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THE APPLICATION OF RADIOFREQUENCY WAVES IN THE MANAGEMENT OF ORAL MAXILLOFACIAL PAIN

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Abstract

Temporomandibular disorders (TMDs) represent a common and complex clinical condition, defined by the American Academy of Orofacial Pain as a group of muscular and neuromuscular disorders which affect the temporomandibular joint (TMJ) and those structures connected to it. These disorders involve orofacial pain, limitation of mandibular movements and the presence of joint sounds.

Epidemiological studies indicate a significant prevalence of TMDs in the European population, with relevant impact on the patients' quality of life. The diagnosis of TMDs relies on several tools, including the Diagnostic Criteria for Temporomandibular Disorders (DC/TMD), a standardized system based on a biopsychosocial model that allows an integrated assessment of the physical (Axis I) and psychosocial (Axis II) aspects of the disorder, providing detailed information on pain intensity, frequency and duration, as well as on its functional and emotional impact.

The nature of TMDs which are frequently chronic, makes their therapeutic process quite complex, this has stimulated research into alternative and non-invasive approaches, including the use of radiofrequency diathermy.

Diathermy employs high-frequency electromagnetic waves to generate endogenous tissue heating, promoting muscle relaxation, reduction of infection, and improvement of blood circulation, with potential benefits for pain relief and joint function. Compared with other therapeutic options, such as occlusal splints, botulinum toxin injections, or systemic pharmacotherapy, diathermy is generally well tolerated, non-invasive and associated with rapid symptomatic relief. However, the studies available are still limited and defined by considerable heterogeneity in treatment protocols and this makes comparisons between different studies quite difficult and hinders the identification of standardized parameters for performing and safe results.

Within this context, the present study aims to evaluate the efficiency of radiofrequency diathermic stimulation in handling the pain and muscle dysfunction associated with TMDs in patients without internal displacement of the joint disc. This will be achieved using the DC/TMD questionnaire as a standardized tool for outcome assessment. Specifically, the objective is to analyze the impact of the treatment on pain symptoms, mandibular function and patients' subjective perception, thereby contributing to filling existing gaps in the literature regarding standardized therapeutic protocols and the short-term duration of diathermy effects.

CHAPTER 1

INTRODUCTION

Orofacial pain represents one of the most significant pain conditions worldwide and constitutes a clinical issue which is troublesome for a great number of patients. It has been reported that orofacial pain affects approximately 25% of the general population¹, with an etiology that may be either odontogenic or non-odontogenic. This condition involves a wide range of clinical manifestations from intraoral pain of dental or periodontal origin to more complex conditions such as orofacial myofascial pain, temporomandibular joint (TMJ) pain, and orofacial pain due to traumas or diseases of the cranial nerves ².

The management of orofacial pain plays a central role in both medical and dental practice, as this symptomatology is closely related to the patient's quality of life. Specifically, pain associated with the inflammation of the TMJ has an estimate between 7% and 10% of the world population and has a significant negative impact on the quality of life in relation to oral health ³⁻⁵.

The term temporomandibular disorders (TMDs) includes a heterogeneous group of pathological conditions characterized by pain in both the masticatory muscles and temporomandibular joints; as well as headaches, alterations in mandibular movements and the presence of joint sounds during mouth opening and closing ⁶.

The etiopathogenesis of TMDs depends on various elements and includes traumatic, systemic, iatrogenic, occlusal, and psychosocial factors. In most cases, symptoms are related to increased tension of the masticatory muscles, frequently exacerbated by the presence of parafunctional habits. The treatment of TMDs is therefore complex and requires a multidisciplinary approach, which may include specific therapeutic exercises for muscle rebalancing, the use of occlusal devices, manual therapy and

pharmacological support. The main goals of treatment are pain control, prevention of disease progression, and restoration of compromised mandibular function⁷⁻¹¹.

Among conventional therapeutic options for painful intracapsular TMJ disorders there are intra-articular injections of anti-inflammatory drugs which represent a strategy used to improve joint function and reduce pain. Pharmacological therapy is primarily aimed at controlling inflammation and pain at both joint and muscular levels and includes the use of muscle relaxants, non-steroidal anti-inflammatory drugs, analgesics, tricyclic antidepressants, benzodiazepines, and corticosteroids¹²⁻¹⁶.

In recent years, radiofrequency has found widespread application in the medical field for pain management, including in the head and neck region^{17,18}. Medical radiofrequency is based on the use of electromagnetic waves capable of inducing a controlled increase in tissue temperature; the thermal energy associated with these procedures can induce ablation or biological stimulation phenomena, promoting the production of new fibroblasts, functional restoration of muscle fibers, and activation of the microcirculation. These effects contribute to modulation of pain transmission at the neural level and to vasodilation, with a consequent increase in oxygen delivery to tissues¹⁹.

Radio waves are electromagnetic waves characterized by frequencies ranging from 3 Hz to 3 THz and are used in physiotherapy, particularly for muscle relaxation, within the range of 3 to 6 MHz. The rehabilitative effects of these waves are based on the principle of diathermy, achieved through the application of rapid alternating current at high voltage. The conversion of electromagnetic energy into heat allows muscle tissues to be heated to temperatures close to 45 °C, promoting increased local blood flow, stimulation of regenerative processes, reduction of muscle tension, and the achievement of an analgesic effect²⁰⁻²⁵.

The clinical use of diathermy has been relatively widespread for about two decades, although its clinical efficiency has only been systematically evaluated more recently. Most available studies report promising results in terms of pain reduction and

functional improvement in various musculoskeletal conditions. Diathermy devices allow the delivery of two distinct treatment modalities, capacitive and resistive, generally administered via medical-grade stainless steel electrodes. These modalities induce different tissue responses depending on the impedance of the target tissue²⁶⁻²⁹.

In particular, the capacitive mode acts selectively on soft tissues with low impedance and high water content, such as adipose, muscular, cartilaginous, and lymphatic tissues. Whereas the resistive mode allows heat generation in deeper and more resistant layers characterized by lower water content, such as bone, tendons, and specific muscle groups. Devices based on capacitive–resistive electric transfer are able to stimulate tissue metabolism and lymphatic drainage, improve oxygenation and local metabolism, and are therefore indicated both in acute inflammatory processes and in subacute healing phases.

Furthermore, as reported in the literature, these devices promote increased hemodynamic flow and vasodilation, reduce tissue tension, and improve tissue compliance, making them particularly useful in chronic conditions as well as during tissue repair and regeneration phases. However, the use of diathermy presents specific contraindications, including pregnancy, the presence of metallic implants, neoplastic diseases, epilepsy, skin diseases, and open wounds, which must be carefully considered in clinical practice³⁰⁻³⁷.

CHAPTER 2

TEMPOROMANDIBULAR DISORDERS

Temporomandibular Disorders (TMDs) comprise a group of conditions involving the temporomandibular joints (TMJs), the masticatory muscles and the anatomical structures connected to them.

The three pathognomonic signs and/or symptoms of this disorder are pain in the mandibular or preauricular area, limitations or deviations in mouth opening, and mandibular clicking.³⁸⁻³⁹

Thirty-nine percent of the population presents at least one sign or symptom of temporomandibular disorder (TMD); among these, mandibular clicking is the most frequent manifestation, followed by mandibular pain.⁴⁰

However, most of these individuals are considered within normal limits and do not require treatment. According to the study by Lipton et al., only 6–12% of the population presents clinical symptoms of TMD, and about 5% shows signs and symptoms sufficiently significant to require therapeutic intervention.⁴¹

A review of the literature shows that temporomandibular disorders more frequently affect individuals aged between 20 and 40 years. Cases are more prevalent during adolescence, a period in which a significant number of individuals undergo orthodontic treatment.⁴²⁻⁴⁵

The most recent data from the National Health Interview Survey (NHIS) indicate a higher incidence rate of facial or jaw pain in women (5.8%) than in men (3.4%).⁴⁶ Cross-sectional studies confirm that the occurrence of TMD ranges from 0–10% in men and 2–18% in women, with women being 1.5–2 times more likely to develop these disorders.^{47,48}

On the other hand, results from the OPPERA study (Orofacial Pain Prospective Evaluation and Risk Assessment) on demographic patterns show that the prevalence of

clinically verified mandibular disorders is only slightly higher in women than in men (hazard ratio: 1.3; $p = 0.06$). The same study also highlights that, six months after the onset of temporomandibular disorder, the condition persists in 41% of men and 54% of women.⁴⁹

The reasons for this different prevalence between the sexes are not yet fully understood; however, available studies suggest a possible role of hormonal factors, which may promote the development of TMD in the period preceding menopause.⁵⁰

Alongside hormonal aspects, psychosocial factors and certain emotional issues, such as depression and anxiety, are also more common in females and appear to be correlated with the onset of TMJ disorders.⁵¹

In conclusion, the studies reviewed indicate that temporomandibular disorders, both muscular and articular in nature, are more frequently found in women. In most cases, however, these are mild conditions that do not result in significant disability for the patient. Only a small proportion of individuals with temporomandibular pain develops chronic pain, with consequent disability and other negative effects.^{38,52}

2.1 Classification of temporomandibular disorders

The most common temporomandibular disorders include both painful forms (such as myalgia, headache attributed to TMD, and arthralgia) and disorders affecting the temporomandibular joint, in particular disc displacements and degenerative joint diseases.⁵³

The most widely used classification system for TMD is the Research Diagnostic Criteria for Temporomandibular Disorders (RDC/TMD).⁵⁴

These criteria were developed to meet the need for a diagnostic system capable not only of distinguishing cases from non-cases for both epidemiological and clinical purposes, but also of defining and diagnosing the most common TMD subtypes.³⁸

Developed in 1992, the RDC/TMD system is based on reliable and validated criteria used for examination, diagnosis, and classification of the most common musculoskeletal forms of temporomandibular disorder.⁵⁴

This system is based on the biopsychosocial model of pain,⁵⁵ and includes two assessment domains:

- **Axis I**, which provides a physical diagnosis through standardised and reliable diagnostic criteria;
- **Axis II**, which assesses psychosocial status and the degree of pain-related disability.

The aim is not only to formulate a physical diagnosis, but also to identify patient characteristics that may influence the clinical expression of TMD and the related healing process.⁵³

The Axis I protocol (Table 1) includes the main temporomandibular disorder conditions, including myalgia, arthralgia, headache attributed to TMD, four different disc displacement conditions, degenerative joint diseases, TMJ subluxation, and TMJ dislocation.

The Axis II protocol (Table 2) consists of 8 items and is recommended as a screening tool for evaluating psychosocial and behavioral factors that may affect the patient's response to treatment.

Before applying these protocols, it is necessary to exclude dental pathologies and other painful disorders that may involve the masticatory system.⁵⁶

This classification played a crucial role because, for the first time, temporomandibular pain was framed as a biopsychosocial condition, recognizing that psychological factors such as anxiety and depression can significantly contribute to the clinical profile of the patient with TMD.³⁸

Evidence from research has therefore led to a shift from a strictly biomedical interpretative model to a multifactorial biopsychosocial model. The latter shows many similarities with other chronic musculoskeletal disorders, such as low back pain, chronic headache and fibromyalgia.⁵⁷

TABLE 1

<u>AXIS I</u>		
DISORDER	HISTORY	SIGNS and SYMPTOMS
Myalgia	Pain in the masticatory structures modified by mandibular movements or parafunctions	Pain in the temporalis and/or masseter muscle on palpation or at maximum assisted/unassisted opening
Arthralgia	Pain in the masticatory structures modified by mandibular movements or parafunctions	TMJ pain on palpation or at maximum assisted/unassisted opening, lateral or protrusive movements
Myofascial Pain	Pain in the masticatory structures modified by mandibular movements or parafunctions	Pain in the temporalis and/or masseter muscle on palpation or pain in adjacent muscles
Headache attributed to TMD	Headache in the temporal area modified by mandibular movement or parafunctions	Headache in the temporal area with pain on palpation of the temporalis muscle or at maximum assisted/unassisted opening, lateral or protrusive movements
Disc displacement with reduction	Joint noises	Noises such as click, pop, snap present during opening/closing movements or during opening, closing, lateral, and protrusive movements

Disc displacement with reduction with intermittent locking	Joint noises and locking in opening with subsequent unlocking	Noises such as click, pop, snap present during opening/closing movements or during opening, closing, lateral, and protrusive movements
Disc displacement without reduction with limited opening	Joint locking with limited opening and severe limitation interfering with the ability to eat	Maximum assisted opening < 40 mm
Disc displacement without reduction without limited opening	Joint locking with limited opening and severe limitation interfering with the ability to eat	Maximum assisted opening > 40 mm
Degenerative joint disorder	Joint noises	Crepitus present during maximum active opening, passive opening, right and left lateral movements, or protrusion

TABLE 2

<u>AXIS II</u>		
QUESTIONNAIRES	ITEMS	PURPOSE
Graded Chronic Pain Scale (GPCS)	7	Pain intensity component: pain amplification and central sensitization Pain-related disability component: reduced functioning due to pain
Pain Drawing	1	Distinguish between local, regional, and widespread pain; evaluates other painful conditions; may indicate pain amplification, central sensitization, and dysregulation
Jaw Functional Limitation Scale (GFLS)	8-20	Quantifies impact on jaw mobility, chewing, verbal and emotional expression

Patient Health Questionnaire-4 (PHQ-4)	4	Identifies psychological distress (depression and anxiety)
Patient Health Questionnaire-9 (PHQ-9)	9	Identifies depression: contributes to chronicity
Generalized Anxiety Disorder-7 (GAD-7)	7	Identifies anxiety: contributes to stress reactivity and parafunctions
Patient Health Questionnaire-15 (PHQ-15)	15	Measures physical symptoms: evaluates specific functional somatic disorders
Oral Behaviors Checklist (OBC)	21	Measures parafunctions: contributes to the onset and perpetuation of pain

According to the 1992 RDC/TMD system, temporomandibular disorders are grouped into three main categories based on common signs and symptoms. Other TMJ disorders, considered rare or lacking sufficiently reliable diagnostic criteria at the time, were not included in this classification.

Group I - Muscle disorders

The Myofascial pain

Muscle pain localized to the mandible, temples, preauricular region, face, or ear, which is influenced by mandibular movement.

It is reproducible through provocation tests of the masticatory muscles (palpation and/or mouth opening). Pain may:

- remain confined to the palpated point (local myofascial pain), or

- radiate beyond the boundaries of the examined muscle (myofascial pain with spreading).

I.b Myofascial pain with limited opening

Myofascial pain as described in I.a, associated with reduced mouth opening, defined as maximum unassisted and non-painful opening < 40 mm.

Group II - Articular disc displacements (DD)

Disc displacements are intracapsular biomechanical disorders affecting the disc-condyle complex. The disc most frequently displaces anteriorly, but displacement may also occur medially or laterally. Magnetic resonance imaging is the diagnostic gold standard for these conditions.

II.a Disc displacement with reduction

With the mouth closed, the disc is displaced anteriorly relative to the mandibular condyle, but returns to the correct position during opening.

Reduction is often accompanied by a sharp joint noise (mandibular click), which usually occurs during opening at an interincisal distance ? 5 mm (more rarely during closing).

This click disappears when opening is performed with the mandible slightly protruded.

II.b Disc displacement without reduction with limited opening

With the mouth closed, the disc is displaced anteriorly and, unlike the form with reduction, does not return to position during opening.

The condition is associated with persistent limited opening (maximum unassisted opening ? 35 mm) and may be accompanied by an uncorrected deviation toward the ipsilateral side during opening. It is also known as "closed lock".

II.c Disc displacement without reduction without limited opening

With the mouth closed, the disc is displaced anteriorly and does not reduce during opening, but in this case, there is no significant limitation of oral opening (maximum unassisted opening > 35 mm).

Group III – Other common TMJ disorders

III.a Arthralgia

Joint-origin pain modulated by mandibular movement.

Pain is reproducible by TMJ provocation tests (joint palpation and/or mouth opening).

III.b Osteoarthritis

Degenerative joint disorder (DJD) characterized by deterioration of joint tissues and bony changes of the mandibular condyle and/or articular eminence, associated with arthralgia.

III.c Osteoarthrosis

Degenerative joint disorder with the same structural features described for osteoarthritis (III.b), but not accompanied by arthralgia.

The evolution of the RDC/TMD system was completed about twenty years after its introduction, following an extensive process of research and data analysis. This pathway led to the new Diagnostic Criteria for Temporomandibular Disorders (DC/TMD), which provides reliable and validated diagnostic criteria not only for the most common TMDs, but also for less common ones.^{53, 56}

In summary, compared with the RDC/TMD, the DC/TMD system shows higher validity for clinical use, as it is based on a broader and more up-to-date body of scientific evidence.³⁸

Taxonomic classification including all disorders.^{56,58}

- **1. Temporomandibular joint disorders**

- (a) Joint pain**

- **Artralgia:** : pain involving a joint and periarticular tissues.
 - **Artrite:** inflammatory process affecting the joints, which may be acute or chronic depending on the clinical course.

- (b) Joint disorders**

- **Disc disorders**

- **Reducible disc displacement:** the disc is displaced from its physiological position between the condyle and articular eminence (anterior and/or mediolateral), but returns to position during opening.
 - **Reducible disc displacement with intermittent locking:** the disc is displaced; during opening it is sometimes recaptured and sometimes not, causing intermittent limitation of mandibular opening.
 - **Non-reducible disc displacement with limited opening:** the disc is displaced and is not recaptured during opening, and mandibular opening is limited.
 - **Non-reducible disc displacement without limited opening:** the disc is displaced and not recaptured, but no significant limitation of mandibular opening is present.
 - **Hypomobility disorders other than disc disorders**
 - **Adhesions and adherences:** fibrous tissue bands forming between tissue structures, for example after trauma or surgery. They may be considered "internal scars" connecting anatomical areas that normally should not be in contact.

- **Ankylosis:** severe limitation or complete permanent abolition of joint movements.
- **Fibrous ankylosis (even false):** due to proliferation of fibrous tissue within the joint.
- **Bony ankylosis (even true):** characterized by fusion of the joint bones due to proliferation of bone tissue (synostosis).

Hypermobility disorders

- **Displacement:** loss of the physiological position of an articular structure.
- **Subluxation:** partial loss of normal anatomical relationships between joint surfaces.
- **Luxation (dislocation):** complete loss of normal anatomical relationships, associated with capsular tearing.

(c) Joint pathologies

- **Degenerative disc diseases**
- **Osteoarthrosis:** non-inflammatory degenerative condition of the joint characterized by structural alterations of joint surfaces and underlying bone, secondary to excessive mechanical load.
- **Osteoarthritis:** inflammatory process (acute, subacute, or chronic) affecting articular bony surfaces, found in some infectious or autoimmune diseases.
- **Other joint pathologies**
- **Systemic arthritides**
- **Idiopathic condylar resorption:** loss of bone tissue at the condyle of unknown cause.
- **Osteochondritis dissecans:** osteochondrosis characterized by necrosis of a portion of the articular surface.

- **Osteonecrosis:** necrosis of bone tissue due to insufficient or absent blood supply.
- **Neoplasm:** pathological process characterized by abnormal proliferation of tissue cells.
- **Synovial chondromatosis:** rare benign condition affecting the synovial membrane.

(d) Fractures

Interruption of bone continuity.

(e) Congenital or developmental alterations

- **Aplasia:** absence or arrest of bone tissue development.
- **Hyperplasia:** non-neoplastic increase in the number of normal cells, presenting as localized or diffuse enlargement of the mandible.
- **Hypoplasia:** incomplete or reduced development of tissue.

2. Masticatory muscle disorders

(a) Muscle pain

- **Myalgia**
 - **Localized myalgia:** muscle-origin pain perceived exclusively at the palpation site.
 - **Myofascial pain (spreading):** muscle-origin pain extending beyond the palpation site but remaining within the anatomical boundaries of the involved muscle.
 - **Referred myofascial pain:** muscle-origin pain projected outside the limits of the palpated muscle.
- **Tendinitis:** inflammatory process affecting the paratenon.
- **Myositis:** inflammatory process affecting muscle tissue.

- **Spasms:** tonic, involuntary, and painful contraction of central origin, associated with local muscle conditions and deep pain.

(b) Contractures

Chronic shortening of a muscle due to fibrosis of fascia, tendons, or muscle fibers.

(c) Hypertrophy

Increase in muscle tissue volume due to abnormal increase in the volume of its constituent cells.

(d) Neoplasm

Abnormal and uncontrolled growth of muscle tissue, benign or malignant.

(e) Movement disorders

- **Orofacial dyskinesia:** abnormal, uncoordinated movements of oral and/or facial muscles, voluntary or involuntary.
- **Oromandibular dystonia:** alteration of muscle tone or neuromuscular control affecting oromandibular muscles.

(f) Masticatory muscle pain attributable to systemic/central pain disorders

- **Fibromyalgia:** idiopathic syndrome, probably autoimmune, characterized by chronic widespread muscle pain, associated with stiffness, asthenia, sleep disorders (especially insomnia), and reduced serotonin levels.

3. Headache

- **Headache attributed to TMD:** headache localized predominantly in the temporal region, secondary to pain related to temporomandibular disorders.

4. Associated structures

- **Coronoid hyperplasia:** increase in volume of the coronoid process of the mandible.

2.2 Etiology of temporomandibular disorders

The most recent scientific evidence indicates that temporomandibular disorders (TMDs) cannot be attributed to a single etiological factor, but rather result from the complex interaction of multiple elements. TMDs are multifactorial conditions that manifest not only through physical signs and symptoms, but often also involve the individual's psychosocial dimension. Such involvement may lead to alterations in behavior, mood, and relational dynamics, with a significant impact on quality of life.⁴⁹

Numerous studies have shown that the etiology of temporomandibular pain is multifactorial, with several risk factors that can predispose to disease onset or contribute to persistence and chronicity. These include traumatic events, dental occlusal alterations, specific anatomical morphological characteristics, oral parafunctions, biological factors such as the influence of sex hormones, and psychosocial factors including stress and pain catastrophizing processes.^{59,60}

In this context, the OPPERA study (Orofacial Pain: Prospective Evaluation and Risk Assessment), previously mentioned, has represented an innovative and highly relevant contribution, configuring itself as a systematic and multidisciplinary approach aimed at identifying the main risk factors associated with TMDs. This study integrated clinical, genetic, somatosensory, and psychological variables, providing a more in-depth overview of the pathogenetic mechanisms underlying these complex disorders.⁶¹⁻

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Some authors have hypothesized the involvement of female sex hormones, particularly estrogens, in the modulation of temporomandibular pain.⁶⁴ It has been suggested that these hormones may influence inflammatory processes at the temporomandibular joint (TMJ) level and modulate nociceptive responses of both the peripheral and central nervous systems.⁶⁵ However, overall, a systematic review has highlighted that evidence supporting a direct association between estrogen levels and the onset of temporomandibular disorders (TMDs) is currently limited and inconclusive.⁶⁶

A large cross-sectional study conducted in the general population reported that 83% of subjects affected by temporomandibular pain present at least one comorbid condition, while 59% present two or more. Among the most frequently associated comorbidities are migraine, neck pain, and lower back pain.^{67,68} Consistently, the OPPERA study demonstrated that the incidence of TMD increases progressively in the presence of one or more comorbidities, being significantly higher in subjects with two or more concomitant conditions compared with those with one or none.⁶⁹ Moreover, the same study identified the presence of five or more episodes of lower back pain as a relevant risk factor for the development of TMD.

Further investigations have confirmed that individuals with temporomandibular pain are 2.6 to 5 times more likely to suffer from lower back pain than asymptomatic subjects.^{67,70} Similarly, according to the study conducted by Ohrbach et al., patients with temporomandibular pain show up to 8.8 times higher odds of reporting headache than individuals without pain; in most cases, such headache can be classified as tension-type headache or migraine.⁷¹

Some studies have also examined the association between cigarette smoking and TMDs, finding a higher incidence of these disorders in habitual smokers than in non-smokers.⁷² A dose–response relationship between tobacco consumption and the intensity of temporomandibular pain has also been observed, suggesting a possible role of smoking as a modifiable risk factor.⁷³

Traumas, both macro- and micro-traumatic, represent additional potential predisposing factors for the development of temporomandibular pain.⁷⁴ Macro-trauma may occur following direct traumatic events or prolonged mouth opening, such as during dental procedures or orotracheal intubation.⁷⁵ Microtraumas, on the other hand, are mainly associated with so-called oral parafunctions.

The term “parafunctions” refers to a set of behaviors characterized by improper and often unconscious use of the masticatory system. The main parafunctions include

bruxism—understood as involuntary hyperactivity of the masticatory muscles while awake or, more frequently, during sleep—as well as habits such as lip or cheek biting, nail biting, and thumb sucking. Although stress-related parafunctions do not necessarily cause TMJ alterations, they are frequently associated with TMDs. In particular, it has been observed that individuals with a score above 24 on the Oral Behaviors Checklist have a 75% higher probability of developing a TMD compared with those scoring below 17.⁷⁶⁻⁷⁸

Numerous studies have also highlighted the determining role of psychological factors in the onset and chronicity of TMDs. In particular, mood and personality disorders are strongly associated with muscle-origin TMDs rather than disc or joint disorders.⁷⁹ Subjects with temporomandibular pain have significantly higher levels of stress, anxiety, depression, pain catastrophizing, and kinesiophobia compared with pain-free populations.^{80,81}

A prospective cohort study demonstrated that catastrophizing and depression contribute significantly to the progression of chronic pain and disability associated with TMDs, highlighting the need to consider these factors in clinical evaluation and therapeutic planning.⁵¹ The OPPERA study also identified several psychological predictors of TMD onset; in particular, the analysis by Fillingim et al. identified the frequency of generic somatic symptoms, such as fatigue and dizziness, as the strongest psychosocial predictor of TMD incidence.^{81,82} Individuals reporting such symptoms at least once a month show an incidence rate about double that of those with less frequent somatic symptoms. These results derive from a multifaceted analysis that evaluated a total of 26 psychosocial factors.⁸²

The relationship between dental occlusion and TMD remains debated in scientific literature. However, several systematic reviews based on population studies have not found a significant association between malocclusion and temporomandibular symptoms.⁸³ Similarly, large cross-sectional epidemiological surveys conducted in adult populations have shown little evidence supporting an etiological role of occlusal

factors in TMDs, encouraging the abandonment of the traditional gnathological paradigm that attributed a central role to malocclusion in the genesis of these disorders.^{84,85}

In conclusion, temporomandibular disorders are complex clinical conditions resulting from the dynamic interaction of multiple genetic, biological, environmental, and psychosocial factors. Numerous risk factors may act for years before clinical manifestation of the disorder, while others influence disease evolution toward recovery or remission. As stated by Slade et al., TMD is rarely an isolated condition, but rather the result of a combination of risk determinants that occur simultaneously or in a specific individual sequence, making it unlikely that a single factor alone is sufficient to cause the disorder.⁴⁹

It is also implied that TMDs tend to develop more frequently in individuals with compromised general health, such as smokers or those affected by systemic comorbidities. Consequently, promoting a healthy lifestyle plays a crucial role in the primary prevention of temporomandibular disorders.⁴⁹

Joint sounds

Definition

Joint sounds of the temporomandibular joint (TMJ) are clinical signs frequently found in temporomandibular disorders and can be distinguished into different types based on their acoustic and temporal characteristics.

Click

The click is a sharp joint sound of very short duration, characterized by a clearly identifiable onset and end. It is generally perceived as a dry snap during mandibular movement. For clinical evaluation, the click should be considered significant only if it is reproducible in at least two of three consecutive opening and closing cycles.

Gravel-like crepitus

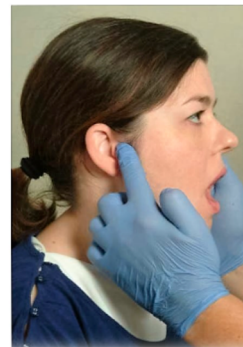
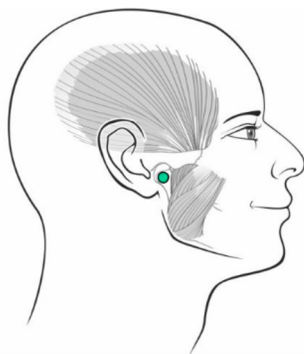
Gravel-like crepitus is a continuous joint sound of longer duration than a click, occurring over a more prolonged period during mandibular movement. It may present as a series of overlapping sounds and is not attenuated on listening. Perceptually, it is often described as a sound similar to the rubbing of bony surfaces against each other, comparable to the noise produced by rubbing one stone against another.

Sandy crepitus

Sandy crepitus is also a continuous sound, longer in duration than a click, present for a larger part of the opening and closing movement. It may manifest with overlapping sounds and is generally described as an acoustic sensation of friction on a rough surface, less pronounced than gravel-like crepitus.

2.3 Clinical evaluation procedure

To evaluate joint sounds, the examiner should place the index finger of both hands over the temporomandibular joint at the preauricular area, anterior to the tragus. The patient is then asked to slowly perform a maximum mouth opening movement, even in the presence of pain, followed by closing until maximum intercuspation is reached. This sequence should be repeated for three consecutive cycles in order to assess the presence, type, and reproducibility of joint sounds.⁸⁶



Assessment of joint sounds

Opening click

An opening click is defined as the perception of a joint sound during the opening phase, when movement is performed starting from maximum intercuspation. To be considered clinically significant, the click must be detected in at least two of three consecutive opening cycles.

Closing click

A closing click is identified by the presence of a joint sound during the closing phase, also detectable in at least two of three consecutive movements.

Reciprocal click

Reciprocal click is characterized by the presence of a click during both opening and closing. A distinguishing element of this condition is the disappearance of both sounds when the patient is asked to perform opening and closing movements starting from a protruded mandibular position.

Palpation

One of the main innovations introduced with the publication of the DC/TMD diagnostic criteria is the reproduction of painful symptomatology during the physical examination, both at the joint and muscle levels.⁸⁷

Under physiological conditions, muscle function or palpation does not evoke pain; therefore, the onset of a painful response during palpation suggests the presence of a tissue alteration, potentially attributable to trauma or functional overload.

Muscle and joint palpation may be performed by applying pressure with the tip of the index and middle finger on the site of interest, or by using the distal phalanx of the index finger with a “flat” contact surface. To ensure greater stability during the

examination, it is recommended to place the contralateral hand on the opposite side from the one being assessed.⁸⁸

The applied pressure should be standardized: a force of approximately 1 kg is recommended for extraoral palpation and approximately 500 g for intraoral palpation.⁸⁹ During the examination, the patient's mandible should be maintained in the resting position, with the teeth slightly apart. To correctly identify the muscle belly, it may be useful to ask the patient to lightly clench the teeth and then relax the musculature.

Joint pain

At the temporomandibular joint level, assessment of palpation-evoked pain focuses on two main anatomical sites.

Lateral pole

The examiner places the index finger immediately anterior to the tragus and superior to the TMJ. The patient is then asked to perform a slight mandibular opening, allowing the examiner to perceive anterior translation of the lateral pole of the condyle and evaluate any onset of pain.

Pericondylar region

Palpation is extended to the area surrounding the lateral pole of the condyle to identify the presence of pain in the pericondylar region.

Muscle pain

- Extraoral sites

Temporalis muscle

The temporalis muscle is divided into three distinct functional areas—anterior, middle, and posterior—each of which can be assessed by palpation independently.

Palpation of the predominantly vertical fibers of the anterior portion is performed above the zygomatic arch, anterior to the temporomandibular joint. The transversely oriented fibers of the middle portion are palpated over the area above the temporomandibular joint. Finally, palpation of the predominantly horizontal fibers of the posterior portion is performed superiorly and slightly posteriorly to the auricle.

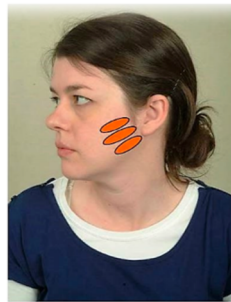


Masseter muscle

Similarly to the temporalis muscle, the masseter is assessed by palpation at three distinct sites. During the examination, one hand is used to stabilize the mandible, while the other palpates the different muscle segments.

Palpation begins at the origin area of the muscle, located inferior to the lower margin of the zygomatic process, starting in the region immediately anterior to the mandibular condyle. Next, the central portion or muscle belly is assessed, located anterior to the earlobe. Finally, the insertion area of the masseter is palpated, located anterior to the mandibular angle.

The sternocleidomastoid and trapezius muscles, although not directly involved in mandibular movements, are included in the clinical assessment as they are frequently symptomatic in patients with temporomandibular disorders (TMD) and are often involved in the genesis of headache patterns associated with TMD.



Sternocleidomastoid muscle

Palpation is performed bilaterally. Examination begins at the mastoid insertion, located posterior to the auricle, and proceeds along the entire course of the muscle belly by progressively sliding the fingers until reaching the inferior insertions, located at the middle third of the superior surface of the clavicle and the anterosuperior surface of the sternal manubrium.

Alternatively, assessment may be performed using “pincer” palpation, grasping the muscle belly between the fingers along its entire extent to identify areas of tenderness, increased tension, or pain radiation.



Trapezius muscle

Palpation is performed assessing both origin and insertion sites. In particular, examination involves the occipital area and the cervicothoracic spine for muscle origins, and insertion sites at the lateral third of the clavicle, the spine of the scapula, and the acromion.

Assessment is conducted by sliding the fingers along the entire extent of the muscle belly or using “pincer” palpation, gently grasping the muscle between the thumb and index finger to identify areas of tenderness, increased tone, or pain radiation.



- **Intraoral (supplementary) sites**

Lateral pterygoid area

To assess the lateral pterygoid area, the patient is asked to open the mouth and deviate the mandible toward the side opposite to the one being examined to facilitate access. The examiner places the index finger on the lateral surface of the alveolar ridge at the level of the upper molars. The finger is then advanced distally, superiorly, and medially, allowing palpation of the lateral pterygoid area and assessment of any evoked pain or discomfort reported by the patient.⁹⁰



Temporalis tendon

After completing palpation of the lateral pterygoid muscle, the index finger is rotated laterally near the coronoid process. The patient is asked to slightly open the mouth, allowing the examiner's finger to slide toward the anterior margin of the coronoid process. Palpation is performed on the upper portion of the process to assess the tendinous structures of the temporalis muscle.



Joint play

The joint play maneuver allows, within physiological limits, separation of the condyle–disc complex from the temporal component through forced passive mobilization of the temporomandibular joint in the three planes of space. This procedure must be performed with extreme gradualness and gentleness, avoiding the provocation of pain, in order to prevent protective reactions characterized by reflex contraction of the jaw elevator muscles. It is therefore essential that the mandible be maintained in a state of maximal muscle relaxation and that the patient's head be adequately supported, stabilized, and kept still against the examiner's body.

Assessment of joint play is carried out through distraction and translation maneuvers, with the aim of analyzing the elasticity of intra- and periarticular soft tissues, especially the capsule and ligaments, and identifying any intra-articular alterations.

The operator stands behind the patient, stabilizing the head against their own chest. The thumb of the dominant hand is placed on the premolars and molars of the hemi-arch corresponding to the side being examined, while the rest of the hand supports the mandible. At the same time, the temporomandibular joint is palpated using the middle finger of the hand stabilizing the head. Through the thumb positioned intraorally, the examiner applies a force directed inferiorly to assess distraction joint play, or a force oriented anteriorly and mesially to assess translation joint play.

The perception of reduced excursion, associated with sensations of joint stiffness or roughness, as well as any reproduction of the pain symptoms reported by the patient, are indicative of the possible presence of joint pathology.

CHAPTER 3

PAIN

Pain is one of the most complex phenomena to define comprehensively, particularly because of its marked subjective component.

The first definition formalized by the IASP (International Association for the Study of Pain) described it as:

"An unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage."

Subsequently, this definition was updated to:

"An unpleasant sensory and emotional experience associated with, or resembling that associated with, actual or potential tissue damage" (IASP).

Whereas in the original formulation pain was conceived as an unpleasant experience that could also merely be "described" in terms of tissue damage, the new definition recognizes that it can also simply be "similar" to that which occurs in the presence of tissue damage (ISAL Foundation), regardless of the individual's ability to describe it verbally.

This change, apparently subtle but conceptually significant, was introduced because the term "described" implied the subject's ability to verbalize their painful experience. This effectively excluded entire categories of individuals, such as newborns, people with severe cognitive deficits, or animals who, despite being unable to express their condition in words, nonetheless experience pain.

Through this revision, the IASP pursued the goal of developing a definition of pain that is as inclusive as possible, capable of encompassing both the subjective and communicative dimensions of the pain experience and the implicit, non-verbalizable

ones, recognizing pain as a complex, multidimensional phenomenon that cannot be reduced to verbal description alone.

To complement the definition, the scientific society added six key points that further specify and broaden its meaning:

- Pain is always a personal experience, variably influenced by multiple biological, psychological, and social factors. Therefore, it cannot be fully understood outside the individual's biopsychosocial context.
- Pain and nociception are distinct phenomena. Pain is the subjective experience—i.e., the sensation or perception of irritating, burning, or unbearable stimuli arising from a specific body region. Nociception, by contrast, is the sensory process through which signals are generated and transmitted that may later be interpreted as pain. Nociceptive receptors may be activated acutely or continuously, whereas the pain experience may occur intermittently⁹¹.
- Over the course of life, individuals learn the concept of pain through personal experiences, observing others, and social and cultural interactions. This learning helps shape how pain is perceived, interpreted, and communicated.
- The subjective report of the pain experience must be considered and respected. The patient's narrative about their pain is central to clinical assessment and cannot be minimized or questioned without adequate reasons.
- Although in most cases pain has an adaptive function—signaling actual or potential tissue damage and promoting protective behaviors—it can also take on a maladaptive dimension. In such circumstances, it can significantly impair overall functioning as well as psychological well-being and social life.
- Verbal expression of pain is only one way in which the pain experience can be communicated. The absence of verbal communication, or the inability to communicate verbally, does not exclude the possibility that a human being, as well as a non-human animal, feels pain. Therefore, pain assessment must also consider non-verbal behaviors and other observable indicators.

31. Physiology of pain

The sensory process responsible for detecting and transmitting painful signals is called "nociception," from the Latin nocere (to harm). Nociceptive stimuli share the ability to threaten the organism's integrity and to activate a specific set of sensory organs, the nociceptors ⁹².

Nociceptors are free, branched, unmyelinated nerve endings belonging to peripheral sensory neurons. They signal that a bodily tissue has been, or is at risk of being, damaged. They are activated by potentially harmful stimuli capable of causing tissue damage. Their membrane expresses ion channels sensitive to different categories of noxious stimuli (mechanical, thermal, chemical), whose activation triggers the generation of the action potential ⁹¹.

Pain perception requires central integration of the peripheral noxious stimulus and unfolds in several phases:

- Transduction: the noxious stimulus is converted into an electrical impulse at the level of nociceptors.
- Transmission: the electrical impulse is carried along peripheral afferent nerve fibers and along the main ascending pathways to the thalamus and sensory cortical areas.
- Perception: at the cortical level, the stimulus is processed and recognized as a painful experience.

Nociceptors thus transform the injurious stimulus into an action potential that propagates along the axons of peripheral afferent nerve fibers to the central nervous system. The main fibers involved in pain transmission are myelinated A-delta fibers and unmyelinated C fibers. A-delta fibers transmit an acute, well-localized pain, mainly related to noxious mechanical stimuli and cold temperatures; C fibers convey a duller pain, less well defined topographically but more persistent over time. These features allow distinguishing the so-called "first pain" (fast, sharp, A-delta) from the "second pain" (slow, burning, C) ⁹³.

After being conveyed by A-delta and C fibers through the first sensory neuron, the painful stimulus reaches the dorsal horns of the spinal cord, where it synapses with the second neuron. Transmission of the impulse between the first and second neuron is mediated by neurotransmitter release at presynaptic terminals.

Cells in the dorsal horn are organized into laminae (Rexed laminae). Neurons in lamina I receive direct afference from A-delta and C fibers and are modulated by inhibitory interneurons located mainly in lamina II (substantia gelatinosa) ⁹⁴.

From the dorsal horn, nociceptive information reaches supraspinal structures through various ascending pathways, consisting of fiber bundles that can be schematically summarized as:

1. Primary afferent nociceptive pathway
2. Multisynaptic ascending system
3. Accessory pathways, i.e., alternative routes of pain-signal transmission

Nociceptive information carried by these pathways reaches the thalamus, where it synapses with the third neuron, which then projects to the cerebral cortex.

Conscious perception of pain results from the interaction between the transmission and perception system (ascending system) and the modulation system (descending system), which can exert both facilitatory and inhibitory effects on pain-signal conduction. This bidirectional dynamic helps determine the intensity, quality, and subjective meaning of the pain experience.

3.2 Classification of pain

Pain can be defined as the conscious perception of an unpleasant sensation or one that threatens the organism's integrity. It represents the conscious sensory effect deriving from integration of the nociceptive message within the sensory, cognitive, and affective spheres of the central nervous system.

From a clinical standpoint, pain may present as a "symptom," i.e., a manifestation of an underlying pathological process, or as a true "disease" when the lesion and pathological processes activate mechanisms that amplify and maintain pain, interfering significantly with the patient's quality of life both physically and psychologically.

Pain can be classified according to different criteria.

Classification by duration

Acute pain:

A short-lasting pain directly related to tissue damage. It has a mainly adaptive and alarm function, signaling the presence of dangerous or potentially dangerous stimuli both in the environment and in the organism.

A cause-effect relationship between the injurious stimulus and pain perception is generally recognizable. Pain tends to resolve when the stimulus ceases or when the damage that caused it is repaired.

Persistent pain:

Pain related to persistence or repetition of the noxious stimulus. It retains the clinical features of acute pain (qualitative modality, link with the stimulus) but, in terms of duration and course, does not yet show the traits of chronic pain.

Chronic pain:

Defined by duration (longer than 3 months) and/or persistence of painful symptoms despite resolution of the initial cause. In chronic pain, a clear link with the original pathogenic event is no longer recognizable.

The chronification process results from maintenance of the algic stimulus and may be accompanied by a progressive increase in pain intensity. It is often associated with increased anxiety and depression levels, which contribute to persistence and amplification of the pain experience ⁹⁵.

Chronic pain is further distinguished into:

- Primary chronic pain: associated with marked psychosocial disturbances and severe functional disabilities, in the absence of an organic pathology that can fully explain the symptoms.
- Secondary chronic pain: associated with an underlying pathology of which it represents a symptom. Psychosocial disturbances and varying degrees of disability are often present, resulting both from persistence of the symptom and from damage caused by the underlying disease, although these aspects are generally less marked than in primary chronic pain ⁹⁵.

The onset and maintenance of chronic pain are linked to multiple mechanisms, including peripheral sensitization and central sensitization.

Peripheral sensitization:

Sensitivity of peripheral nociceptive terminals is increased due to reduced activation threshold and increased membrane excitability. Clinically, it manifests as:

Primary allodynia ⁹⁶: pain evoked by a low-intensity stimulus that would not cause pain in other areas ⁹⁵.

Primary hyperalgesia ⁹⁶: an excessive response to a truly noxious stimulus, perceived as more painful than in regions not affected by the inflammatory process responsible for lowering the nociceptive threshold ⁹⁵.

Central sensitization:

Characterized by increased and prolonged hyperexcitability of the central nociceptive system (and, broadly, also peripheral), generally reversible, which produces clinical pictures of hypersensitivity. In this context, secondary allodynia is observed: mild cutaneous stimuli applied within the painful area are perceived as unpleasant or painful. This indicates that, at the spinal synapse, some neurons have lowered their activation threshold, so that tactile signals normally not painful are integrated into the hyperexcited circuit, generating a sensation reported as bothersome or painful ⁹⁷.

Breakthrough pain (episodic pain):

Defined as a transient increase in pain intensity in a patient whose baseline pain is adequately controlled by fixed-schedule analgesic therapy. It is characterized by rapid onset, severe intensity, and an average duration of about 30 minutes per episode.

It can be further classified as:

- Incidental: related to specific triggers (e.g., movement or certain activities).
- Spontaneous: not associated with particular activities or evident stimuli.
- End-of-dose: related to an inadequate analgesic dose or an interval between administrations that is too long.

Classification by origin of the lesion (pathogenetic mechanism)

Nociceptive pain

According to the IASP definition, it is "pain that arises from actual or threatened damage to non-neural tissue and is due to activation of nociceptors."

Pain impulses originate from peripheral nociceptors located in various somatic and visceral tissues ⁹¹ and are characterized by integrity of the somatosensory system ⁹⁷.

Within nociceptive pain, the following can be distinguished:

Inflammatory nociceptive pain: tissue damage triggers release of inflammatory mediators that cause peripheral sensitization of nociceptors. Primary allodynia is typically present.

Mechanical-structural nociceptive pain: anatomical changes (e.g., compressions, structural deformities) determine a local alteration that exposes nociceptors to high-intensity stimuli. The main difference from inflammatory pain is the absence of primary allodynia as a distinguishing feature.

Neuropathic pain

Defined as "pain caused by a lesion or disease of the somatosensory nervous system" (IASP).

It is divided into:

- **Peripheral neuropathic:** when the lesion or disease affects the peripheral nervous system.
- **Central neuropathic:** when the central nervous system is involved ⁹⁷.

In this context, pain impulses are generated at the site of the lesion along the nerve pathway, called the ectopic site, to distinguish it from the normotopic site, corresponding to tissue nociceptors. The ectopic site constitutes a hyperexcitable zone of the nerve pathway from which spontaneous action potentials may arise or be induced by mechanical, thermal, or chemical stimuli. At the same time, the lesion interrupts conduction along fibers, resulting in loss of some peripheral sensory modalities.

Clinical conditions most frequently associated with neuropathic pain include diabetic polyneuropathy, neuropathies related to HIV infection or chemotherapy treatments, radiculopathies, and various forms of peripheral neuropathies. In clinical practice, presentations characterized by hypersensitivity to stimuli with reduced pain thresholds are common.

Nociplastic pain

According to the IASP definition, nociplastic pain is "pain that arises from altered nociception despite no clear evidence of actual or threatened tissue damage causing activation of peripheral nociceptors, nor evidence of disease or lesion of the somatosensory system that would account for the pain" ⁹⁵.

In this case, the nociceptive system is dysfunctional in central processing and modulation, despite the absence of an evident structural lesion, producing often complex clinical pictures in which pain has a marked diffuse and persistent component with relevant psychosocial repercussions.

3.3 Temporomandibular pain

Temporomandibular pain can present with different clinical characteristics and is influenced by multiple pathogenetic and contextual factors. From a mechanistic standpoint, it can be divided into three main types: peripheral nociceptive pain, peripheral neuropathic pain, and centralized pain.

Peripheral nociceptive pain is related to nociceptor activation following actual or potential tissue damage or an inflammatory process. It is characterized by pain and/or tenderness localized at, or in close proximity to, the injured area. Diffuse hyperalgesia is not present. Pain intensity is proportional to the degree of nociceptive stimulation and is modulated by individual sensitivity, which varies significantly among subjects⁹⁸.

Peripheral neuropathic pain is instead due to lesion or dysfunction of primary nociceptive afferents. In this case, pain is typically referred to the dermatomal area innervated by one or more damaged nerves. Diffuse hyperalgesia is not observed; pain may radiate, be stabbing in nature, and often shows marked variability in intensity and temporal course⁹⁸.

Centralized pain refers to conditions in which pain is perceived chronically in the absence of an adequate counterpart of peripheral nociceptive stimulation. It reflects maladaptive adaptations of the central nervous system, which abnormally amplify ascending peripheral input, generating and maintaining the pain experience even with minimal or absent nociceptive drive. This picture is attributed to dysregulation of pain-processing circuits at the spinal cord and/or supraspinal brain structures.

Clinically, centralized pain may present as regional or radiating pain; patients often report difficulty precisely localizing the pain site. It is characterized by diffuse hyperalgesia and by lack of correlation between the extent of any peripheral damage and the intensity of perceived pain. This type of pain is frequently found in a group of idiopathic chronic pain conditions-such as fibromyalgia (FM), chronic fatigue syndrome (CFS), irritable bowel syndrome (IBS), several forms of headache, and

temporomandibular disorders (TMD)-which show moderate-to-high heritability and comorbidity⁹⁹⁻¹⁰¹.

Distinguishing predominantly peripheral pain from centralized pain is crucial, as the underlying mechanism significantly affects both prognosis and therapeutic strategies. The similarity between these clinical pictures, aside from the body regions involved, suggests the existence of multiple common elements in their pathophysiological bases⁹⁸.

Central sensitization in temporomandibular disorders (TMD)

The term central sensitization (CS) is used to describe a process of amplification of pain signals within the central nervous system that may develop and persist in the presence of chronic pain. 98 Alterations of CNS processing of nociceptive input are considered a key factor in the onset and maintenance of pain in patients with chronic TMD¹⁰².

Although the pathophysiology of CS in TMD is not yet fully clarified, numerous experimental and clinical findings indicate alterations in pain threshold and modulation.

Among the most frequently observed features in TMD patients are:

- reduced Pressure Pain Threshold (PPT) after mechanical stimulation applied to the face,
- thermal hyperalgesia,
- increased Temporal Summation (TS) of pain. ¹⁰³

Pressure Pain Threshold

Pressure Pain Threshold (PPT) is defined as the minimum level of applied pressure capable of evoking a painful sensation. It is an indicator of the nervous system's sensitivity to pain and is commonly measured using pressure algometry in different body regions.

When reduced PPT is found also at body sites distant from the temporomandibular dysfunction area, this suggests possible central nervous system involvement in terms of central sensitization¹⁰⁴.

Many studies have investigated hyperalgesia in TMD patients by measuring PPT¹⁰⁵⁻¹⁰⁶. In particular, pain sensitivity has been compared between groups affected by TMD and asymptomatic control groups, often assessing distal painful sites as indirect indicators of central sensitization. Despite variable distances and measurement points, ten of the twelve studies considered showed a statistically significant reduction in PPT values in subjects with TMD compared with healthy controls¹⁰⁷⁻¹¹⁵, supporting the hypothesis of altered central pain modulation in this population.

Thermal hyperalgesia

Thermal hyperalgesia indicates increased sensitivity to pain evoked by thermal stimuli. It is mainly studied through two parameters:

- Cold Pain Threshold (CPT): the temperature at which the sensation of cold becomes painful;
- Heat Pain Threshold (HPT): the temperature at which the sensation of heat becomes painful.

CPT was used as an index of hyperalgesia in TMD patients in two clinical studies that demonstrated significant cold hyperalgesia (reduced CPT) in subjects with temporomandibular dysfunction¹¹⁶⁻¹¹⁷. Moreover, the extent of cold hyperalgesia in the region innervated by the trigeminal nerve was correlated with the duration and intensity of TMD pain symptoms,¹¹⁶ suggesting a link between chronicity and central amplification of the pain response.

Regarding HPT, the literature reports non-univocal results. Three studies found no significant differences in sensitivity to painful heat between TMD subjects and asymptomatic controls,¹⁰⁸⁻¹¹⁸⁻¹¹⁹ whereas five other studies reported greater sensitivity to thermal pain (reduced HPT) in TMD patients compared with pain-free subjects.¹⁰⁷⁻¹¹¹⁻¹¹⁶⁻¹²⁰⁻¹²¹ Overall, these data suggest that, at least in a subgroup of patients, the

thermal component of pain is modulated in a hyperalgesic direction, consistent with a picture of central sensitization.

Temporal Summation

Temporal Summation (TS) refers to the progressive increase in perceived pain in response to repeated nociceptive stimuli delivered over a given time interval. Increased TS is generally considered indicative of spinal hyperexcitability and, more broadly, of central sensitization.¹²²

Five studies found greater mechanical TS in TMD patients than in controls,^{102,112,123-125} confirming a tendency toward pain amplification after repeated stimulation. Sarlani et al. showed that following repeated painful stimulation, TMD patients exhibited more pronounced temporal summation and greater intensity of post-stimulation pain sensations, even in remote areas of the face and head, compared with controls.¹⁰²

In contrast, Sato et al. did not observe a significant increase in TS in any of the examined groups; however, participants consistently reported post-stimulation pain sensations,¹²⁶ suggesting that central plasticity mechanisms may not emerge in the same way across different experimental paradigms.

Further differences emerged by sex. Sarlani et al. found that neither asymptomatic subjects nor male TMD patients showed significant TS of mechanically evoked pain,¹²⁷⁻¹²⁸ whereas this phenomenon was more evident in women, supporting a possible role of hormonal, genetic, and psychosocial factors in modulating central sensitization.

Psychosocial factors

Several lines of evidence indicate that, in each individual, at least two overlapping sets of somatic traits coexist:

- those related to pain and sensory amplification,
- those related to mood and affectivity¹⁰⁰

Some studies suggest that sensory and affective amplification of pain is a distinctive feature of centralized pain, whereas psychological factors seem to influence the affective component of pain experience to a greater extent.

In TMD patients, high levels of depression and/or the presence of somatic disorders-often associated with chronicity of the disorder, orofacial tenderness, and reduced pain tolerance-are consistent with a picture of centralized pain.¹²⁹⁻¹³¹

For the dentist treating these patients, it is therefore essential to consider AXIS II dimensions, i.e., psychosocial characteristics, recognized as important predictors of rehabilitation treatment outcomes. The literature documents that high levels of depression, catastrophizing, and somatic disorders are strong predictors of a negative treatment response. Moreover, the presence of alexithymia and marked psychosocial stress can directly influence the onset, persistence, and intensity of TMD symptoms, acting on neural substrates of pain and/or indicating shared etiological factors that promote psychological vulnerability and development of chronic pain.¹³²⁻¹³⁴

Clinically, in the assessment and treatment of TMD patients, centralized pain and psychological dysfunction should be considered closely interconnected aspects. Their interaction may be related to shared etiological factors, including altered functioning of specific neurotransmitter systems and early psychological trauma associated both with the onset of psychological disorders and the development of chronic pain conditions.¹³⁵⁻¹³⁶ It follows that the dentist should integrate into clinical analysis further components of the biopsychosocial model, such as:

- personality traits,
- stress levels,
- presence of depression, anxiety, and catastrophizing.

Personality

Personality refers to the relatively stable set of traits and characteristics that distinguish an individual, determining habitual patterns of thought, emotional experience, and behavior across different life contexts.

In TMD patients, some personality traits are associated with bruxism, defined as a repetitive jaw activity manifested by clenching, tooth grinding, or mandibular thrusting movements.¹³⁷ Bruxism is generally divided into sleep bruxism and awake bruxism.

In both forms, it is closely related to temporomandibular joint disorders and overload of masticatory muscles.¹³⁸

Pingitore et al.¹³⁹ found a higher frequency of bruxism in individuals described as ambitious, aggressive, competitive, and emotionally tense. Conversely, Fisher et al.¹⁴⁰ observed in subjects with bruxism a more markedly introverted and shy personality profile. These heterogeneous data indicate a link between specific personality characteristics and parafunctional behaviors that may contribute to the development or maintenance of TMD.

Stress

Stress can be defined as the result of an imbalance between environmental demands and the individual's perceived resources. It is often accompanied by intense negative emotions and determines not only psychological but also somatic manifestations affecting various body districts, including the stomatognathic system.

Emotions typical of stress states are associated with physiological changes (neuroendocrine, autonomic, and muscular) that can facilitate or accelerate development of somatic disorders. A prolonged state of emotional tension frequently induces increased muscle activity, including in masticatory muscles. Moreover, chronic tension is associated with increased parafunctional activities, considered among the main triggering factors for TMD.¹⁴¹

Parafunctions are defined as unconscious, abnormal, and habitual activities of the masticatory apparatus occurring outside its physiological functions (mastication, swallowing, phonation). They may occur both during wakefulness and during sleep. Stress, together with anxiety, depression, and specific personality traits, seems to favor a higher frequency of abnormal contacts between dental arches.¹⁴²

Some studies have shown a significant association between stress levels and daytime bruxism, whereas insufficiently robust data are available to confirm with the same strength a link between stress and nocturnal bruxism.¹⁴³

Within this framework, recognition and management of psychosocial factors-especially stress, dysfunctional personality traits, depression, anxiety, and catastrophizing-are fundamental elements for comprehensive care and for setting physiotherapeutic interventions that are truly effective in TMD patients.

Depression

Depression is classified as a major mental health factor. The most frequently observed symptoms include low mood, anhedonia (loss of interest in usual activities), reduced activity level, easy fatigability, difficulty concentrating, low self-esteem, sleep disturbances, and a pessimistic view of the future.¹⁴⁴

The association between depression and numerous painful disorders, including temporomandibular disorders (TMD), is widely documented.¹⁴⁵⁻¹⁴⁷ In patients with both depression and chronic pain, pain intensity and degree of functional limitation are higher than in non-depressed subjects. Moreover, response to analgesic treatments tends to be less favorable in patients with depressive symptoms.

TMD patients with concomitant stress and depression show greater activation of masticatory muscles, accompanied by increased abnormal occlusal contacts.¹⁴²⁻¹⁴⁸ The study by Lajnert et al.¹⁴⁷, conducted on patients with chronic TMD, shows a significant association between this condition and depressive symptoms and somatization. This comorbidity can reduce tolerance thresholds to pain and stress. Higher levels of

depression and somatization were found in patients with chronic painful TMD compared with those with acute temporomandibular symptoms.

Anxiety

Anxiety is defined as a state of psychosomatic tension, generally perceived as a condition of distressing passivity toward potentially harmful events that the subject believes are about to occur or fears might happen.

The scientific literature reports numerous findings supporting a direct correlation between anxiety disorders and painful TMD.¹⁴⁸ Somatization related to myofascial pain may involve bony and muscular structures of the temporomandibular joint (TMJ), with particular involvement of masticatory muscles. It has been shown that the influence of somatization on chronic painful TMD is not, contrary to past hypotheses, directly associated with increased parafunctional activity during sleep.¹⁴⁹

Some anxiety manifestations may induce compulsive behaviors, frequently found in specific forms of TMD. These compulsive behaviors are often associated with recurrent obsessive thoughts and drive the individual to perform "relieving" ritual actions that are repetitive and stereotyped. These rituals are neither useful nor pleasant for the patient and are often related to psychological problems. Some may constitute parafunctional activities involving the masticatory system; among these, the most common is onychophagia.

Onychophagia, classified as a psychodermatosis, consists of the habitual behavior of biting one's nails, often related to psychological disorders. It can arise in childhood or adulthood, with typical onset between 4 and 6 years and increasing frequency with age.¹⁵⁰ Mild and severe forms are distinguished. Etiology is multifactorial and frequently associated with psychological distress, emotional hypersensitivity, low self-esteem, psychological complexes, and apprehension states.

The literature also reports among possible causal factors the sucking habit adopted by children with unmet emotional needs, lack of affection, insecurity, as well as alterations

in blood levels of iron, copper, and vitamins. Prolonged nail biting can lead to numerous aesthetic and health complications, including dental problems such as malocclusions, temporomandibular joint disorders, masseter muscle hypertrophy, wear of dental surfaces, and inflammatory processes affecting gingival tissues.¹⁵¹

Catastrophizing

Catastrophizing is a cognitive distortion frequently found in depressive disorders and anxiety disorders. In particular, pain catastrophizing is defined as a set of excessively negative cognitive and affective responses associated with a real painful experience or its anticipation. It typically affects individuals who tend to formulate irrational and disproportionate negative predictions about future events. Pessimism and worry are closely intertwined with pain experience, making it difficult for the subject to shift attention away from pain.¹⁴⁴

Catastrophizing is frequently observed in individuals with multiple musculoskeletal disorders and can cause a marked increase both in perceived pain intensity and in related functional limitations. In addition, in subjects with high levels of catastrophic thoughts, analgesic treatments tend to be less effective than in patients who do not display this cognitive style.¹⁵²

These findings are confirmed by an experimental study in which a 5% hypertonic saline solution was injected into the masseter muscle: patients with higher catastrophizing tendency reported significantly higher pain intensity than those with low catastrophizing tendency.¹⁵²

A possible explanation is the hypothesis of more pronounced central sensitization in subjects with marked catastrophizing tendencies, influencing central nervous system activity at higher levels, including the prefrontal cortex, which is involved in the control of complex emotions.¹⁵³

Integrating standardized tools for assessment and diagnosis of catastrophizing into the clinical examination allows early identification of patients with this specific cognitive

vulnerability, enabling selection of a more targeted and appropriate therapeutic approach. ¹⁵⁴⁻¹⁵⁵

Sleep

The relationship between sleep and pain can be described through two main interpretive models. In acute pain, the relationship follows a linear model: pain causes a transient impairment of sleep quality, which tends to recover once the algic condition resolves.

In chronic pain conditions, the relationship becomes circular: disturbed sleep is followed by days characterized by intense and fluctuating pain, with consequent failure to achieve the restorative effect of sleep. ¹⁵⁶

Quartana et al. ¹⁵⁶ show that sleep disturbance symptoms reported by patients are the best predictors of subsequent pain experience, rather than the reverse. Other authors, in studies comparing TMD subjects with healthy individuals, have investigated the role of sleep in modulating pain perception and central nervous system processing, suggesting that poorer sleep quality may predispose to increased pain intensity. ¹⁵⁷⁻¹⁵⁹

Sanders et al. ¹⁶⁰, within the "Orofacial Pain Prospective Evaluation and Risk Assessment study" (OPPERA), observed that subjective sleep quality progressively worsens before onset of symptoms attributable to TMD pain. The study involved participants aged 18 to 44 years, without significant TMD history, without relevant medical disease, no recent facial trauma or surgery, and not undergoing orthodontic treatment. Participants who reported poor sleep quality already at baseline developed TMD earlier, with double the incidence compared to those reporting good sleep quality. The authors also found no evidence that poor sleep quality was associated with changes in sensitivity to pain stimuli under experimental conditions, nor that the effect of poor sleep quality on TMD risk was mediated by laboratory-induced pain sensitivity.

Ribeiro Da Silva et al. ¹⁵⁸ confirm the correlation between impaired sleep quality and TMD. Lyngberg et al. ¹⁶¹ and Boardman et al. ¹⁶¹ further suggest that sleep alteration is a more robust and reliable predictor of pain onset than other factors.

Disturbed sleep can lower pain thresholds and increase sensitivity. Insomnia is associated with increased risk of developing future chronic pain disorders in initially healthy individuals, whereas pre-existing pain does not emerge as a significant predictive factor for new insomnia onset.

Overall, available evidence shows an association between painful TMD and sleep quality: the presence of pain appears to significantly impact sleep quality in TMD patients.

Genetic and immune factors

Scientific literature indicates that genetic factors contribute approximately 50% to individual variability in pain sensitivity and that the same genes may increase predisposition to developing chronic pain. Mutations have been identified in five main genetic groups: sodium and potassium channel genes, GTP cyclohydrolase genes, adrenergic receptor genes, and genes regulating catechol-O-methyltransferase (COMT), an estrogen-sensitive enzyme that may partly explain sex differences observed in chronic pain.¹⁶³⁻¹⁶⁷

The current hypothesis is that factors associated with so-called "centralized pain" contribute to defining a pain-prone phenotype which over time favors development of multiple chronic pain conditions. This framework could help predict which subjects are at higher risk of transition from acute to chronic pain following injuries or specific environmental stress factors.

The OPPERA study is, to date, the largest and most methodologically rigorous investigation of genetic risk factors associated with TMD and related conditions.¹⁶⁸

It is a prospective study conducted on over 2,700 individuals aimed at identifying single-nucleotide polymorphisms (SNPs) in genes commonly implicated in pain perception, affective processes, and inflammatory mechanisms, to determine those associated with higher likelihood of developing TMD. No SNP was identified as a

primary cause of TMD; however, some polymorphisms were predictive of intermediate phenotypes related to centralized pain.

Main associations identified include:

- between nonspecific orofacial pain and a single SNP in the SCN1A gene, involved in initiation and propagation of action potentials in afferent fibers, with possible impairment of inhibitory function mediated by gamma-aminobutyric acid (GABA) in the central nervous system; ¹⁶⁹
- between general psychological symptoms and a single SNP in the COX1 gene, a key regulator of central neuroinflammation processes; ¹⁷⁰
- between temporal summation of pain sensation induced by thermal stimuli and a single SNP in the MPDZ gene, which codes for proteins associated with G-protein-coupled receptors for various neurotransmitters, including GABA. ¹⁷¹

Sex and age

To date, it is not yet fully clear which specific biological, psychological, or sociocultural aspects make women more susceptible to developing TMD than men. Sex differences may be related to hormonal factors, cultural and social elements, higher levels of occupational stress, and differences in pain sensitivity. ¹⁷¹⁻¹⁷⁹

Depression and anxiety disorders affect women at nearly twice the frequency observed in men, which could help explain part of the prevalence differences of TMD between sexes. ¹⁷⁶

Regarding age, TMD are particularly widespread in the adolescent population, as documented by recent epidemiological studies on large general-population samples. ¹⁸⁰⁻¹⁸¹ Despite heterogeneity of results among studies, most authors found that the predominant proportion of subjects with TMD symptoms are women, similar to findings in adult populations.

However, only a few works have specifically examined sex as a risk factor for developing TMD in adolescence. In one study, authors observed a 37% increase in

prevalence ratio in women for developing TMD symptoms and a 76% increase for myofascial pain.¹⁸¹ Another publication reported a 30% increased risk of developing symptoms attributable to temporomandibular dysfunctions and a 50% increase for myofascial pain, without significant sex differences for two other clinical signs, arthralgia and osteoarthritis.¹⁸¹

The increase in pain intensity seems to affect women more than men both in adult and adolescent populations.¹⁷⁶ Overall, available data indicate that females have more than double the probability of developing TMD. Nonetheless, other determinants must also be considered, such as general health status, presence of chronic pain disorders, age, ethnicity, and psychosocial and genetic factors.

In conclusion, the differing incidence of TMD in women compared with men remains an area requiring further research in order to improve understanding of underlying mechanisms and to orient toward a more targeted and personalized clinical approach in managing and treating these conditions.

3.4 Pain assessment scale

Given the centrality and clinical relevance of pain, Italian legislation-Article 7 of Law no. 38/2010 requires that healthcare documentation record pain characteristics and their evolution over time, the analgesic technique used, administered drugs with dosages, and the analgesic outcome achieved.

Using suitable, reliable, and validated instruments for pain measurement is one of the fundamental aspects of correct pain management. The ability to ensure adequate treatment depends both on the coherence of the assessment tool with the patient's cognitive and communicative abilities and on correct interpretation of scores by healthcare professionals¹⁸². Systematic pain measurement allows appropriate therapeutic interventions, prevention of secondary disability, and improvement of quality of life.

Pain measurement scales thus play a central role in clinical practice and are essential for global assessment and management of a care need that profoundly affects the lives of assisted persons.

The literature describes numerous pain assessment scales, traditionally divided into:

- unidimensional scales, which mainly detect pain intensity and are preferred self-assessment tools;
- multidimensional scales, which consider different components of the pain experience, such as sensory, motivational, affective, and cognitive-evaluative dimensions ¹⁸².

Many of these instruments require, as a prerequisite, the possibility of establishing verbal communication with the patient and the patient's ability to understand instructions and the meaning of proposed items.

Among the most widely used unidimensional scales are:

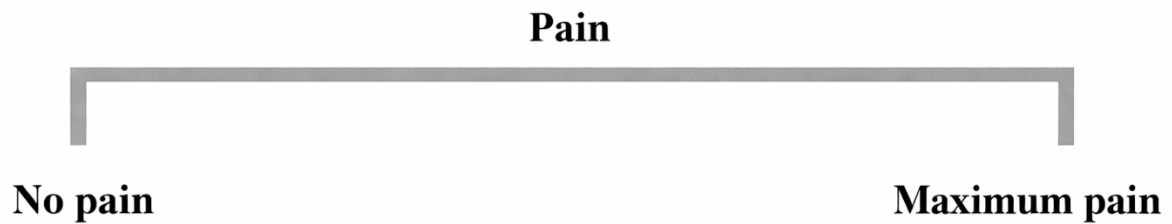
VAS - Visual Analogue Scale

VAS consists of a straight line, generally 10 cm long, without intermediate numerical indications. The patient is asked to mark on the line the point that best represents the intensity of pain perceived at the time of evaluation. The left end corresponds to "no pain," while the right end indicates "worst imaginable pain."

The score is obtained by measuring, in millimeters, the distance between the left end of the line and the point marked by the patient ¹⁸².

VAS has some applicability limits, particularly in elderly populations and in subjects with perceptual impairment and/or cognitive decline, in whom abstract understanding of the scale and adequate task execution may be difficult.

VAS SCALE – VISUAL ANALOG SCALE



NRS - Numeric Rating Scale

NRS is a unidimensional numerical scale, generally ranging from 0 to 10, in which:

0 corresponds to no pain,

10 represents the maximum imaginable pain.

It is a tool for measuring pain intensity in which the assisted person is invited to express their pain level by indicating a number between 0 and 10 that best describes their perception of pain, or by circling the chosen number ¹⁸².

When the scale is administered exclusively verbally, it is often referred to as the Verbal Numeric Rating Scale (VNRS) or Verbal Numeric Scale (VNS). In this modality, the clinician asks the patient to quantify pain on a scale from 0 to 10 and records the reported value.

NRS SCALE – NUMERIC RATING SCALE



VRS - Verbal Rating Scale

VRS is a unidimensional scale that represents pain through a series of verbal descriptors associated with corresponding numerical values. Visually, it is represented as a segment on which 5 or 6 pain adjectives are arranged in increasing order, each linked to a numerical score quantifying intensity.

A typical progression from left to right is:

0 = no pain

1 = mild pain

2 = moderate pain

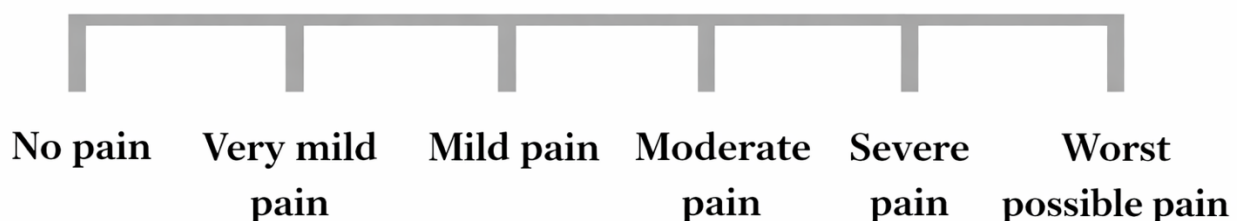
3 = severe pain

4 = very severe pain (medical pain)

5 = unbearable pain

The patient is invited to choose the adjective that best describes their pain experience; the healthcare provider then records the corresponding numerical value. This scale is particularly useful in subjects who have difficulty using abstract or numerical representations of pain, enabling a more immediate and intuitive assessment of pain intensity.

VRS SCALE – VERBAL RATING SCALE



Multidimensional scales

MPQ - McGill Pain Questionnaire

The McGill Pain Questionnaire (MPQ) combines use of a VRS-type scale with detection of pain location through questions addressed directly to the patient and completion of body diagrams. These diagrams depict the human figure in anterior and posterior views, on which the patient is asked to precisely mark the point or area where pain is perceived.

It is a multidimensional scale considering three main components of the pain experience:

- Sensory, relating to quantitative and qualitative characteristics of pain (e.g., type, duration, intensity);
- Affective-emotional, referring to the emotional and affective impact of pain;
- Evaluative, concerning the patient's overall judgment of pain intensity.

Each descriptive item is assigned to a subclass, for a total of 20 distinct terms, each associated with a numerical value. The sum of obtained scores provides an overall index contributing to defining subjective pain perception.

BPI - Brief Pain Inventory

The Brief Pain Inventory (BPI) is a multidimensional tool assessing two fundamental aspects:

- pain intensity,
- degree of pain interference with daily activities and the patient's life.

Pain intensity and related disability are measured using 0-10 numerical scales associated with questions exploring both physical areas (e.g., mobility, self-care, work activities) and psychosocial dimensions (mood, interpersonal relationships, sleep).

Patients are also asked to answer a set of questions referring to the previous 24 hours, aiming to document pain course, temporal variations, and overall impact on daily functioning.

PAINAD - Pain Assessment in Advanced Dementia

PAINAD is a multidimensional scale specifically designed to assess pain in non-cooperative patients with severe cognitive impairment, such as those with advanced-stage dementia (Sanitary AssoCareNews Periodical).

The scale considers five key observational parameters:

- breathing
- vocalizations (moaning, groaning, crying out)
- facial expression
- body language (agitation, rigidity, antalgic postures)
- reassurance (response to comfort attempts)

Each item is assessed and monitored with a specific score; the sum generates a total score from 0 to 10, interpreted as:

- 0: no pain
- 1-3: mild pain
- 4-6: moderate pain
- 7-10: severe pain

In addition to these tools, specific scales are available for identifying a neuropathic component of pain:

DN4 (Douleur Neuropathique en 4 questions)

NPSI (Neuropathic Pain Symptom Inventory)

DN4

The DN4 questionnaire¹⁸³ consists of 10 items related to sensory characteristics and clinical signs typical of neuropathic pain. Each positive answer scores 1, each negative answer scores 0. The sum yields a total score from 0 to 10.

A value that equals or is higher than 4 indicates a high probability that pain is neuropathic in origin.

NPSI - Neuropathic Pain Symptom Inventory

NPSI is a questionnaire specifically developed to assess and describe different symptomatic manifestations of neuropathic pain. It includes 10 descriptors, each rated on a 0-10 numerical scale according to reported intensity.

The 10 items are grouped into five main categories:

- superficial pain / burning
- deep pain
- paroxysmal pain
- paresthesias / dysesthesias
- allodynia / hyperalgesia

The questionnaire can be further complemented by adding two additional items aimed at evaluating overall pain duration and frequency of episodes of maximum pain intensity ¹⁸⁴.

Taken together, these multidimensional tools allow a more in-depth and articulated characterization of pain, enabling better therapeutic planning and more accurate monitoring of clinical evolution.

3.5 Available treatments

In the context of orofacial pain, therapeutic intervention is generally set according to a multimodal approach integrating pharmacological and non-pharmacological strategies.

In addition to drug therapy, widely used interventions include:

- physiotherapy specifically targeting head-neck-mandibular structures;
- laser therapy with analgesic and anti-inflammatory aims;
- transcutaneous electrical nerve stimulation (TENS) as a non-invasive pain modulation technique;
- use of occlusal splints, often combined with counseling and behavioral re-education;
- hyaluronic acid infiltrations, particularly in temporomandibular joints;
- botulinum toxin injections into selected muscle groups, when appropriate.

Integrating these therapeutic modalities allows more effective pain control, improved orofacial function, and reduced risk of chronicity.

Gnathological therapy

Gnathological therapy refers to the set of treatments aimed at modifying mandibular position and/or the pattern of dental occlusal contacts. In temporomandibular disorders (TMD), the main goal is restoration of physiological mandibular kinematics.

Specifically, treatment of temporomandibular joint (TMJ) disorders should aim to achieve:

- minimal muscle activity and a physiological disc-condyle position at rest;
- coordinated, harmonious activity of masticatory muscles and physiological joint kinematics during extreme mandibular movements and main oral functions (mastication, swallowing, phonation);
- balanced distribution of occlusal contacts ensuring stable intercuspation;

- correct organization of occlusal guidance to allow interference-free chewing cycles.

One etiopathogenetic theory of cervical and masticatory musculoskeletal pain attributes symptom onset to body misalignment due to altered cervical posture, pelvic imbalance, or leg-length discrepancies, capable of generating or perpetuating painful conditions. According to the dental kinesiology approach, such misalignments would cause pathological head protrusion, followed by compensatory mandibular protrusion, with consequent changes in dental intercuspation and onset of muscle pain.

However, as highlighted by Widmer, the hypothesis that musculoskeletal pain is directly caused by anatomical anomalies is based on an ideal theoretical model and would require demonstration of a clear cause-effect relationship to be confirmed. In reality, some postural misalignments are extremely frequent even in asymptomatic individuals. For example, forward head posture, observed in up to 93% of young women, has such prevalence that it can be considered a biologically common variant. Similarly, comparative tomographic studies have shown that variations in mandibular condyle position within the glenoid fossa are comparable in healthy subjects and in patients with TMD.

Another theory links orofacial pain to an alleged misalignment of cranial bones and stress on cranial sutures. This hypothesis is based on empirical observation of musculoskeletal symptom improvement following cranial manipulations. However, based on current knowledge, there is no scientific evidence demonstrating real mobility of cranial bones in adults, nor have randomized controlled clinical trials confirmed effectiveness of such manipulations.

Beyond specific etiopathogenetic theories, gnathological therapy is still often simplistically reduced to the use of an occlusal splint alone. Although this device has numerous clinical indications, its isolated use is rarely sufficient to produce significant and stable improvement of symptoms and clinical signs. Depending on diagnosis, gnathological therapy must therefore be integrated with other interventions, such as

definitive occlusal therapy, functional re-education, and, when indicated, pharmacological treatment.

Occlusal splint therapy

Clinical indication for occlusal splints can be schematically traced to the need to interrupt or modify dental intercuspation in the presence of temporomandibular joint dysfunctions and alterations (intracapsular pathologies), pain syndromes with mainly muscular involvement (extracapsular pathologies), and to protect dentition from wear and occlusal trauma in subjects with parafunctions such as bruxism and clenching.

Occlusal splints can be made for either the upper or lower arch. Upper splints generally provide greater stability and allow easier creation of occlusal stops and guiding planes. Lower splints are less bulky, interfere less with the tongue, and adapt more easily to the occlusal plane, requiring minimal build-ups.

Materials used must be easily modifiable over time, allowing grinding and relining, except for splints intended for long-term stabilization. Occlusal contacts may be partial or total and may have smooth surfaces, only centric indentations, or eccentric guides. It should be emphasized that partial contacts maintained for prolonged periods can cause extrusion of teeth not involved in occlusion. In particular, exclusive anterior contacts can induce upward displacement of the condyle within the glenoid cavity with joint compression phenomena, whereas exclusive posterior contacts can promote downward condylar displacement, resulting in joint distraction.

Clinically, several types of occlusal splints are distinguished:

- disengagement splints;
- anterior repositioning splints;
- stabilization splints;
- posterior pivot splints.

Disengagement splints, also known as relaxation or unloading splints, have smooth surfaces and lack intercuspations. They are indicated mainly in muscular-origin pathologies, as they interrupt intercuspation, allow the mandible to move freely in all directions, eliminate incongruent occlusal contacts, and reduce muscle hyperactivity. These devices are commonly used in initial phases of treatment of craniomandibular disorders to deprogram the neuromuscular system, followed by splints with indentations in therapeutic occlusion or stabilization devices. They are also indicated for control of bruxism and occlusal parafunctions.

Anterior repositioning splints are designed to guide and maintain the mandible in a predetermined therapeutic position through inclined guiding planes. They are used mainly in cases of condyle-disc incoordination such as clicking and mild locking, aiming to promote disc recapture. In such conditions, the mandible is positioned anteriorly in bilateral clicking, or shifted 1-2 mm contralaterally to the click side in unilateral clicking.

Stabilization splints aim to modify and maintain mandibular position and movement trajectories. Their main purpose is to stabilize the mandible in a therapeutic centric position and guide eccentric movements through guiding planes. They are indicated in patients with joint noises, with or without other symptoms, both in early treatment phases and as continuation after disengagement or repositioning splints. These devices cover all occlusal surfaces and incisal edges to prevent unwanted orthodontic effects and distribute occlusal contacts uniformly over the opposing arch, contributing to stabilization of mandibular position. Stabilization splints are also indicated in prolonged therapies for conditions such as non-compensable clicking, chronic locking, TMJ osteoarthritis, neuromuscular disorders, and rheumatoid arthritis.

Posterior pivot splints, through localized occlusal build-ups at molars, disclude anterior teeth, shift condyles caudally with a joint distraction effect, and rotate the mandible counterclockwise. Main indications include unilateral or bilateral joint block due to

loss of the condyle-disc relationship, typically consequent to posterior disc displacement (the most common) or lateral displacement.

Occlusal therapy

Indication for occlusal therapy is limited to patients in whom occlusal alterations are identified as etiological or contributory factors of musculo-articular dysfunction. The main goal is achieving stable intercuspation and creating harmonious functional guides compatible with individual morphological and functional characteristics of the stomatognathic system.

In occlusal adjustment techniques, therapeutic intervention consists of sequential procedures aimed at rebalancing dental contacts. First, occlusal prematurities are eliminated, followed by redistribution of intercuspation bilaterally, ensuring simultaneous contacts of equal intensity. Next, occlusal pathways during retrusion, lateral movements, and protrusion are corrected to eliminate functional interferences. Another goal is correct distribution of incisal contacts over an adequate number of anterior teeth to prevent localized overload and reduce risk of tooth migration, particularly in patients with periodontal disease.

Operational modalities involve minimal, selective corrective interventions on dental surfaces, aimed at restoring physiological functional conditions while avoiding unnecessary structural changes. When reconstructive conservative or prosthetic therapy is indicated, it must be planned and performed to re-establish stable intercuspation and reconstruct adequate functional guides, respecting slopes and individual patient characteristics.

Pharmacological therapy

Pharmacological therapy can significantly contribute to controlling symptoms associated with temporomandibular disorders (TMD), provided the patient is informed that medication has mainly symptomatic aims and does not eliminate the underlying cause of the disorder. Within an appropriate clinical framework, pharmacological management is more effective when integrated with physical measures and causal interventions (functional and/or occlusal), as part of a multimodal therapeutic strategy.

A crucial aspect concerns choice of active ingredient and prescription modality. Because TMD often have a fluctuating course with exacerbations and remissions, "as-needed" prescriptions may favor inappropriate or potentially abusive use, up to development of physical or psychological dependence, especially with opioid analgesics and sedative-anxiolytics. Such drugs may sometimes produce brief periods of subjective well-being, inadvertently reinforcing medication-taking behavior. Moreover, prolonged use is associated with reduced perceived effectiveness and more frequent pain cycles. Therefore, for drugs indicated in TMD treatment, regular dosing for defined periods is generally preferable (e.g., three administrations/day for about two weeks), with clinical reassessment at the end of the cycle.

Main pharmacological classes used include: analgesics, non-steroidal anti-inflammatory drugs (NSAIDs), corticosteroids, anxiolytics, muscle relaxants, tricyclic antidepressants, and local anesthetics. In general, analgesics, corticosteroids, and anxiolytics are indicated mainly in the acute phase; NSAIDs, muscle relaxants, and local anesthetics are used both in acute conditions and persistent presentations; tricyclic antidepressants are used predominantly in chronic orofacial pain management.

Analgesics

Analgesics may play a relevant role in symptomatic treatment of TMD, particularly in mild-to-moderate pain. However, cases in which pain reflects a deep and cyclical mechanism (e.g., recurrent muscular pain) should be distinguished: in such conditions,

analgesia is not an etiological treatment but a temporary support aimed at improving symptom tolerability.

Non-opioid analgesics, although pharmacologically heterogeneous, share effectiveness in controlling mild-to-moderate pain and specific tolerability profiles. Among these, acetaminophen (paracetamol) is often used due to good clinical manageability and relative limitation of adverse effects at therapeutic doses.

Opioid analgesics act on specific receptors in the central and peripheral nervous system, with CNS depressant effects; for this reason, they should be reserved for short periods and for moderate-to-severe pain, with careful assessment of dependence risk. In selected situations, codeine combinations (e.g., with paracetamol) may be considered, always on a regular schedule and for limited duration, to reduce dependence risk.

NSAIDs

NSAIDs are a first-line resource for controlling pain associated with TMD, especially when an inflammatory component is present and in musculoskeletal presentations. To obtain a true anti-inflammatory effect, intake should follow a continuous rather than sporadic regimen, generally for at least two weeks, respecting scheduled times.

Response to NSAIDs varies among individuals; prolonged use is associated with risk of gastric irritation up to ulcer complications, requiring careful assessment of the patient's risk profile.

Corticosteroids

Corticosteroids have strong anti-inflammatory activity, but systemic use in TMD is limited by side effects. They may be indicated in selected conditions such as acute inflammation involving muscles or joints, especially in polyarthritis contexts.

Oral administration often uses regimens with an initially relatively high dose followed by gradual tapering, useful to reduce complications and limit secondary infection risk. Intra-articular infiltration (e.g., hydrocortisone) has been proposed to reduce pain and

functional limitation: although a single infiltration can be effective in some cases, multiple injections have been associated with possible structural damage and should be considered with caution. In certain presentations (e.g., TMJ osteoarthritis or rheumatoid arthritis), favorable clinical outcomes have been reported, particularly for acute symptom control.

Anxiolytics

In patients where emotional stress and anxiety are considered relevant factors in maintaining symptoms, anxiolytics may offer benefit for associated pain and parafunctions, especially nocturnal ones. Diazepam is widely used, but its high dependence potential requires short cycles (typically no more than 10 consecutive days) or use of a single evening dose for limited periods, prioritizing muscle relaxation and reduction of parafunctional activity.

Muscle relaxants

Muscle relaxants have historically been prescribed in TMD; however, their clinical effectiveness is often modest, likely because many muscular pain conditions are not necessarily related to persistent hyperactivity.

Cyclobenzaprine, structurally similar to tricyclics, may reduce muscle pain (especially morning pain) when taken in the evening; an additional daytime dose may be useful in selected cases, but sedation is a frequent adverse effect.

Antidepressants

Tricyclic antidepressants, originally developed for depression treatment, are now established in chronic pain as well. At low doses (e.g., evening amitriptyline), they can exert analgesic effects independent of antidepressant action and are more useful in persistent pain than acute phases. These drugs also influence sleep architecture (reducing awakenings and modifying sleep phases), which may be beneficial in patients with chronic pain, sleep disorders, and some forms of nocturnal bruxism.

Local anesthetics

Local anesthetics have both diagnostic and therapeutic roles. Infiltration into a muscle or suspected joint area as the pain source can produce transient symptom resolution, supporting etiological framing. Therapeutically, infiltration at trigger points can reduce pain until drug metabolism.

In chronic presentations, local anesthetics may help temporarily interrupt the "pain cycle," allowing reduction of central sensitization; consequently, when the nociceptive stimulus reappears, reported pain intensity may be attenuated for a variable period. Commonly used drugs include lidocaine 2% and mepivacaine 3%; for intramuscular infiltrations it is preferable to avoid vasoconstrictors. If longer anesthesia is needed, bupivacaine 0.5% may be indicated, bearing in mind its myotoxicity, which discourages intramuscular injection.

Infiltrative procedures (hyaluronic acid and botulinum toxin)

In addition to systemic pharmacological therapy, management of temporomandibular disorders (TMD) also uses infiltrative procedures more selectively targeting joint and/or muscle structures involved in pain genesis.

For articular-origin pain, intra-articular hyaluronic acid (H.A.) injections are mainly used when problems are directly related to the TMJ. Based on available evidence, H.A. supplementation appears associated with short-term benefits in pain reduction and improvement of functional limitations typical of TMD.¹⁸⁵ Although more effective in reducing joint pain, favorable effects on muscular components have also been reported. Moreover, H.A. supplementation seems to positively impact maximal mouth opening and mandibular mobility in lateral and protrusive movements.

Another infiltrative approach involves botulinum toxin type A (BTX-A), whose effectiveness has been reported in TMD treatment. Freund et al.¹⁸⁶ documented significant pain reduction, increased mouth opening, and improvement in frequently

associated disorders such as bruxism, clenching, and headache. In a study by Bentsianov et al.¹⁸⁷ on TMD patients, a positive response rate of 70% was observed after BTX-A infiltrations into masseter, temporalis, and lateral pterygoid muscles; therapeutic response was defined as pain reduction and/or reduction in pain frequency of at least 50%.

Pharmacokinetic studies indicate persistence of clinical effect up to about 12 weeks, with notable interindividual variability and need in some patients to adjust administration frequency based on optimal dosage. In a subset of subjects, symptomatic benefit may persist beyond expected pharmacokinetic duration, suggesting that BTX-A action may not be exclusively peripheral but could also involve neuromodulatory mechanisms at the central nervous system (CNS) level.¹⁸⁸

Physiotherapy

Physiotherapy includes a set of supportive interventions that, together with etiological treatment, contribute substantially to therapeutic success in many TMJ disorders. Physiotherapy procedures can be grouped into two macro-areas: technical procedures and manual techniques.

Technical procedures

Technical procedures include locally applicable physical modalities such as thermotherapy, cryotherapy, ultrasound, phonophoresis, iontophoresis, electrical stimulation, acupuncture, and low-level laser therapy.

Thermotherapy: application of heat aims to increase local blood perfusion. In some models of muscle pain, reduced tissue perfusion could be an initiating factor; heat-induced vasodilation can reduce symptoms.

Cryotherapy: cold application can reduce pain and promote relaxation of spasming muscles. Ice should be moved over the skin in circular motions without compression; application is brief (about 5-7 minutes) and often repeatable after a warming phase.

Ultrasound: produces deeper heating than superficial thermotherapy and may increase perfusion, connective tissue flexibility, and collagen fiber gliding.

Phonophoresis: uses ultrasound to deliver substances through a transcutaneous way (e.g., topical corticosteroids or other local-use drugs), enhancing their effect.

Iontophoresis: introduces drugs locally via low-voltage electric current, limiting systemic effects; used mainly for anesthetics and anti-inflammatory agents.

Electrogalvanic stimulation (EGS): rhythmic electrical impulses induce muscle contraction and relaxation to reduce spasm and increase perfusion.

TENS: transcutaneous sensory stimulation with low-intensity currents aimed at modulating pain perception. Portable devices may be useful especially in chronic pain patients.

Acupuncture: stimulates antinociceptive circuits and may promote endorphin release, reducing pain in some masticatory apparatus conditions.

Low-level laser therapy: proposed for chronic musculoskeletal pain with hypothesized effects on vascularization and tissue repair; literature reports sometimes favorable results, though not always supported by methodologically robust studies.

Manual techniques

Manual techniques aim to reduce pain and dysfunction and include: soft tissue mobilization, joint mobilization/stretching, and muscle conditioning.

Soft tissue mobilization: superficial massage can modulate pain through cutaneous stimulation; deep massage, performed by experienced operators, aims to improve perfusion, tissue mobility, and treat trigger points, and is often more effective if preceded by moist heat.

Joint mobilization (stretching/distraction): may reduce intra-articular pressure and increase range of motion, also helping manage transient adhesions and disc

mobilization. The maneuver must be gentle and non-painful; pain suggests an inflammatory component and requires suspension or reassessment.

Muscle conditioning: includes active and assisted techniques aimed at functional recovery: passive stretching, assisted stretching, resisted exercises, and postural exercises.

Passive stretching involves slow movements within non-painful limits, avoiding cyclical pain. In intracapsular disorders it may be unrealistic or inappropriate to impose "straight-line" opening trajectories; patients often develop functional compensations that reduce friction or disc interference.

Assisted stretching is progressive, intermittent, and non-traumatic; useful in myofascial pain and post-operative phases to prevent adhesions and stiffness.

Resistance exercises, based on reciprocal inhibition, can promote relaxation of elevator muscles during opening against resistance; they should be stopped if they induce pain.

Postural exercises are proposed especially in subjects with forward head posture; although a contribution of cranio-cervical posture is biologically plausible, evidence is not univocal. As simple and non-invasive exercises, they can nonetheless be integrated when appropriate.

Manual therapy and therapeutic exercise are therefore among the most widely adopted rehabilitation approaches in managing temporomandibular disorders (TMD). Numerous scientific findings support their effectiveness, particularly manual therapy, which can be used for multiple therapeutic aims: improving local blood circulation, reducing muscle spasm, promoting relaxation of periarticular musculature, decreasing adhesions, increasing joint range of motion, and above all alleviating pain symptoms.

Beyond biomechanical and neurophysiological effects, manual therapy has specific relevance in the patient-physiotherapist relationship. Manual contact is often perceived by the patient as a particularly meaningful interaction, capable-through tactile

stimulation-of evoking sensations coherent with care expectations. Such sensory stimulation can strongly activate the nervous system, contributing to analgesic effects and modulation of emotions, affective states, and mental representations related to pain experience.

Within the biopsychosocial model, counseling also represents a potentially effective conservative therapeutic option in managing orofacial pain. This approach mainly targets psychological domains, promoting adaptive behaviors and coping strategies. Education, behavioral change promotion, and avoidance of parafunctions are central elements in determining observed positive effects. Counseling strategies can also modulate pain experience by influencing intensity and perception through cognitive and emotional mechanisms, contributing to more global and integrated management of TMD.

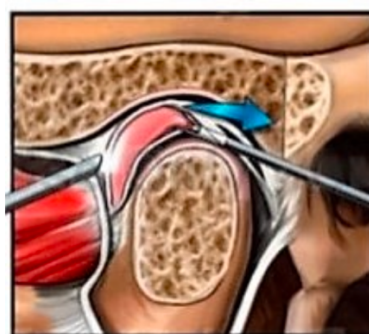
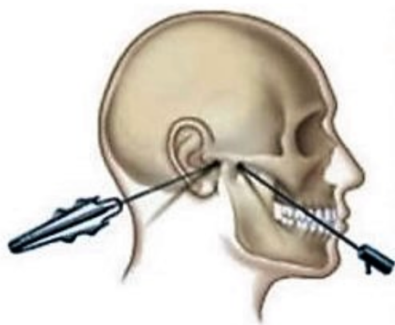
Minimally invasive therapy: arthrocentesis

Arthrocentesis is a minimally invasive procedure described as lavage of the upper joint space of the TMJ, performed under local anesthesia by introducing two needles (inflow/outflow) and irrigating with saline solution or lactated Ringer's. Conceptually derived from arthroscopy, it is characterized by greater technical simplicity and lower invasiveness.

Many clinical observations suggest that a substantial part of the benefit attributed to arthroscopy derives not so much from complex maneuvers (e.g., disc repositioning or fixation) as from the joint lavage effect, which allows: removal of catabolites and inflammatory mediators, reduction of intra-articular hydraulic pressure (also in relation to negative pressure phenomena on the disc), decrease of friction between joint surfaces, and improvement of synovial fluid rheological properties. Consequently, arthrocentesis acts on both chemical and mechanical determinants of joint pain.

For landmark identification, the Holmlund line (from the tragus to the lateral canthus of the eye) is commonly used: the first access point is about 10 mm anterior to the midpoint of the tragus and 2 mm inferior to the line; the second is about 20 mm anterior and 10 mm inferior. It has also been proposed that procedure effectiveness increases by combining lavage with intra-articular hyaluronic acid injection.

Reported outcomes in the literature indicate, across several studies, improvement in maximal mouth opening and pain reduction in patients with intracapsular disorders, with maintenance of benefit at medium- to long-term follow-up. Complications are generally local and transient: preauricular swelling due to tissue imbibition, temporary cutaneous hypoesthesia, and sometimes transient weakness of terminal branches of the facial nerve. There may also be temporary disclusion of ipsilateral molars and premolars due to condylar lowering induced by fluid pressure, as well as temporary persistence of solution in the external auditory canal; such events tend to resolve spontaneously within a few hours. Exceptionally, more lasting sequelae have been described, attributed to puncture injuries of regional nerve structures (e.g., the auriculotemporal nerve).



CHAPTER 4

CAPACITIVE AND RESISTIVE DIATHERMY

Diathermy is a physical procedure that uses radiofrequency. The use of radiofrequency (CRT) is documented in general medicine for pain management, including in the head and neck region¹⁸⁹.

The term radiofrequency describes an electromagnetic wave whose frequency falls between 30 kHz and 300 GHz. It is an electrical and magnetic disturbance that propagates through space, even in a vacuum, carrying energy and information from one point to another. It consists of the simultaneous oscillation of an electric field and a magnetic field around their initial equilibrium position.

Applying an artificial electromagnetic field to biological tissue, appropriately modulated in terms of power, frequency and wavelength, allows the transfer of energy to the cellular substrate, contributing to the restoration of the electrochemical balance altered by the pathological process.

The devices used in Resistive and Capacitive Therapy are based on a specific mode of electromagnetic interaction founded on the physical model of the capacitor¹⁹⁰.

A capacitor is a physical system composed of two conductors separated by an insulating material (or by a vacuum), between which a regime of complete electrostatic induction is established. The two conductors act as the capacitor plates: one becomes positively charged, the other negatively charged and the interposed insulating material is called the dielectric.

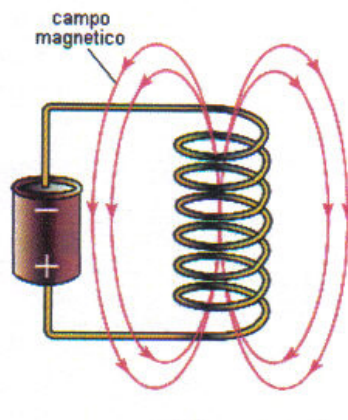
By virtue of its configuration—two plates carrying charges of opposite sign—the capacitor can store electrostatic energy, i.e. the energy associated with the electric field generated by the distribution of charges on the plates. A specific potential difference (ΔV) is established between them.

The fundamental physical quantity that characterizes a capacitor is its capacitance (C), which expresses the system's ability to accumulate electric charge in relation to the applied potential difference. For the same potential difference between plates, a capacitor with greater capacitance can store a greater amount of charge than one with lower capacitance.

Capacitance mainly depends on:

- the shape and size of the plates;
- the distance separating them;
- the characteristics of the interposed dielectric (dielectric constant).

In summary, a capacitor is an electrical device capable of storing and releasing electrostatic energy in a controlled way, based on its geometric characteristics and the insulating medium used.



Devices used in Resistive and Capacitive Therapy transfer energy to tissues through capacitive and resistive mechanisms, producing endogenous heat and enabling the treatment of pathological conditions previously unreachable with conventional methods. Depth effectiveness is achieved without the skin surface being directly involved by energetic projections, thus preserving the integrity of superficial tissues¹⁹¹⁻

Any form of energy—electrical, chemical, or mechanical—can be converted into thermal energy (heat). Heat transfer from one body to another can occur through three main physical mechanisms:

- **Radiation (thermal radiation):** occurs even in a vacuum and is mediated by electromagnetic waves. These radiations, interacting with matter, can induce vibration of atoms and molecules, with consequent conversion of the radiated energy into thermal energy.
- **Conduction:** heat transfer that occurs between solid bodies (or between parts of the same body) in direct contact. It is a molecular phenomenon: the thermal agitation of molecules in the hotter body is progressively transmitted to molecules in the cooler body, leading to heat diffusion.
- **Convection:** typical of fluids, both liquids and gases. Here heat is transferred by the movement of portions of matter from one area to another, producing convective currents that carry thermal energy.

The convective phenomenon is the basic principle underlying more or less traditional hyperthermia systems, including classical hyperthermia and tecartherapy/diathermy methods.

Local heating of a body region not only produces direct effects on the treated area but may also induce indirect effects in surrounding regions, which may have different properties and physiological responses. In this context, it is particularly important to distinguish between superficial heating and deep heating, as they involve different mechanisms of action, heat distribution, and biological consequences.

With superficial heating, vasodilation is mainly observed at the skin level as a homeostatic response to the local temperature increase. This is indirectly associated with moderate vasoconstriction of arterioles and deeper organs, with a consequent—though partial—reduction in their functional activity.

This effect is typical of conventional heating systems based on convection, conduction, radiation, or endogenous heat production. All these methods cause, to varying degrees, a more marked thermal increase at the superficial level (skin and subcutaneous adipose tissue). In the case of endogenous heat production, however, the thermal gradient between superficial and deep tissues may be less pronounced than with other modalities.

In many clinical settings, however, achieving the desired therapeutic effects on the musculoskeletal system requires marked deep vasodilation, obtainable only through significant heating of internal tissues. This must occur while avoiding excessive superficial temperature increases, which could cause unwanted side effects (e.g., skin irritation, pain, intolerance to treatment).

From this perspective, recent technological innovations have made available electromagnetic energy sources capable of inducing selective deep heating, allowing an effective increase in the temperature of internal tissues while providing relative protection to superficial tissues. These systems represent a significant evolution compared with traditional thermotherapy methods, enabling more targeted control of heat distribution for therapeutic purposes.

4.1 Features of Capacitive–Resistive Therapy

Capacitive and resistive therapy (C.R.T.) allows the performance of a form of thermotherapy/hyperthermia consisting of raising the temperature of target tissues and maintaining it, for a defined time interval, at values significantly above baseline levels in the specific body area to be treated.

A distinctive element of this method is the ability to induce endogenous hyperthermia through the controlled application of electromagnetic energy. Electromagnetic energy is also used in more traditional diathermy systems such as shortwave and microwave generators; in those cases, however, it is predominantly a form of radiative energy of a

static nature. In conventional diathermy instruments, the radiated energy is largely absorbed by the more superficial cellular layers, limiting penetration into deeper layers. Consequently, energy density progressively decreases with increasing tissue depth, producing heat mainly at the surface and only partially involving deep structures, which are often the primary target in physiatric and orthopedic practice.

To obtain adequate deep heating with traditional diathermy systems, very high power levels would be necessary, with a real risk of thermal damage or true burns to superficial tissues, while still failing to produce sufficient temperature increases in deep endogenous tissue.

C.R.T. technology overcomes these limitations because it applies energy in radiative form but in direct contact with the body region to be treated via specific electrodes. The use of a frequency of about 500 kHz allows deeper penetration than that achievable with traditional microwaves and shortwaves, promoting heat generation directly within tissues—both superficial and deep—along the path of electromagnetic energy diffusion and its conversion into heat.

The main innovations introduced by C.R.T. can be summarized as follows:

- Targeted energy delivery to the body, capable of modulating cellular and subcellular metabolic thresholds, with bio stimulating and regulatory effects on tissue functions;
- The possibility of modulating the action according to the characteristics of the tissue traversed, thanks to the complementary use of resistive and capacitive modes, which make it possible to selectively direct energy toward tissues with different impedance (more fluid-rich or more resistive);
- Documented therapeutic efficacy also in tissues physiologically more resistant to traditional treatments, such as fibrous, tendinous, or deep joint structures.

Thanks to the peculiarity of endogenous diathermy, C.R.T. can effectively involve deep cellular layers while largely protecting superficial tissues from excessive thermal

overload. This advantage is closely linked to the use of frequencies around 500 kHz, representing an optimal compromise between penetration and safety in biological tissues.

C.R.T. action is localized and confined to the anatomical region where the electrode is applied, with predominant involvement of the tissue layers immediately beneath it.^{195, 196}

C.R.T. can be fully defined as a method that is:

- **Non-invasive**, as treatment is performed using specific handpieces able to transfer energy selectively within tissues, without surgical access or skin penetration.
- **Painless**, as the procedure is configured as an assisted manual treatment during which the delivered energy is perceived mainly as a pleasant, well-tolerated warmth, without pain or significant discomfort.
- **Effective**, as it can integrally involve the different structures affected by the pathological process, promoting muscle relaxation, marked lymphatic drainage with anti-edema action, increased local blood flow, and a reduction in pain symptoms.
- **Long-lasting in its effects**, as it does not limit itself to a merely symptomatic action but stimulates the body's natural physiological responses, promoting regulation and functional rebalancing processes that can persist beyond a single treatment session.

4.2 Application Modes: Resistive and Capacitive

Capacitive mode

In capacitive mode, the operating principle of C.R.T. equipment is comparable to that of an electrical capacitor. The mobile electrode, coated with an insulating material, behaves as one capacitor plate, while the biological tissue - continuously connected to the neutral electrode - acts as the second plate. The use of high voltage and high frequency allows electric charges to overcome the insulating layer (dielectric barrier) and penetrate the tissues beneath the mobile electrode, producing a selective energy transfer.

This mode provides a highly localized treatment, particularly indicated for soft tissues (especially muscles and vasculo-connective tissues). The resulting heating is moderate and confined to the area near the active electrode, with a predominantly superficial and sub-superficial thermal distribution. It is used mainly for skin, connective tissue, and small muscles.

The main effects according to the absorbed energy dose are:

- **Low level:** bio stimulation increases energy transformations at the intracellular level and intensifies cellular metabolism.
- **Medium level:** bio stimulation increases energy transformations at the intracellular level and intensifies cellular metabolism. These processes are associated with improved capillary and precapillary microcirculation, with a consequent increase in endogenous tissue temperature.
- **High level:** activation of local blood flow mechanisms promotes an increase in regional temperature and accelerates lymphatic drainage processes, contributing to improved cellular trophism and optimization of tissue metabolic exchanges.

Resistive mode

During resistive mode, the active electrode is made of a conductive material and is applied directly to the body surface. Electricity, with a frequency of about 480 kHz, crosses the anatomical district between the active and neutral electrodes and then disperses toward the neutral electrode.

Thermal energy is generated due to the resistance offered by biological tissues to the passage of electric charges: the greater the tissue impedance, the greater the heat production. The highest concentration of charges—and consequently the greatest thermal development—occurs in structures with higher electrical resistance along the current path, such as bone, tendons, ligaments, and myotendinous fasciae.

In this way, a true displacement of electric charges is produced within biological tissue, strongly involving deep layers and favoring a relatively homogeneous distribution of the deep endothermic response. This makes it particularly suitable for treating deep osteo-tendinous and capsulo-ligamentous structures. It is indicated for cartilaginous tissues, joints, tendons, fibrotic tissues, and bony structures.

The main effects according to the absorbed energy dose are:

- **Low level:** a low-intensity energy level, characterized by an almost athermal action, can induce a cellular bio stimulation effect, promoting increased oxygen consumption and activation of intracellular energy transformation processes, particularly the conversion of adenosine diphosphate (ADP) into adenosine triphosphate (ATP).
- **Medium level:** increased endogenous temperature activates biological processes involving bone structure, favoring the establishment of an osteo-reparative response.
- **High level:** in addition to the metabolic stimulation induced by energy transformations related to ATP-to-ADP conversion, a marked increase in local blood flow is observed, accompanied by vasodilation and increased lymphatic

drainage. At the same time, modulation of nociceptive sensitivity occurs, resulting in reduced pain perception and improved tissue response to repair processes.

4.3 Physiological effects

Physiological responses induced by C.R.T. include:

- Local vasodilation, with increased blood flow and increased capillary pressure;
- Increased permeability of cell membranes, facilitating intracellular and interstitial exchanges;
- Increased local metabolic reactions, with activation of biochemical processes at the tissue level;
- Modulation of sensory nerve conduction, with a possible effect on pain stimulus processing;
- Increased extensibility and elasticity of tendon, muscle, joint capsule, and scar tissue.

Thanks to these effects, the produced heat stimulates tissue regeneration phenomena and promotes reduction of edema and muscle spasm, contributing significantly to decreased muscle and tendon pain. In general terms, increasing local temperature leads to greater oxygen delivery to tissues, activation of reparative processes, and partial inhibition of pain transmission.

C.R.T., thanks to the possibility of using continuous and pulsed emissions, offers considerable therapeutic versatility, allowing modulation of treatment from completely athermal conditions to progressive hyperthermia levels.

Thermal effects, achievable with both continuous and pulsed emission, allow:

- generating deep heat;
- increasing blood supply;
- increasing local metabolism;
- improving tissue elastic properties;
- inducing a muscle-relaxant effect;
- promoting enzyme interaction and activity;
- reducing pain symptoms.

Athermal effects, obtained mainly with low-intensity pulsed emission, allow:

- reducing edema;
- reducing lymphatic edema;
- promoting functional recovery of superficial tissues.

Among the various effects, one of the most important is increased local blood flow. In this sense, performing the method in association with massage further enhances blood supply to the treated region. To obtain a significant increase in blood flow, it is necessary to reach relatively high temperatures, close to 41 °C—values considered clinically effective but also close to potentially risky thresholds if not properly controlled.

From a biophysical standpoint, the biological effect linked to the attraction and concentration of charges near the active mobile electrode develops selectively at the application site, enabling targeted intervention on areas with a therapeutic indication. The deep layers of biological tissue involved in C.R.T. are affected fairly uniformly by the thermal increase.

This phenomenon is closely related to capacitive and resistive transfer modes. The temperature increase is associated with displacement currents of electric charges and is directly proportional to their intensity. Electrically, the current intensity is mainly determined by the electric field generated by the active electrode, which is controlled by the voltage applied by the generator, rather than by the tissue's resistive properties. Thus, in the area beneath the electrode, current intensity tends to remain almost constant, ensuring homogeneous heat development across the tissue layers traversed.

In capacitive transfer, the current is not carried by direct contact between electrode and tissue, but by the attraction and repulsion movement of the tissue's intrinsic electric charges. Energy transfer, even under athermal or near-athermal conditions, is appreciable also in deep layers. In this context, the presence of metal prostheses is not an absolute contraindication to treatment: metal is an excellent conductor with near-zero resistance and therefore does not develop significant heat. For this reason, there is no real risk of overheating prosthetic materials when C.R.T. is correctly used with appropriately controlled settings¹⁹⁰.

4.4 Contraindications of C.R.T.

The main contraindications for capacitive and resistive therapy are related both to the application of heat and to the presence of sensory deficits, which prevent the patient from perceiving and/or reporting any painful sensation related to the treatment.

Some relative contraindications, such as the presence of metal prostheses, may be partially overcome—after medical evaluation and indication—by predominantly using the capacitive mode and appropriately reduced energy parameters.

Treatment is not recommended:

- in pregnant women;

- in patients with pacemakers or other implanted life-sustaining electronic devices;
- in individuals with Parkinson's disease;
- in the presence of known or suspected neoplasms.

Particular caution is also required when the method is applied at the cervical level, because changes in blood flow in this region may cause mild dizziness or a sense of instability.

If, during treatment, a flare-up of pain is observed, this may sometimes represent a physiological reaction to energy transfer at the pathological site. In such cases it is advisable to:

- reduce output power;
- decrease application duration.

If, despite these measures, symptoms persist or intensify, treatment must be interrupted and the prescribing physician should be consulted for case reassessment.

If skin irritation attributable to the treatment occurs (persistent erythema, burning, blisters, or other skin alterations), C.R.T. must be suspended and the patient should be referred to a physician for appropriate clinical assessment and possible modification of therapy.^{195_197}

CHAPTER 5

STUDY DESIGN

The retrospective and non-randomized study was designed with the aim of evaluating the clinical outcomes of the diathermic stimulation technique using the intraoral Velvet TMJ™ handpiece (Top Quality Medical, Castello, Italy).

Outcome assessment was conducted using a selected set of questions taken from the DC/TMD questionnaire (Diagnostic Criteria for Temporomandibular Disorders), a validated and specific tool for the evaluation of patients affected by temporomandibular disorders (TMD).

The subjects included in the study were identified, based on predefined inclusion criteria, from a database of patients with temporomandibular joint (TMJ) disorders who had undergone treatment with the Velvet TMJ™ device during the period between February 2022 and February 2024.

The retrospective evaluation of the outcomes of rehabilitative treatment for TMJ disorders was approved by the Ethics Committee of Area 2 of Milan (Prot. No. 575-2018; approval date: July 17, 2018) and is part of a broader ongoing research project. The study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki. All patients involved had provided written informed consent prior to the execution of the therapeutic procedures.

5.1 MATERIALS AND METHODS

Inclusion and exclusion criteria

The inclusion criteria comprised patients classifiable within Axes I and II who were affected by temporomandibular joint (TMJ) disorders, in the absence of internal derangement of the articular disc. In cases in which this condition was not clinically detectable, the symptoms were nonetheless predominantly attributable to a dysfunction of muscular origin. Additional inclusion criteria included adequate oral hygiene conditions, the ability to understand the study requirements and to comply with the instructions and schedule of therapeutic sessions, as well as the ability to provide written informed consent prior to the initiation of treatment. Only patients in good general health, as assessed through medical history and physical examination, were included.

The exclusion criteria comprised patients with oncological diseases, individuals with a positive history of alcohol or substance abuse, patients with major psychiatric disorders (including psychosis) or endocrine diseases, individuals unable to maintain adequate standards of home oral hygiene, women who were pregnant or breastfeeding at the time of enrollment, and patients of developmental age.

The study included exclusively patients with a diagnosis of temporomandibular disorders (TMD) in the absence of internal disc displacement; this diagnosis was confirmed by a physician experienced in the field. All patients presented with bilateral TMJ pathology of muscular origin and underwent diathermic treatment using the Velvet TMJ™ handpiece. No additional therapies were associated during the observation period.

Diathermic stimulation technique

Diathermic stimulation was performed using a metal plate designed to deliver radiofrequency waves generated by the handpiece at controlled frequency and

direction. To reduce impedance, a conductive gel was interposed between the metal plate and the skin surface. All patients underwent bilateral extraoral diathermy applications involving the cheek region, the mandibular angle, and the TMJ area.

Prior to application, the treatment area was carefully prepared by verifying skin integrity and ensuring that the skin was clean and dry. Fisiowarm cream (Golden Star Srl, Rome, Italy) was applied to the treatment area to improve electrode contact and glide; throughout the entire procedure, the presence of the cream between the electrode and the skin was maintained in order to optimize treatment effectiveness. Subsequently, the therapeutic protocol was carried out in accordance with the manufacturer's instructions.



The Velvet TMJ™ device handpiece was used to stimulate only one side of the TMJ during each session. Each monopolar resistive application (frequency 1000 kHz) lasted 6 minutes and was performed at 7% power, using the mode referred to as “fracture consolidation treatment.” All treatments were carried out by the same operator, who

followed the parameters provided by the manufacturer for the delivery of capacitive and resistive stimulations, aimed respectively at stimulating tissues with high water content (such as muscle fibers) and those with low water content (such as tendons and ligaments).

Diathermic stimulation sessions were performed on a weekly basis according to the following timeline: T0 (baseline, before the first diathermy simulation), T1 (second session), T2 (third session), T3 (fourth session), and Tf (final simulation). Each patient completed a therapeutic cycle consisting of 20 minutes of capacitive applications and 20 minutes of resistive applications, once per week, for a total of five weeks of treatment. Therapeutic outcomes were evaluated in relation to the different time points, using T0 as the baseline reference for the analysis of clinical improvements.

DC/TMD Questionnaire

At the end of the diathermic treatment, all patients affected by temporomandibular disorders (TMD) in the absence of internal disc displacement completed a set of 47 items selected from the DC/TMD questionnaire (Diagnostic Criteria for Temporomandibular Disorders).

The questionnaire was completed independently and voluntarily during follow-up visits, based on the two assessment axes provided by the DC/TMD model. The standard versions of the questionnaire are originally available in English; however, for the present study, the officially translated versions available were used.

The original DC/TMD questionnaire items were integrated into dedicated evaluation software (www.questionwriter.com), which uses a scoring system consistent with the original scoring tools. The questions were randomly selected by the software and automatically assigned to specific reference categories; subsequently, they were organized into sections according to the international criteria established by the DC/TMD protocol.

For each patient and for each evaluation session, the sequence of questions was further randomized by means of an artificial intelligence–based system, in order to reduce the risk of bias related to the recognition of the investigated domains and to the memorization of previously provided answers. The questionnaire results were processed using a homogeneous scoring system across all phases of the study and for each response (Table 1).

Table 1

ANX: Anxiety disorder score (This category of questions is based on the diagnoses of anxiety disorders in a general sense. Its value represented the degree of perception and psychological suffering and the degree of impact on social and relationship life. This indicator can be influenced by systemic factors and psychiatric diseases as an etiological factor of TMD but is also useful for identifying psychological risk factors in patients subject to occasional stress).
PHQ: Depression Disorder (This score is based on the degree of disability from a depressive disorder. The questions were extracted for mental health and hidden depression which are known as contributory causes of TMD).
DC: Chronic Pain Grading (A score on chronic pain reported in the last 30 days with a VAS scale of 0-10. Chronic pain analysis was used to visualize the intensity of pain, and the risk of chronic pain associated with some forms of TMD. Its value expresses both the subjective level of perception of pain and the possibility that the chronic pain itself is the cause of TMD).
LM: Mandibular Functional Limitation Scale (Score on the functional limitation perceived by the patient to recognize the degree of limitation of the masticatory system. The scale showed the degree of limitation in carrying out the main daily activities. Mild or moderate chewing parafunctions).
Co: Bruxism and score on oral behavior associated with bruxism as perceived by the patient (Involuntary parafunctions such as bruxism or clenching which can be occasional and spontaneous).
G1: Score relating to muscle pain present in the last 30 days. (The TMJ joint is free from obstacles but there may be slight overload phenomena without anatomical damage. In the presence of

limitations in mandibular movement or other daily activities of the mouth, involvement of the nearby pterygoid muscles is considered as muscular pain).

G2A: TMJ sounds (Score on the noise of the temporomandibular joints).

G2B: TMJ blocking (Score on the tendency to block the temporomandibular joints).

The maximum achievable score was set at 26, which was considered indicative of the worst clinical condition; improvement percentages were calculated accordingly for outcome evaluation. All scores were analyzed both as absolute values (with 26 as the maximum value) and as percentage changes relative to baseline (T0). The complete list of DC/TMD questionnaire items related to TMD management used in the present study is reported in Table A1.

Table A1

QUESTIONS	1	2	3	4	5	CATEGORY
In the last 2 weeks, how often have you gotten angry or had worries that you couldn't control?	Never	Sometimes	More than half of the days	About every day	-	ANX
In the last 2 weeks, how often have you had trouble relaxing?	Never	Sometimes	More than half of the days	About every day	-	ANX
Over the past 2 weeks, how often have you had trouble concentrating?	Never	Sometimes	Often	Always	-	ANX
In the last 2 weeks, how often have you had little or too much appetite?	Never	Sometimes	Often	Always	-	ANX
In the last 2 weeks, how often do you find yourself worrying too much about different things?	Never	Sometimes	More than half of the days	About every day	-	ANX

In the last 2 weeks, how often have you felt bored or irritable?	Never	Sometimes	More than half of the days	About every day	-	ANX
In the last 2 weeks, how often have you felt nervous or anxious?	Never	Sometimes	More than half of the days	About every day	-	ANX
In the last 2 weeks, how often have you felt afraid about what might happen?	Never	Sometimes	More than half of the days	About every day	-	ANX
In the last 2 weeks, how often have you found yourself talking or moving so slowly that other people noticed?	Never	Sometimes	Often	Always	-	PHQ
Over the past two weeks, have you felt tired and low on energy?	Never	Sometimes	Often	Always	-	PHQ
In the last 2 weeks, how often did you have low self-esteem or feel like a failure?	Never	Sometimes	Often	Always	-	PHQ
In the last 2 weeks, how often have you been so nervous that you can't sit still?	Never	Sometimes	More than half of the days	About every day	-	PHQ
In the last 2 weeks, how often have you had trouble staying asleep or sleeping too much?	Never	Sometimes	Often	Always	-	PHQ
In the last two weeks, how many times have you been bothered by the thought that it would be better to be dead or to be hurt in some way?	Never	Sometimes	Often	Always	-	PHQ

In the last 2 weeks, how often have you felt little interest or pleasure in doing things?	Never	Sometimes	More than half of the days	About every day	-	PHQ
In the last 2 weeks, how often have you felt down, depressed, or hopeless?	Never	Sometimes	More than half of the days	About every day	-	PHQ
How often do you play musical instruments that require the use of your mouth?	Never	Sometimes	Often	Always	-	CO
How often do you chew gum?	Never	Sometimes	Often	Always	-	CO
How often do you knock, press, or hold your teeth together in addition to chewing?	Never	Sometimes	Often	Always	-	CO
How often do you press your tongue to your teeth or put your tongue between your teeth?	Never	Sometimes	Often	Always	-	CO
How often do you clench or grind your teeth when you are half asleep or during the day?	Never	Sometimes	Often	Always	-	CO
How often do you sleep in a position that compresses your jaw (e.g., on your side or your stomach)?	Never	1-2 nights/month	1-3 nights/week	4-7 nights/week	-	CO
How often do you bite your tongue or play with your tongue or cheeks?	Never	Sometimes	Often	Always	-	CO
How often do you bite objects like pens, pencils, and nails?	Never	Sometimes	Often	Always	-	CO
How often do you bring your hand to your jaw so that it is held in a rigid position?	Never	Sometimes	Often	Always	-	CO

In the last 30 days, how would you rate your facial pain as the worst? Use a 0-10 scale, where 0 is "no pain" and 10 is "the pain is as bad as it gets".	0-10	DC
In the past 30 days, how much has facial pain interfered with your daily activities? Use a 0-10 scale, where 0 is "no interference" and 10 is "unable to carry on activities"	0-10	DC
How do you rate your facial pain right now? Use a 0-10 scale, where 0 is "no pain" and 10 is "the pain is as bad as it gets".	0-10	DC
In the past 30 days, how much has facial pain interfered with your family and social activities? Use a 0-10 scale, where 0 is "no interference" and 10 is "unable to carry on activities"	0-10	DC
Please indicate your level of limitation in opening your mouth wide enough to eat or drink during the last 30 days. Please mark 10 if the activity was completely avoided.	0-10	LM
Please indicate your level of restriction for chewing soft food during the last 30 days. Please, if the activity was completely avoided mark 10.	0-10	LM
Please indicate your level of restriction in swallowing for the last 30 days, please mark 10 if the activity has been completely avoided.	0-10	LM

Please indicate your level of restriction for chewing hard food during the last 30 days, please, if the activity has been completely avoided mark 10.						
	0-10					LM
In the last 30 days, have you had headaches that include the temple area?	Never	A single episode	Often, about 1 day/week	Always, a few days a month without pain	-	G1
In the last 30 days, have you had pain when chewing hard or challenging foods?	Never	Sometimes, but I can finish chewing	Often, I need to be careful when chewing	Always, I have to avoid foods that are too demanding	-	G1
In the last 30 days, have you had pain when opening your mouth or moving your jaw forward or sideways?	Never	Sometimes	Often	Always	-	G1
In the last 30 days, have you had pain or discomfort in your mouth upon waking up?	Never	Sometimes	Often	Always	-	G1
In the last 30 days, how long have you had pain or discomfort in your jaw, temples, or elsewhere?	No pain or suffering	The pain comes and goes	The pain is always present		-	G1
In the past 30 days, have certain activities such as kissing, talking, or yawning increased your pain or discomfort?	Never	Sometimes	Often	Always	-	G1

In the last 30 days, when you open your mouth wide, have you experienced loud noises or clicking sounds, or has your mouth ever felt stuck so you couldn't close it completely?	No, never	Sometimes, just a big fool but without pain or limitations	Often a big crash with pain and limitations that slowly resolve	All the time, my jaw locks and I have to reposition it by hand	-	G2A
In the last 30 days, have you experienced any clicking or popping in your jaw joints when you move or use your mouth?	No, never	Sometimes but not always	Often	Always	-	G2A
In the last 30 days, has your jaw ever locked so that you couldn't open it fully, even for a moment, and then unlocked so that it came back free?	Never blocked	Yes, spontaneous unlocks	Yes, it unlocks with the help of my hands	No, never unlocked again	-	G2B
Has your jaw ever been locked or stuck so much that it interfered with your ability to eat?	Never	Sometimes	Often	Always	-	G2B
Have you ever had your jaw lock or hold, even for a moment, so much so that you couldn't open it fully?	Never	Sometimes	Often, also with joint pain	Always, I'm stuck right now too	-	G2B
Observe your mouth opening and closing movement by looking at yourself in the mirror. Indicate the mouth-opening path that best matches your natural movement.	Straight and linear movement	Slight lateral skids but substantially linear	Deviated to one side and then returned aligned to maximum opening	Permanently deviated to one side	-	G2B

Measure your mouth-opening ability
 using your fingers as a reference as
 shown in the figure.

5
 fingers

4 fingers

3 fingers

2 fingers

1 finger

G2B

Statistical methods

Statistical data analysis was performed using GraphPad Prism software, version 5.03 (GraphPad Software, San Diego, CA, USA). Pre- and post-treatment scores were collected at five distinct time points: T0 (baseline, before the first diathermy simulation), T1 (second session), T2 (third session), T3 (fourth session), and Tf (final diathermy session).

The maximum index score ($n = 26$) represented the worst clinical condition, whereas a value of zero indicated the absence or minimal presence of temporomandibular joint (TMJ) disorders, pain, and functional limitations. A reduction in the score compared with the baseline value was interpreted as indicative of an improvement in the clinical condition of the TMJ. In order to allow data normalization and comparison across different time intervals, results were converted into percentages.

Trends over time in percentage scores, as well as mean values and standard deviation (SD), were analyzed. A $p\text{-value} \leq 0.05$ was considered statistically significant. Given the small sample size and the non-normal distribution of the data, a non-parametric approach was adopted, applying the Friedman test for repeated measures followed by Dunn's post-hoc test for multiple comparisons.

5.2 Results

A total of ten patients (8 women and 2 men), aged between 22 and 50 years, were included in the study in accordance with the inclusion criteria. Over the observation period, a general trend toward improvement was observed across all analyzed categories, with a progressive reduction in mean scores over time. No adverse events were recorded in any of the treated subjects.

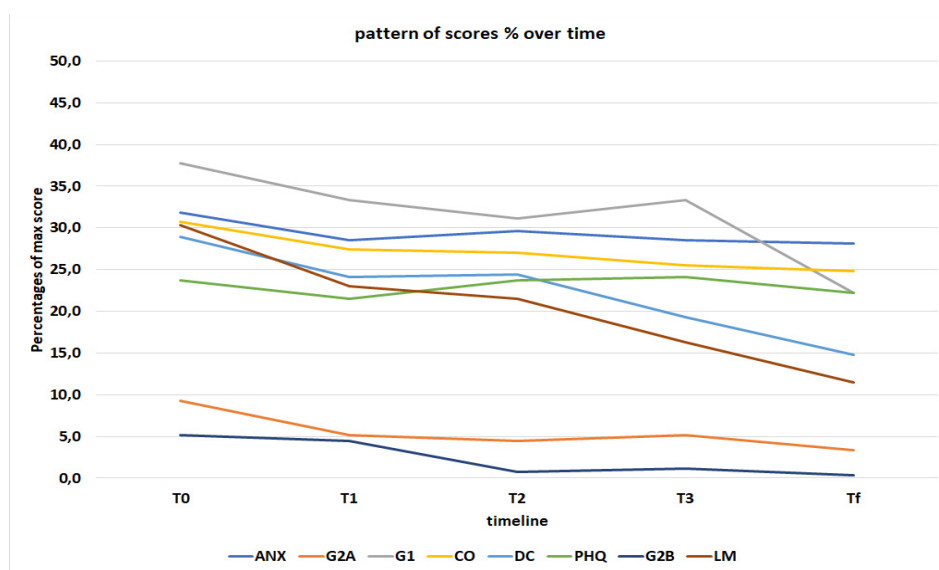


Figure 1. The graph shows the mean values (MV) and standard deviations (SD) for percentage scores of various health parameters related to temporomandibular disorder (TMD) across five-time points during a five-week diathermy treatment. These parameters include anxiety (ANX), TMJ sounds (G2A), muscle pain (G1), oral behaviors (CO), chronic pain grading (DC), depression disorder (PHQ), TMJ blocking (G2B), mandibular functional limitation (LM).

As illustrated in the graph, several clinical parameters—including anxiety (ANX), TMJ joint noises (G2A), muscle pain (G1), degree of chronic pain (DC), TMJ locking (G2B), and mandibular functional limitation (LM)—showed a decreasing trend over time, suggesting an overall improvement in the patients' clinical conditions.

Table 2

Time	Health Parameters															
	ANX		G2A		G1		CO		DC		PHQ		G2B		LM	
	MV	SD	MV	SD	MV	SD	MV	SD	MV	SD	MV	SD	MV	SD	MV	SD
T0	31.85	18.33	9.26	9.44	37.78	14.05	30.74	13.97	28.89	23.86	23.70	12.49	5.19	10.06	30.37	20.57
T1	28.52	16.66	5.19	7.03	33.33	13.52	27.41	12.85	24.07	18.66	21.48	14.38	4.44	8.52	22.96	16.64
T2	29.63	16.47	4.44	4.88	31.11	14.42	27.04	14.19	24.44	21.25	23.70	16.49	0.74	2.34	21.48	16.27
T3	28.52	14.40	5.19	5.58	33.33	13.18	25.56	13.69	19.26	16.45	24.07	17.31	1.11	3.51	16.30	13.09
Tf	28.15	12.37	3.33	4.77	22.22	11.04	24.81	10.34	14.81	11.84	22.22	15.71	0.37	1.17	11.48	10.25
<i>p-value</i>	0.9686		9.1665		0.3731		0.7783		0.4736		0.999		0.2805		0.0216*	

ANX: Anxiety disorder score, PHQ: Depression Disorder, DC: Chronic Pain Grading, LM: Mandibular Functional Limitation Scale, Co: Bruxism and score on oral behavior associated with bruxism, G1: Score relating to muscle pain present in the last 30 days, G2A: TMJ sounds, G2B: TMJ blocking, *significant.

In particular, the anxiety score (ANX) showed a moderate reduction from 31.85% at T0 to 28.15% at Tf. TMJ joint noises (G2A) exhibited a more marked decrease, dropping from 9.26% to 3.33%, suggesting a potential positive effect of diathermy on joint-related symptoms. Similarly, muscle pain (G1) and the degree of chronic pain (DC) showed a progressive reduction over time, with muscle pain decreasing substantially from 37.78% to 22.22%, indicating a relevant symptomatic relief.

Parameters related to associated headaches (CO) and the Patient Health Questionnaire (PHQ) remained largely stable throughout the treatment period, showing only minor fluctuations. This finding suggests a lower sensitivity of these variables to diathermy or a possible influence of confounding factors.

The G2B parameter, indicative of TMJ locking, showed a marked reduction, decreasing from 5.19% at T0 to 0.37% at Tf, suggesting a significant improvement in joint mobility. Among all the variables analyzed, only mandibular functional limitation (LM) showed a statistically significant reduction ($p = 0.0216$), indicating a relevant improvement in mandibular function following diathermic treatment. Specifically, the LM score decreased from 30.37% to 11.48%.

The remaining parameters, despite showing a favorable trend, did not reach statistical significance, likely due to the limited sample size and the high interindividual variability in treatment response. Mean values, standard deviations, and p-values for each parameter are reported in Table 2.

5.3 Discussion

First-line treatment of temporomandibular disorders (TMD) is mainly based on non-invasive approaches, particularly physical therapy, which includes exercises aimed at improving mandibular mobility, muscle strengthening, and reduction of pain-related symptoms. Additional commonly adopted strategies include the use of occlusal devices (splints or bite appliances), which help stabilize the mandible, reduce muscle tension, and prevent bruxism. In association with these interventions, supportive pharmacological therapy comprising analgesics, muscle relaxants, and anti-inflammatory drugs is frequently prescribed.

Although pharmacotherapy may provide short-term symptomatic benefit, it is not free from potential side effects, such as gastrointestinal disturbances, and the use of opioids requires particular caution due to the risk of dependence. When conservative measures fail, minimally invasive procedures, such as arthrocentesis or intra-articular corticosteroid injections, may offer temporary symptomatic relief. Surgery represents the last therapeutic option and is reserved for the most severe cases of TMD, such as structural abnormalities or degenerative joint diseases; however, these interventions are associated with a higher risk of complications, including infections and neurological damage, with possible repercussions on mandibular function.

Among emerging non-invasive options, several modalities of instrumental physical therapy including ultrasound, radiofrequency, electrical stimulation, and diathermy have been proposed to reduce edema, relieve pain, and improve joint function. These techniques appear to provide moderate clinical benefit and are often used in combination with other conservative treatments, such as cognitive-behavioral therapy. In the present pilot study, diathermic stimulation using radiofrequencies of 500 kHz and 1 MHz was employed. Diathermy represents a form of endogenous thermotherapy that exploits controlled increases in tissue temperature for therapeutic purposes; it is painless and non-invasive and potentially effective in the treatment of tissues of the stomatognathic system and TMD.

The mechanism of action of diathermy is based on an increase in local temperature, which promotes enhanced blood flow, reduction of inflammation, and decreased muscle stiffness, thereby facilitating healing processes. In the context of TMD, this therapeutic approach aims to decompress the temporomandibular joint, relax the involved musculature, and contribute to improved mandibular positioning. However, improper application may carry the risk of thermal tissue injury. Moreover, diathermy has some contraindications, including the presence of pacemakers, metallic implants, and skin conditions (such as acne, infections, or neoplasms), which limit its widespread use.

Most available studies on diathermy and ultrasound therapy focus on general musculoskeletal disorders, whereas evidence related to TMD treatment remains limited and often characterized by insufficiently robust methodology. In a comparative study, Talaat et al. evaluated the effectiveness of shortwave diathermy versus ultrasound therapy and a pharmacological combination of methocarbamol and acetylsalicylic acid, finding comparable results between diathermy and ultrasound, but a superiority of diathermy over pharmacological therapy in post-treatment pain relief. Although there is evidence supporting the short-term effectiveness of diathermy in muscle relaxation and pain reduction, long-term effects on TMJ function and patient-reported outcomes remain unclear.

A recent systematic review and meta-analysis conducted by Pollet et al. analyzed the effectiveness of electromagnetic diathermy including shortwave electrical transfer, microwave therapy, and resistive-capacitive techniques in the treatment of musculoskeletal disorders. The review, which included 68 studies involving a total of 4,892 patients, evaluated pain, function, quality of life, and adverse effects as primary outcomes. The results suggest that diathermy may contribute to pain reduction, functional improvement, and enhanced quality of life, likely through increased blood flow, improved tissue oxygenation, and possible modulation of the central nervous system.

Previous studies by Adly et al., Gray et al., and Dhanasekaran et al. also assessed the effectiveness of shortwave diathermy in the management of TMJ disorders, highlighting faster symptomatic relief compared with other therapeutic modalities. In line with these findings, the present study observed a progressive reduction in muscle pain and degree of chronic pain over time, with muscle pain decreasing from 37.8% to 22.2%, indicative of a significant analgesic benefit.

In a clinical study, Adly et al. evaluated the reduction of joint clicking through stethoscope auscultation and observed a more marked decrease in the placebo group than in the treated group immediately after therapy²¹⁰. The authors concluded that shortwave diathermy is effective in controlling TMJ pain but less so in treating joint noises. In contrast, in the present study, the G2A parameter showed a significant reduction, decreasing from 9.26% to 3.33%, suggesting a potential favorable effect of diathermy on TMJ joint sounds.

Furlan et al. conducted an integrative review on the use of superficial thermotherapy in the management of TMD, analyzing application modalities, duration, treated areas, and clinical benefits²¹⁸. Although moist heat was the most commonly used technique and was associated with improvements in pain and mandibular function, the included studies showed considerable methodological heterogeneity and frequent use in combination with other conservative treatments. Moreover, superficial thermotherapy presents limitations related to application complexity, reduced adherence, and potentially lower effectiveness compared with more advanced techniques such as diathermy²¹⁸. In the present study as well, diathermy demonstrated overall favorable results.

The results of this pilot study highlighted a progressive improvement across all analyzed categories, with a statistically significant reduction in mandibular functional limitation (LM; $p = 0.0216$), suggesting improved mandibular mobility and reduced functional restrictions. Although the other parameters did not reach statistical significance, they nonetheless showed favorable trends, supporting the potential role

of diathermy in the management of TMD, in agreement with findings reported in the literature²⁰⁸⁻²¹¹.

A distinctive feature of the present study is its focus on patient-reported outcomes, assessed using a specific questionnaire, which allows integration of the subjective perception of therapeutic benefit into the overall evaluation of diathermy effectiveness. This approach may contribute to the optimization of therapeutic protocols and to defining the role of diathermy within the multidisciplinary management of TMD.

This study has some limitations. First, the evaluation was limited to the short term and conducted on a small sample size, consistent with the pilot nature of the study, whose primary aim was to collect preliminary data useful for planning future prospective investigations. In addition, the included patients presented with TMD in the absence of internal disc displacement, and outcomes were mainly based on self-assessment measures. Further limitations derive from the retrospective, non-randomized, non-blinded design and the absence of a control group. Future studies should be designed as prospective, randomized, double-blind, controlled trials in order to confirm these preliminary findings and provide more robust scientific evidence.

CHAPTER 6

CONCLUSIONS

The aim of this study is to evaluate the efficacy of diathermic stimulation in the treatment of pain associated with temporomandibular disorders (TMD), mandibular mobility, and the related impact on the patients' quality of life. Taking into account the limited availability of studies on this subject and the absence of standardized therapeutic protocols, the results obtained should be interpreted as preliminary ones. Nevertheless, they may help to fill certain knowledge gaps regarding the methods of diathermy application and the short-term duration of its therapeutic effects.

The results do not allow the claiming of a complete resolution or definitive remission of symptoms, nor do they find a cure for temporomandibular disorders. However, based on the data collected, diathermic stimulation may be considered a potentially useful intervention for reducing both pain and functional limitations of the temporomandibular joint. This clinical improvement could lead, in the short term, to a benefit of the quality of life of patients affected by TMD without internal displacement of the articular disc.

Further clinical studies with larger sample sizes and longer follow-up periods are required in order to confirm the effectiveness and safety of diathermy in the treatment of temporomandibular joint disorders. In particular, future research should include patients with more severe clinical conditions and assess the use of diathermy both as a standalone treatment and in combination with other conservative therapies in order to allow for a more comprehensive clinical evaluation and generalized results. Moreover, the use of control groups and additional objective assessment tools could help strengthen the scientific evidence supporting this therapeutic modality.

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