

# Description of low-threshold mechanisms of consciousness and occlusal dysesthesia: diagnosis and therapy through active functional rehabilitative repositioning bite

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## Abstract

The regulatory factors controlling the low-threshold mechanisms of consciousness within the stomatognathic system at the central nervous level are not yet fully understood, as they are difficult to evaluate. Neuroplasticity, neuro signatures, engrams, and praxis patterns—which manage complex regulatory pathways potentially involved in occlusal hypervigilance, dysesthesia, and possibly even in mandibular clenching and bruxism—remain only partially known. As a result, these phenomena are often overlooked or mismanaged. Clinicians tend to focus more easily on visible malocclusions and muscular tension, often choosing invasive occlusal treatments that address peripheral symptoms. This article examines the low-threshold mechanisms of consciousness in the stomatognathic system that can affect the tone of masticatory muscles and change the interocclusal rest space to improve clinical understanding. Special emphasis is given to occlusal hypervigilance and occlusal dysesthesia, helping to achieve more accurate diagnoses and suitable therapeutic plans to avoid unnecessary or invasive treatments. A therapeutic protocol backed by previously published clinical trials is suggested. This protocol is minimally invasive, conservative, and designed to be cost-effective, in accordance with current international guidelines.

**Keywords:** TMD diagnosis, TMD therapy, dysesthesia, phantom bite, neuroplasticity, occlusal hypervigilance

## Introduction

### *Low-threshold mechanisms of consciousness*

The masticatory system mainly operates below conscious awareness. Most routine rhythmic functions—such as chewing, swallowing, phonation, and parafunctional activities—are performed automatically. Likewise, the mandibular rest position, characterized by a natural interocclusal free space maintained for about 23.3 hours each day, is regulated subconsciously, although it remains dynamically changing, as shown by Palla in 2001.

Maximal occlusal contact between dental arches should only occur during swallowing and mastication. Under normal physiological conditions, this contact is limited to an estimated total duration of about 30 minutes per 24-hour cycle. This regulation helps prevent overload of the stomatognathic structures. The free space and vertical dimension (VD) result from peripheral and central neuromuscular adaptations that involve neuroplasticity and neurosignatures.

This automatic activity, mostly occurring without conscious thought, is controlled by central processes and sensory inputs from neuromuscular, joint, and periodontal receptors. These sensory signals travel to the cortex via the principal sensory nucleus and the mesencephalic nucleus of the trigeminal nerve, then through the



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## How to Cite

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thalamus to reach the cognitive cortex, which can influence voluntary muscle responses. Additionally, thalamic signals are relayed to the reticular formation, extrapyramidal system, medulla oblongata, spinal cord, and limbic system.

If stress-related, psychological, hormonal, biochemical, or neurological factors disrupt the neuromuscular system—factors that may vary among individuals and within the same individual over time, depending on genetic and constitutional predispositions—the duration of dental contact may extend beyond physiological limits, leading to a reduction in the interocclusal rest period. This shift results in increased and repetitive proprioceptive occlusal stimulation, which activates intensified afferent neural flow to central neurological structures.

Prolonged exposure to such intense afferents causes structural changes in the central nervous system, boosting receptor density and neurotransmitter activity. This process, known as central sensitization and neuroplasticity, results in the formation of new adaptive neural pathways—“preferred channels” or “neuro signatures”—as described by Melzack (2001–2004).

These neuroadaptive patterns become ingrained as unconscious motor schemes, prolonging the duration of interocclusal contact. They operate similarly to praxis movements—automatic gestures coordinated toward specific goals that do not need conscious supervision. According to Marbach (1976), the repeated contact of cusps and fossae during mastication creates a neuroimprint within the neuromatrix or engram. As a result, these neuroimprints and praxis patterns become deeply rooted, reinforcing repetitive behaviors that extend dental contact beyond normal physiological limits.

As discussed by Behr and colleagues, neuroplasticity might explain certain aspects of bruxism, wakeful clenching, and sleep bruxism. This highlights the automatic nature of these parafunctional behaviors, which usually occur below conscious awareness and only become clinically relevant when they cause symptoms or pathological signs.

### *Neurophysiological, Chemical, and Central Metabolic Mechanisms*

Among the known mechanisms capable of causing the changes described above persistently and unconsciously, amplification plays a key role in the continuous repetition of neural impulses originating from peripheral receptors. These afferent signals can trigger central chemical and metabolic processes, leading to structural changes such as neuronal hyperexcitability—or central sensitization—and the increasingly recognized phenomenon of neuroplasticity. Multiple neurochemical pathways underlie these processes, including those mediated by metabotropic transmembrane receptors (indirect synapses), which differ from ionotropic transmembrane receptors (direct synapses). Metabotropic receptors exert their effects through intracellular second messengers, triggering biochemical reaction cascades beyond the cell membrane. Although slower because of the need for intracellular activation, these receptors cause more lasting changes in membrane permeability and cellular

metabolism. These effects support the structural modification of neurons, resulting in an increase in both the number and diversity of receptors and neurotransmitters. Similar mechanisms are involved in chronic conditions such as persistent pain, heterotopic pain, myofascial syndromes, fibromyalgia, and hyperalgesia.

In contrast, ionotropic receptors linked to direct synapses quickly open ion channels and create faster but short-lived effects because they do not induce intracellular metabolic changes.

Thus, strong and sustained action potentials arriving from peripheral receptors—transmitted through the main sensory and mesencephalic trigeminal nuclei to the thalamus and then to the reticular formation, extrapyramidal system, medulla oblongata, spinal cord, and limbic structures—produce significant central responses.

Thalamic connections with the reticular formation, including the locus coeruleus (a key modulator of noradrenaline and other neurotransmitters), influence muscle spindle receptors to regulate postural tone. This interaction increases the tonicity and contraction of the mandibular elevator muscles, potentially worsening clenching behavior. Such neuromuscular overload may activate the ascending reticular activating system (ARAS), leading to functional and, over time, structural damage to masticatory muscles.

Furthermore, the thalamus closely communicates with the hypothalamus, a diencephalic structure responsible for neuroendocrine and visceral-motor regulation, along with the amygdala and hippocampus, both components of the limbic system. The limbic system controls emotions, mood, and self-awareness, and it influences behavior. It also integrates autonomic and neuroendocrine functions, creating a complex interplay with emotional, perceptual, cognitive, and behavioral domains.

The extrapyramidal connections are especially complex: neural information travels from the thalamus to the striatum, which organizes motor impulses and sends them to downstream extrapyramidal nuclei. These nuclei control descending pathways that manage rhythmic and semi-automatic movements performed below conscious awareness, such as arm swinging during walking, facial expressions, chewing, speech, swallowing, posture, and overall body alignment.

### *Neurofunctional Implications and Responses*

Under normal physiological conditions, the human body maintains a high threshold for adaptation, keeping automatic functions below conscious awareness. For example, when occlusion is suddenly changed due to dental procedures—whether minor or extensive—the proprioceptive system quickly alerts the cortex and other relevant neural structures. As a result, these occlusal changes are immediately perceived consciously.

However, if the occlusal alteration aligns with the individual’s usual function and the person has adequate physiological control of interocclusal space and mandibular rest position—without signs of clenching, bruxism, or other neuromuscular issues—adaptation is

likely to happen within a variable period. Function will then return to its automatic regulation below conscious awareness. Yabushita (2006), who demonstrated the remarkable plasticity of masseter muscle spindles after changes in the occlusal vertical dimension (OVD), showed this capacity for adaptation.

Conversely, in individuals with preexisting dysfunctions—such as impaired autoregulation of the mandibular rest position or reduced interocclusal space due to parafunctional activities like clenching or bruxism—even minor occlusal changes may not be accommodated. In such cases, increased sensitivity to the occlusal change may develop, often focused on perceived “prematuities” or “interferences.” This ongoing perception causes discomfort and obsessive attention to the occlusion, resulting in occlusal hypervigilance.

Occlusal hypervigilance can reflexively activate the neuromuscular system, worsening proprioceptive and muscular discomfort and leading to a subjective feeling of occlusal disharmony—called occlusal discomfort or simply discomfort. The patient may repeatedly try to find a more comfortable mandibular position but fails to do so.

In individuals with impaired autoregulation of interocclusal space and low adaptive thresholds, the continued presence of this condition can lead to three compounding phenomena, further worsening the clinical situation.

### **1. Central Sensitization**

At the central level, the area of sensory hypersensitivity expands due to sustained and intense afferent stimuli from peripheral receptors. These stimuli generate prolonged action potentials that facilitate neural transmission along the trigeminal pathways, inducing central chemical and metabolic modifications—including glial activation and potential changes in COMT metabolism. These changes contribute to neuroplastic remodeling and may involve activation of the reticular formation, particularly the locus coeruleus, which enhances spindle receptor sensitivity and further increases mandibular elevator muscle tone and contraction.

### **2. Peripheral Muscular Hyperactivity**

Due to central changes and the sensation of occlusal instability, the masticatory muscles may show increased basal tone, leading to hyperactivation of the gamma efferent system. This can cause muscular contractures or co-contractions and, in some cases, mandibular clenching, a subtle form of bruxism where the masticatory muscles stay contracted without dental contact. Additionally, localized muscular contractures may prevent the patient from avoiding contact at rest, decreasing the free interocclusal space and worsening symptoms—potentially reinforcing the persistence of parafunction.

### **3. Psychological Consequences**

Due to central sensitization, even minor stimuli are perceived as excessively intense. This increased sensory perception of occlusal discomfort—where microscopic contact is seen as a significant problem—combined with peripheral neuromuscular hyperactivity, can trigger

psychological effects. Patients might develop secondary anxiety, worsened discomfort, and worsening symptoms over time. This biopsychosocial burden can lead to secondary depression, adding psychological distress to physical suffering.

## **Occlusal Dysesthesia**

Occlusal dysesthesia—also known as phantom bite in the literature—can be partly understood as a sign of occlusal hypervigilance. It usually occurs after conservative dental treatments, prosthetic procedures, or occlusal adjustments, even when no objectively detectable occlusal issues are present. The patient, however, reports an intense, persistent sense of occlusal discomfort lasting at least six months, despite dental interventions being performed accurately and precisely. Fortunately, this condition is relatively rare.

Patients with occlusal dysesthesia frequently present not only localized occlusal complaints but also exhibit concomitant neuromuscular disturbances, often accompanied by psychological symptoms and anxiety-related components. 1976 Marbach described the condition as “the patient’s perception of an irregular occlusion even when the dentist finds no objective irregularity.” Due to the absence of observable occlusal alterations, Marbach coined the phantom bite, aligning the condition with somatoform disorders and phantom phenomena.

In 2012, Hara and colleagues systematically reviewed the literature, analyzing 84 publications, of which only 13 were included in the final synthesis. Only four of these studies applied occlusal dysesthesia diagnostic criteria (DC). These findings defined the condition as “a persistent complaint of occlusal discomfort for at least six months, without any identifiable physical abnormalities related to the occlusion, dental pulp, periodontium, masticatory muscles, or temporomandibular joints. The symptoms cause significant distress and drive patients to seek repeated dental treatments.”

In the same study, Hara et al. described therapeutic strategies reported in the literature, including psychotherapy, cognitive-behavioral therapy (CBT), splint therapy, and the prescription of antidepressants or anxiolytic medications. However, most evidence was derived from limited case reports.

The etiology of occlusal dysesthesia remains poorly understood. It likely involves an interplay of psychological variables—such as underlying anxiety, depressive tendencies, and somatoform disorders—possibly coexisting with functional receptor-level changes or central neurological dysfunction. Diagnostic evaluation must pay careful attention to patient history. When a patient reports persistent sensations of “prematuities” or “interferences” lasting longer than six months, with no objective confirmation, occlusal dysesthesia should be considered.

These patients frequently present lengthy clinical histories marked by unsuccessful treatment attempts and may attribute therapeutic failure to the perceived incompetence of their providers. Sometimes, they may pursue legal action, further complicating the therapeutic relationship.

## Scientific Foundations of Neuroplasticity and Myelinogenesis

Neuroplasticity refers to the central nervous system's (CNS) intrinsic capacity to permanently alter its structural and functional organization in response to external stimuli. This adaptive capability enables the formation of new synaptic connections and the reorganization of neural circuits.

The foundational work in this field began with the pioneering studies of Rita Levi-Montalcini, Nobel Laureate in 1986, who identified the nerve growth factor (NGF). Her research demonstrated that neural cells possess the capacity to differentiate and regenerate—an unprecedented discovery at the time, as the CNS had long been considered immutable.

Eric Kandel made subsequent advances and was awarded the Nobel Prize in Medicine in 2000 for his work elucidating the neurochemical basis of neuroplasticity. Kandel's experiments on the sea slug *Aplysia*—an organism with a simple nervous system consisting of only 24 sensory neurons and 6 motor neurons—demonstrated that repeated stimuli can induce structural changes in the neural pathways. Specifically, the repeated stimulation of a specific body area triggered the withdrawal of a gill in a protective reflex. This reflex results from the activation of particular genes that promote the growth of new synaptic connections between sensory and motor neurons.

Thus, repetitive stimulation has been shown to induce gene expression and foster the development of novel neuronal connections. Furthermore, beyond synaptic reorganization, repeated practice and motor activity stimulate oligodendrocytes—a type of glial cell—to produce myelin. Myelination dramatically increases the conduction velocity of action potentials along axons by as much as a factor of one hundred. A myelinated neural circuit can function up to 3,000 times more efficiently than its unmyelinated counterpart.

Accordingly, repetitive stimuli, training exercises, and functional movements are crucial for neuroplastic remodeling and optimizing neuromotor efficiency through enhanced myelinogenesis. Within the stomatognathic system, such principles directly apply to therapeutic strategies, especially those aimed at re-educating and reorganizing neuromuscular coordination.

## Functions of Bite Appliances

The therapeutic effects of a bite appliance depend heavily on its structural design and mode of use. The distinction between passive splints and active functional repositioning devices is of paramount importance.

### 1. Passive Mandibular Deprogramming Bite (e.g., Michigan Splint, Full-Coverage Devices)

These devices are generally not intended to alter mandibular position significantly. Consequently, they do not affect leverage mechanics or muscle force vectors. Their main functions include:

- A. Increasing vertical dimension
- B. Eliminating occlusal interferences
- C. Modifying habitual occlusal schemes
- D. Redistributing occlusal loads

### E. Protecting parafunctional activity

### 2. Mandibular Repositioning Bite (e.g., Farrar, NTI-tss, Lingual Ring RIPARA)

These appliances promote an anterior repositioning of the mandible, influencing the muscular leverage system and neuromuscular activation patterns. In addition to the five functions listed above, they perform the following:

- F. Anterior repositioning of the mandible, modifying leverage, and muscular force vectors
- G. Modulation of muscular activity
- H. Reduction of muscular tension related to clenching

### 3. Active Functional Rehabilitative Bite

When the bite is used to perform specific cognitive-functional and motor training exercises, it no longer serves merely a passive role. It becomes a cognitive-active positional reeducation device, stimulating peripheral and central systems. This enables the bite to exert:

- I. Active functional reeducation effects via exercises
- J. Promotion of new motor and functional patterns
- K. Stimulation of neuroplastic mechanisms
- L. Placebo and suggestive effects, enhancing compliance and cognitive engagement

### 4. Other potential central neurological effects not yet fully elucidated

An essential component of this protocol involves the tongue, which serves as a dynamic proprioceptive-perceptual organ. By engaging the tongue in exercises and positioning strategies, additional neuromuscular and postural rebalancing can be achieved, particularly concerning the suprahyoid and infrahyoid muscular chains.

Therefore, any therapeutic plan addressing these clinical pictures should integrate a functional rehabilitative repositioning bite. This device should not be viewed solely as a tool to disengage occlusion or protect against parafunctions, but as a neuromuscular and cognitive reeducation instrument. Only after a comprehensive deconditioning and neurofunctional retraining phase should further therapeutic or occlusal interventions be considered.

## Patient Education and Functional Reeducation Strategy

The therapeutic protocol must focus on deconditioning and active functional reeducation to reestablish neuromuscular and cognitive balance. Initial occlusal modifications should be strictly avoided.

The clinician has a pivotal role in guiding the patient through four fundamental reeducation tasks designed to restore proprioceptive awareness, cognitive control, and neuromotor coordination:

### 1. Maintaining Dental Disclusion

The patient must be taught to keep the teeth apart as much as possible to cognitively and neuromotorly reorganize the occlusal-free space. This instruction aligns with cognitive-behavioral therapy (CBT) principles,

which target habitual parafunctional behaviors and enhance self-regulation.

## 2. Masticatory Muscle Relaxation

Patients should learn to relax the masticatory muscles, including using self-massage techniques. By palpating the masseter and temporalis muscles, patients can become aware of muscle tone and distinguish between contraction and relaxation. This facilitates biofeedback and reduces neuromuscular hyperactivity.

## 3. Elevated Tongue Posture

Instruction should include maintaining the tongue tip against the “spot,” i.e., the palatal area immediately behind the upper central incisors. This positioning enhances cognitive-perceptual, proprioceptive, and neuromotor self-control and supports correct orofacial posture.

## 4. Execution of Functional Exercises

Patients should perform a structured set of cognitive-functional and motor exercises involving:

- Mouth opening and closing with the tongue maintained against the spot, promoting muscle stretching and sensorimotor reeducation;
- Swallowing actions while keeping the tongue on the spot, without clenching the teeth (aided by small sips of water if necessary);
- Phonation exercises (e.g., counting aloud to 100 or engaging in spoken tasks) with the tongue on the spot and the bite in place, enhancing cognitive-sensorimotor integration;
- Deep nasal inhalations followed by brief breath-holding and oral exhalation, all performed with the tongue in its elevated position and without occlusal contact.

## The Role of the Bite in Reeducation

These four core therapeutic actions are initially challenging, as the patient must actively counteract muscular hyperactivity and the compulsion to seek habitual occlusal contact. In this context, the functional rehabilitative repositioning bite—such as the RIPARA lingual ring—becomes a critical therapeutic tool.

This bite device is not merely protective; rather, it facilitates:

- The peripheral biomechanical functions (actions 1–8);
- The central biochemical and neurofunctional effects (actions 9–13);
- A platform for cognitive-functional exercises carried out daily with the bite in place.

The RIPARA lingual ring has been used successfully at the authors’ clinical center for several years. It is specifically designed to support and enhance the therapeutic actions described above.

## Summary of the Therapeutic Actions of the Bite Device

The functional rehabilitative repositioning bite performs synergistic actions encompassing both peripheral biomechanical effects and central neurofunctional stimu-

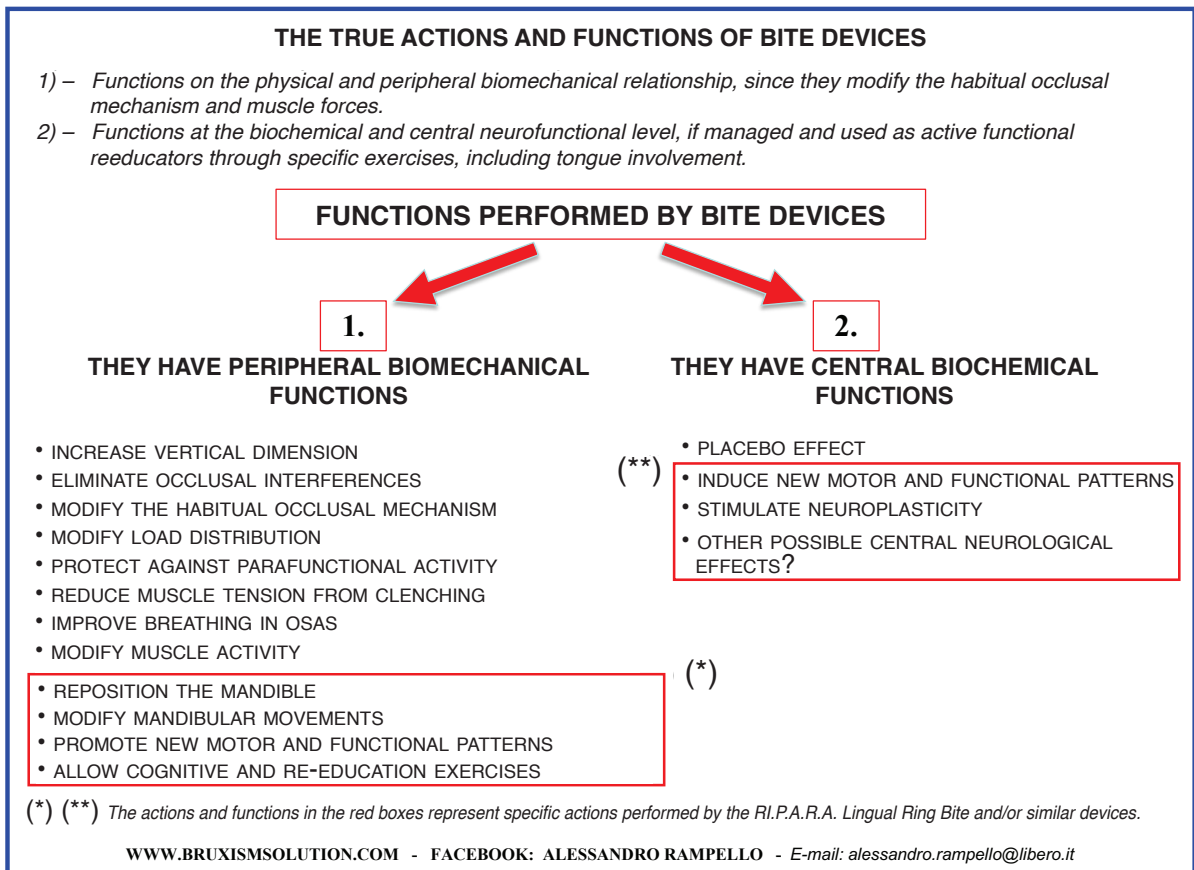
lation, promoting global neuromuscular reorganization.

The following are the principal actions:

1. Disengagement from habitual occlusal contacts and modification of vertical dimension.
2. Distraction from habitual occlusal awareness exploits cognitive and proprioceptive mechanisms and potential placebo effects, contributing to systemic deconditioning and gradual neuromuscular realignment.
3. Anterior mandibular repositioning with condylar forward translation produces muscle elongation and alters muscular force vectors. This is achieved in synergy with anterior and superior tongue positioning, facilitating hyoid bone elevation. Such repositioning helps counteract the paradoxical increase in occlusal vigilance sometimes reported with conventional bite splints (as noted by Reeves and Merrill) by fundamentally altering the stomatognathic neuromuscular equilibrium.
4. Protection from awake clenching and nocturnal bruxism is considered repetitive masticatory muscular activities, varying according to circadian phenotypes.
5. Psychotherapeutic action, through enhanced patient understanding and engagement in cognitive-behavioral therapy, aims to mitigate stress-related and psychological comorbidities.
6. Neuro-occlusal-muscular proprioceptive reeducation, supported by the soft silicone material of the bite, continuously stimulates dental proprioceptors to facilitate fine motor adjustments and modulate occlusal pressure through anterior propulsion and vertical dimension changes.
7. Neuromuscular coordination training requires the patient to maintain the bite device in place using tongue posture, not occlusal pressure. This dynamic “balancing act” makes the tongue a central element of control, transforming it into a functional and proprioceptive tool.
8. Execution of active exercises with the lingual ring in place, including:
  - Mouth opening and closing with the tongue held on the spot, promoting both muscle stretching and sensorimotor reeducation;
  - Swallowing actions without lifting the tongue or clenching the teeth;
  - Phonation (e.g., counting aloud, reading) with the tongue stabilized against the spot and without tooth contact;
  - Controlled nasal breathing exercises with the tongue in the elevated position and the bite in place.

## Conclusions

Understanding the neurophysiological role of the central nervous system (CNS) in controlling neuromuscular, neurosensory, neuroendocrine, and proprioceptive-occlusal-articular processes is essential. These interactions may contribute to poorly understood conditions such as occlusal hypervigilance, occlusal dysesthesia, neuromuscular disorders, mandibular clenching, and bruxism, all of which can



**Table 1.** Overview of the functions of bite appliances: Peripheral Biomechanical Actions and Central Biochemical Effects.

significantly affect the stomatognathic system, the temporomandibular joint (TMJ), and the patient’s biopsychosocial health.

A thorough understanding of these mechanisms is vital for proper diagnosis and for implementing noninvasive, conservative, and cost-effective treatment strategies aligned with international best practices. This method supports the restoration of allostasis and homeostasis while preventing unnecessary and potentially harmful occlusal modifications.

Contemporary gnathological therapy should extend beyond occlusal analysis or static night-time splints. Instead, it should include functional and neurofunctional approaches, using bite devices as protective tools

during sleep and as active reeducation aids during the day to reorganize neuromuscular behavior and central processing.

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**Figure 1 – Tongue Positioning on the “Spot” with the Ri.P.A.Ra. Lingual Bite** Frontal view of the device in place: the patient performs the exercise by maintaining the tongue tip against the palatal area just behind the upper central incisors (“spot”), which is essential for functional reeducation. The device promotes anterior mandibular repositioning and neuromuscular activation.



**Figure 2 – Active Functional Exercise with the Ri.P.A.Ra. Bite** The patient performs dynamic exercises (mouth opening/closing, swallowing, phonation) with the device in place and the tongue positioned on the spot. The goal is to promote neurofunctional reorganization through proprioceptive stimulation and cognitive-motor control.

Details of how to insert and position the tongue on the “spot” using the Lingual Ring Bite Ri.P.A.Ra. This is a functional rehabilitative repositioning device designed to alter the posture of the mandible, condyles, tongue, and ultimately, the entire vector and force system of the masticatory muscles. It is not just a simple interocclusal spacer. The patient should not use the Ri.P.A.Ra. Bite passively or statically at night; instead, they should actively perform specific exercises with the device in place, as outlined in the text.

## References

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1. Jankelson B, Hoffman GM, Hendron JA; The physiology of the stomatognathic system - JADA, 46, 375-386, 1952 <https://doi.org/10.14219/jada.archive.1953.0070>
2. Jankelson B.: Physiology of the human dental occlusion - J.A.D.A., 50, 664-680, 1955 <https://doi.org/10.14219/jada.archive.1955.0130>
3. Woda A, Pionchon P, Palla S; Regulation of Mandibular Postures: Mechanisms and Clinical Implications. Critical Reviews Oral Biology & Medicine 2001; 12: 166-178. <https://doi.org/10.1177/10454411010120020601>
4. Jankelson B; Neuromuscular aspects of occlusion: Effects of occlusal position on the physiology and dysfunction of the mandibular musculature. Dent Clin North Am 1979; 23: 157-168. [https://doi.org/10.1016/S0011-8532\(22\)03188-3](https://doi.org/10.1016/S0011-8532(22)03188-3)
5. Fayz F, Eslami A; Determination of occlusal vertical dimension: a literature review JPD 1988; 59: 321-323 [https://doi.org/10.1016/0022-3913\(88\)90182-5](https://doi.org/10.1016/0022-3913(88)90182-5)
6. Garnick J, Ramfjord SP; Rest position: An electromyographic and clinical investigation. JPD 1962; 12, 895-911 [https://doi.org/10.1016/0022-3913\(62\)90043-4](https://doi.org/10.1016/0022-3913(62)90043-4)
7. Ismail YH, George WA; The consistency of the swallowing technique in determining the occlusal vertical relation in edentulous patients. JPD 1968; 19, 230-236 [https://doi.org/10.1016/0022-3913\(68\)90083-8](https://doi.org/10.1016/0022-3913(68)90083-8)
8. Swerdlow, H. Vertical dimension literature review. JPD 1965; 15, 241-247 [https://doi.org/10.1016/0022-3913\(65\)90093-4](https://doi.org/10.1016/0022-3913(65)90093-4)
9. Landa JS; The free-way space and its significance in the rehabilitation of the masticatory apparatus. JPD 1952; 2, 756-779 [https://doi.org/10.1016/0022-3913\(52\)90104-2](https://doi.org/10.1016/0022-3913(52)90104-2)
10. Konchak PA, Thomas NR, Lanigan DT, Devon RM; Free-way space measurement using mandibular kinesiograph and EMG before and after TENS. Angle Orthod. 1988; 58:

- 343-350.11. Johnson A, Wildgoose DG, Wood DJ; The determination of freeway space using two different methods; J. Oral. Rehabil. 2002; 29: 1010-1013 <https://doi.org/10.1046/j.1365-2842.2002.00950.x>
12. Lundeen HC, Gibbs CH; Mandibular movement and its clinical significance. Phillip J Restaur Zahnmed. 1987; 4: 87-97.13. Sessle BJ, Adachi K, Avivi-Arber L, Lee J, Nishihara H, Yao D, Yoshino K.; Neuroplasticity of face primary motor cortex control of orofacial movements; Arch Oral Biol. 2007; 52: 334-337. <https://doi.org/10.1016/j.archoralbio.2006.11.002>
14. Melzack R; Pain and the neuromatrix in the brain. J Dent Educ. 2001; 65: 1378-1382. <https://doi.org/10.1002/j.0022-0337.2001.65.12.tb03497.x>
15. Melzack R,Coderre TJ, Katz J, Vaccarino AL; Central neuroplasticity and pathological pain. Ann N Y Acad Sci. 2001; 933: 157-174. <https://doi.org/10.1111/j.1749-6632.2001.tb05822.x>
16. Melzack R; Evolution of the neuromatrix theory of pain. The Prithvi Raj Lecture: presented at the third World Congress of World Institute of Pain, Barcelona 2004. Pain Pract. 2005; 5: 85-94. <https://doi.org/10.1111/j.1533-2500.2005.05203.x>
17. Dellow PG, Lund JP; Evidence for central timing of rhythmic mastication. J Physiol 1971; 215: 1-13. <https://doi.org/10.1113/jphysiol.1971.sp009454>
18. Nozaki S, Iriki A, Nakamura Y; Localization of central rhythm generator involves cortically induced rhythmic masticatory jaw-opening movement in the guinea pig. J. Neurophysiology 1986; 55: 806-825. <https://doi.org/10.1152/jn.1986.55.4.806>
19. Nozaki S, Iriki A, Nakamura Y; Trigeminal premotor neurons in the bulbar parvocellular reticular formation participating in induction of rhythmic activity of trigeminal motoneurons by repetitive stimulation of the cerebral cortex in the guinea pig. J. Neurophysiology 1993; 69: 595-608. <https://doi.org/10.1152/jn.1993.69.2.595>
20. Johnson, L. R. (2012). Essential medical physiology. 3rd Edition: 2003, English Book edition.21. Lund JP, Sasamoto K, Murakami T, Olsson KA; Analysis of rhythmic jaw movements produced by electrical stimulation of motor-sensory cortex of rabbits. J Neurophysiol. 1984; 52: 1014-1029. <https://doi.org/10.1152/jn.1984.52.6.1014>
22. Achari NK, Thexton AJ; Diencephalic influence on the jaw reflex in the cat. Arch Oral Biol. 1972; 17: 1073-1083. [https://doi.org/10.1016/0003-9969\(72\)90182-3](https://doi.org/10.1016/0003-9969(72)90182-3)
23. Hikosaka O; GABAergic output of the basal ganglia. Prog Brain Res. 2007; 160: 209-226. [https://doi.org/10.1016/S0079-6123\(06\)60012-5](https://doi.org/10.1016/S0079-6123(06)60012-5)
24. Okeson J P; Il trattamento delle disfunzioni e dei disordini temporomandibolari. 2015, Italian Book: VII Ediz. Martina BO.25. Molina M, Valsesia C, Pisa W, Magnano G, Pirazzi Maffiola A, Meersseman G, Miglio C; Concetti Fondamentali di Gnatologia Moderna. 1988 Milano: ILIC Editrice;26. Shaw SM, Martino R. The normal swallow:

- muscular and neurophysiological control. *Otolaryngol Clin North Am.* 2013; 46: 937-956. <https://doi.org/10.1016/j.otc.2013.09.006>
27. Schwartz LL; Pain associated with the temporomandibular joint. *J. Am. Dent. Assoc.* 1955; 51: 394-397. <https://doi.org/10.14219/jada.archive.1955.0223>
  28. Schwartz LL; A temporomandibular joint pain-dysfunction syndrome; *J Chronic Dis.* 1956; 3: 284-293. [https://doi.org/10.1016/0021-9681\(56\)90123-0](https://doi.org/10.1016/0021-9681(56)90123-0)
  29. Schwartz LL, Cobin HP; Symptoms associated with the temporomandibular joint; a study of 491 cases. *Oral Surg Oral Med Oral Pathol.* 1957; 10: 339-344. [https://doi.org/10.1016/0030-4220\(57\)90156-1](https://doi.org/10.1016/0030-4220(57)90156-1)
  30. Laskin DM; Etiology of the pain-dysfunction syndrome. *J Am Dent Assoc* 1969; 79: 147-153. <https://doi.org/10.14219/jada.archive.1969.0234>
  31. Graber G; Neurologic and psychosomatic aspects of myoarthropathies of the masticatory apparatus. *ZWR* 1971; 80: 997-1000.32. Molin C; From bite to mind: TMD - a personal and literature review. *Int J Prosthodont* 1999; 12: 279-288.33. Türp J C, Greene C S, Strub J R; Dental occlusion: a critical reflection on past, present and future concepts. *J Oral Rehabil.* 2008; 35: 446-453. <https://doi.org/10.1111/j.1365-2842.2007.01820.x>
  34. Manfredini D, Lobbezoo F Relationship between bruxism and temporomandibular disorders: a systematic review of literature from 1998 to 2008. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.* 2010; 109: 26-50. <https://doi.org/10.1016/j.tripleo.2010.02.013>
  35. Türp J C, Schindler H; Review Article: The dental occlusion as a suspected cause for TMDs: epidemiological and etiological considerations; *J Oral Rehabil.* 2012; 39: 502-512. <https://doi.org/10.1111/j.1365-2842.2012.02304.x>
  36. Chisnoiu AM, Picos AM, Popa S, Chisnoiu PD, Lascu L, Picos A, Chisnoiu R; Factors involved in the etiology of temporomandibular disorders - a literature review; *Clujul Med.* 2015; 88: 473-478. <https://doi.org/10.15386/cjmed-485>
  37. Manfredini D, Perinetti G, Stellini E, Di Leonardo B, Guarda-Nardini L; Prevalence of static and dynamic dental malocclusion features in subgroups of temporomandibular disorder patients: Implications for the epidemiology of the TMD-occlusion association; *Quintessence Int.* 2015; 46: 341-349.38. Manfredini D, Lombardo L, Siciliani G; Temporomandibular disorders and dental occlusion. A systematic review of association studies: end of an era? *J Oral Rehabil.* 2017; 44: 908-923. <https://doi.org/10.1111/joor.12531>
  39. Behr M, Hahnel S, Faltermeier A, Bürgers R, Kolbeck C, Handel G, Proff P; The two main theories on dental bruxism; *J Ann Anat.* 2012; 194: 216-219. <https://doi.org/10.1016/j.aanat.2011.09.002>
  40. Ferrucci M, Fornai F. Anatomia funzionale della formazione reticolare nel tronco encefalico dell'uomo. *Manuali Medicina Pisa University Press* 03/201741. Nieuwenhuys R, Voogd J, van Huijzen C, Papa M; *Il sistema nervoso centrale*; Springer Int Pub Sci 2010. <https://doi.org/10.1007/978-88-470-1140-3>
  42. Anastasi G, Balboni G, De Caro R; *Anatomia Umana. Trattato vol. III*, Milano, Edi. Ermes, 2010.43. Willard FH, Holt JA; *Neuroanatomia Medica*; Futura Publishing Society; 2012.44. Janig W, McLachlan EM, Baron R; *Il Sistema Neurovegetativo Regolatore delle funzioni integrative nelle dinamiche corporee*. Futura Publishing Society; 2013.45. Lemon RN, Johansson RS, Westling G; Corticospinal control during reach, grasp, and precision lift in man. *J Neurosci.* 1995; 15: 6145-6156. <https://doi.org/10.1523/JNEUROSCI.15-09-06145.1995>
  46. Kakei S, Hoffman DS, Strick PL; Muscle and movement representations in the primary motor cortex. *Science.* 1999; 24; 285: 2136-2139. <https://doi.org/10.1126/science.285.5436.2136>
  47. Yabushita T, Zeredo JL, Fujita K, Toda K, Soma K; Functional adaptability of jaw-muscle spindles after bite-raising. *J Dent Res.* 2006; 85: 849-853. <https://doi.org/10.1177/154405910608500914>
  48. Suzuki A, Stern SA, Bozdagi O, Huntley GW, Walker RH, Magistretti PJ, Alberini CM; Astrocyte-neuron lactate transport is required for long-term memory formation. *Cell.* 2011; 144: 810-823. <https://doi.org/10.1016/j.cell.2011.02.018>
  49. Manfredini D; *Il bruxismo nella clinica odontoiatrica*; Quintessenza Edizioni. 2015
  50. Lobbezoo F, Ahlberg J, Glaros AG, Kato T, Koyano K, Lavigne GJ, de Leeuw R, Manfredini D, Svensson P, Winocur E; Bruxism defined and graded: an international consensus. *J Oral Rehabil.* 2013; 40: 2-4. <https://doi.org/10.1111/joor.12011>
  51. Davis PJ, Reeves JL, Hastie BA, Graff-Radford SB, Naliboff BD; Depression determines illness conviction and pain impact: a structural equation modeling analysis. *Pain Medicine.* 2000; 1: 238-246. <https://doi.org/10.1046/j.1526-4637.2000.00032.x>
  52. Imeri L, Mancia M; *Testo atlante di fisiologia umana: Neurofisiologia*. Milano: Casa Editrice Ambrosiana. 2006.53. Tsukiyama Y, Yamada A, Kuwatsuru R, Koyano K; Biopsychosocial assessment of occlusal dysaesthesia patients. *J Oral Rehabil.* 2012; 39: 623-629. <https://doi.org/10.1111/j.1365-2842.2012.02317.x>
  54. Marbach JJ; Phantom bite. *Am J Orthod.* 1976; 70: 190-199. [https://doi.org/10.1016/S0002-9416\(76\)90319-5](https://doi.org/10.1016/S0002-9416(76)90319-5)
  55. Marbach JJ; Phantom bite syndrome. *Am J Orthod.* 1978; 135: 476-479. <https://doi.org/10.1176/ajp.135.4.476>
  56. Marbach JJ, Varoscak JR, Blank RT, Lund P; Phantom bite. Classification and treatment. *J Oral Rehabil.* 1983; 49: 556-559 [https://doi.org/10.1016/0022-3913\(83\)90322-0](https://doi.org/10.1016/0022-3913(83)90322-0)
  57. Hara ES, Matsuka Y, Minakuchi H, Clark GT, Kuboki T; Occlusal dysesthesia: a qualitative systematic review of the epidemiology, aetiology and management. *J Oral Rehabil.* 2012; 39: 630-638. <https://doi.org/10.1111/j.1365-2842.2012.02300.x>
  58. Dworkin SF, Leresche L; Research diagnostic criteria for temporomandibular disorders: review, criteria, examinations and specifications, critique. *J Craniomandib Disord.* 1992; 6: 301-355
  59. Schiffman E, Ohrbach R, Truelove E, Look J, Anderson G, Goulet JP, List T, Svensson P, Gonzalez Y, Lobbezoo F, Michelotti A, Brooks SL, Ceusters W, Drangsholt M, Ettlin D, Gaul C, Goldberg LJ, Haythornthwaite JA, Hollender L, Jensen R, John MT, De Laat A, de Leeuw R, Maixner W, van der Meulen M, Murray GM, Nixdorf DR, Palla S, Petersson A, Pionchon P, Smith B, Visscher CM, Zakrzewska J, Dworkin SF; Int RDC/TMD Consortium Network, Int Ass for Dental Research; Orofacial Pain Special Interest Group, Int Ass for the Study of Pain. Diagnostic Criteria for Temporomandibular Disorders (DC/TMD) for Clinical and Research Applications: recommendations of the International RDC/TMD Consortium Network\* and Orofacial Pain Special Interest Group. *J Oral Facial Pain Headache.* 2014; 28: 6-27 <https://doi.org/10.11607/jop.1151>
  60. Steenks Mh, Türp JC, De WiJer A; Reliability and Validity of the Diagnostic Criteria for Temporomandibular Disorders Axis I in Clinical and Research Settings: A critical appraisal.; *J Oral Facial Pain Headache.* 2018; 32: 7-18 <https://doi.org/10.11607/ofph.1704>
  61. Rampello A, Falisi G, Panti F, Di Paolo C. A new aid in TMD Therapy: the Universal Neuromuscular Immediate Relaxing appliance "UNIRA". *Oral Implantol (Rome)* 2010 Jan;3 (1):20-32. Epub Nov 19. 2010