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Etiology and Mangement of median Diastema

A project Submitted to the council of the College of Dentistry, of Dentistry, Department of Orthodontic University of Baghdad in partial fulfillments of Requirements for B.D.S Degree

Done by

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Introduction

"Diastema" is space between adjacent teeth . Midline diastemata (or diastemas) occur in approximately 98% of 6 year olds, 49% of 11 year olds and 7% of 12–18 year olds. In most children, the medial erupting path of the maxillary lateral incisors and maxillary canines, as described by Broadbent results in normal closure of this space. In some individuals however, the diastema does not close spontaneously. The continuing presence of a diastema between the maxillary central incisors in adults often is considered an esthetic or malocclusion problem. Midline diastema's can be physiological, dentoalveolar, due to a missing tooth, due to peg shaped lateral, midline supernumerary teeth, proclination of the upper labial segment, prominent frenum and due to a self-inflicted pathology by tongue piercing. Angle and Sicher stated that an abnormal frenum is a cause of midline diastema, while Tait in his study reported that frenum is an effect and not a cause for the incidence of diastemaThe extent and the etiology of the diastema must be properly evaluated. In some cases interceptive therapy can produce positive results early in the mixed dentition. Proper case selection, appropriate treatment selection, adequate patient cooperation, and good oral hygiene all are important.8-10 Eruption, migration and physiological readjustment of the teeth, labial and facial musculature, development into the beauty conscious teenage group, the anterior component of the force of occlusion and the increase in the size of the jaws with accompanying increase in tonicity of the facial musculature all tend to influence closure of the midline dental space. The mandibular diastema is not a normal growth characteristic. The spacing, though seen less frequently than maxillary diastema, often is more dramatic. No epidemiologic data have been published on its prevalence. The primary etiologic factor in mandibular diastema is tongue thrust in a low rest position. Many patients seek closure of a diastema for aesthetic reasons. In the case of normal physiological development, diastemas

of less than 2mm in nine-year-old children generally close spontaneously. If they do not do so, small diastemas (less than 2mm) can be closed with finger springs on a removable appliance or with a split Essix plate, as described by Sheridan. In adults with wider diastemas, fixed appliances are required for correction so that crown and root angulations are controlled. The etiology, pathogenesis and diagnosis of maxillary median diastema have been somewhat controversial over the years. The purpose of this paper is to review the published information and controversies regarding the etiology and treatment of the midline diastema in order to give the practitioner an overview to direct effective diagnosis and treatment. (**U Hussain,2013)**.

2.1 Occlusion:

Normal occlusion:

Mentioned that the precise definition of normal occlusion and malocclusion has been difficult to produce but there is a general agreement that an individual occlusal status described by two major characteristics: **(Proffit, 2000)**

1. The relationship of the teeth with each arch to a smoothly curving line of occlusion.
2. The pattern of occlusal contact between the upper and lower teeth.

six keys of normal occlusion are: (Andrew's, 1972; Warotayanont PR, 2011).

- **Molar relationship:**

The distal surface of the distobuccal cusps of the upper first permanent molar made contact and occluded with the mesial surface of the mesiobuccal cusps of the lower second molar.

- **Crown angulation "The mesiodistal tip ":**

The gingival portion of the long axis of each crown was distal to the incisal portion varying with the individual tooth type but within each type the tip pattern was consistent from individual to individual.

- **Crown inclination (labiolingual or buccolingual inclination) :**

The inclination of all the crowns has a consistent scheme:

Anterior teeth (central and lateral incisors):

Upper and lower anterior crown inclination is sufficient to resist over eruption of anterior teeth.

Upper posterior teeth (canines through molars):

A lingual crown inclination existed in the upper posterior crown was a constant and similar from the canines through the second premolar and was a constant, similar and slightly more pronounced in the molars.

Lower posterior teeth (canines through molars):

The lingual crown inclination in the lower posterior teeth progressively increases from the canine through the second molar.

- **Rotation:**

There are no rotations.

- **Spaces:**

There are no spaces with tight contact point.

- **Occlusal planes:**

The plane of occlusion varied from generally flat to a slight curve of spee.

2.2 Malocclusion:

Definition:

Said that malocclusion may be defined as those irregularities of the teeth beyond the accepted range of normal. **(Houston, 1976; Hashim MH, 2006)**

A malocclusion is a misalignment or incorrect relation between the teeth of the two dental arches when they approach each other as the jaws close **(Gruenbaum et al., 2010).**

2.3 The median diastema:

Definitions:

Defined it as a wide space between the maxillary central Incisors (Median diastema). It is often present in childhood during ugly duckling stage which spontaneously closed (If it is less than 2 mm space) after eruption of permanent canines. Although this space tends to close, over 6% of youths and adults still have a noticeable diastema **(Proffit and Field, 1993)**.

If the upper arch is spaced or the lateral incisors are diminutive or absent, there is less pressure forcing the upper central incisors together and the diastema will tend to persist (**Mitchell et al, 2004)**.

2.4The etiological factors of median diastema:

The main etiological or contributing factors of median diastema are as follows:

1. Physiological**:** Spacing of the permanent central incisors is normal at 7(1/2) and 12 Years of age when the teeth erupt (ugly duckling stage). The diastema tends to close with eruption of the permanent laterals and canines (**Walther, 1967; Kumar M, 2011).**
2. Abnormal frenum labii: Frenum is abnormally attached to soft and bony tissues between incisors at the site of median diastema. Figure (1) **(Edward, 1977; Popovich et al, 1977; Agarwal A, 2012)**.

Fig 1 Abnormal frenum

3. Disproportion between teeth sizes and dental arches length i.e. small teeth in large jaw **(Bolton, 1958; Olayemi AB, 2011).**
4. Median diastemas also occur when one or more incisors are congenitally missing or extracted or canine impaction.fig (2),(3) **(U Hussain,2013)**

Fig 2 Impacted canine

Fig 3 Missing lateral

5. Discrepancies in incisors position: like labially position of incisors to mandibular incisors with overjet larger than normal or one or more incisors are severely rotated. Fig (4),(5) **(Taylor, 1939; Gardiner, 1967; and Popovich and Thompson, 1974; Hashim MH, 2006)**.

Fig 4 severely rotated incisor

Fig 5 increased in overjet

6. Ethnic and racial background, blacks are more than twice as likely to have a median diastema as white. It may be due to facial divergence, cranial rotation which differs according to genetic factors that can be modified by environmental factors. **(OM Tanaka,2015)**
7. Peg-shaped lateral incisors **(Whalter, 1967; wall LG, 2013)**.
8. Presence of supernumerary tooth or teeth lying between incisors. Fig (6) **(Mitchell et al, 2004)**

Fig 6 Supernumery tooth

9. Median cyst **(Gass et al, 2003)**.
10. Parafunctional habits such as thumb-sucking, mouth-breathing and tongue-thrusting. **(Muhamad Abu-Hussein et al,2016)**.
11. Macroglossia **(Nainar and Gnanasundaram, 1988; Tarvade SM, 2015)**.
12. Others, like: generalized spacing, closed bite, inter-premaxillary suture and ankylosed central incisors **(P Nagmode et al,2012)**.
13. Periodontal disease.

Divided diastemas into two categories: (Bishara, 1972; Korkut B, 2015)

A. Those present before the beginning of orthodontic treatment:

1. In deciduous dentition diastemas are a common and normal occurrence. These spaces are called primate spaces.
2. In the permanent dentition diastemas may be the result of one or any combination of the following factors:

a. Diastemas usually are a normal stage in the development of the dentition, specifically between the ages of 7 and 12 years.

b. Diastemas due to genetic factors may be localized or generalized.

3. Ethnic or racial characteristics, such as interincisor diastemas which are more frequently seen in Negroes than in Caucasians. This may or may not be related to the tendenc***y of Negroes to have bimaxillary dental protrusion***

4. Labial frenums preventing the maxillary central incisors from moving toward each other, even after eruption of the remaining dentition.
5. Rotated teeth, which may cause diastemas to appear.
6. A supernumerary tooth or supernumerary teeth (for example, mesiodens which are frequently discovered between the separated maxillary central incisors).
7. Certain pathologic condition, such as extensive proximal decay, may give the appearance of a diastema. Periodontitis, cysts, and tumors may cause separation of teeth. Spaces might be caused by the removal of teeth, followed by migration of the neighboring teeth into the extraction space.
8. Thumb-sucking, lip biting, and tongue-thrusting habits may cause maxillary and / or mandibular anterior teeth to tip labially.

B. Diastemas associated with or appearing after orthodontic treatment:

1. Diastemas may result from the removal of teeth to obtain stable results.
2. Orthodontic treatment often necessitates the removal of dental units from the maxilla and / or mandible. The sizes of the teeth removed are not necessarily equal either in the same arch or between opposing arches. The amount of the discrepancy varies, and consequently the amount of residual spaces varies. Such discrepancies are usually apparent toward the end of orthodontic treatment.

2.5 *Familial and heritability aspects of median diastema:*

Suggest that median diastema is more heritable in the white population than in the black sample. If the white sample had more uniform environmental influence or greater genetic variance, it would be reflected in a higher heritability for the trait. (**Gass et al, 2003).**

2.6 The incidence and prevalence of median diastema :

Found that the incidence of median diastema is more than the incidence of mandibular diastema. **(Keene, 1939; Al-Rubayee MA, 2013)**

2.7 The superior labial frenum:

A diastema between the maxillary central incisors is a relatively common finding during the deciduous and mixed dentitions. The majority of these closes spontaneously by the time the maxillary canines appear, but a few persist into the mature permanent dentition. **(Popovich et al, 1977; Kotlow LA, 2013).**

2.8Definitions and classifications:

The superior labial frenum can be defined as a fold of mucous membrane consisting of highly vascularised connective tissue covered with epithelium. **(Noyes, 1935; Mohan R, 2014).**

The frenum can be classified into six types, namely: thick and thin and either high, medium or low attachments.

Type 1: were high, and thin attachments.

Type 2: were high, and thick attachments.

Type 3: were medium and thin attachments.

Type 4: were medium and thick attachments.

Type 5: were low, and thin attachments.

Type 6: were low, and thick attachments.

Low is taken to be at just above the gingival margin and high is taken to be high up on the alveolus approximately at the level of the apices of the incisor teeth **(Popovich et al, 1977; Mohan R, 2014).**

2.9 Development and growth of superior labial frenum :

Embryonically, the superior labial frenum appears to be developed from the frontonasal process, and it begins to take form in the fetus at a relatively early stage.

Within the first few months of fetal life, it emerges as a part of the oral cavity, along with the lips and the cheeks. As growth and development progress, a prominence begins to appear in the middle part of the inner zone of the upper lip, and this becomes the tuberculum. About this time, another prominence forms on the anterior part of the palate and develops into the palatine papilla (Which defined by **(Dewel, 1946; Hashim MH, 2006)** as an oval tuft of tissue located later in life in the interproximal area immediately adjacent to the central incisors ligually. However, recessive tendencies that are characteristic of the structure throughout its life soon begin to operate). A continuous fold of tissue, the tectolabial frenum (which its appearance at between the second and third months in utero), connects the tuberculum with the palatine papilla **(Ceremello, 1953; Hashim MH, 2006)**.

The manner of infant feeding -breast, artificial, or partly breast and partly artificial has no influence on the development of the frenum and its resulting diastema **(Tait, 1924; Hashim MH, 2006).**

2.10 Histological and anatomical view:

Histologically, **(Noyes, 1935; Balogh MB, 2011)** studied newborn infants and found that the frenum is composed mostly of connective tissue, with a few striated muscle fibers which arise from the muscle bundles of the lip on either side of midline and pass in a diagonal direction medially and posteriorly but do not reach the alveolar process. The loose character of the fibrous connective tissue becomes more regular in arrangement with strands lying in an anteroposterior direction as it nears the alveolar attachment. In the labial portion there are mucous glands in the subcutaneous tissue on either side of a central artery and vein that lie near the muscle bundles of the lip. This artery and vein have branches which are given to the frenum and these travel in an anteroposterior direction, providing the blood supply of the structure. Nerve filaments accompany the vessels. The posterior fibersterminate by ramifying

with the connective tissue of the alveolar crest and its anterior surface **(Dewel, 1946; Anderson and George, 1948; Hashim MH, 2006)**.

2.11Clinical view:

The normal appearance of the frenum at maturity is that of a thin, triangular, knifeedge fold of mucous membrane which has a relatively wide origin on the inner surface of the upper lip, extends posteriorly to the midline attachment on the labial aspect of the alveolar process and terminates at a point 4 or 5 mm above the interproximal gum septum between the central incisors, beneath its lower attachment, the gingival tissues appear to be continuous **(Dewel, 1946; Thosar N, 2017)**.

2.12 Variations of the normal frenum:

As with any other anatomic structure, the normal frenum is subject to certain recurring variations in form, size and position. some frenums are broad and sturdy; others are thin and pliable. These variations may be seen not only in the same child at different ages, but also in different children of the same age **(Coleman, 1941; Jindal V, 2016).**

Factors in variations of the normal frenum (Dewel, 1946,* Hashim MH, 2006*):

A truly abnormal frenum is rare and must not be confused with a frequently recurring enlarged frenum that either has not had the time to atrophy or persists because of other conditions present. In both of these conditions, there is an association to:

1. Disturbance in growth and development throughout the entire premaxillary area.
2. Arch form: This may be abnormal, so that the lateral incisors and cuspids are prevented from producing sufficient mesial pressure on the central incisors to close the space in a normal manner.
3. Eruption of permanent teeth

4. Malocclusion: Ordinarily, restoration of normal occlusion will release whatever developmental forces are necessary for atrophy of the frenum and closure of the space.

2.13 The abnormal superior labial frenum:

Described the abnormal frenum as elongated at its alveolar end into a sheet like process that extended lingually between the central incisors, cutting through the interproximal fibers of the periodontal membrane, and ending in the central papilla of the rugae. He added that by its presence it prevented the permanent incisors from approximating each other **(Strang, 1950; Baart JA, 2004)**.

Diagnostic symptoms of an authentic abnormality of the frenum:

(Dewel, 1946; Hashim MH, 2006):

Abnormal frenum is found to be markedly enlarged, retaining in exaggerated from most of the coarsened and thickened characteristics of its earliest developmental stages. It have a wide fan shaped attachment to the upper lip, which tapers downward to a distinct sheet like process extending between central incisors to a definite union with palatine papilla.

2.14 The relationship between the superior labial frenum and median diastema:

The most probable reason for the existing confusion and bewilderment on the subject is the insistence of some to associate the median diastema and frenum as almost synonymous terms, rather than to look up on them as more or less separate entities. It is not necessary that these etiologic conditions be either pathologic or abnormal. They may just as readily be developmental. This is true because the ultimate size and position of the frenum depends for the most part on the normal development of the adjacent dental and premaxillary structures. For this reason, careful diagnosis requires that no frenum be resected without allowing adequate time to elapse between the first examination and the final decision. Observation should extend at least over a period of months to

determine whether the natural forces of growth and development are not sufficient to close the space in a normal manner. Ultimately, it will be observed that this process is the rule rather than the exception **(Dewel, 1946; Diaz-pizan ME, 2006).**

2.15 Surgical correction of abnormal frenum labii:

Advised the surgical removal of all questionable frenal with a suitable lancet or bistoury, a deep incision is made between the teeth splitting the ligament, after which an electrocautery knife, at white heat, is passed through the incision. No pain will be occasioned if, preliminary to the operation, the tissue be locally anesthetized with a proper solution of cocaine, applied by means of a pledget of cotton for about ten minutes.eads to a reduction of median diastema. **(Angle, 1907; Hashim MH, 2006)**

Suggested the principle objections of the operation are as follows: **(Dewel, 1946; Hashim MH, 2006)**

1. It is unnecessary.

2. Scar tissue is usually left between the teeth.

3. The possible creation of an unsightly point of tissue under the fold of the lip.

4. The danger of severing the transceptal fibers.

5. The resultant lack of control of the lip and the loss of the normal vermilion border.

6. Many orthodontists maintain that it is more difficult to close the space and retain the position in those who have been operated up on than in those who have not been operated up on. This may be due to the severing of the transceptal fibers or to scar tissue or both.

7. There is a better way when there is a malocclusion present and it should be treated as such, close the space by means of routine orthodontic procedure and the objectionable band of tissue is eliminated by pressure atrophy.

2.16 *Skeletal dental base rotation:*

Until longitudinal studies of growth using metallic implants in the jaws were carried out in the 1960s, primarily by Bjork et al in Copenhagen (1968), the extent to which both the maxilla and mandible rotate during growth was not appreciated, the reason is that the rotation that occurs in the core of each jaw, called internal rotation, tends to be masked by surface changes and alterations in the rate of tooth eruption. The surface changes produce external rotation. Obviously, the overall change in the orientation of each jaw, as judged by the palatal plane and mandibular plane, results from a combination of internal and external rotation. The terminology for describing these rotational changes is itself confusing. **(Proffit, 2000).**

2.17Tooth eruption and rotation of jaw:

It is essential to take into consideration that the rotation of the jaws during growth exerts an influence on the path of eruption of the teeth and hence on the occlusion and tooth spacing. In addition to the variation in direction, importance attaches also to the location of the center of rotation. According to **Bjork's studies (1947, 1955, 1960, 1969, Bjork and Skieller, 1972 and Perera, 1987; Hashim MH, 2006):** there is different ways in which the mandible can rotate in relation to maxilla during growth, which are:

A. Forward rotation:

The center for this rotation was located in the anterior part of the dental arch, here the eruption of the molars was greater than that of incisors.

Types of forward rotation:

Type I:

1. Forward rotation in which the center at the incisors.
2. This type of rotation is the most suitable one.
3. The lower molars erupted farther than the incisors.
4. No increase in overbite.

Type II:

1. Here the center of rotation at the premolars.
2. Here the lower incisors were tipped forward 11.3 degrees on the jaw base to compensate for a rotation of the mandible of the same magnitude.
3. Collapse of the anterior part of the dental arch as the result of an extreme compensation in inclination lead to the development of crowding in the lower arch.
4. When anterior occlusion is instable, the forward rotation of the mandible can give rise to basal deep overbite.

B. Backward rotation:

It is less frequent than forward rotation. Two types have been recognized by **Bjork (1962, 1968, 1969)**:

Type I:

1. The rotation of the mandible may be described as a rolling of the mandible against the maxilla about a center that moves back toward the posterior occluding molars.
2. The chin then swings downward and backward which an increase in the anterior lower face height. The posterior face height, on the other hand, becomes relatively low.
3. The incisors erupting further than the molars.
4. The compensatory eruption of the incisors was to some extent prevented by the position of the tongue and the tendency of open-bite increased further.
5. The lower anterior teeth were tipped backward on the jaw base, while there was simultaneous reduction in the alveolar prognathism. This lead to crowding in the anterior segment.
6. The lower molars were tipped back in relation to the jaw base. However, since in this type of rotation the eruption of the molars is diminished, this compensatory movement of the molars will often be slight.

Type II:

1. Here the center of backward rotation lies in the temporomandibular joints.
2. Increase in the anterior face height.
3. This type of rotation also occurs in connection with growth of the cranial base. In the case of flattening of the cranial base, the middle cranial fossae are raised in relation to the anterior one, and then the mandible is also raised.
4. This underdevelopment of the posterior face height leads to backward rotation of the the mandible, with overdevelopment of the anterior face height and possibly open-bite as a consequence.
5. The mandible is, in principle, normal.

2.18 The relationship between the skeletal dental base rotation and median diastema:

Growth rotation are most obvious and have their greatest impact on the mandible, their effect on the maxilla are small and are almost completely masked by surface remodeling. Growth related rotation may also occur in the mid-face. The nature of it may be determined along the palatal plane and expressed by the angle of inclination, mid-face rotation is only partly due to growth as it may also be affected by mechanical forces. Occlusal forces act in the cranial, gravity act in the caudal direction. These forces may have an effect on the inclination of the maxilla. Both jaws may rotate in the same direction (horizontally or vertically), or in opposite direction (maxilla vertically, mandible horizontally, or vice versa) **(Rakosi 1982; Hashim MH, 2006)**.

The genetic factors give the growthof the face their general orientation but it may be attenuated or accentuated by the environments which react in a direction.

So, mandibular rotation ***can be influence by:*** **(You et al, 2001).**

1. Digit sucking and lips sucking which attenuate the anterior rotation and aggravating the posterior rotation.

2. Mouth breathing lead to extrusion of the molars and so, aggravating the posterior rotation.
3. Tongue thrust aggravating posterior rotation.
4. Bruxism cause anterior rotation.
5. Any diminution of dental system is a factor of anterior rotation and these are:
   - Genetic (microdontia, hypodontia)
   - Pathological (extraction, caries)
   - Physiological (shedding of deciduous teeth)
   - Orthodontic intervention.

2.19 Facial divergence:

The first definition of the divergence was coined by **Hellman (1921**) who defined it as an anterior or posterior inclination of the lower face relative to the forehead.

A steep mandibular plane angle has often used as an indication of a hyperdivergent facial pattern, and conversely a low mandibular plane angle has been used as an indication of a hypodivergent skeletal facial pattern. The facial skeletal type was also be determined by using the jaraback analysis. Subjects were considered to be facially hyperdivergent if the posterior – anterior face height ratio (Sella-Gonion / Nasion-
Menton) was 59% or less, while the lower half of the gonial angle (Nasion – Menton) was 76ؑ or more, subjects were considered to be hypodivergent face if the posterior – anterior face height ratio was 65% or more while the lower gonial was 69ؑ or less (**Giradot, 2001**).

2.20The relationship between facial divergence and median diastema:

In clinical orthodontics the assessment of space in the dental arch is of importance in the diagnosis and treatment planning. There are a number of factors, intrinsic and extrinsic in origin that may influence the space conditions,

as a discrepancy between tooth size and the size of the jaw, the width of the dental arches, the inclination of the teeth, muscle function and occlusal relationships. This complexity frequently makes it difficult for the orthodontist in planning adequate treatment. It is therefore relevant to ask if the basal parts of the facial cranium may form the basic for differentiating types of lack of space, and if this relationship will manifest itself in spite of the influence of local factors **(Leighton and Hunter, 1982; Hashim MH, 2006)**.

2.21 Mesio-distal angulation:

Published an article, in which he wrote that the mean of mesiodistal angulation estimated from the non orthodontic normal people for maxillary central incisors is equal to 5. (**Andrews, 1976; Hashim MH, 2006)**

Occlusal factors:

Tooth agenesis:

Definition:

- Tooth agenesis: can be defined as the congenital absence of at least one permanent tooth, is the most frequently encountered dental anomaly. (**Tavajohi-kermani et al, 2002).**
- Hypodontia: is a congenital absence of one or more teeth **(Stewaer, 1982; mKingston, 1985; Proffit, 2000),** while **Foster (1990)** defined the hypodontia as the developmental absence of one or more teeth from the dentition.
- Oligodontia: is a congenital absence of six or more teeth excluding the third molar **(Schalk et al, 1994; Proffit, 2000)**.
- Anodontia: is the total failure of development of all teeth. **(Jones and Oliver, 2000; Proffit, 2000)**.

2.22 Causes of tooth agenesis:

1. The hereditary factor plays a very important role in hypodontia. **(Grahnen, 1956; Gravely and Johnson, 1971**; **Graber, 1978; Tai, 1981; Kirdelan et**

al, 1998; Hashim MH, 2006), all suggested that, the hypodontia is an inherited characteristic and can be transmitted by one of the following:

a. Autosomal dominant gene.

b. Autosomal recessive gene.

c. X-linked gene.

d. Multi-factorial inheritance.

e. Genetic mutation and homobox genes.

2. There are some environmental factors which can damage developing teeth and could cause hypodontia like birth trauma, radiation, tumor, rubella virus, scarlet fever, Thalidomide and glandular dysfunction **(Grahnen, 1956; Gullikson, 1975; Hashim MH, 2006)**.
3. Reduction in jaw size, making the jaw unable to accommodate the original complement of teeth **(Hattab and Angmar- Mansson, 1997; Hashim MH, 2006)**.

2.23The prevalence of tooth agenesis:

A large number of studies have been carried out on the prevalence of hypodontia of permanent teeth. The earliest of these studies was by **(Leroyd' Eliolles, 1851; Hashim MH, 2006)** in a short communication to oral biology society, representing the most ancient reference in hypodontia. In Iraqi study found that the prevalence of hypodontia was higher in male than females but the number of conginittaly missing teeth in females was higher than males. perincisors. **(Mahmoud, 2004)**

2.24 The effects of hypodontia

Mention that hypodontia causes spacing and median diastema. Missing upper lateral incisors affects the position of the canine because it is the guideline for the eruption of the canine. It mainly produces midline diastema. (**Mitchell et al, 2004).**

The management:

Patient with unilateral or bilateral congenitally missing lateral incisors have 2 major treatment alternatives: closing the space or opening it for prosthetic replacements or implants. Either of these may compromise esthetics, periodontal health, and function.

(Robertson and Mohlin, 2002)

2.25 Dental arch size and permanent teeth sizes:

In a study of comparison between group that died in 1810 and a group registered about 1970; the mesiodistal tooth size from the first permanent molar to the first permanent molar was compared, the skull group was found to have 4.3% smaller teeth in the maxilla and 3.3 % smaller teeth in the mandible than the modern group. And this is due to that: **(Lindsten et al, 2002)**

1. Stature has increased during the last century, because of improved socioeconomic situation and nutrition. **(Hauspie et al, 1997)**
2. Prenatal conditions also affect tooth size. Smaller teeth are associated with children of short birth length and low birth weight; other maternal conditions were associated with larger or smaller teeth in children **(Garn et al, 1979; Hashim MH, 2006)**.
3. A reduction in mesiodistal tooth size of 2% to 8% was found due to vitamin deficiencies in pregnant rats **(Paynter and Grainger, 1956; Hashim MH, 2006)**.
4. Postnatal experiments in pigs subjected to protein-deficient or a calorie-deficient diet showed that tooth was affected. Under nutrition was extreme; the size of first permanent molars was not affected, but that of the later forming teeth was **(Luke et al, 1979; Hashim MH, 2006)**.
5. In children with advanced hypodontia, tooth size is smaller than in children without advanced hypodontia. **(Rune and Sarnas, 1974; Schalk et al, 1996)**.

2.26 Impaction:

Definition:

Impacted teeth can be defined as those with a delayed eruption time or that are not expected to erupt completely based on clinical and radiographic assessment **(Bass, 1967; Rayne, 1969**; **While Bishara, 2001)** defined the impacted tooth as a tooth blocked from eruption by a physical barrier such as another tooth.

2.27 The prevalence:

The prevalence of impacted maxillary canines is 1-2% in the general population.

The palataly impacted canine forms about 76% and in about 65% the upper canine come out into the oral cavity with a slight rotation about 45؛ with the dental arch and 10% of these case with 180؛ of rotation (the lingual surface become labially) , while 25% of these cases the canine come out in good alignment. Palatally erupting or impacted maxillary canines occur twice as often in females than males, have a high family association and are 5 times more common in Caucasians than Asians **(Peck et al, 1997; Bishara, 2001**).

2.28Treatment of median diastema:

Initiation of orthodontic treatment to close diastemas should be done after close evaluation of certain aspects of the problem. The specific question to be answered is:

Does the diastema affect esthetic and speech, and is the patient aware of its presence? If he is self-conscious about it, does he want to do something about it? Next, is the diastema self-cleansing, or are food impaction and inflammation of the interdental tissues present? Finally, if spaces are mechanically closed, would they stay closed or would they relapse? **(Suri and Utreja, 2003).**

Golden ratio

The search for perfect proportions has engaged people for a long time. Even the ancient Greeks tried to find the ideal proportion which rules the world. The look of a smile is affected by two ratios: the first is the proportion of the width of upper front teeth in relation to each other and the second ratio is the height-to-width proportion The proportions of the teeth in relation to each other in a smile are described by the golden proportion otherwise called the divine proportion . Because of the curvature of the dental arch, the visible widths of upper front teeth are different then real widths. According to the theory of the aesthetics of the smile, the best considered width of the lateral incisors should be 62% of the width of the central incisor and the canine apparent width — 62% of the width of the lateral incisor. Ideal central incisor width-to-height proportion should be 75–78% or 80% and it depends on teeth length . In this study the scope of the W/L ratios was higher and amounted to 92–100%.(**R. Chałas**,**2015**)

A. Treatment of diastemas not caused by orthodontic treatment:

1. Diastemas present as part of the manifestation of the normal development stage of the dentition should not be closed for fear of deflecting the roots of the maxillary central incisors in the path of eruption of the lateral incisors.
2. Diastemas caused by supernumerary teeth, cysts between the teeth, or the presence of a fibrous epulis pushing the teeth apart should not be closed before removal of the pathologic condition.
3. The prognosis for diastemas caused by habits (thumb- and finger-sucking, lipbiting and tongue-thrusting) may range from good to poor, depending on whether the patient is willing and / or able to stop his deleterious habit.
4. If an abnormal labial frenum is diagnosed as the cause of the malocclusion, surgical excision or permanent retention by splinting together of the teeth should be considered**(Nikolaos Gkantidis et al,2008)**.

5. Diastemas resulting from migration of teeth in residual spaces from congenitally missing or extracted teeth may be treated by one of two different approaches:
   a. Use of an orthodontic appliance to open the space for the missing tooth, followed by bridging of the space.
   b. Orthodontic closure of all spaces in the arch.

(Bishara, 1972; Hashim MH, 2006).

6. Peg-shaped maxillary lateral incisors may cause diastemas to open between the neighboring teeth through lack of proximal support. The peg-shaped lateral incisors may be extracted if the root is short or resorbing and then treated as stated above, or, if the roots are of good size and shape, a crown can be constructed after orthodontic movement of the teeth to their proper positions. In the latter case, very little or no relapse should be expected**(SK LN, P Nagmode et al,2012)**.

7. Diastemas caused by rotated teeth are difficult to retain after closure because of the tendency of the rotated teeth to return to their original positions.

8. Generalized diastemas between the teeth caused by microdontia are difficult to close and retain. In general, complete closure of spaces is not advised. Two alternatives are suggested: no treatment or full mouth reconstruction if it is esthetically or functionally needed and feasible.

B. Treatment of diastemas associated with or appearing after orthodontic treatment:

1. Teeth which have been tipped into extraction sites during treatment will tend sometimes to upright when the wires are removed.
2. Constricting the dental arches and encroaching on the tongue space by moving the teeth into an unbalanced position between the lingual and buccal musculatures is an invitation to relapse and space opening. It should be remembered that the teeth in malocclusion are in a state of balance, and

unless their new position is in another state of balance with the surrounding environment post treatment changes are to be expected.

Suggested that the relapse may be a major problem following closure of midline diastema that persist after the eruption of permanent canines. To minimize relapse, permanent retention in the form of bonded wires, restorative options and even small powerful magnets bonded to the mesiopalatal surface of central incisors has been proposed. **(Dixon et al, 2002)**

The ultimate success in keeping the spaces closed after removal of the active appliance and retainers will depend upon the following factors:

1. Removal of the cause of the diastema, when possible.
2. The inherent tendency of the tissues, whether periodontal or muscular, to regain their original shape and position, either partly or completely.
3. Whether the new tooth position is in balance with the surrounding tissues and also on whether retention time was enough to allow adaptive changes in the tissues to occur, thus stabilizing teeth in their newly acquired positions.
4. The fulfillment of all orthodontic treatment objectives, including the positioning of the teeth in a balanced position with optimal interdigitation and axial inclinations. These three factors, separately or combined, will greatly influence prognosis of median diastema treatment***(According to Bishara, 1972 Hashim MH, 2006)***

2.29.Alternative approach for diastema closure other than orthodontic treatment:-

Treatment of diastema varies and it requires correct diagnosis of its etiology, and early intervention relevant to the specific etiology. Correct diagnoses include radiological and clinical examinations and possibly tooth size evaluation.

• No treatment is usually done, if the diastema is physiological/transient as it spontaneously closes after the eruption of permanent maxillary canines.

Spontaneous correction of a childhood diastema is most likely when it width is not more than 2 mm.

• Pathological causes like supernumerary teeth midline soft tissue anomalies can be removed surgically and spaces are closed orthodontically. Oral habits such as thumb sucking and tongue thrusting should be corrected before closure of the space.

• Esthetic approach: patient demand for aesthetic dentistry with minimally invasive procedures has resulted in the extensive utilization of freehand bonding of composite resin to anterior teeth.

When the teeth are in proper orthodontic alignment, no preparation of the tooth structure is necessary. If there is an alignment problem, minor tooth preparation will be necessary to achieve proper arch form(**LN Sunil Kumar et al,2013**)

2.30 Retention of the result and prognosis:-

Relapse of the maxillary midline diastema appears, according to Sullivan et al. (1996),30 in almost 34% of cases, while ,according to Shashua and Artun (1999) this rate rises to50%. The reason for relapse is the placement of teeth in a position where no equilibrium exists with their functional environment. In most of these cases, the factor disturbing this equilibrium is still present after treatment .Shashua and Artun (1999) concluded that the most important risk factors for relapse are the increased pretreatment width of the midline diastema, the presence of a family member with a similar condition, and the presence of more than one diastemas in the maxillary anterior region .However, according to Sullivan et al. (1996), no pre-treatment predictors of relapse can be established. As a general rule, treatment is unlikely to produce assured and stable results, thus the use of permanent retention for a considerable period of time or even for life, is essential in almost every case. The most appropriate method for achieving long-term retention after orthodontic treatment is through the use of palatally bonded multi-stranded stainless steel wire retainers, which

allow teeth to maintain their physiologic mobility and are easy to fabri-cate. In cases where the retainer interferes in functional movements of the mandible, it can be bonded cervically or within a shallow rim constructed in the enamel of teeth. **(NikolaoA et al,2008).**

Reference

1. Agarwal A, Kapahi R. Labial Frenectomy through Z Plasty. J Clin Diagnost Res. 2012; 6(3):537-8.
2. Al-Rubayee MA. Median Diastemain a College Students Sample in the Baghdad City. Medical Journal of Babylon. 2013; 10(2):400-6.
3. Baart JA, Bosgra JF. A too short superior labial frenum. Ned Tijdschr Tandheelkd. 2004; 111(5):174-8.
4. Balogh MB, Fehrenbach, MJ. Illustrated Dental Embryology, Histology, and Anatomy. Philadelphia: Elsevier; 2011:PP.41.
5. Bishara SE. Text book of orthodontics. WB. Saunders Company, 2001, p.13.
6. bKumar M. Midline Diastema. Slid share. dentist at Bapuji Dental College & Hospital & college of dental sciences, davangere. Feb 16, 2011. https://www.slideshare.net/kumarmanish0001/midline-diastema
7. Diaz-pizan ME, Lagravere MO, Vallena R. Midline Diastema and frenum morphology in the primary dentistry. J dentistry. 2006; 26:11-14.
8. Dixon V, Reud MJF, O'Brien KD, Worthington HV, Mandail NA. A randomized clinical trait to compare three methods of orthodontic space closure. Am J Orthod-Dentofac Orthop 2002; 29: 31-6.
9. Gass JR, Manish V, Hemant K, Tiwari HK, Hans MG, Elston RC. Familial correlations and heritability of maxillary midline diastema. Am J Orthod-Dentofac Orthop 2003; 123(1): 35-9.
10. Giradot RA. Comparison of condylar position in hyperdivergent and hypodivergent facial skeletal types. Angle Orthod 2001; 71(4): 240-246.
11. Gruenbaum, Tamar. Famous Figures in Dentistry Mouth – JASDA 2010;30(1):1

12. Hashim MH. CONTRIBUTING FACTORS OF MEDIAN DIASTEMA IN IRAQI ADULTS. Thesis: Master. Iraq: University of Baghdad, College of Dentistry, Department of Orthodontics; 2006: PP.1-124.
13. Jindal V, Kaur R, Goel A, Mahajan A, Mahajan A, Mahajan A. Variations in the frenal morphology in the diverse population: A clinical study. Indian Society of Periodontology. 2016; 20(3):320-3.
14. Korkut B, Korkut D. Anterior Aesthetic Build-Up and Diastema Closure Procedures Supporting Orthodontic Treatment: 2 - Case Reports. PARIPEX -INDIAN JOURNAL OF RESEARCH. 2015; 4(11):14-16.
15. Kotlow LA. Diagnosing and understanding the maxillary lip-tie (superior labial, the maxillary labial frenum) as it relates to breastfeeding. J Hum Lact. 2013; 29(4):458-64.
16. Lindsten R, Ogaard B, Larsson E. Dental arch space and permanent tooth size in the mixed dentition of a skeletal sample from the 14th to 19th centuries and 3 contemporary samples. Am J Orthod-Dentofac Orthop 2002; 122(1): 48-58.
17. LN Sunil Kumar, P Nagmode, V Tambe… - Journal of Dentofacial …, 2013.
18. Mahmoud FS. Hypodontia of primary teeth in a sample of children aged 2½-5½ years in Baghdad city. A Master thesis, College of Dentistry, Baghdad University. 2004.
19. Mitchell L, Carter NE, Doubleday B. An introduction to orthodontics. 2nd Ed. Oxford medical population, 2004, p. 29, 89.
20. Mohan R, Soni PK, Krishna MK, Gundappa M. Proposed classification of medial maxillary labial frenum based on morphology. Dental Hypotheses. 2014; 5(1):16-20.
21. Muhamad Abu-Hussein*, Nezar Watted* IOSR Journal of Dental and Medical Sciences (IOSR-JDMS) e-ISSN: 2279-0853, p-ISSN: 2279-

0861.Volume 15, Issue 6 Ver. II (June. 2016), PP 116-130 www.iosrjournals.org

22. Nikolao Gkantisdis , Olga-Elpis Kolokitha, Nikolaos Topouzelis ***The Journal of clinical pediatric dentistry 32(4):265-72 · February 2008 with 2,379 Reads
23. Nikolaos Gkantidis , Olga-Elpis Kolokitha ,Nikolaos Topouzelis… in The Journal of clinical pediatric dentistry 32(4):265-72 · February 2008 with 2,383 Reads
24. Olayemi AB. Assessment and determination of human mandibular and dental arch profiles in subjects with lower third molar impaction in Port Harcourt, Nigeria. Ann Maxillofac Surg. 2011; 1(2): 126–130.
25. OM Tanaka, AYK Morino, OF Machuca… - Case reports in …, 2015
26. P Nagmode, V Tambe, S Gonmode… - MIDLINE DIASTEMA: …, 2012 - jemds.com
27. Peck S, Peck L, Hirsh G. Mandibular lateral incisor-canine transposition in monozygotic twins. ASDC J Dent Child 1997; 64(6): 409-13.
28. Proffit WR, Fields HW JR, Sarver DM. Contemporary Orthodontics. 4th ed. Philadelphia: Elsevier; 2007.
29. Proffit WR. Contemporary orthodontics. 3rd Ed. Mosby year book, 2000, p. 10-12, 101-106, 166-170, 170, 586-587.
30. R. Chałas, MD, PhD, Department of Conservative Dentistry and Endodontics, Medical University of Lublin, ul. Karmelicka [Received 2 November 2014; Accepted 18 January 2015]
31. Robertson S, Mohlin B. The congenitally missing upper lateral incisor: A retrospective study of orthodontic space closure versus restorative treatment. Europ J of Orthod 2000; 22: 697-710.

32. SK LN, P Nagmode, V Tambe, S Gonmode… - MIDLINE DIASTEMA: …, 2012 - jemds.com
33. Suri S, Utreja A. Management of a hyperdivergent classIII malocclusion, maxillary midline diastema, and infected mandibular incisors in a young adult. Am J Orthod- Dentof Orthop 2003; 124(6): 725-734.
34. Tarvade SM, Ramkrishna S. Tongue thrusting habit: A review. International Journal of Contemporary Dental and Medical Reviews (2015), Article ID 151214, 5 Pages.
35. Tavajohi-Kermani H, Kapur R, Sciote J. Tooth agenesis and craniofacial morphplogy in an orthodontic population. Am J Orthod-Dentofac Orthop 2002; 122(1): 39-47.
36. Thosar N, Murarka P, Baliga S, Rathi N. Assessment of maxillary labial frenum morphology in primary, mixed, and permanent dentitions in Wardha district. European Journal of General Dentistry. 2017; 6(1): 14-17.
37. U Hussain, A Ayub, M Farhan - Pakistan Orthodontic Journal, 2013 -
38. U Hussain, A Ayub, M Farhan - Pakistan Orthodontic Journal, 2013
39. Wall LG. Treatment options for peg-shaped laterals using direct composite bonding. INT DENTI SA. 2013; 12(1):26-33.
40. Warotayanont PR. 6 keys to normal occlusion. (UCSFPEDO) UCS pediatric dentistry. Jan 30, 2011. https://ucsfpedo.wordpress.com/2011/01/30/6-keys-to-normal-occlusion/
41. You ZH, Fishman LS, Rosenblum RE, Subieiny JD. Dentoalveolar changes related to mandibular forward growth in untreated CL II persons. Am J Orthod- Dentofac Orthop 2001; 120 (6): 598-607.