Review Article

Midline diastema

ABSTRACT

Midline diastema is a space between the maxillary and/or mandibular central incisors. Midline diastema can be due to various causes such as genetic, environmental, and so on. Proper history taking and correct diagnosis of the etiology of the diastema is essential to ensure that the orthodontic correction is successful, and no future relapse takes place. The presence of diastema between the central incisors in the adult patient has esthetics and malocclusion concerns.

Key words: Etiology; midline diastema; stability.

Introduction

Angle described the dental midline diastema as a rather common form of incomplete occlusion characterized by a space between the maxillary and less frequently the mandibular central incisors.[1]

Broadbent described the maxillary midline diastema in growing children as unaesthetically pleasing and termed it as the "ugly duckling" stage of dental development. He considered this stage as a phase which underwent spontaneous closure with the complete eruption of lateral incisors and canines.[2]

For some individuals, the diastema does not close spontaneously.[3]

The extent and the cause of the diastema must be properly evaluated. Proper case selection, appropriate treatment selection, adequate patient cooperation, and good oral hygiene are crucial to the treatment success.[4]

Clinical diagnosis is important and should compulsorily include a radiographic examination. During the "ugly duckling" phase, the long axes of the roots of the maxillary central and lateral incisors converge toward[5] and which often misguides practitioners to a diagnosis of a diastema caused by a hypertrophic labial frenum.[6]

Enlarged labial frena have been considered to be a contributing factor for a majority of persistent diastemas, but this has now been attributed only to the small proportion of cases. Other etiologies related to diastema include oral habits, muscular imbalances, physical obstructions, abnormal maxillary arch structure, and various dental anomalies.[4]

According to Taylor, 98% children 6 years of age presented with a midline diastema. As age increased, the percentage of diastema significantly decreased being 48.7% of children in the age group of 10–11 years old and 7% in the age group of 12–18 years.[7]

Effective treatment of diastema requires an accurate diagnosis of its etiology and a treatment plan that is related to that specific etiology, including medical and dental

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histories, radiographic and clinical examinations and probably tooth-size evaluations.[4]

Etiology and Effective Treatment Options

Timing of the treatment is important to achieve satisfactory results. Most of the researchers do not advise tooth movement until the eruption of the permanent canines.[8] However, in selected cases, where very large diastemas exist, early treatment can be contemplated.

Genetics

"Heritability" is defined as the ratio of the total genotypic diversity to the total phenotypic diversity with values ranging from 0 to 1.[9]

Gass *et al*. noted that heritability of midline diastema was 0.32% for the white population and 0.04% for the black population.[10]

Incomplete palatal fusion and cystic formations have been attributed to the cause of midline diastema.[11]

Many other authors like Gardiner[12] and Schmitt *et al*.[13] suggested that genetics could be a cause for midline diastema.

Hypertrophic Labial Frenum

The most common factor associated with maxillary midline diastema is a hypertrophic labial frenum.[14,15]

A maxillary midline diastema may be caused by the attachment of the labial frenum into the notch in the alveolar bone so that a band of heavy fibrous tissue lies between the central incisors.[15] The two central incisors may erupt widely parted from one another, and the rim of bone surrounding each tooth may not extend till the median suture. In such cases, bone is not deposited inferior to the frenum. A V-shaped bony cleft exists between the two central incisors, and an "abnormal" frenum attachment typically results.[16] Transseptal fibers fail to multiply across the midline cleft, and space might never close.[17]

According to Angle,[18] the maxillary midline diastema is caused by a high labial frenum, but the stability of space closure is not influenced by frenum excision. Sicher[19] and Gardiner[12] also supported this view.

This was contradicted by Tait who stated that high frenum is an effect and not a cause for the incidence of diastema.[20]

Ceremello compared the frena of two groups, one with diastemas and the other without.[18] He found no association between frenum attachment and diastema width, between frenum width and diastema, or between frenum height and frenum width. Dewel established the same results in a similar study.[21]

The blanching test is a simple investigative assessment to predict whether a normal tight contact is a present between the central incisors.[22]

Peg Laterals

Bolton discrepancy (tooth-size discrepancy) is an alternative cause of midline diastema regularly reported in the literature. Bishara,[23] Becker,[24] and Oesterle and Shellhart[25] termed tooth-size discrepancy as one of the main causes for maxillary midline diastema. According to them, the presence of peg-shaped lateral incisors results in distal tipping of central incisors, hence producing the midline diastema.[23-25]

Midline diastema caused due to peg laterals can be closed, and the space required for restoration of the peg-shaped lateral can be obtained by moving the peg lateral into position between the central incisor and the cuspid.[26,27]

Anterior Traumatic Bite

Excessive anterior overbite is another chief causative factor for midline diastema.[28] Trauma to the maxillary anteriors from the mandibular incisors causes the maxillary incisors to procline resulting in an increase in the upper arch circumference, leading to diastema.

When there is no Bolton discrepancy and the patient has an Angle's Class I occlusion, an increase in the anterior overbite outcome will either increase the upper arch circumference leading to diastema or anterior mandibular crowding. This occurrence is due to the wedge-shaped lingual surface of upper central incisors.[28] Excessive anterior overbite can be due to a disproportionate vertical alveolar growth of the mandibular or the maxillary incisors, the insufficient vertical dimension of posterior occlusion (molars) and skeletal conditions such as augmented ramal height.[28]

Oral Habits

Finger sucking and/or abnormal tongue movement may result in interincisal spacing.

According to Proffit and Fields,[29] tongue position at rest may have a bigger impact on tooth position compared to tongue pressure, as the tongue only temporarily contacts the lingual surface of the anterior teeth while thrusting. The tongue

pushes the anterior teeth to a forward position, increasing the circumference which results in spacing.

Supernumerary Teeth

A mesiodens is a supernumerary tooth which occurs in the midline between the two maxillary central incisors.[30] A mesiodens accounts for 80% of all supernumerary teeth.

The presence of a mesiodens can inhibit the close approximation of the central incisors resulting in a midline diastema and can also give way to several other complications such as impaction, delayed and ectopic eruption of adjacent teeth, crowding, axial rotation, displacement, radicular resorption of adjacent teeth, and dentigerous cyst.[31]

Russel and Folwarczna have recommended the extraction of a mesiodens in the early mixed dentition period. According to them, this will aid in improved alignment of teeth and will also minimize the requirement for orthodontic treatment.[32] However, some authors such as Mitchell and Bennett prefer the late extraction of mesiodens when the adjacent permanent incisors have finished their root formation.[33]

Developmental cysts in the orofacial midline

An odontogenic keratocyst can develop in the maxilla and can dislodge teeth, leading to spacing in the anterior region.[34] A median palatal cyst is another midline structure which is a rare cyst commencing from the epithelium trapped along the line of fusion of the lateral palatal maxillary process during growth.[35,36]

Abnormal maxillary arch structure

Tooth size discrepancies are caused by disproportionately large maxillary arch or bony defects that impede approximation of the incisors.[4] The presence of large jaws and normal or small teeth can be attributed to inherited characteristics. However, in a few cases, it can be due to endocrine imbalances. Conditions such as acromegaly can cause unusually large jaws comparative to the teeth size. Normal-sized jaws and small teeth can also result in generalized spacing.[37]

Muscular imbalances in the oral region

The dentition is in equilibrium between the various forces from the intraoral and extraoral soft tissues. The muscular imbalance in the oral region can disrupt this balance and cause the teeth to move until the forces achieve a new equilibrium. In patients with hypotonic lips, the teeth may drift and remain in a labial or buccal position owing to the tongue pressure which leads to wide, ovoid arches deprived of interproximal teeth contact.[38]

Diagnosis and Treatment

Because of the likelihood for multiple etiologies, the diagnosis of a diastema must be founded on systematic medical/dental history, clinical inspection, and radiographic assessment. A nominal diastema treatment requires the precise diagnosis of its etiology and a medication that is appropriate to that specific etiology including medical and dental histories, radiographic and clinical examinations and also tooth-size evaluations.[4] Diagnostic study models also may be essential for analysis.

The treatment objectives are principally attributed to esthetic and psychological reasons rather than functional reasons. Although it is frequently the case, treatment plan should not be selected empirically but should rather be based on adequate scientific documentation. The ideal treatment should deal not only with the diastema but also with the cause of the diastema. Regardless of the selected treatment, of the stability of treatment results has always been deliberated.[39]

Various techniques can be used for diastema closure. Some of the methods that have been proposed for the closure of unaesthetic diastemas involve the usage of fixed or removable appliances, elastics, composite build-ups and brass wires placed around the central incisors and gradually tightened until the diastema is closed.[40]

Stability after diastema closure

Relapse is a major factor to be considered in the treatment of midline diastema. Meticulous diagnosis and elimination of the etiology is the key to gaining a stable result. Long-term use of retainers or the use of permanent bonded lingual retainers have been encouraged, especially in cases with large diastema.[41-44] Large pretreatment diastema and the existence of at least one family member with a related condition increases the risk of relapse.[45]

Conclusion

Considering the different views related to uneventful dental development, it is concluded that an initial presence of midline diastema is not a matter of concern. However, when the diastema is larger than 2.7 mm even after the eruption of lateral incisors, orthodontic intervention may be necessary. Timing often is significant to achieve satisfactory results. Several etiological factors are conveyed and debated in literature, and no single etiological factor is decided upon for the development of a midline diastema. Elimination of the etiologic agent usually can be commenced on diagnosis and after the adequate development of the central incisors. Tooth movement usually is postponed until the eruption of the

permanent canines, but can begin premature in certain cases with very large diastemas. Retention procedure should be subject to the size and the etiology of the midline diastema.

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