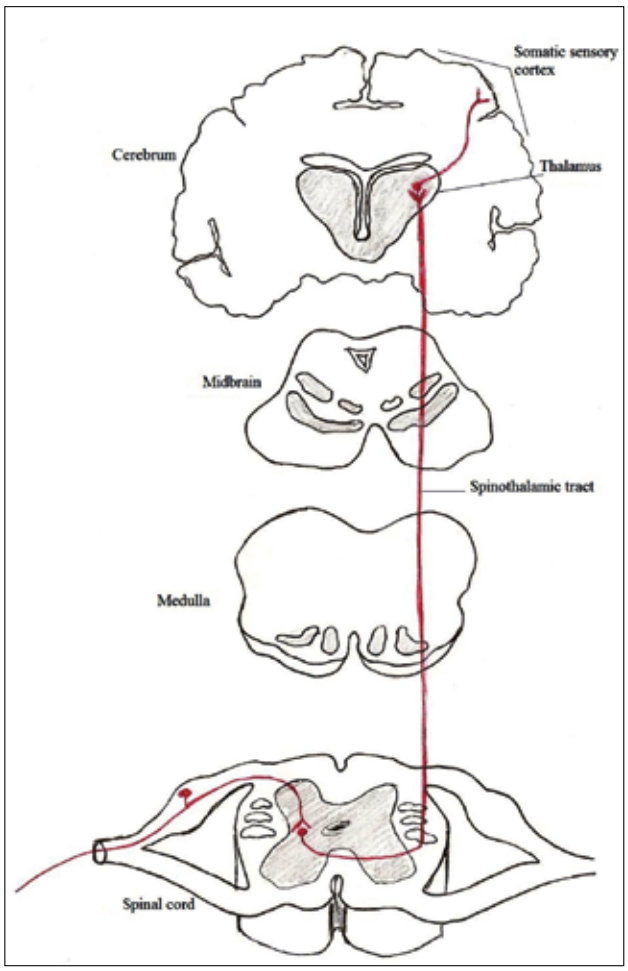


Figure 2 Spinothalamic tract

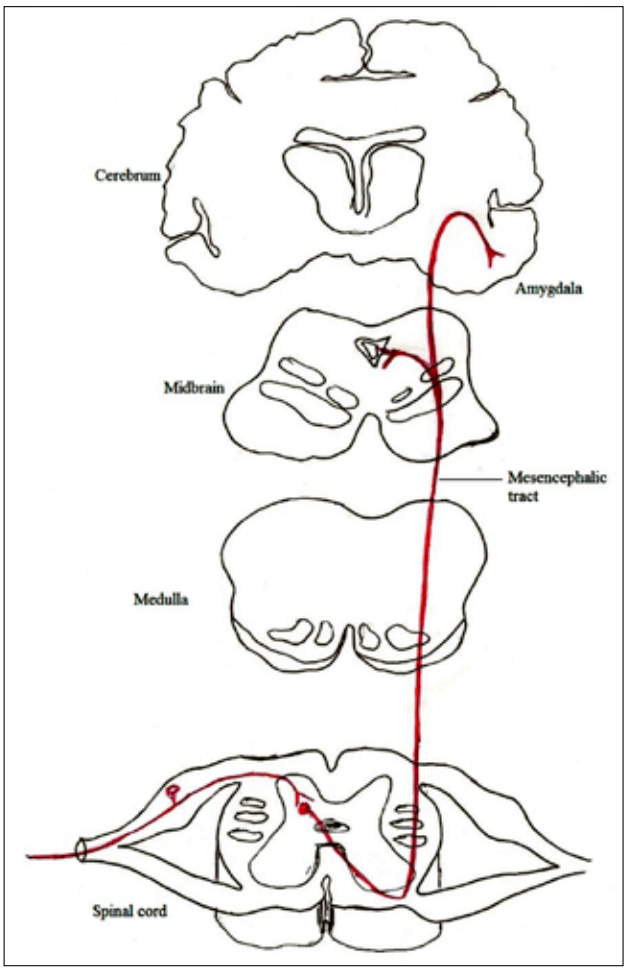


Figure 3 Mesencephalic tract

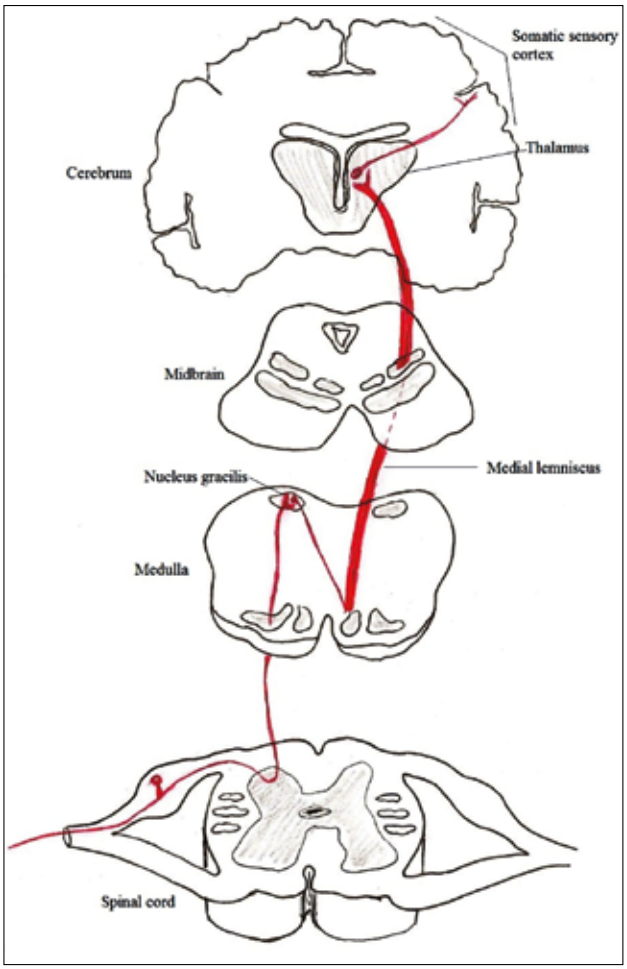


Figure 4 Medial lemniscus

nent assesses each person's learned behaviour when facing pain.

Pain is the phenomenon in the occurrence of which different mechanisms are involved. Experts in this field claim that, according to their clinical characteristics, pain syndromes can be divided in: nociceptive, neuropathic, idiopathic or psychogenic.

Nociceptive pain

The systems responsible for the sensation and perception of pain can be divided into three segments: afferent pathways, central nervous system and efferent pathways. The afferent pathways consist of nociceptors, afferent nerve fibers and the transmission network at the level of the spinal cord[3] (Figure 1).

There are two types of peripheral nociceptive fibers: A-delta for the well-localized pain that is rapidly transmitted and C-type fibers for transmitting dull, diffuse pain. Fitzgerald and Woolf consider that C-fibers are the first sensors to determine the release of serotonin, prostaglandins and leukotrienes, by generating an electrical impulse[4]. Nerve fibers respond to noxious stimuli in the skin, muscles, joints and viscera[5].

After depolarization, information is transmitted along the axonal fiber to the spinal cord. The first neuron of the afferent pathway is located in the posterior horn of the spinal cord. Neuromodulatory substances of these processes of neurotransmission are numerous, being represented by endorphins, neurokins, prostaglandins, GABA, neurotensin, etc.[6,7]. From this level, the impulse is transmitted to the brain, through connexions:

- The spinothalamic tract – the main pathway of transmission that reaches the ventral posterolateral nucleus of the thalamus (accurate information about the location of pain is transmitted) (Figure 2);
- The spinoreticular tract – has an ascending trajectory along the spinal cord up to the right and left intralaminar nuclei in the thalamus. From this level, the information is transmitted to the cingulate nucleus (it mediates emotion), the amygdala (it mediates memory and emotion) and the hypothalamus.
- The spinomesencephalic tract – reaches the midbrain, in the superior colliculus; from this level, the information is transmitted to the cerebral trunk and then to the amygdala, the hypothalamus, as well as the limbic system (Figure 3);
- The dorsal column of the medial lemniscus transmits pain information directly to the thalamus[8] (Figure 4).

At a central level, pain is projected at the subcortical and cortical levels. Subcortical structures are represented by the thalamus, the caudate nucleus of the hypothalamus, the amygdala, the hippocampus or the cerebellar vermis[9].

Until now, it hasn't been described an area of accurate projection of pain in the cortex. There are several areas in the cortex (based on Brodmann's classification) where the projection of pain syndromes would be made: the sensory and motor areas, the parietal cortex, the frontal, cingulate or occipital cortex[10,11].

Perception, description and location of pain occur at the level of the thalamus and sensory cortex. When it comes to diffuse pain, both the thalamus and the reticular formation participate in its perception. The latter, together with the limbic system, are involved in the emotional and affective control of pain.

Neurons in these areas of the cortex synapse with the neuronal cells of the efferent pathways, ending at the level of the posterior horn of the spinal cord.

Because of these connections, awareness of pain is always associated with an autonomic response.

As in any system of nerve transmission, on the transmission route of the painful stimulus, there are mechanisms that facilitate or inhibit pain perception, phenomena underlying the analgesia. This is the role of the reticular formation, which is under the influence of cortico-reticular impulses. From the reticular for-

mation leave fibers, which later make connections between the spinal cord and the pontine dorsolateral tegmentum or the hypothalamus.

The descending transmission of nociception has a noradrenergic and serotonin mechanism. Endorphins (enkefalins) are peptide mediators of pain, being components of the endogenous opioid family. Serotonin, a neuramin derived from tryptophan, is present both in the central nervous system and in the peripheral nervous system. Its synthesis is made in the neurons from the bulbopontine area and plays the role of a mediator; its involvement in the nociceptive process is not univocal. It may have an antinociceptive or pronociceptive action, depending on the receptors involved.

Neuropathic pain

Neuropathic pain is the syndrome produced by damage to or dysfunction of the peripheral or central nervous system. The nervous control mechanism is altered, and the nerve lesion constantly produces nociceptive stimuli[12]. Pain that occurs can be spontaneous or caused by contact (e.g. swallowing), with flashing, burning or distension manifestations, following the topography of a nerve trajectory. This type of pain may be accompanied by hyperalgesia, allodynia (pain caused by a painless stimulus, such as exposure to light) or hypoesthesia.

Neuropathic pain may take various clinical aspects[13]:

- The peripheral component is predominant (e.g. radiculopathy, polyneuropathy)[13,14];
- Changes in the spinal cord or the brain (e.g. spinal cord injuries, cerebrovascular accidents, etc.).

Changes at the peripheral level can be complex and may be due to demyelination of nerve fibers or reverse transport of anterograde and retrograde mediators.

Nevertheless, there is no evidence or information to explain neuropathic syndromes of central origin, despite brain neuroplasticity (e.g. phantom pain)[15].

Idiopathic and psychogenic pain

There is a close relationship between the psyche and pain perception; patients may present mood changes (depression, anxiety), lack of cooperation, etc.[16]. In these cases, symptomatology is polymorphic, cenestopathic, the balance of investigations being normal. Modern psychiatry defines this category as "somatomorphic disorders".

CLINICAL INTERPRETATION OF PAIN

A proper treatment of the pain syndrome involves a complete and correct diagnosis.

A fair assessment of a pain syndrome must follow several steps[17-19]:

Table 1
Characteristics of pain[19]

Characteristic of pain	Description
Temporal framing	Acute or chronic Persistent or recurrent Moment of occurrence
Intensity	Pain scale score Time of day when the intensity increases or decreases
Topography	Localized or diffuse Superficial or deep nature of pain Radiating area
Quality of pain	Stinging, burning, etc.
Exacerbating/relieving factors	

1. Complete history of the patient, by finding the pain history;
2. Clinical examination, including a neurological examination;
3. Previous diagnoses and treatments;
4. How the patient perceives pain, the impact it has on his social and family life, on his mental status or sleeping.

Pain is entirely a subjective symptom, being perceived differently by each and every person. The description of this syndrome should include information about the nature, intensity, location, duration of pain, or if there are factors that may enhance or diminish it (Table 1).

From a clinical point of view, pain can be acute (symptomatic pain) or chronic (pain as a disease). Acute pain has a sudden, recent onset, with variable intensity and does not stop for more than a few hours or days. It may be accompanied by anxiety, tachycardia, hypertensive spikes, mydriasis or profuse sweating.

Pain is considered chronic when it lasts more than 3-6 months or meets one of the criteria:

- It lasts more than a month after healing from the disease;
- It is associated with some chronic diseases;
- It has periods of recurrence at short intervals.

Chronic pain intensity is variable and may be accompanied by irritability, depression and less frequently by anorexia, insomnia or weight loss. Chronic pain can be classified into malignant pain, produced by neoplasias, or non-malignant (neurological, muscular, skeletal, cephalalgia, vascular or psychogenic pain).

Regardless of the acute or chronic type of the pain syndrome, quantification of its intensity should be part of the diagnostic protocol. There are many scales that can be used to measure pain, being indicated to use any of them (e.g. VAS, The McGill Pain Questionnaire, The Brief Pain Inventory, Neuropathic Pain Scale)[20-23].

PAIN IN OTORHINOLARYNGOLOGY

In specialized ambulatory practice, one can notice that about 50% of those presenting for consultation invoke pain as the primary reason. Pain in otorhinolaryngology is multifactorial, complex and frequent, its complexity deriving from the extremely rich innervation and vascularization of the cephalic extremity.

Acute pain syndrome

Pain that occurs in case of acute illness is closely related to the triggering disorder and disappears with its treatment. We can talk about various ear (Table 2), nose (Table 3), oral cavity (Table 4), cervical region (Table 5), pharyngeal region (pharyngoamygdalitis, epiglottitis, Ludwig's angina, retropharyngeal abscess, laryngotracheal trauma) disorders or we can speak of acute cephalalgia, which can be posttraumatic, hemorrhagic, determined by intracranial infections, cerebrovascular diseases, brain tumors or encephalitis.

Chronic pain syndromes

Cephalalgia is a very common syndrome, with various clinical manifestations, which determines frequent general practitioner or specialist consultations. From a clinical point of view, chronic cephalalgia may be primary or secondary. Secondary cephalalgias occur as a result of comorbidities (e.g. brain tumor, craniocerebral trauma, etc.).

International Headache Society (IHS) divides headaches, cephalalgia, in several groups[25,26]:

I. Primary cephalalgia:
   1. Migraine
   2. Tension cephalalgia
   3. Cluster cephalalgia or other trigeminal autonomic cephalalgias

II. Secondary cephalalgia:
   1. Cephalalgia associated with head and neck traumas
   2. Cephalalgia associated with vascular diseases
   3. Cephalalgia associated with some non-vascular intracranial disorders
   4. Toxic, withdrawal cephalalgia
   5. Cephalalgia associated with homeostasis disorders
   6. Cephalalgia associated with disorders of the neck, eyes, ears, nose, teeth, mouth or other cranial structures
   7. Cephalalgia associated with non-cephalic infections
   8. Cephalalgia associated with psychiatric disorders

III. Cranial neuralgias, deafferentation pain

Moreover, IHS divides facial pain in: neurological, non-neurological or of psychological origin[27].

Migraine is characterized by periodic attacks of cephalalgia accompanied by vegetative phenomena[28]. The underlying mechanism of migraine is a neuronal one, found in the hypothalamus, where appears a painful vasodilatation through release of neuropeptides. This vascular response, translated by the release of some vasoactive algogene substances (histamine, bradykinin, prostaglandins, substance P), occurs due to some triggering factors like stress, hormonal status, genetic factors, etc. Extension of this vasodilator process to bulbar structures triggers the occurrence of accompanying vegetative phenomena – nausea, vomiting and photophobia.

Approximately 12 to 17% of adults suffer from migraines[28], the most affected being women and may involve all social categories.

From a clinical point of view, there are several types of migraine: common migraine, without aura; migraine with aura; child migraine; rare migraines – familial hemiplegic migraine, ophthalmoplegic migraine, retinal migraine, malaise migraine and migrainous infarction.

Table 2
Acute auricular pain

Auricle	External auditory canal	Middle ear	Irradiated pain
Cellulitis	Acute external otitis (Figure 7)[24]	Acute otitis media (Figure 8)[24]	Dental disorders
Traumas	Furunculosis	Acute suppurative otitis (Figure 9)[24]	Angina
Sebaceous cyst (Figure 5)[24]	Malignant external otitis	Barotraumas	Lingual amygdalitis
Perichondritis		Acute mastoiditis	Peritonsillar abscess
Frostbite			ATM
Burns			Thyroiditis
Shingles			Parotitis
Herpes simplex (Figure 6)[24]			

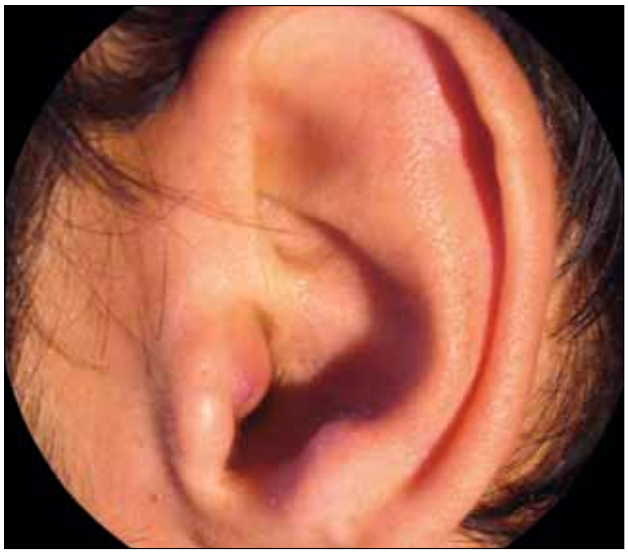


Figure 5 Tragus sebaceous cyst

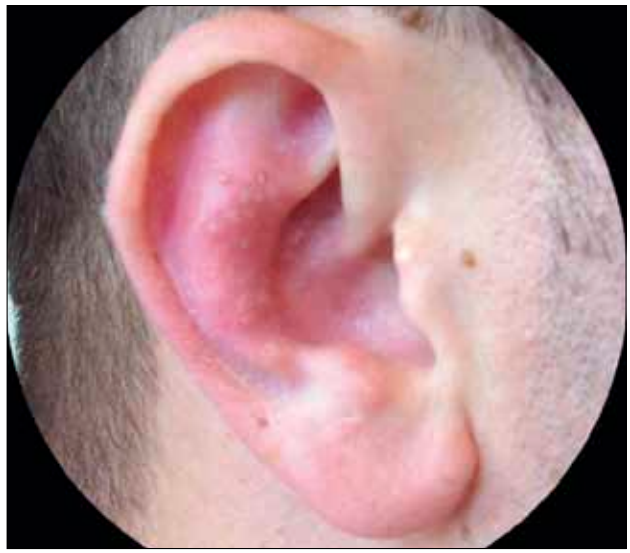


Figure 6 Herpes simplex – acute erythematous plaque with well defined edges located in the right ear pavilion, with pustules and vesicles on the surface

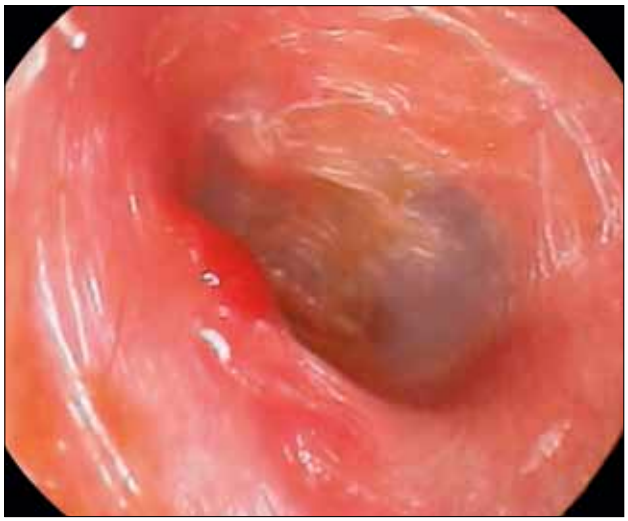


Figure 7 Acute diffuse external otitis media (left ear)

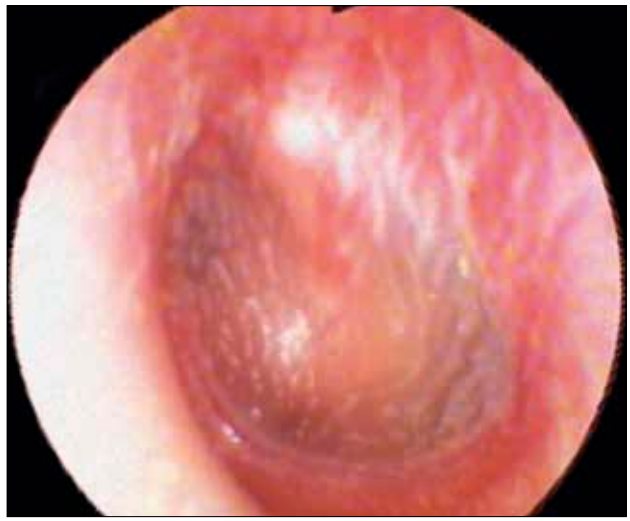
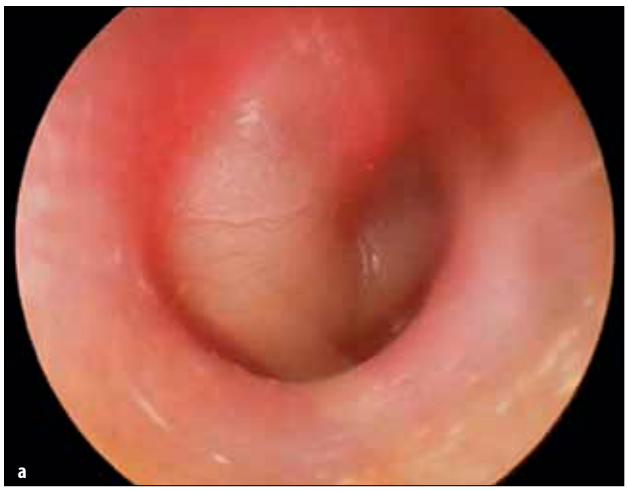


Figure 8 Acute otitis media (left ear)



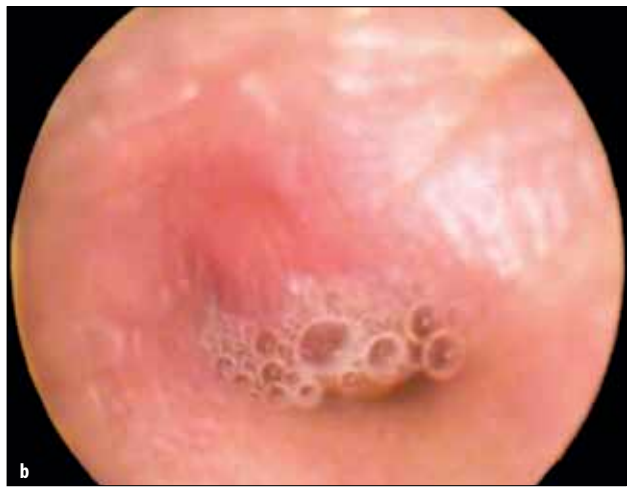


Figure 9 Acute suppurative otitis media (left ear) – **a.** congestion and bulging tympanic membrane; **b.** spontaneous perforation

Table 3
Acute nasal pain

Nasal pyramid	Nasal fossae
Folliculitis	Rhinopharyngitis
Furunculosis	Acute rhinosinusitis (Figure 10)[24]
Nostril fissures and ulcers	Intranasal foreign bodies (Figure 11)[24]
Herpes	
Traumas (septal hematoma, nasal pyramid fractures, septal fractures)	
Relapsing polychondritis	

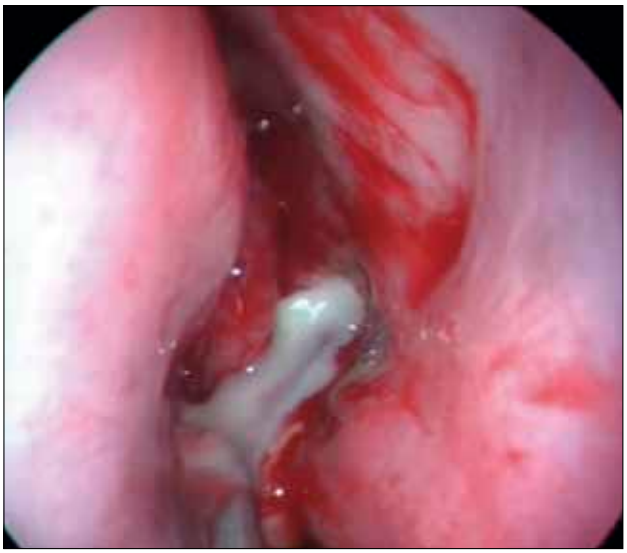


Figure 10 Acute rhinosinusitis

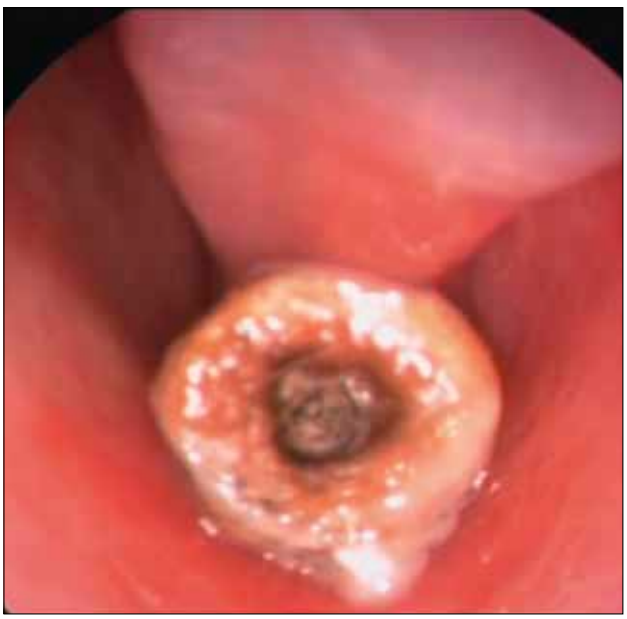


Figure 11 Intranasal foreign body – left nostril – nasal endoscopic examination

The migraine without aura is characterized by seizures lasting between 4 and 72 hours, initially with unilateral location, in the temporal region, and it may subsequently become diffuse. Seizures occur most often in the morning or at night and may be accompanied by nausea, vomiting, phono- and photophobia. It is a throbbing pain, synchronous with the pulse, with moderate to severe intensity, which may be enhanced by physical activity. Severe attacks may force the patient to isolate in a quiet and dark environment.

The migraine with aura is a less common disorder, being characterized by reversible neurological phenomena, aura, which can predict pain, accompany it or occur in its absence[29]. This syndrome can be manifested in various forms and can last between 5 and 20 minutes:

- Visual symptoms – are the most frequent[30]; the patient could report scotomas, photosphenes, reduction of the visual spectrum; visual distortions, such as micropsia or macropsia, are more common in children[31];
- Sensitive manifestations – paresthesias.

The familial hemiplegic migraine is a particular form of migraine with aura, an autosomal dominant disease characterized by triad cephalalgia of migrainous type, hemiplegic deficit, lasting several hours and having a familial nature[31,32].

Retinal migraine is characterized by migraine crises with scotomas or monocular cecity, lasting several hours. Differential diagnosis must be made with the *ophthalmoplegic migraine*, in whose context appears paresis or palsy of nerves II, IV or VI, being much more frequent than the first[33].

Association of one or more symptoms of the aura type with a demonstrated ischemic brain injury is known as migrainous infarction[27].

Child migraine is taken into account when the specialist is faced with:

1. At least 5 seizures lasting between 1 and 48 hours;
2. Cephalalgia is throbbing, of moderate or severe intensity;
3. During the crisis may appear: vertiginous episodes, nystagmus, nausea, vomiting, phono- and photophobia;
4. Normal neurological, audiometric and vestibular examinations between seizures;
5. EEG within normal limits[27].

Tension cephalalgia is the most frequent form of primary headache, with a prevalence of 31 – 74%, but without having a significant impact on patient's quality of life compared to migraine[34-36]. According to IHS, tension cephalalgia may be episodic (less than 15 episodes per month) or chronic (more than 15 episodes per month)[25].

Pain that characterizes tension cephalalgia is localized bilaterally, of moderate or severe intensity and,

Table 4
Acute pain in the oral cavity

Oral cavity	Tongue	Lips
Dental pain	Glossitis	Cheilitis
Traumas	Glossodynia	Impetigo
Burns	Burns	Steven-Johnson Syndrome
Stomatitis	Contact allergy	Labial erosion
Candidosis	Bite	
Leukoplakias	Candidosis (Figure 12)[24]	
Kaposi's disease		

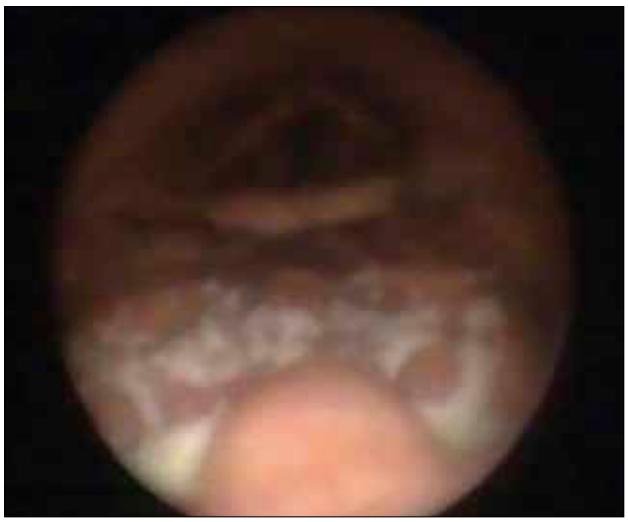


Figure 12 Lingual candidosis – white fungal deposits on the lingual tonsil - laryngofibroscopic examination

unlike the migraine, is not aggravated by physical activity. In case of episodic headache, pain intensity increases by palpation of the skull, electromyography of cranial muscles being pathological but not pathognomonic[37]. The differential diagnosis of this pathology should be made with craniomandibular disorders, psychosocial stress, anxiety, depression, muscle contractures or painkiller overdose.

Cluster headache is a **trigeminal autonomic cephalalgia**, characterized by severe, unilateral pain, with orbital, supraorbital or temporal localization, being accompanied by ipsilateral vegetative phenomena (nasal congestion, miosis with or without palpebral ptosis, etc.). It occurs much more frequently in men, the average age of onset being 27 – 31 years[38,39]. Pain reaches its intensity peak within maximum 10 – 15 minutes, and can last up to 60 minutes. Between the seizures, a background pain similar to migraine may persist.

Only 10 – 20% of patients with cluster headache have a chronic form, without remission for a year, or with remission for less than a month[26].

In patients with *paroxysmal hemicrania*, pain has a small duration (2 – 45 minutes), unilateral localization, accompanied by ipsilateral vegetative phenomena and a very fast response to Indomethacin[40,41].

Post-traumatic headache mostly occurs as a result of cranio-cervical trauma. If the headache occurs more than three months after the accident, it

Table 5
Acute pain in the cervical region

Anterior region	Posterior region	Lateral region
Thyroid gland	Meningitis and meningism	Parotitis
Irradiated pain – cardiac, vascular, mediastinal, pulmonary	Vertebral disc disorders	Cervical lymphadenitis
Abscess of the anterior cervical triangle	Cervical osteoarthritis	Latero-pharyngeal suppurations
Hyoid bone syndrome	Cervical osteomyelitis	SCM disorders
Traumas	Atlanto-axial subluxation	Trapezius muscle myofasciitis
Esophagitis	Cervical myositis	Infected cysts
Esophageal cancer	Subarachnoid hemorrhage	Irradiated pain – AMI, acute pericarditis, dissecting aneurysm of the aorta

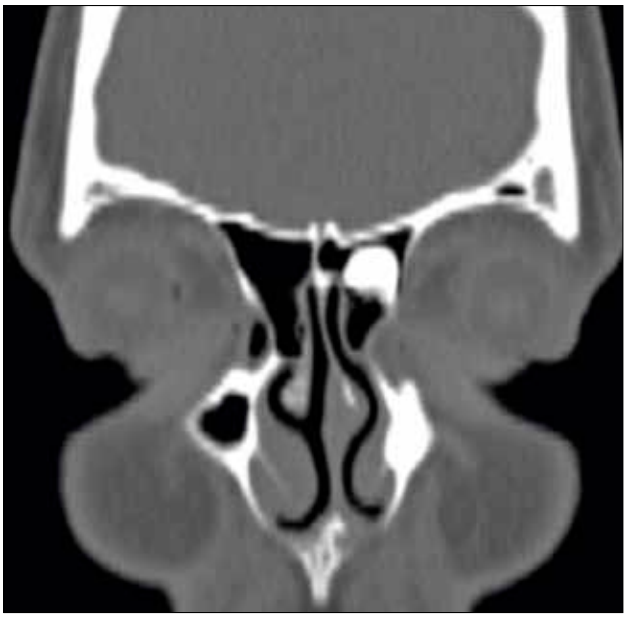


Figure 13 Left ethmoidal osteoma

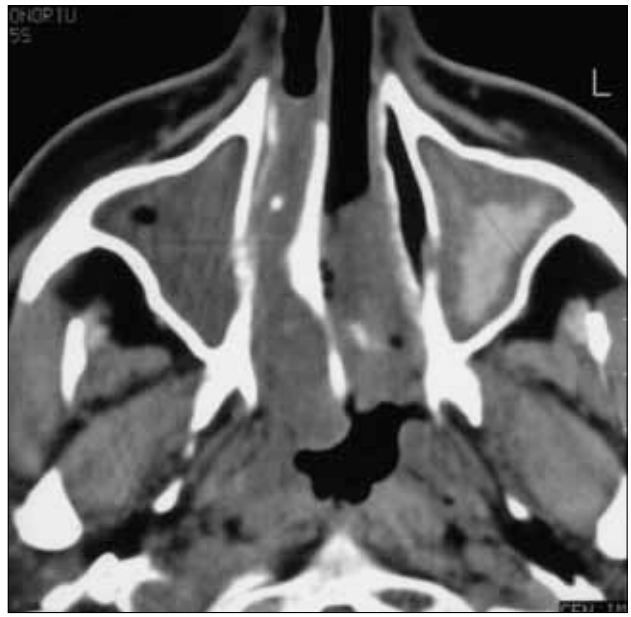


Figure 14 Bilateral chronic rhinosinusitis

is an acute posttraumatic headache[42]. The mechanism of occurrence may be by whiplash, with acceleration of the cervical rachis, or by compression or elongation of the algogene structures of the cervical rachis.

Cephalalgia associated with vascular diseases includes painful syndromes that are closely related to:

1. Stroke[43]
2. Vascular malformations[44]
3. Arteritis (ex. Horton's disease – temporal arteritis with giant cells, temporal cephalalgia, uni- or bilateral, irradiating in the occipital region, accompanied by tegument swelling in the temporal region, and highlighting of the temporal artery trajectory and painful swelling)[45]
4. Artery dissections[46]
5. Cerebral venous thrombosis[47].

Medication overuse headache affects about 1% of the population, daily consumption of drugs representing a major risk of developing a chronic headache. Acute exposure to ergotamine, alcohol, opioids and combinations of analgesics, triptan or caffeine may aggravate or foster a headache[48].

Chronic cephalalgia due to rhinosinusitis must be distinguished from neuralgias, migraine syndromes or neurological or ophthalmologic disorders. In rhinosinusal disorders, the classic symptomatic triad consists of nasal obstruction, rhinorrhea and headache. The latter may be localized either at the top of the head, suggesting damage to the sphenoidal sinus or the posterior ethmoid cells, or in the region of the glabella or of the eyeballs, suggesting damage to the anterior ethmoid cells or the frontal sinus. Cephalalgia can be associated with a sensation of facial pressure or a full head feeling, enhanced with bending the head[49] (Figure 13, Figure 14).

Neuralgias are painful syndromes that occur after an irritation of the sensitive fibers on the trajectory of a particular nerve[49]. Clinically, neuralgias may be essential, characterized by paroxysmal crises arising in the absence of objective, or symptomatic, with a permanent painful background.

Trigeminal neuralgia (Figure 15) may be symptomatic or essential. Symptomatic neuralgia can be determined by:

- dental disorders – pain occurs on the path of the maxillary and mandibular nerves[51];
- Maxillary neuralgia – tumors or proliferative processes in the maxillary bone;
- Raeder's syndrome – the cervical apex syndrome – hypoesthesia in the territory of the ophthalmic nerve (branch of the trigeminal nerve); may be determined by tumors, inflammatory causes, aneurysms, etc.;
- Tumors of the pontocerebellar angle[52];
- Vascular compressions – arterial or venous[53].

Essential trigeminal neuralgia or Fothergill's disease[54] is a pathological entity referring to trigeminal algiae that meet six criteria: they are paroxysmal, provocable, unilateral (they occur bilaterally extremely rarely), they are strictly localized in the territory of the trigeminal nerve, they aren't accompanied by signs of organic distress and no residual pain occurs between crises, unlike the symptomatic form, where paresthesias persist. It is a pathology that manifests most frequently in the second half of life, generally after 50 years, and it mostly affects females (women: men = 2:1)[55].

The pathogenic mechanism seems to act both intra-

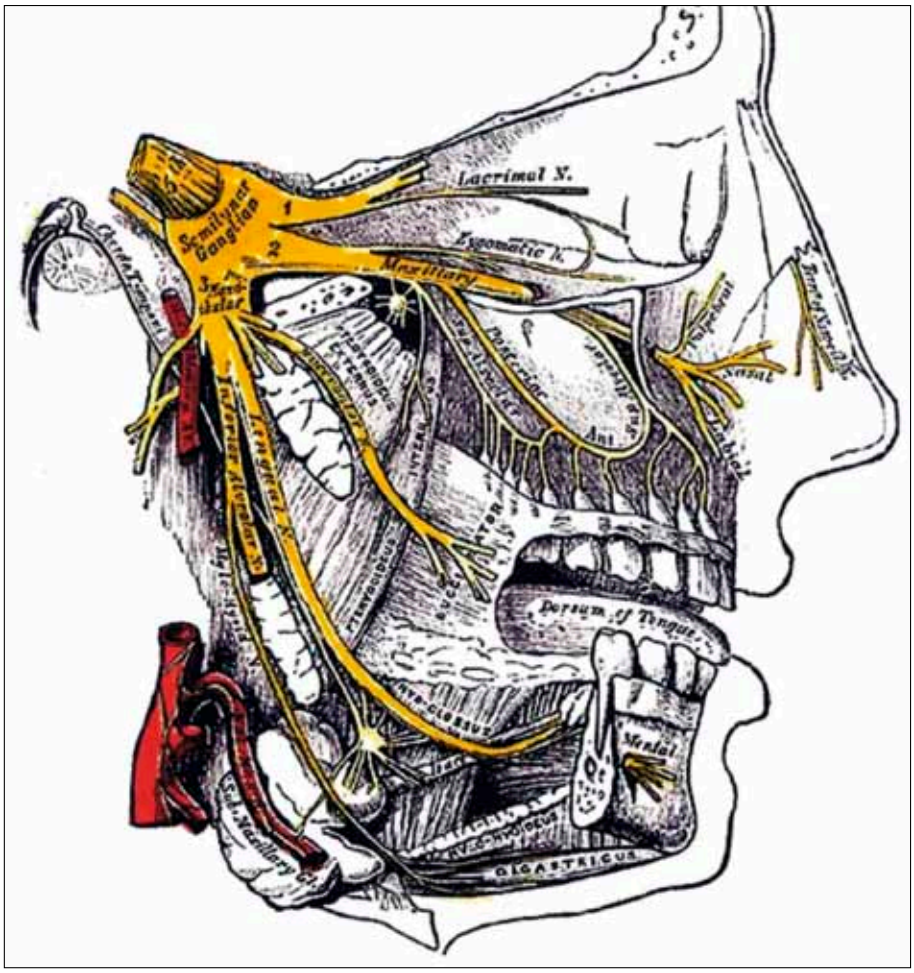


Figure 15 Trigeminal nerve distribution (reproduction after Gray's Anatomy of the Human Body[50])

and extraneuraxially and is represented by a transaxonal short-circuit of the action current along the nerve fibre[56-58]. A curious fact, but still unexplained, is predisposition for the second branch of the trigeminal nerve (the maxillary branch) and for the right side of the face, on the second place being the third branch (the mandibular branch) and only then the first branch (the ophthalmic branch).

From a clinical point of view, pain is paroxysmal, of a flashing type, lasting several seconds, and it could be described as knife stabs or electrical shocks. The number of crises varies, since they may occur from one to dozens per day. Paroxysms do not succeed with certain regularity; they can occur during the day or at night. The violence of the crises frequently causes a rictus on the face, or a palpebral or labial spasm ipsilateral to pain, reason for which the trigeminal neuralgia has also been called painful facial twitching.

Pain may be caused by tactile, vibratory (a stream of air) stimuli or even by chewing or speech movements. Trigger areas, called trigger zones, may be single or multiple – facial skin, labial mucosa, gingival or buccal mucosa, tongue movements, swallowing, hiccup, etc. The presence of these trigger zones automatically implies a characteristic behaviour, attitude from the patient, who tends to avoid any stimulus suspected of being a triggering factor.

Essential neuralgia pain is unilateral and never exceeds the midline of the face. It may be accompanied by vegetative phenomena such as tearing, salivation, nasal hypersecretion or skin congestion.

An important characteristic is the absence of residual pain or paresthesias in the interparoxysmal periods.

Diagnosis is based on characteristic symptoms and lack of objective sensitivity disorders or impaired reflexes in the trigeminal territory. A correct and complete investigation involves ENT, stomatologic, neurological examinations, cranial radiographies, CT or MRI scans.

Glossopharyngeal nerve neuralgia (Figure 16) is characterized by painful crises with characteristics similar to those from the "painful facial twitching", but with different topography and triggers. Pain is located at the base of the tongue, in the region of the palatine tonsils, and may have otic irradiation (depending on

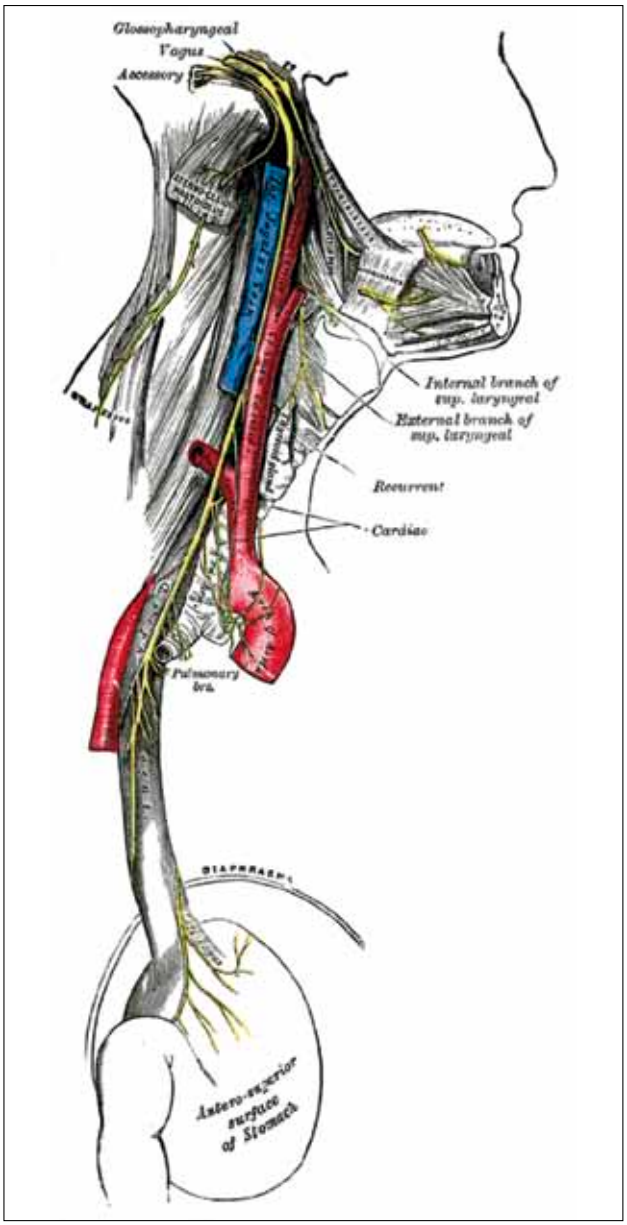


Figure 16 Glossopharingeal nerve distribution (reproduction after Gray's Anatomy of the Human Body[50])

branches or nerve trunk damage); triggering stimuli can be represented by swallowing, mastication, cough, smoking or phonation[59]. It is a disorder common in people over 40 years, with an incidence of about 0.7/100.000 people /year[60].

The etiology of the glossopharyngeal neuralgia is unknown, the most supported theory being intracranial vascular compression of the glossopharyngeal nerve. Idiopathically, it may appear after a surgical intervention (e.g. tonsillectomy).

Due to the vegetative branches of the nerve, during painful crises, changes in heart rate and blood pressure can occur; patients may have heart palpitations, extrasystoles or bradycardia or even loss of consciousness.

Sluder's syndrome[61] or sphenopalatine neuralgia is a form of essential neuralgia characterized by pain in the peri- and suborbital territory, the nose wing, with radiation to the upper jaw or the zygomatic region. The painful syndrome may be accompanied by nasal congestion with rhinorrhea, tearing, sneezing or eyelid edema.

Charlin's syndrome or anterior ethmoid nerve neuralgia is characterized by pain in the inner corner of the eye, accompanied by rhinorrhea, epiphora and nasal congestion[62].

Supraorbital nerve neuralgia is an algic syndrome appeared in the innervation area of the supraorbital nerve, branch of the frontal nerve. It is characterized by pain in the area of the eyebrow arch and of the forehead; it can be triggered by touching or contact with painful stimuli (à frigore, secondary to certain surgical interventions or post-zosterian).

Another type of neuralgia is *Eagle syndrome* or *stilalgia* caused by the abnormal elongation of the styloid process or calcification of the stylohioid ligament. Clinically, there is a direct compression on the nerves V, VII, IX, X, on the carotid; this results in odynophagia, pain located in the lower pole of the palatine amygdala, with radiation to the ear or pharyngeal foreign body sensation. Differential diagnosis must be made with sphenopalatine neuralgia, glossopharyngeal or trigeminal neuralgia, temporomandibular arthritis. Conventional radiography can confirm the diagnosis.

Superior laryngeal nerve neuralgia is manifested by pain in the cervical region, in the submandibular area, with auricular irradiation. The painful syndrome is exacerbated by swallowing, turning of the head, upper airway infections, etc.

TREATMENT

Treatment of pain syndromes is made according to disease stage, previous treatments, to the pain topography and location of the trigger area, as well as depending on the physical and mental condition of the patient. The first step is pharmacological treatment, the surgical one being reserved for cases refractory to the first therapeutic strategy.

There are several principles that underlie pain treatment:

1. Any pain can be reduced by etiological treatment.
2. Pain must be labelled "organic pain" only after proving this.
3. Pain is a simple symptom – one must always find the cause.
4. Treatment does not address the symptom, but the patient.
5. Blind treatment of pain is wrong before deci-

phering its mechanism.
6. Analgesics are used in nociceptive pain, complying with the three levels recommended by the World Health Organization.
7. Prescription of analgesics is not made depending on disease prognosis, but according to the intensity of pain.

Pharmacological treatment

According to numerous studies in the literature, first-line drug treatment of pain syndromes is represented by antiepileptics – carbamazepine and oxcarbazepine[63,64]. Although the first has proved more effective, oxcarbazepine has a much higher safety profile[65,66]. To these can be added diphenylhydantoin, clonazepam or neurontin.

Garcia-Callejo reported in a study the increased efficacy of treatment for 2 to 16 months with gabapentin as monotherapy or in association with carbamazepine, in seven out of nine patients with glossopharyngeal neuralgia rebellious to treatment[67].

Muscle relaxants are part of the second-line pain therapy. Baclofen, an agonist of GABAB receptors, has proved its efficacy in 70% of the patients included in a double-blind study, who were administered 10-60mg/day[68,69]. Instead, lamotrigine acts on sodium channels, by stabilizing the cell membrane and blocking the excitatory effect of neurotransmitters[69].

Phenothiazide neuroleptics, such as chlorpromazine or levomepromazine, have proved worth taking into consideration for painful syndromes.

Botulinum neurotoxin type A is increasingly supported as a method for treating pain, by its capacity to release neuropeptides like substance P, glutamate, thus inhibiting central and, probably, peripheral sensitization[69-71].

Non-steroidal anti-inflammatory drugs, acetaminophen or ketamine, can also be used successfully. They act by blocking cyclooxygenase and inhibit thereby prostaglandin synthesis.

Over time, a scale of the pharmacologic treatment of pain has been developed:

- first level – mild persistent pain (intensity score 1-4) – non-opioid analgesics, sedatives;
- second level – average persistent pain (intensity score 5-7) – minor opioid analgesics (codeine, tramadol, dihydrocodeine) associated or not with non-opioid analgesics and / or sedatives;
- third level – severe persistent pain (intensity score 8-10) – major opioid analgesics (morphine, hydromorphone, fentanyl) associated or not with non-opioid, co-analgesic or sedative ones[72].

In migraines, prompt treatment is essential, treatment being initiated with paracetamol or salicylic acid and metoclopramide. In case of failure, a more aggressive treatment is needed; it is represented by triptans (Imigran), selective serotonin receptor agonists, or ergotamine tartrate with vasoconstrictor action. Background treatment must be done for at least 6 months and addresses both the neurovascular and the metabolic systems (dihydroergotamine, antiserotonin, beta blockers).

Surgical treatment

Surgical treatment is chosen in cases refractory to medical treatment. The type of intervention must be chosen depending on the clinical aspects of the painful syndrome and not necessarily on imaging[73].

Chemical blockage of peripheral trigeminal branches with alcohol, anesthetic or opioid could be the first step in the surgical treatment of cranial painful syndromes, especially neuralgias. Administration of lidocaine 8% by intranasal injection in the sphenopalatine ganglion resulted in partial reduction of pain[74]. Opioid analgesia of the superior cervical ganglion may have a beneficial effect on cranial and facial neuralgia[75].

Another strategy would be cutting the nerve root. This technique was performed for the first time in 1920 by Sicard and Robin in glossopharyngeal neuralgia[76,77]. They sectioned the glossopharyngeal nerve root and the pharyngeal branches of the vagus nerve. In trigeminal neuralgia, partial resection of the nerve root may be taken into consideration.

Alcoholization, electrocoagulation or thermocoagulation of the Gasserian ganglion require local anesthesia, have a very low mortality rate[63,64] and may represent the first-line therapy in trigeminal neuralgia.

Many new methods, such as magnetic and electrical stimulation, radiofrequency stimulation, are used when pain is resistant to other treatments[78]. The electro-magnetic stimulation method was used for the first time in 1918, and in 1965 the first stimulation of the infraorbital nerve was performed[79]. The effect is represented by blocking of sodium channels, by modifying the release of substance P, GABA, adrenaline and serotonin[79,80].

Alternative treatment

MEOPA – an equimolar mix of oxygen and nitrous oxide – is a gas composed of 50% oxygen and 50% nitrous oxide. This mixture causes a state of euphoria and drowsiness with amnesia for pain.

Behavioral therapy, acupuncture or heat therapy can be used as methods of conservative treatment of painful syndromes[81].

CONCLUSIONS

Pain syndromes in the head and neck areas may accompany an acute disease, but, most commonly, they are chronic. Clinical and paraclinical diagnosis, as well as the accurate differential diagnosis may prevent conversion of pain in an incapacitating syndrome.

Proper treatment, carried out according to the level

of pain intensity, represents the first strategy in approaching a cranio-cervico-facial painful syndrome.

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