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Muscle physiology in orthodontics

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Abstract
Craniofacial development and orthodontic treatment outcomes are significantly influenced by the physiology and functional dynamics of orofacial muscles. This review explores the structural, physiological, and reflex-based roles of skeletal muscle in orthodontics. Key themes include muscle tissue types, contraction mechanisms, neuromuscular coordination, and therapeutic implications. Additionally, the role of muscle training, neuromuscular disorders, and orthodontic appliance interaction with musculature are discussed. A multidisciplinary understanding of muscle physiology enhances orthodontic diagnosis, appliance design, and post-treatment stability.

Keywords: Muscle physiology, orthodontics, craniofacial muscles, muscle fatigue, neuromuscular reflexes

Introduction

Physiology is the branch of biology dealing with the functions and vital processes of living organisms, their parts and organs. The origin of the word (from Greek ~ucns = nature and Myos = rule, or maybe logic) comes close to the Chinese meaning of physiology = logic of life. The development of physiology is closely related to the development of anatomy. To cite J.W. Goethe, "Funktion ist Form in Tatigkeit gedacht" ("function is form in action") [1].

The practice of contemporary orthodontics involves an understanding and application of biomechanical principles that enable the orthodontist to achieve planned and visualised dentofacial outcomes. The craniomandibular function is determined by the complex and interrelated components comprising the morphology and biomechanics of the muscles, joints, dentition and the neuromuscular system indicating the close association between dental occlusion and the outcome of the action of the muscles of mastication, i.e., the functional jaw movements.

While orthodontic forces guide tooth movement, it is the adaptive capacity of muscle and bone that governs long-term outcomes. Malocclusion, facial asymmetry, and relapse are not merely dental phenomena, but often stem from muscular imbalances and dysfunctions. This review highlights the biological and clinical aspects of muscle physiology relevant to orthodontic practice, emphasizing the necessity of integrating muscle-based diagnostics and therapy into treatment planning.

Molecular Mechanisms of Muscle Contraction

The term “contraction”, as used in muscle physiology, does not necessarily mean ―shortening. It refers to the activation of the force-generating sites within muscle fibers. Some of the mechanisms through which force and tension are generated causing muscular contraction are as follows –

a. Sliding-Filament Mechanism/Ratchet Mechanism

According to H.E. Huxley and Hanson [2], and A.F Huxley and Niedergerke [3], when force generation produces shortening of a skeletal muscle fiber, the overlapping thick and thin filaments in each sarcomere move past each other, propelled by movements of the cross-bridges. During this shortening of sarcomeres, there is no change in the lengths of either the thick or thin filaments. This is known as the sliding-filament mechanism or the Ratchet mechanism of muscle contraction which has been prevailing since the mid-1950s.

During shortening, each myosin cross-bridge attached to a thin filament actin molecule moves in an arc much like an oar on a boat. This swivelling motion of many cross-bridges forces the thin filaments attached to successive Z lines to move toward the center of the sarcomere, thereby shortening the sarcomere.

One stroke of a cross-bridge produces only a very small movement of a thin filament relative to a thick filament. This motion continues as long as a muscle fiber remains activated, thereby resulting in large displacements of the filaments. The sliding during muscle contraction occurs when the myosin heads bind firmly to actin, bend at the junction of the head with the neck, and then detach. This power stroke depends on the simultaneous hydrolysis of ATP. Myosin-II molecules are dimers that have two heads, but only one attaches to actin at any given time. (Fig 1)

The sequence of events that occur between the time a cross-bridge binds to a thin filament, moves, and then is set to repeat the process is known as a cross-bridge cycle. Each cycle consists of four steps –

1. Attachment of the cross-bridge to a thin filament
2. Movement of the cross-bridge producing tension in the thin filament
3. Detachment of the cross-bridge from the thin filament and
4. Energising the cross-bridge to allow re-attachment

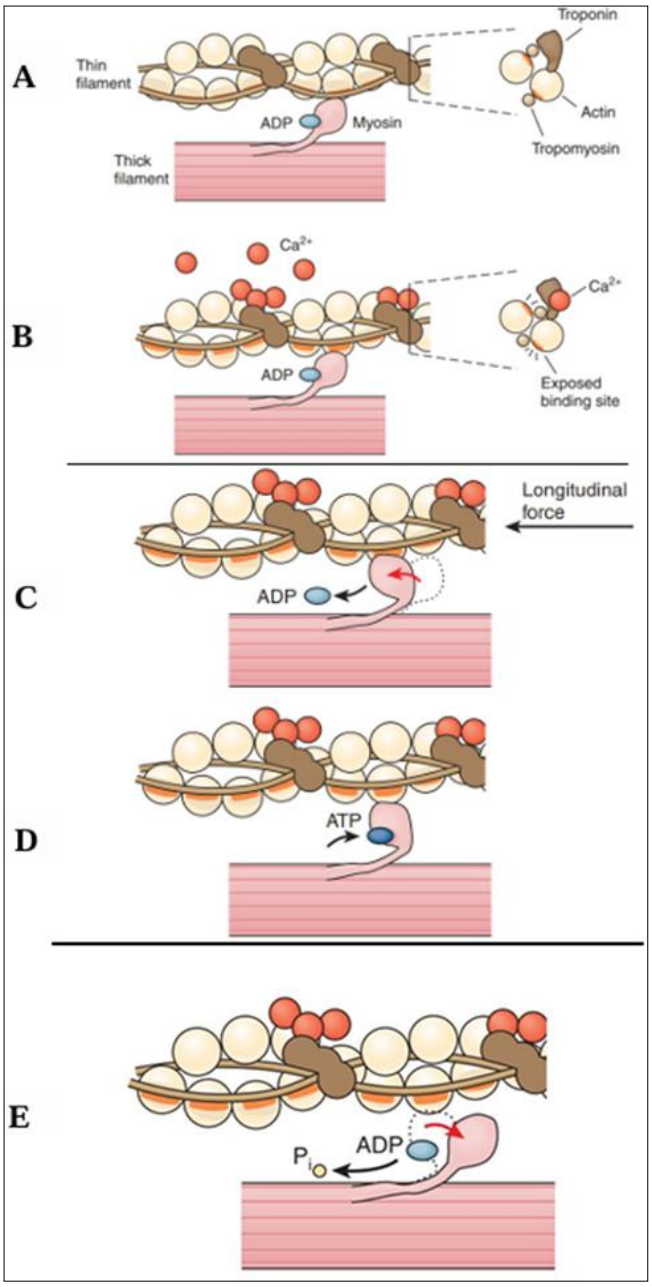

Fig 1: Power stroke of Myosin in Skeletal Muscle

(A) At rest, myosin heads are bound to adenosine diphosphate. (B) Ca 2+ bound to the Troponin–tropomyosin complex. (C) Myosin heads rotate, move the attached actin and shorten the muscle fiber, forming the power stroke. (D) At the end of the power stroke, ATP binds to a now exposed site. (E) ATP is hydrolysed into ADP and inorganic phosphate (Pi)

Role of Troponin, Tropomyosin and Calcium ions in contraction [4].

Tropomyosin is a rod-shaped molecule composed of two intertwined polypeptides with a length approximately equal to that of seven actin monomers. Chains of tropomyosin molecules are arranged end to end along the actin filament. These molecules partially cover the myosin-binding site on each actin monomer, thereby preventing the cross-bridges from contacting actin.

Each tropomyosin molecule is held in this blocking position by smaller globular proteins known as Troponin. Troponin, which has three subunits namely I inhibitory, T-tropomyosin-binding and C-calcium binding, interacts with both actin and tropomyosin.

One molecule of Troponin binds to each molecule of tropomyosin and regulates the access to myosin-binding sites on the seven actin monomers in contact with tropomyosin. The binding of calcium produces a change in the shape of Troponin, which relaxes its inhibitory group and allows tropomyosin to move away from the myosin-binding site on each actin molecule. Conversely, the removal of the calcium ions from Troponin reverses the process, turning off the contractile activity.

b. Excitation-Contraction Coupling [4]

The discussion of skeletal muscle physiology begins with the process of muscle activation itself. It is well known that peripheral nerves innervate skeletal muscles and that neural activation precedes muscle contraction. The precise process by which this neural activation signal culminates in muscle contraction is known as excitation contraction coupling.

Excitation-contraction coupling is a sequence of microscopic events, each of which is necessary for contraction to occur. If any single step in this process is impaired, muscle contraction does not occur normally. This impairment might be interpreted as muscle paralysis or fatigue. The steps are as follows – (Fig 2)

1. Generation of action potential

The first step in excitation-contraction coupling chain is the generation of the peripheral nerve action potential. An action potential results from activation of the peripheral nerve axon that innervates the muscle.

2. Acetylcholine release

Following nerve activation, the resulting depolarization causes a quantum or unit of ACh to be released into the synaptic cleft. ACh then diffuses across the synaptic cleft and binds to the ACh receptor, which is integrated into the muscle membrane.

3. Involvement of the transverse tubular-system and sarcoplasmic reticulum

The action potential is conducted deep into the fiber by the T-system. The interface between the muscle fiber and the contractile apparatus occurs when the T system signals the sarcoplasmic reticulum (SR) to release calcium.

4. Release of calcium resulting in muscle contraction

After the T-system signals the SR that the fiber has been activated, the SR releases calcium ions in the region of the myofilaments. This release process is much faster that the resulting contraction/relaxation cycle.

5. Calcium uptake resulting in muscle relaxation

As long as neural impulses arrive at the NMJ and calcium concentration remain high in the region of the myofilaments, force generation continues. However, when the impulses cease, calcium levels drop and force decreases as calcium is pumped back into the SR by the calcium-activated adenosine triphosphate enzyme (ATPase)

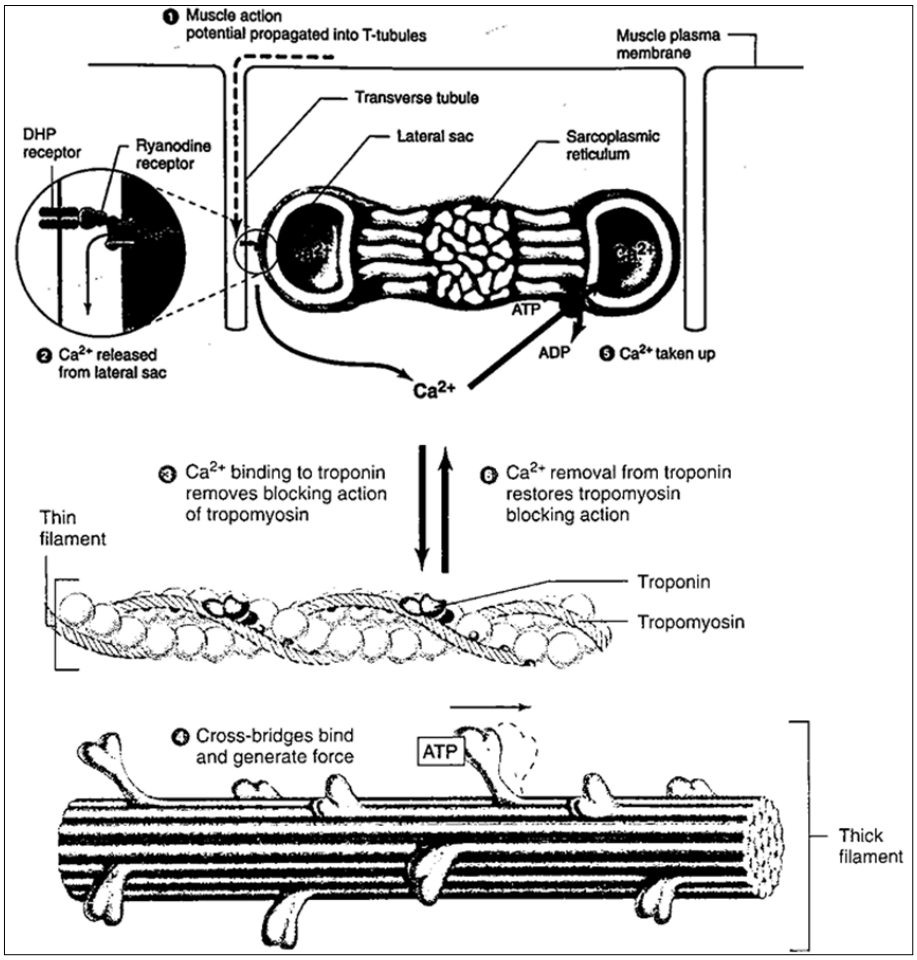

Fig 2: Release and uptake of calcium ions by sarcoplasmic reticulum during contraction

Types of Reflexes

1. Myotatic Reflex [5].

The myotatic/stretch reflex is a monosynaptic neuromuscular mechanism that maintains muscle tone and posture. It involves muscle spindles, sensory neurons, motor neurons, and muscle fibers, and triggers muscle contraction in response to passive stretching.

When a muscle stretches, the muscle spindle is activated, increasing Ia afferent firing, which stimulates alpha motor neurons to contract the muscle and resist further stretch. Reciprocal inhibition ensures relaxation of the antagonist muscle, helping maintain muscle length. (Fig 3)

In the masticatory system, this reflex is seen in the masseter muscle when a sudden downward force (e.g., with a rubber hammer) is applied to the chin. This stretches the spindles, sending afferent signals via the trigeminal mesencephalic nucleus to the trigeminal motor nucleus, which then activates alpha motor neurons causing the masseter to contract. Clinically, this is observed as a reflex elevation of the jaw and tooth contact following a downward tap on the relaxed chin.

This property of the elevator muscles counteracts the effect of gravity on the mandible and maintains the articular surfaces of the joint in constant contact. The myotatic reflex is a principal determinant of muscle tonus in the elevator muscles. As gravity pulls down on the mandible, the elevator muscles are passively stretched, which also creates stretching of the muscle spindles. (Fig 4)

Since the 1930s, functional appliances have been in use, Andresen and Haupl [6], who had introduced the use of the activator, believed that a repetition of the new path of mandibular closure pattern brought about a musculoskeletal adaptation and resulted in the re-education of the orofacial musculature. According to their concept, depending on the construction of the appliance, the activator can initiate a myotatic reflex activity, inducing isometric muscle contractions. This causes stimulation of the protractor muscles and inhibition of the retractor muscles of the mandible. This muscle force thus generated, is transmitted by the appliance to the surrounding structure to bring about a change in the stomatognathic system.

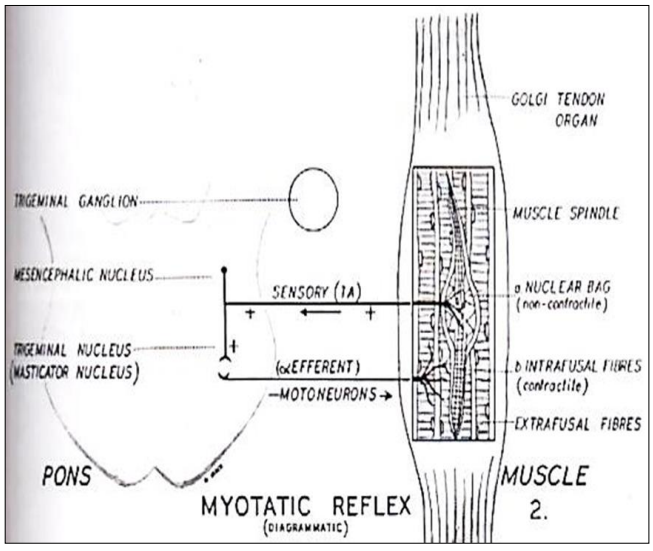

Fig 3: Myotatic Reflex

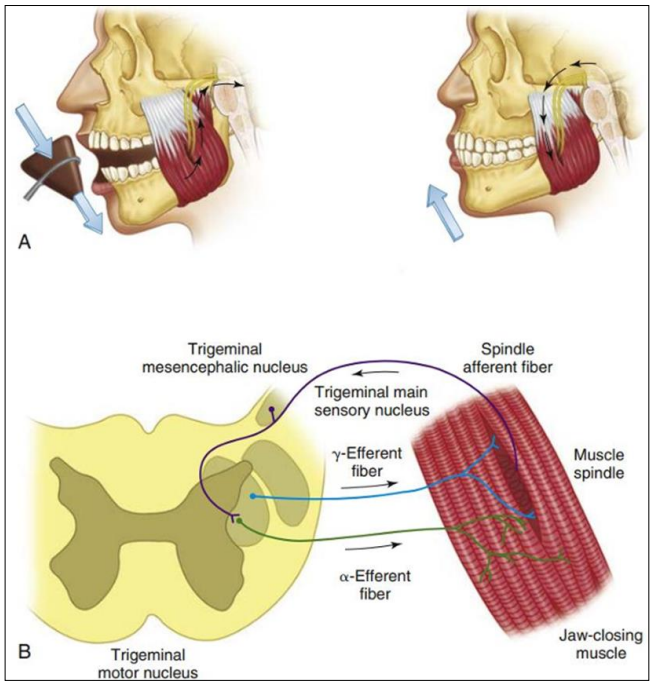

Fig 4: Activation of Myotatic Reflex in the masticatory system

(A) The myotatic reflex is activated by a sudden application of downward force to the chin with a small rubber hammer. This results in contraction of the elevator muscles (masseter). This prevents further stretching and often causes an elevation of the mandible into occlusion. (B) The pathway is as follows: Sudden stretching of the muscle spindle increases the afferent output from the spindle. The afferent impulses pass into the brainstem by way of the trigeminal mesencephalic nucleus. The afferent fibers synapse in the trigeminal motor nucleus with the alpha efferent motor neurons that lead directly back to the extrafusal fibers of the elevator muscle, which was stretched. The reflex information sent to the extrafusal fibers is to contract.

2. Clasp Knife Reflex [5].

The clasp-knife reflex is a stretch reflex characterized by a sudden decrease in resistance during passive limb movement, resembling the snap of a spring-loaded clasp knife. It is thought to protect muscles and tendons from excessive force.

This reflex indicates an upper motor neuron lesion**,** where damage disrupts normal inhibition, leading to increased muscle tone. Upper motor neurons originate in the cortex and influence lower motor neurons in the spinal cord, which control muscles. Also called autogenic inhibition**,** the reflex is mediated by Golgi tendon organs in the muscle tendons. These receptors inhibit excessive contraction to prevent injury. The reflex helps explain increased mandibular displacement with functional appliances in orthodontics. (Fig 5)

Clasp-knife inhibition is evoked by stretch of active muscle. Because both muscle force and length increase during stretch, either might be responsible for evoking clasp-knife inhibition. However, this argument is no longer compelling because muscular free nerve endings, which are not simple force transducers, most likely mediate clasp-knife inhibition.[7] Rymer *et al* [8]. also implicated muscle force by showing that the magnitude of clasp-knife inhibition covaried with initial muscle force, but an apparent dependence on force could also arise from central nonlinearities or decay of the crossed-extension reflex used to set the initial level of force.

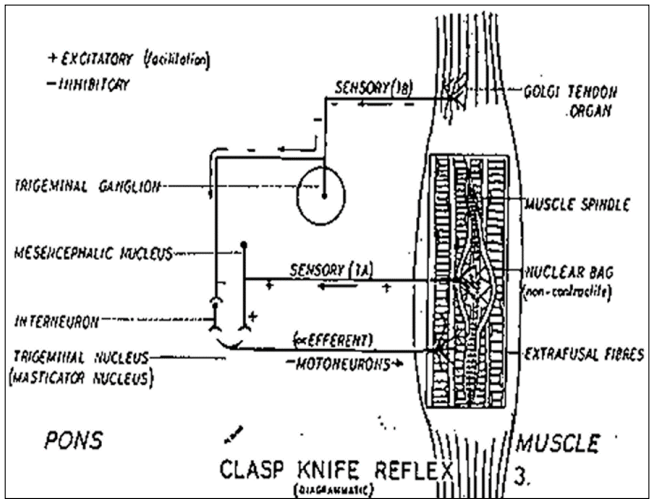

Fig 5: Clasp Knife Reflex

Muscle Fatigue [4, 9, 10].

Muscle fatigue plays a significant role in orthodontics as it influences treatment planning, stability of tooth movement, and long-term retention of results. The neuromuscular system, particularly the orofacial and masticatory muscles, continuously adapts to orthodontic forces. Understanding muscle fatigue in this context helps orthodontists optimize appliance designs, predict treatment outcomes, and enhance patient comfort.

1. Influence on Orthodontic Tooth Movement
   Excessive or prolonged muscle fatigue can disrupt the balance between muscle and orthodontic forces, potentially leading to altered tooth positioning and occlusion.
2. Impact on Myofunctional Therapy and Functional Appliances
   Muscle fatigue reduces the effectiveness of appliances and myofunctional therapy by limiting force output and delaying skeletal or dental changes.
3. Postural and Neuromuscular Adaptations
   Fatigue in jaw and neck muscles may cause compensatory movements, discomfort, and TMJ issues during orthodontic adaptation.
4. Stability of Orthodontic Results and Retention
   Fatigued muscles may not adapt well to new occlusions, increasing relapse risk. Retainers must counter residual muscle imbalance.
5. Implications for TMD Muscle overuse during treatment can lead to TMD symptoms such as pain, headaches, and restricted jaw movement. Early detection of fatigue is crucial.
6. Clinical Considerations for Appliance Design
   Appliances should consider muscle fatigue limits—excessive force can cause discomfort, while insufficient force reduces effectiveness. Intermittent forces improve comfort and outcomes.

Muscles and Its Role in Craniofacial Development and Malocclusion

Role in Craniofacial Development

Moss and Salentijn hypothesized that human facial growth occurred as a response to functional needs and was mediated

by the soft tissue. Masticatory muscle function has a considerable influence on craniofacial morphology. Several earlier reports have suggested that biting force and muscle activity are correlated with jaw size and morphology.
Ringqvist [11]. showed that the size and shape of the mandible, including mandibular body length, ramus height and gonial angle were correlated with bite force. Miller [12]. found a strong correlation between craniofacial morphology and masticatory muscle activity during chewing, swallowing, and maximum clenching. Furthermore, several studies have focused on the relationship between masticatory muscle activity with the mandible at rest and craniofacial morphology. Muscle fiber types have been associated with variation in skeletal facial morphology.
Kitai *et al* suggested that, although genotype is important, mechanical stress brought about by the force and volume of certain masticatory muscles might influence the size of locally adjacent craniofacial skeletal regions. Additionally, Moss understood that genetic factors played a role in skeletal growth, but proposed that growth was also linked to the underlying muscular matrix. The sustained forces of facial expression and speech from the orbicularis oris may contribute to the position of the maxilla.
Earlier studies have described a relationship between the cross-sectional area of the mandibular muscles and facial morphology. A common finding has been that the masseter and medial pterygoid muscles have large cross-sections in people with shorter anterior face heights and relatively smaller gonial angles. This has recently been confirmed with three dimensional computed tomographic studies of subjects with different underlying patterns.

Role in Maocclusion

Malocclusions are generally considered alterations in the normal field of craniofacial growth and morphology. Due to its different possibilities of etiological factors, it is often difficult to determine its specific cause [13]. There are four main structures that can cause malocclusions:

a. the craniofacial skeleton,
b. the teeth,
c. the orofacial neuromuscular system
d. and other soft tissues.

An example of how the musculature can generate malocclusion is the constantly open mandible in the mouth breathing leading to a constant anteriority of the tongue, which may force the incisors or prevent the eruption of the mandibular incisors. Another example is that if a patient has a tongue with anterior posture during rest, the duration of this pressure, even if very light, may interfere with the eruption process or move the anterior teeth, resulting in an open bite [14]. There are also environmental influences, such as oral habits and balance between the orofacial musculature and the teeth, both at rest and during the functions [15]. Mouth breathing is one of these conditions. It causes muscle posture alteration that changes the balance in the oral cavity and modifies the forces exerted on teeth and bones, impacting facial growth and tooth positioning. Other muscles that can influence tooth morphology are masseter and temporalis, which are jaw elevators. When they are active during rest or sleep, it is considered as bruxism, and may cause tooth wear.

Orofacial Forces on The Teeth

There are four main factors responsible for the dental balance:

a. intrinsic forces of the tongue, lips, and cheeks
b. extrinsic forces such as oral habits or orthodontic appliances
c. forces of the dental occlusion d. and forces of the periodontal membrane, as for example, the eruption of teeth.

Among these, the most important are the resting position of tongue and lips, in addition to the periodontal forces, since they have a long duration. When one of these forces stands out, tooth movement occurs, and the teeth are susceptible to adaptations when subjected to some pressure or force.
The balance between the pressure of the tongue, lip, and cheek contributes to the maintenance of the teeth in their positions. The forces exerted by these structures are lighter than those of the masticatory function, but longer in duration. Even though the magnitude of force is low, it can cause a movement in the teeth when applied for a sufficient amount of time [16]. The final position of the tooth, responsible for final shape of the dental arches, results from the balance between the perioral musculature represented by the mechanism of the buccinator and the intraoral pressure exerted by the musculature of the tongue.

Impact of Tongue

The tongue is a mobile muscular organ that composes the stomatognathic system and is located on the mouth floor. This structure assists in the functions of chewing, swallowing, sucking, and speaking.
The most common tongue dysfunctions are those involving selective outer pressure/pressing and tongue biting. Tongue thrusting can be anterior, posterior or combined. The consequences of the localization of aberrant pressures depend on the area of the applied pressure [17].

a. Anterior open bite – anterior tongue thrust
b. Lateral open bite or deep overbite – lateral tongue thrust or postural spread that causes infra-occlusion of the posterior teeth.
c. Edge-to-edge incisal and cuspal relationship of the teeth in the buccal segments – combined thrust aka complex tongue thrusting.

The consequence of tongue posture and function abnormalities in the dentoalveolar region also depends on the skeletal pattern. In a horizontal growth pattern, the forward tongue thrust or posture can result in a bimaxillary protrusion. With the tongue pressing against the lingual surfaces of both the upper and lower incisors simultaneously, there is often spacing in the incisor segments. In a vertical growth pattern, the tongue thrust can open the bite and the lower incisors may be tipped lingually. During the abnormal functional and postural forward positioning, the tip of the tongue lies between the dental arches, in contact with the lower lip which the patient constantly sucks. Thus, the incisors are tipped lingually.

Impact of Bruxism on Dentition [18].

According to the international consensus on the assessment of bruxism, sleep bruxism is a rhythmic or non-rhythmic masticatory muscle activity during sleep. Awake bruxism is a masticatory muscle activity during wakefulness that is characterized by repetitive or sustained tooth contact and/or by bracing or thrusting of the mandible. Both cannot be

considered as a movement or sleep disorder in otherwise healthy individuals.

Bruxism is not a disorder in healthy individuals but might be defined as a motor behaviour with multifactorial causes. It can be a risk factor for negative oral health consequences, but can also be a potential protective factor when associated with other clinical conditions (e.g., sleep apnea or other sleep disorders) or symptoms (e.g., xerostomia) without a cause-and-effect relationship. Or it can be harmless behaviour (not risk or protective factor) in terms of consequences.

Clinical signs of bruxism include masticatory muscle hypertrophy, indentations on the tongue or lip, and/or a linea alba on the inner cheek. However, these signs can also be present on orofacial myofunctional disorders, such as atypical swallowing. Damage to the dental hard tissues, repetitive failures of restorative work, or mechanical wear of the teeth may also be indicators of awake bruxism and sleep bruxism. Yet, it does not assure that it is still active.

Orofacial Muscle Reponse To Orthodontic Treatment

Orthodontic treatment results are achieved by the application of forces or the stimulation and redirection of the functional forces within the craniofacial complex. These functional forces arise from the orofacial musculature.

a. Muscle Response to Vertical Dimension Alterations

Orthodontists routinely raise and lower the vertical dimension of occlusion; bite blocks can be incorporated in various treatment plans. Carlsson *et al* [19]. increased vertical dimension by approximately 4 mm at the incisor region with acrylic splints.

Although some symptoms were reported during the first days, objective examination of the muscles indicated no change in tenderness or movement capabilities. Electromyographic evaluation also showed that masticatory muscle activity was generally less with bite - raising appliances in place. This is consistent with findings reported by Kovaleski and DeBoever [20]. and Greco *et al* [21].

On evaluating the changes in electrical activity of the postural muscles of the mandible upon varying of the vertical dimension Manns *et al* [22]. and Rugh and Drago [23]. concluded that as the vertical dimension was increased, muscle activity generally decreased until a point of minimal activity was observed.

b. Muscle Response to Intramaxillary, Intermaxillary And Extraoral Forces

In the treatment of malocclusion with orthodontic fixed appliances, various forces are directed to the teeth. These forces are primarily derived from active arch wire and elastic elements, although extraoral forces may be added. During the course of treatment, patients usually experience various degrees of discomfort and pain resulting from tooth movement.[24] It has been demonstrated that pain reduces muscle-biting strength. It has also been shown electromyographically, that a decrease in the activity of the masseter muscle occurs following arch wire changes. In a study, conducted over a longer period of time, it was demonstrated that the decrease was greater at the initial levelling period, and also following the removal of the multibracket appliance [25].

Thilander and Filipsson [26]. demonstrated that there was a fairly low response at rest position, i.e., without spontaneous movements of the mandible. On the other hand, in the presence of functional movements muscle activity was intense, with the exception of the masseter muscle whose activity was least. This increase in muscle activity was attributed to the swallowing movements induced by the increment in salivary secretion. Ngan *et al* [27]. assessed electromyographically the masseter muscle activity before, during and after treatment with maxillary protraction headgear and found no significant differences.

c. Muscular Adaptation to Functional Appliance Therapy

Functional appliances evolved from the idea that modification of functional stimuli may influence skeletal development through reorganization of the shape and internal structure of bone [28]. It has been suggested that basic objectives of functional appliances are to train the perioral musculature, assist optimal dentofacial development by eliminating abnormal muscle function, relief aberrant muscle forces on the developing dentition and basal structures, and elicit modified neuromuscular activity thus, stimulating normal functional patterns. Changes in mandibular position may have an effect on the excitability of masticatory muscles reflexes. Therefore, a modification of mandibular functional position, by means of the different types of functional appliances, could activate a series of neuromuscular adaptation mechanisms.

Conclusion

Exploring the intricate relationship between muscle physiology and orthodontics unfolds a narrative that goes beyond conventional biomechanical paradigms, delving into a rich tapestry of interconnected factors that shape treatment planning, execution, and long-term stability within the field. This comprehensive examination not only underscores the significance of traditional biomechanical considerations but elevates the discourse to encompass a broader understanding of the complex interplay between craniofacial muscles and orthodontic outcomes.

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