Australian Society of Orthodontists

University of Sydney

Myofunctional Therapies & Appliances

PART 1

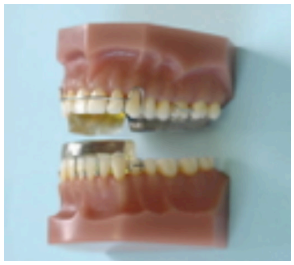
Creating Brighter Futures

Myofunctional Therapies & Appliances PART 1

Definition

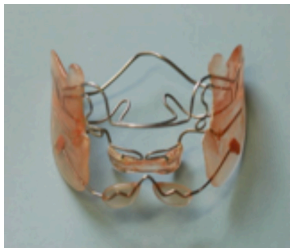
Myofunctional therapies and appliances are used to treat malocclusions by influencing the effect the musculature has on facial and dental growth and development. These include effects on the dentition, guidance of eruption and growth modification of skeletal structures. Myofunctional therapies and appliances are prescribed to treat a variety of malocclusions, however they are of limited use in non-growing patients and patients with severe skeletal malocclusions.

Functional or orthopaedic appliances use to be referred as myofunctional appliances. Functional appliances are used to treat Class II, and very occasionally Class III, skeletal and dental malocclusions in growing patients, and include appliances such as the Twin Block, Activator, Bionator, Frankel and Herbst appliances [1]. Recently the nomenclature has been used to describe appliances such as The Trainer for Kids (T4KTM, Myofunctional Research Co, Australia).



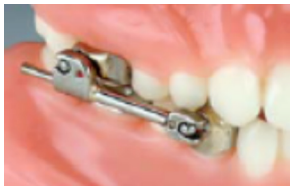
Twin Block

Bionator



Frankel T4K™

Bionator



Herbst Appliance

In order to use myofunctional appliances and therapies to correct a malocclusion, it is crucial to understand the aetiology of malocclusion and also the timing and magnitude of growth.

Aetiology of malocclusion

The aetiology of malocclusions is multifactorial in nature with many factors contributing to its formation. The tongue and soft tissues do play a role in the development of a malocclusion but are not the sole factor.

The growth of the craniofacial skeleton is dependent on complex genetic, epigenetic and environmental interactions. For normal occlusion to develop, proportional growth between the cranial base, the maxilla and the mandible and a harmonious relation between skeletal bases and soft tissues (perioral musculature, lips and tongue) must occur[2].

In the literature heritability of skeletal structures is high, meaning that there is strong genetic control, whereas occlusal variables, or the position of teeth on the basal bone, has lower heritability, meaning strong environmental influences [3]. Interestingly siblings may have similar malocclusions not only due to shared genetic and environmental factors but also shared genetic information affecting how teeth and supporting structures respond to the environmental factors [4].

The aetiology of malocclusion can be classified as:

1) Hereditary (Genetic) Factors
2) Non-Hereditary Factors:
    - (a) Transitional
    - (b) Specific causes:
        - Trauma
        - Muscle Dysfunction
        - Pathology-Disturbances of Dental Development
    - (c) Environmental influences:
        - Equilibrium theory and effect on the dentition
        - Functional influences:
            - (1) Sucking and other habits
            - (2) Tongue thrusting
            - (3) Abnormal tongue posture, Respiratory pattern and Functioning spaces

You may wish to share this issue of Brighter Futures with your hygienists and other staff members.

(4) Abnormal tongue volume
(5) Masticatory muscle strength
(d) Iatrogenic factors

Hereditary/Genetic Factors

Most malocclusions are polygenetic in nature, which refers to the inheritance of a phenotypic trait that can be attributed to two or more susceptible genes and their interactions with the environment. However there are malocclusions that arise from a monogenetic model, where a single mutated gene follows the Mendelian pattern of inheritance.

The general morphology of craniofacial bones and teeth are mainly genetically determined, although some variation may be partly due to environmental factors [5-8]. Many craniofacial abnormalities develop during embryonic craniofacial morphogenesis, in which genetic mechanisms predominate, indicating that genetic factors play a role in some malocclusions. Our genome determines the response to environmental factors in the development of a malocclusion or phenotype. Epigenetic factors are thought to also modify this interaction.

The ability of a patient's skeletal growth and development to respond to change such as a change in oral musculature activity is dependent on their genetic susceptibility.

The question a clinician has to ask is whether treatment aimed at changing the environment is significant enough to exact a change? If so what is the duration of treatment required to create a stable change? The environmental and genetic factors that influenced a developing malocclusion may not be the same ones that will influence how a patient responds to treatment. An environmental modification may alter the development of a phenotype at a particular moment, but a gross structural morphology may not change unless the environmental modification is significant enough to alter the pre-existing structure. Timing of the intervention is important as less mature structures are more susceptible to future stimuli and more mature structures are less susceptible due to the cumulative effects of previous stimuli [9].

There is strong evidence that Class II division 2 and Class III malocclusions have a strong genetic component [10-13]. Previously it was thought to be through a polygenetic inheritance, however, more recent studies suggest a monogenetic model of inheritance [13-15]. There is also a significant genetic component to hypodontia, cleft lip and palate and cleidocranial dysostosis [10].

The investigation of how much genes play a role in malocclusion can be performed by (in ascending order of strength of evidence)

(1) Twin studies
(2) Linkage studies
(3) Gene Association studies
(4) Genome wide association studies

Non-Hereditary Factors

Environmental Influences

Environmental factors influencing the development of a malocclusion are thought to be habits, tongue posture, tongue volume, tongue thrust, breathing pattern and masticatory muscle strength. To this we can add various medical, dental and orthodontic/orthopaedic treatments. These factors can be classified as either functional or stationary.

Equilibrium Theory

Teeth sit in equilibrium between the sustained and resting pressure from the lips, cheeks and tongue. The supporting structures of teeth (periodontal ligament, gingival fibres and alveolar bone) also play a pivotal role in the position of teeth as they resist forces placed on the teeth. If the equilibrium of the dentition is altered, such as by sustained orthodontic treatment or a change in the resting position of the tongue, teeth will move to a new equilibrium. Teeth will also experience functional forces from the tongue during mastication, swallowing and speech, however such intermittent short duration pressures are unlikely to significantly impact on tooth position.

Habits

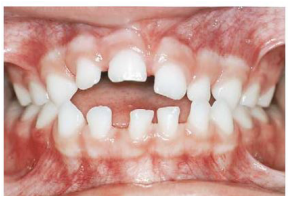
The most prevalent habits of young children are thumb sucking, digit sucking, nail biting, tongue sucking, pacifier sucking and tongue thrusting [16, 17]. These habits are correlated with posterior cross-bites, increased overjet, decreased overbite and anterior open bite and increased palatal depth [18-20]. If the duration of the habit is sufficient enough to alter the equilibrium via the tongue, lips and cheek then a malocclusion forms by guiding the eruption of teeth and interfering with growth and development. The habits cause largely dentoalveolar changes with only minor effects on the skeletal pattern [21].

Pacifier habits are associated with increased mandibular arch width, greater prevalence of posterior cross-bite and anterior open bite, whereas digit and thumb sucking are associated with greater overjet, greater maxillary arch depth and constriction and anterior open bite [22, 23].

There is a high prevalence of cross-bites among children in the primary dentition who suck their thumb, fingers and pacifier [18, 19, 24, 25]. However, most cross-bites can self-correct if the habit ceases before the transition from primary to the early mixed dentition. Most children with finger habits after the transitional dentition do not have cross-bites after the age of 9 years [20, 26].

Once the habits have ceased spontaneous correction of the malocclusion can occur, therefore treatment is aimed at eliminating the habit. However, some malocclusions such as increased overjet and narrow maxillary width may persist into the mixed dentition [22, 27, 28].

The aim of orthodontic treatment is to initially eliminate habits to reduce habit related malocclusions. This can be done by counselling the patient and parents, and can also be supplemented with reminder devices. If the malocclusion does not self correct after the cessation of the habit active orthodontic treatment can be undertaken. There are no studies looking at the relationship between myofunctional appliances and therapies and the reduction of habits.



Anterior open bite common in children with prolonged digit habits.

Tongue

The tongue can produce forces on the teeth and alveolus when in function (tongue thrust) or when static (resting tongue posture). A tongue thrust swallow is defined as the placement of the tongue tip forward between the incisors during swallowing. It is seen in 2 circumstances: (1) as a transition between normal physiologic maturation and as(2) a physiologic adaptation to form an anterior oral seal by bringing the lips together and placing the tongue between separated anterior teeth [29]. It is controversial whether a

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Correspondence is welcome and should be sent to:

Department of Orthodontics
University of Sydney
Sydney Dental Hospital
2 Chalmers Street,
Surry Hills NSW 2010

AUTHOR & EDITORS

Dr Peter Duc Hoang
PRINCIPAL AUTHOR

Dr Chrys Antoniou
Dr Dan Vickers
Prof M Ali Darendeliler
Dr Michael Dineen
Dr Ross Adams
Dr Susan Cartwright
Dr Vas Srinivasan

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tongue thrust, during swallowing or speech causes a malocclusion (anterior open bite and proclined incisors) or simply just adapts to one.

On average we swallow 585 times a day, with each swallow lasting 2 seconds [30]. This equates to approximately 19 and a half minutes of swallowing per day. Considering that there are 1440 minutes during a day it is unlikely forces from the tongue during function can alter the oral equilibrium sufficiently to cause a malocclusion. Although almost every patient with an anterior open bite has an adaptive tongue thrust swallow the reverse is not true. A tongue thrust swallow is often present in children with good occlusion [17, 31]. During the transition from the mixed dentition to the adult dentition 80% of anterior open bites tend to self correct [32]. Any intervention eliminating a tongue thrust swallow during the mixed dentition must have a higher success rate than 80% for it to be indicated. A more recent study found a correlation between the movement of the tongue during swallow and dentofacial morphology; however this study could not distinguish whether this was a cause-and-effect which lead to a malocclusion or whether it was simply an adaptation to the malocclusion [33].

A resting tongue posture is more likely to alter the position of teeth as it can exceed the minimum of 4-8 hours of force required for tooth movement [34, 35]. Anterior and low tongue posture can result from airway problems in the nose or pharynx. It can also be a transient problem from the mixed to adult dentition. According to Scammon's growth curves the tongue finishes its growth at approximately 8 years of age, where as the mandible does not even begin its peak growth until a few years later and can continue to grow in an individual till after their second decade of life. This results in a comparatively larger tongue in a smaller skeletal housing in those developing years. Lymphatic growth also peaks just before puberty, resulting in comparatively larger adenoids in some patients [36]. In a susceptible patient, enlarged adenoids may create an obstruction to breathing resulting in an anteriorly positioned tongue to allow for a more patent airway.

The tongue plays a role in the development of the occlusion, but the evidence is unclear whether it is a cause of some malocclusions. A low anterior tongue posture seems to play a role in the development of an open bite and sometimes incisor proclination. However many patients with such malocclusions have normal tongue posture and tongue activity. There is no evidence as yet that myofunctional appliances and therapies can change tongue behaviour long term.

Muscles of Mastication

Adults with a long-face craniofacial morphology have considerably reduced masticatory muscle strength compared to subjects with a vertically normal facial form. This reduced muscle strength has been suggested as a major determinant of excessive vertical facial growth [62-66]. On the basis of this observation one can hypothesize that in children weak jaw elevator muscles are a key determing factor of a long face growth pattern due to a diminished vertical restraint on growth, while strong elevator muscles predispose one to a short face [67].

The muscles of mastication also have a complex biomechanical influence on the craniofacial structures. Bone apposition occurs with tensile loading evoked by contracting jaw muscle fibres attached to the periosteum [68]. It can be hypothesized that through the contractions of the masseter and temporalis muscles, being attached laterally to the ramus, the zygomatic arches, and the temporal bones, it will stimulate bone apposition leading to increased craniofacial width [69]. However if we are going to accept this hypothesis then we have to ask ourselves how and where do jaw muscles restrain vertical growth of the craniofacial complex? How strong is this relationship and how much strength is required to effect a biological change?

It is debated whether weak jaw muscles leads to a vertical growth pattern or that a long face bony architecture leads to weak muscles due to an unfavourable geometry [70]. Maximum bite force in long face and short face children are very similar, which is in contrast to adults where long face adults have significantly smaller bite force to normal and short face adults [64, 71, 72]. Long and short craniofacial growth patterns are established early in life [73-75]. The long face craniofacial morphologic phenotype is distinguishable well before a reduced masticatory function. Therefore the evidence only supports that children with a vertical growth pattern (under strong genetic control) predisposes or is an indicator for future reduced masticatory function. This is due to a combination of reduced size of jaw closing muscles, reduced force per unit cross section and unfavourable geometry of muscle orientation [70].

References upon request