


INTEROSSEOUS SEPTUM AND MIDLINE DIASTEMA RELATIONSHIP



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## TABLE OF CONTENTS

|                                    | PAGE |
|------------------------------------|------|
| INTRODUCTION . . . . .             | 4    |
| REVIEW OF THE LITERATURE . . . . . | 6    |
| MATERIALS AND METHODS . . . . .    | 24   |
| FINDINGS . . . . .                 | 27   |
| DISCUSSION . . . . .               | 31   |
| SUMMARY AND CONCLUSIONS . . . . .  | 36   |
| BIBLIOGRAPHY . . . . .             | 39   |
| TABLES I - VII                     |      |
| APPENDIX A                         |      |

## INTRODUCTION

The maxillary midline diastema is frequently observed in the normal dentition. Taylor<sup>1</sup> describes the existence of a maxillary midline diastema as normal in 98% of 6 to 7 year old children. Since this space is often viewed with concern by parents, an orthodontist will see many such cases during his years in practice. Fortunately only about 7% of a 12 to 18 year old population sample retain these diastemas.

Although the majority of midline diastemas resolve uneventfully through normal growth and development, the remaining cases and their treatment are cause for much controversy and confusion. The reason for this is a lack of detailed clinical studies concerning maxillary midline diastema. Most of the literature written and treatments suggested are empiric in nature.

The presence of a midline diastema may be attributed to numerous causes. Careful diagnosis will help to assure that the cause of a diastema is accounted for or removed while under orthodontic supervision.

Although usually not difficult to obtain mechanical closure of the interincisal diastema, it concerns the orthodontist because of a tendency of some of these spaces to relapse.

Many factors can contribute to the reopening of maxillary anterior diastemas. The following have been offered as possible reasons:

- 1) improper axial inclinations of the roots of the central incisors,
- 2) tooth size discrepancies, 3) pernicious habits, 4) deleterious occlusal patterns which would tend to place lateral forces on the central incisors in shifts from centric relation to centric occlusion or during any of the other excursions of the mandible, 5) the actual anatomy of the teeth, 6) possibly even muscular imbalances in the oral region, and 7) the superior labial frenum and/or its associated interdental soft tissues.

The purpose of this investigation is to attempt to study the osseous septum between the central incisors as it relates to the relapse phenomenon observed after the orthodontic closure of diastemas.

## REVIEW OF THE LITERATURE

A form of malocclusion which is characterized by a space between the upper central incisors has long been recognized in orthodontics.

In 1899 Edward Angle<sup>2</sup> summarized his beliefs and treatment methods in the Dental Cosmos. He found closing the space to be a comparatively simple operation, yet admitted that these cases were well known to be annoying cases to treat on account of the difficulty of permanently establishing the teeth in their corrected positions. He felt that in nearly every instance the relapse which a diastema case exhibited could be traced to an abnormal attachment of the frenum labii. Angle stated, "This strong ligamentous cord not only prevents the space from closing by its passive presence, but exerts an active mechanical influence as well, each movement of the lip exerting a lateral tension upon the cord, which tends to separate the teeth still further".

Angle's goal of treatment was to remove or modify the ligament so that it could no longer act mechanically upon the teeth. He found partial success by mere severing of the frenum with a pair of scissors, union of the ends while healing being prevented by occasional manipulation. The more popular and successful method of treatment at the time for removal of an abnormal frenum involved cautery. A deep incision with a cautery knife was made splitting the cord. This was preceded by a

suitable lancet or bistoury, in order to better control the direction of the cautery instrument. After surgery the teeth were drawn together and mechanically supported for several weeks or months.

At the time Angle thought that it was probable that the abnormal frenum might be due to an abnormal suture. From 1905 to 1907 Dr. Ketchum<sup>3</sup> conducted a study of this subject involving 48 patients. The conclusion of Dr. Ketcham was that the attachment of the frenum, whether normal or abnormal, was in no way governed by the normality or abnormality of the suture.

Martin Dewey<sup>4</sup> wrote an article in 1918 in which he discussed what he believed caused a frenum to have an abnormal attachment. His observations led him to believe that the abnormal frenum may be congenital in some and in others it may be acquired. "Where we find the frenum separating the deciduous incisors it may be considered as a congenital condition. In those cases where the frenum seems to be acquired we find that the abnormal frenum makes its appearance about the time of the eruption of the permanent incisors." In both situations Dewey believed that the frenum grew occlusally as the teeth and alveolar process moved occlusally. After examining a number of frenum cases, he found three distinct types based on crown and root location, frenum width, and location of attachments.

Dewey and also Engstrom<sup>5</sup>, who concurrently published a surgical article on frenectomies, believed that the three types of frenum would have to be treated differently, both in regard to operative procedure and orthodontic treatment. One surgical procedure described was performed on a child seven and one half years of age with unerupted lateral incisors.

These articles exemplify the diversity of opinion and confusion that

could be found in the literature on diastema.

In 1925 Harold Vaughan<sup>6</sup> wrote that the usual practice of cauterizing the frenum was not the way to obtain the best results. He recommended submucous excision of the fibrous band as did Engstrom, leaving only a linear incision. Early correction of these cases, at around eight or ten years of age was suggested treatment.

In a 1926 article Patton<sup>7</sup> recommended that the space between the incisors be two-thirds closed before the frenum should be excised surgically. He also advocated cleaning deep down in the fissure with a small curette or spoon excavator.

A number of cases presenting separated central incisors in Cecil Tait's<sup>8</sup> practice led him to an investigation of the median frenum of the upper lip in 1929. At that time he was not able to find any reference as to the exact mode of formation of the frenum. From literature at his disposal, he was able to formulate these general facts. During the second month of fetal life, the two globular processes fuse with each other and also with the maxillary process on either side. The fused globular processes eventually give rise to the middle third of the upper lip and a portion of the premaxillary part of the upper jaw; it is then, in the mass of tissue formed by the globular processes that the frenum is developed. The frenum is composed of mesoderm with a covering of ectoderm. It is still underdeveloped at about the third month. In older stages, two papilla are situated in the median line: a papilla palatina and a papilla labii superioris. These are connected with each other by the frenum tecto-labiale. About the fourth month the well marked frenum joins the papilla palatina through the cleft formed by the as yet



unfused alveolar process of the premaxilla. When the alveolar tissues have become differentiated and bone formation becomes evident, the divided alveolus of earlier months becomes fused. The alveolus is built up pushing the frenum before it.

Normally, the growing alveolar process causes a severance of the continuous fold of tissue, dividing it into a palatal and labial portion. The palatal part corresponds to the palatina papilla, and the labial tissue becomes the superior labial frenum, extending from the lip to the crest of the alveolar ridge.

Upon eruption of the deciduous teeth, the crestal part of the frenum tends to disappear, due not only to pressure, but also to the rapid increase in depth of the alveolus. With eruption of the permanent teeth, the frenum becomes attached higher up on the alveolus.

Tait made clinical observations on frena at different stages from fetus to adult. In 75 fetuses he found that the frenum was present in all but two cases. He found that variations of the frenum can be seen at all stages of development. In youths and adults, all degrees of frena associated with various types of dentition could be observed. The general tendency in youths and adults was for the gradual disappearance of the frenum.

Ketcham<sup>9</sup>, 1932, was also a proponent of first moving the central incisors together, then he would remove the appliance permitting the teeth to separate for an hour or so before the cautery operation, then closure. However, he found that if the centrals were drawn together and retained until the eruption of the permanent canines, the necessity for a frenectomy was often obviated. He would only operate in those cases in which

the centrals separate after removal of the retainer or where the abnormal attachment was very pronounced. He advised against immediate surgical interference in cases of wide separation of deciduous central incisors.

In the same year, 1932, Leroy Johnson<sup>10</sup> wrote, "The space between the incisors is, in most instances, a perfectly normal condition and if not associated with other evidence of malocclusion will usually disappear during the completion of the permanent denture. Most of the men I know, no longer consider an abnormal frenum as a cause of malocclusion." He also said that he would not think of removing a frenum by use of cautery because of the trouble with creation of scar tissue.

Harold Noyes<sup>11</sup> undertook an extensive macro and microscopic study of the frenum labii in 1935 on newborn infants. The tissue of the frenum labii springs from a relatively wide origin on the inner surface of the upper lip; presents a smooth septum which narrows in width as it progresses posteriorly to be inserted at the midline in the outer layer of the periosteum and the connective tissue of the intermaxillary suture and the alveolar process. The ridges of the latter structure are less prominent at this point forming a groove in which the dorsal extremity of the frenum lies. A smaller and somewhat similar structure arises from the incisal papilla of the palate and passes anteriorly to be inserted in this groove. Considerable variation in the size of the frenum was noted. Histologically, Noyes found that the frenum is composed mostly of alveolar and fibrous connective tissue with a few striated muscle fibers which arise from the muscle bundles of the lip on either side of the 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 anterior-posterior direction as it nears the posterior 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 with branches which are given to the frenum. These travel in an anterior-posterior direction, providing the blood supply of the structure. Nerve filaments accompany the vessels. Noyes observed that while in the normal tissue chosen for section, no continuity could be observed in the anterior-posterior fibers of either the labial or lingual structures; they are more intimately related superficially. This he felt supported the hypothesis that the abnormality of a continuous frenum might reveal a continuity of the fibers of these tissues. He stated, "If we are correct in assuming that the adult position of the normal frenum attachment is the result of growth of the alveolar process, it might be further hypothesized that the interference to occlusal growth of a band of connective tissue at this point might cause the diastema between the central incisors and the deformity of the midline suture commonly observed in patients who present this abnormality".

Enayat Shirazy<sup>12</sup> in 1938 commented that the frenum labii superioris may be abnormally developed from hypertrophy with or without the definite separation between the first maxillary incisors. He stated that, "the proper time for correcting this unsightly deformity is after the permanent cuspids have completely erupted." He was aware that others like Schwartz and Abbott<sup>13</sup> disagreed with surgical intervention on account of the scar tissue which follows an operation. This they felt prevented adequate

orthodontic approximation of the separated incisors. Shirazy felt that the scar tissue developing after obliteration of the diastema should aid in keeping the incisors in proper approximation.

Kelsey<sup>14</sup> was of the opinion in 1939 that the fibers of the frenum under no circumstances ever pass through the premaxillary median suture in the prenatal individual, but that they do become enveloped by it as the alveolar process grows downward. "The deep labial furrow which appears between the alveolar sockets of the two central incisors show clearly to my mind that not only have the teeth themselves separated, but they have reconstructed their socket walls. This furrow or groove to a great extent disappears when the frenum has been properly corrected and the anterior teeth have been drawn together."

B. F. Dewel<sup>15</sup> wrote an excellent article in 1946 on differentiation of the normal and abnormal frenum. By this time adequate attention had been given in the literature to the favorable processes of normal development for the frenum and of the natural inclination of the frenum to degenerate with age. An article was really needed at this time which could clear some of the confusion about the frenum in its deviation from the normal. Dewel felt that the frenum presents two different but confusing deviations from the normal. The point is stressed that a truly abnormal frenum is rare and must not be confused with the frequently recurring enlarged frenum that either has not had time to atrophy or persists because of other conditions present. In both of these conditions there is an associated disturbance in growth and development throughout the entire premaxillary area. Dewel stresses that an enlargement of the frenum, instead of being the cause of separation of the central incisors, might actually be the result

of such a separation. The frenum develops in the fetus as a comparatively massive structure. Thereafter, it is characterized by persistent recessive developmental tendencies. No frenum should be considered abnormal until every cause of a separation has been eliminated. Abnormal tongue pressure, lip habits, absence of lateral incisors or presence of peg-shaped lateral incisors, and certain forms of malocclusion are among the causes of separation of the central incisors. Dewel recommended attempting to close space by orthodontic means. If tissue hypertrophy instead of pressure atrophy resulted, convincing evidence was finally obtained for a diagnosis of a true abnormality of the frenum. If present, an indisputable abnormal frenum is found to be markedly enlarged. Other criteria which Dewel lists include a wide fan shaped attachment to the upper lip which tapers downward to a distinct sheetlike process extending between the central incisors to a definite union with the palatine papilla. If by applying tension to the upper lip, blanching and a definite amount of movement of the interdental tissue and palatine papilla is produced, the frenum may be considered abnormal, provided it can be traced distinctly as a continuous structure, that sufficient time has elapsed for normal developmental forces to assert themselves and that all other possible causes of separation have previously been eliminated. Even with present available knowledge, Dewel felt it difficult at times to distinguish between a simple enlarged frenum and an authentic abnormal labial frenum. Dewel did not oppose judicious surgery but he feared little attention was paid to the danger of scar tissue or possible damage of transeptal fibers of the periodontal membrane.

Morven Curran<sup>16</sup> believed that many labial frena were resected unnecessarily and that proper diagnosis should be made emphasizing the location

of the frenum and the age of the patient. A condition which would indicate an abnormal frenum in a patient 11 to 12 years of age when the laterals and cuspids have erupted might be normal in a younger patient. He also felt that the separation of the upper central incisors is not the result of the abnormal frenum per se, but of the constant movement of the frenum and it is necessary to do only a partial resection, severing the connection between the basal portion of the frenum and interseptal tissue.

Baume<sup>17</sup> stated that certain spaces in the deciduous denture are congenital. This might be a factor in some of the midline diastema cases.

Bedell<sup>18</sup> listed many cases of the midline dental space.

In 1952 Robert Moyers<sup>19</sup> wrote an article which attempted to give dentists a broader view of the causes of spacing between the maxillary central incisors. He felt that there has been entirely too much emphasis upon the role of the labium frenum in such cases. His comments concerning diagnosis of a true malposed labium frenum include: "This condition sometimes may be detected by noting the blanching of the soft tissues as the frenum is stretched by the lifted lip. However, it is impossible to diagnose all frenum problems in this manner. The ultimate answer may always be found in the radiogram. The normal osseous septum between the central incisors is V shaped. When the labium frenum inserts on the palatal side of the septum, the fibers run across the bone rounding it over so that the septum is spade shaped. Even when the fibers insert so deeply as not to cause blanching, the condition may be diagnosed in the radiogram."

The radiograph can also be used in diagnosis of another cause of spacing, the incomplete fusion of the median palatine processes. A small

bit of periosteum is invaginated between the central incisors in these cases. This causes a separation of the septal bone which is readily apparent where the septum is seen to be W shaped. This separation may be shallow or it may continue high into the alveolus. Moyers advocated scalpel excision after first bringing the incisors together as treatment for a malposed labial frenum. Treatment for incomplete fusion of median palatine processes is excision of the invaginated periosteum rather than the frenum's fibers. "One satisfactory method is to lift a V shaped mucosal flap directly over the septum. One may then gently insert a surgical fissure or tapered fissure bur into the osseous cleft. With the motor turning at a slow speed the bur will not only remove the invaginated periosteum but will freshen the edges of the bone as well.

Occasionally the frenum will insert into the cleft. The fibers should be excised and the cleft cleaned in the same manner. Always hold the central incisors together until healing has taken place. The radiogram will now reveal a normally shaped septum."

Peter Ceremello<sup>20</sup> studied frena on plaster casts of patients with a diastema and without a diastema present. He found that the frenum has a range of normality. It varies in bulk from a heavy mass to a thin fold of tissue and in attachment height from near the alveolar crest to high above it. His findings stressed the independent character of the frenum and the midline diastema and their lack of correlation. He states that, "more often than not, the midline diastema can be attributed to a normal developmental origin, and that it usually will close with eruption of the succeeding teeth".

He writes that normal or developmental spacing of the maxillary

permanent central incisors may originate at the time the teeth are still in their bony crypts. The crypts of these teeth are often separated by the intermaxillary suture. This bony separation may well act as a barrier, preventing the teeth from approximating each other and causing the teeth to start downward with a space between them when the process of eruption is initiated. Another factor that affects the position of the teeth is that they may be so bunched while in their crypts that the apical portions crowd together causing the coronal portions to flare outward. This places the teeth in position to erupt along a diverging path, and as they erupt, they become more and more spaced. The eruption path of the maxillary canines also affects spacing of the incisors. During the early stage of eruption the crowns of these teeth press mesially on the roots of the lateral incisors creating or increasing space between these teeth. Spacing of incisors may also result from an increase in size of the anterior portion of the alveolar process during the eruption stage of the incisors.

Eruption, migration, and physiological readjustment of the teeth; labial and facial musculature; development of musculature of the cheeks and lips; 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.

Ceremello believed that the abnormal frenum should retain all the characteristics of the tectolabial frenum of fetal life.

The periodontium and its relationship to orthodontic treatment has been considered by many men particularly with respect to relapse.



Kaplan, Aisenberg, and Erickson<sup>21</sup> found that transeptal fibers are remarkably persistent in extraction sites. Elongated transeptal fibers appear in these spaces. When teeth opposite such spaces are brought into approximation the transeptal fibers relax, coil, and then become compressed. No physiologic process exists which shortens or removes the excess of these scarlike fibers. They concluded that transeptal fibers can pull teeth to which they are attached in the direction of an applied force. In 1948, Aisenberg<sup>22</sup> stated that the subepithelial tissues, which ordinarily do not yield to tooth movements, as does bone, constitute a major factor in relapse.

Thompson<sup>23</sup> was the first investigator in recent years to emphasize the potential effect of the supra-alveolar fibers on relapse and to advocate soft-tissue surgery to enhance retention. He stated: "We sincerely believe that the inherent forces of these fibers do exert an influence on our results." Thompson was also one of the first investigators to surgically remove the excess tissue between approximated central incisors following diastema correction. He also made incisions on the distal aspects of both incisors and advocated that the relapse which he experienced could have been due to the undisturbed labial and lingual fiber arrangement.

Following the classic precedent set by Thompson, other investigators began to use surgery as a means to enhance retention. Ewen and Pasternak<sup>24</sup> chided the clinical orthodontist for failure to add soft-tissue surgery to his armamentarium as an adjunct to relapse prevention. They presented five cases which relapsed following closure of central diastemas. They found that only two cases showed mild relapse following retreatment and

surgical removal of the intervening tissues. The surgical method was a reverse bevel incision gingival resection, which included one and one-half teeth beyond the central incisors.

Wiser<sup>25</sup>, Brain<sup>26</sup>, and Boese<sup>27</sup> also found that soft tissue surgery was an adjunct to retention.

Edwards<sup>28,29</sup> advocated gingival surgical techniques to prevent relapse on rotated teeth and in extraction sites in his earlier articles.

Campbell, Moore, and Matthews<sup>30</sup> conducted a histologic investigation of the interincisive soft tissue in an attempt to present some possible explanation for diastema relapse. They concluded that the interincisive soft tissues and their associated fiber arrangement, as well as the contiguous fibrous network of adjacent areas, provide significant resistance to orthodontic tooth movement. When a midmaxillary interincisal diastema is orthodontically corrected, the interincisive soft tissue undergoes a stretching stress. These men believed that oxytalan with elastic qualities is produced in these areas of stress and that it apparently causes the inelastic connective tissues to rebound as if they were elastic. This rebound on both the mesial and distal aspects of the central incisors may contribute to diastema relapse. Their preliminary findings favored a combination of frenectomy and circumferential supracrestal fiberotomy as the method of choice to enhance retention of orthodontically corrected interincisal diastemas since it does not result in loss of fibrous periodontal support and is relatively simple to accomplish.

To shorten the active treatment time in reduction of multiple anterior diastemas, surgical intervention was proposed by Kole<sup>31</sup> in 1959. He

used vertical corticotomies through the labial and palatal cortices to the mesial and distal of the tooth or teeth involved. After surgery, standard orthodontic appliances and techniques were used. Correction was much more rapid than by orthodontic treatment alone.

Immediate closure of a single labial diastema by frenectomy and maxillary ostectomy was outlined by Clark<sup>32</sup> in a 1968 report. This surgical procedure had been used in more than a thousand cases with few complications over an eight year period. Frequently the procedure was performed in conjunction with orthodontic treatment. Within three months, the central incisors were stabilized in juxtaposition.

In 1970, Bell<sup>33</sup> reported a case in which multiple diastemas were closed by surgically repositioning teeth as individual segments. He described the creation of tooth bone segments that could be repositioned to a predetermined alignment. An edgewise orthodontic appliance was used for stabilization.

In the same year, Peterson<sup>34</sup> described an application and extension of Bell's technique. An acrylic lingual splint was used instead of an edgewise orthodontic appliance; the splint served both as an index and as a fixation device.

The definitive treatment of maxillary diastema with use of anterior maxillary osteotomy and midpalatal ostectomy was presented by Cole and Staples<sup>35</sup> in 1973. Custom-made Ticonium arch bars allowed for stable fixation that was esthetic and hygienic.

In 1969, Sanin, Sekiguchi, and Savara<sup>36</sup> introduced a method that permits the clinician to determine the probability of complete closure

of a central diastema. The prediction was accurate in 887 of the cases presented. Their findings suggest that patients with a diastema at completion of the permanent dentition had a larger diastema in the mixed dentition than the patients who completed closure and the spaces between the central and lateral incisors as well as the intercanine distance are considerably larger. They concluded that the earliest age in which the presence of a diastema at completion of the permanent dentition can be predicted was the age at which the central and lateral incisors were fully erupted.

Good reviews of the diastema problem have been written by West<sup>37</sup>, Bishara<sup>38</sup>, and Becker<sup>39</sup>. Bishara categorized diastemas by etiology and presented possible treatment for each type. He stressed the need for removal of the cause and proper methods of treatment mechanics. He also accepted permanent retention and even non-treatment in some cases.

In 1972 Bergstrom, Jensen, and Martensson<sup>40</sup> conducted an investigation into the possible contribution of a superior labial frenum to the occurrence of a persisting midline diastema. Forty school children 8 to 9 years old with prominent frena and midline diastema of more than 1 mm were subdivided into two groups. One group was subjected to frenectomy; the other served as a control group. Two years after the start of the study, the diastemal closure was significantly greater in the frenectomized group. However, the difference in closure between the two groups was gradually reduced, and after ten years there was no longer any significant difference. The investigation revealed that further closure at such a late age as 14 to 19 years can occur.

Higley<sup>41</sup> felt that the slight cleft of interseptal bone did much to

hold the teeth apart, and that removal of the periosteal lining of the cleft would permit bone to develop in the cleft and enable the teeth to come together.

Stubley<sup>42</sup> implicated improper formation of transseptal fibers in a certain type of upper midline diastema which he calls "persistent median diastema". In this condition, the cause is due to the presence of a persisted midline suture which interferes with the normal transseptal fiber arrangement. Instead of passing directly across the interdental space from one tooth to the other, after leaving the mesial surface of each central incisor, the fibers turn upward at right angles to their normal path to enter the suture parallel to those coming from the opposite side. Such an arrangement is equivalent to a break in the transseptal fiber chain. Because the lack of continuity has robbed the mesial transseptal fibers of their balancing pull, the fibers on the distal sides of the developing central incisors can move these teeth a considerable distance apart. Stubley believed that an essential part of treatment of persistent median diastema would be an operation to clear the upper suture of transseptal fibers.

From analysis of histories, dental casts, and cephalograms of 471 children, Popovich, Thompson, and Main<sup>43</sup> drew some of the following conclusions in 1977. The primary cause of persistence of a maxillary diastema appeared to be generalized spacing; with the intermaxillary suture and a related low, thick frenum acting as secondary factors. A low attachment frenum was associated with spacing and with a suture which they described as either type 2, 3, or 4. Type 2: bone with wider than normal open suture. Type 3: spade shaped bone between

centrals bisected by an intermaxillary suture. Type 4: W-shaped bone with a deep open suture. It was their conclusion that the larger the diastema at age 9 years, the less likely it will close by age 16, especially when it is associated with generalized spacing, a low attachment frenum, and a suture of type 2, 3, or 4.

Also in 1977, Edwards<sup>44</sup> attempted to study the maxillary anterior frenum as it relates to the relapse phenomenon observed after closure of diastemas and develop an effective method of eliminating the possible influences of frenal tissue on relapse. He found that the pretreatment relationship between a clinically "abnormal" appearing frenum and a diastema showed a strong but not absolute correlation. Diastema cases in which there were "abnormal" pretreatment frenums demonstrated a stronger potential for relapse after closure. But again there were exceptions. Edwards believed that the orthodontist is apparently unable to distinguish between the diastema cases which will relapse and those which will not. The surgical techniques examined in this study showed a dramatic benefit in reducing the problems of relapse following orthodontic treatment. However, they were not effective in 100 per cent of the patients. The surgical techniques outlined were comprised of three separate procedures. 1) Apical repositioning of the frenum with denudation of alveolar bone, 2) destruction of transseptal fibers between approximated incisors, and 3) recontouring of the labial and/or palatal gingival papillae in cases of excessive tissue accumulation.

A small percentage of diastemas continue to open despite the orthodontist's best efforts after retainers are removed, therefore permanent retention has been suggested by some as a reasonable treatment option.

Andreason and Johnson<sup>45</sup> suggested a cemented nonparallel lingual pin retainer for permanent retention. Baum and Marshall<sup>46</sup> in 1972 suggested a method of rigid retention which is esthetically undetectable, has rigid retention and is completely compatible with good oral hygiene. They demonstrated the use of a cast lingual bar retainer with pins which inserted into parallel holes in prepared inlays in the teeth. A technique which involved bonding teeth together was described by Kaswimer<sup>47</sup> in 1973. Chan<sup>48</sup> and Andreasen described a retention method accomplished by use of lingual staples covered with a composite restorative material. Young<sup>49</sup> suggested use of a fine mesh which is placed between the central incisors on the lingual and bonded in place.

Thus it can be concluded that the differential diagnosis, treatment, and retention of the maxillary central diastema problem is multifactorial and only poorly understood.

## MATERIALS AND METHODS

The materials for this study consisted of study models and periapical x-rays of 22 cases treated in the orthodontic department of the University of Oregon Health Sciences Center. The cases were treated by graduate students using fully banded edgewise (.022 bracket) appliances.

In each case, three sets of study models were examined. The first set of models was taken before orthodontic treatment was instituted (designated time 1). The second set of models was taken after active orthodontic appliances were removed and retainers placed (time 2). A third maxillary model was taken as long after retention had been discontinued as was possible.

For each patient, a pretreatment periapical x-ray of the central incisors was examined to determine the anatomy of the interosseous septum.

The sample consisted of 13 females and 9 males. The breakdown of the sample according to Angle's classification was: Class I - 2 cases, Class II - 16 cases, and between Class I and Class II or end to end - 4 cases.

The mean age of patients at the start of treatment was 12.8 years with a range of 11.0 years to 15.7 years. The mean age at the end of treatment was 15.4 years with a range of 13.2 years to 18.5 years. The mean time of treatment was 2.5 years with a range of 1.8 years to 4.4 years. The mean time in retention was 2.2 years with a range of 0 years for one patient to 3.7 years. The mean time out of retention was 6.2 years with a range of



1.3 years to 13.9 years.

Measurement of the maxillary midline diastema was not taken in cases with impacted teeth, absence of any maxillary anterior tooth, or presence of other midline anomalies. The size of the central diastemas was measured with a John Bull millimeter micrometer accurate to 1/10 of a millimeter. The measurement was taken at the junction of the middle and incisal thirds of the crowns to avoid the rounded mesio-incisal angles. Measurement was taken from a plaster cast obtained by an alginate impression. The gauge was held perpendicular to the labial surfaces of the central incisors.

Patients were divided into three groups according to the anatomy of the osseous septum between their central incisors. A periapical x-ray was studied for each patient and they were classified as belonging to one of three types according to the following definitions:

Type 1 Normal osseous septum which is V shaped between the maxillary central incisors and bisected by the intermaxillary suture, which sometimes is not visible in the radiograph.

Type 2 Osseous septum which is spade shaped between the maxillary central incisors.

Type 3 W-shaped interdental osseous septum in which the separation of the osseous septum may be shallow or continue well into the alveolus. Or bone with wider than normal open suture. The suture is involved in all cases.

#### ERROR OF THE MEASURE

The standard error of the measure was determined from 12 randomly

selected pairs of models. Replicate measurements of the maxillary diastemas were taken on each cast at two different time periods. One set of 12 casts was measured in random order during the first time period. Then the same set of 12 casts was placed in a new random order and measured again during a later second time period. Measurements were taken using a John Bull millimeter micrometer. The standard error of the measure was computed using the following formula:

$$\begin{aligned} \text{S. E. Meas.} &= \sqrt{\frac{\sum (X_1 - X_2)^2}{2N}} \\ &= .09 \text{ mm} \end{aligned}$$

## FINDINGS

In this study 22 patients were divided into three types based on pretreatment x-rays of the osseous septum between the central incisors.

1. Patients were tested for significant difference between type 1, 2, and 3 at each time interval, that is before, treatment, after treatment, and after retention.
2. All patients were also tested for significant difference in diastema size at time periods 1, 2, and 3.
3. Each group of patients was also tested to see if there was any significant difference in frequency of observed diastema relapse between time 2 and time 3.

Table I was the source of data from which all computer computations and statistical analysis were performed.

It was possible to use analysis of variance to test for significant difference in size of the diastema for type 1, 2, and 3 at time 1 because the assumptions of normal distribution and homogeneity of variances in the groups were met. Because of a high incidence of the same value (zero) at time 2 and time 3, necessary assumptions were not met and that data did not lend itself to ANOVA. The results of ANOVA for time 1 are found in summary Table II-A.

The critical table value for F with df of 2 and 19 was found for

alpha = .05 to be 3.52. Since our computed value for  $F = 3.74$ , we must reject the null hypothesis that there is no significant difference in the mean diastema size in the three groups of patients.

Since the ANOVA was significant the various combinations of treatment means were investigated.

The Scheffe test was used to make all possible comparisons between individual pairs of means. It consisted of computing an F statistic for each comparison using the formula:

$$F = \frac{(\bar{X}_1 - \bar{X}_2)^2}{\text{MSE}(n_1 + n_2)/n_1 n_2}$$

Information from Table II-B was used to compute these formulas:

$$\begin{aligned} \bar{X}_1 &= \bar{X}_2 & F &= \frac{(1.3833 - 2.2167)^2}{0.2969(6 + 6)/(6)(6)} \\ & & &= \frac{(-0.8334)^2}{.099} \\ & & &= 7.04 \end{aligned}$$

$$\begin{aligned} \bar{X}_2 &= \bar{X}_3 & F &= \frac{(2.2167 - 1.640)^2}{0.2969(6 + 6)/(6)(6)} \\ & & &= \frac{(0.5767)^2}{.099} \\ & & &= 3.359 \end{aligned}$$

$$\begin{aligned} \bar{X}_1 &= \bar{X}_3 & F &= \frac{(1.3833 - 1.640)^2}{0.2969(6 + 6)/(6)(6)} \\ & & &= \frac{(-0.2567)^2}{.099} \\ & & &= 0.6656 \end{aligned}$$

To obtain the critical value for evaluation of these three Scheffe F's, we multiply  $(K-1)$  by critical table value for original F in ANOVA. Or  $(2)(3.52) = 7.04$  for  $\alpha = .05$ ,  $df = 2, 19$ . From our Scheffe F's we conclude  $\bar{X}_1$  is just barely significantly different from  $\bar{X}_2$ . The other means are not significant.

A nonparametric test, the Friedman analysis of variance by ranks was used to tell if measurements at  $t_1$ ,  $t_2$ , or  $t_3$  were significantly different.

Subjects were first ranked according to the amount of space present at each of the three time periods. See Table III.

The following formula for the Friedman statistic was used:

$$X_r^2 = \frac{12}{KN(K+1)} (\sum R_j^2) - 3N(K+1)$$

With  $df = 3 - 1 = 2$  and  $X_2^2 = 35.04$  computed and a table value of 2.995 for  $\alpha = .05$  or 6.908 for  $\alpha = .001$  we must reject the null hypothesis of no difference in mean diastema size measurement at the different time intervals.

To further investigate, the Friedman statistic was again performed but this time involving only  $t_2$  and  $t_3$ .  $df = 2 - 1 = 1$ ,  $X_r^2$  computed = 1.08. table value at  $\alpha = .05$  was 3.84.

Since the computed value did not exceed the table value we must accept the null of no difference between mean measurements at  $t_2$  and  $t_3$ .

We conclude that the mean diastema width at  $t_1$  is significantly larger than at  $t_2$  or  $t_3$  and there is no difference which is significant between mean measurement widths at  $t_2$  and  $t_3$ .

Another nonparametric test, Chi Square, was used to determine if there was a difference in frequency of relapse between the three

different types from end of treatment ( $t_2$ ) until some time after retainers were removed ( $t_3$ ).

It was not possible to use ANOVA to obtain this information because the high frequency of zeros made it impossible to meet the assumptions necessary for ANOVA.

It was found (Tables IV, V-A, V-B, VI, and VII) a Chi Square value of 1.37. The critical table value of Chi Square is 5.991 for  $\alpha = .05$  and  $df = 2$ .

Since the computed value does not exceed the table value for chosen alpha level, we accept the null of no significant difference in frequency of observed relapse from  $t_2$  to  $t_3$  between patients of types 1, 2, and 3.

## DISCUSSION

The objective of this investigation was to study the osseous septum between the central incisors as it relates to the relapse phenomenon observed after orthodontic diastema closure. Previous articles<sup>19,41,42</sup> have suggested that the interosseous bone may have a definite effect on diastema closure and relapse. It was necessary to divide the sample into three groups based on osseous septum type for study purposes. The three groups were matched fairly well in size and if all other variables were not significantly different, then any difference in size of relapse or frequency of relapse could be expected to be due to the different types of interosseous bone. Frenectomy had not been performed on any of the patients. To our knowledge, only one other clinical investigation in the orthodontic literature had recognized various types of interosseous septum identified from radiographs and used these categories as a basis for part of the investigation. That particular investigation by Popovich et al, however, was not concerned with diastema relapse after orthodontic treatment.

The presence of a midline diastema may be attributed to numerous causes. The following list of possible etiologies may not satisfy all authors but will allow a better appreciation of the wide range of suggested diagnoses. Separation of the central incisors

may be due to a number of factors.

I. Normal

1. Normal growth and development
2. Ethnic and familial norm

II. Deficiency of tooth material in arch

1. Unusually small teeth
2. Congenitally missing lateral incisors
3. Peg shaped lateral incisors
4. Ectopic teeth
5. Extracted teeth
6. Crowding of teeth which in turn causes ectopic eruption, thereby producing a lack of tooth material in the arch.

III. Increased arch length

1. Thumb or finger habits
2. Tongue thrusting
3. Interposing the lower lip between the upper and lower incisors.
4. Closed bite

IV. Physical Impediments

1. Retained deciduous tooth
2. Supernumerary teeth
3. Other midline pathology

V. Artificial causes

1. Rapid palatal expansion
2. Milwaukee Brace



## VI. Other

1. Incomplete fusion of the median palatine processes
2. Enlarged or malposed labial frenum

Careful diagnosis will help to assure that the cause of a diastema is accounted for or removed while under orthodontic supervision.

Possibly more than any other causative factor, the maxillary labial frenum has received the most attention in the orthodontic literature. Angle<sup>2</sup> described the cause of the midline diastema as being the abnormal development and attachment of the frenum. Although this may be true in the case of a truly abnormal frenum, these cases have been considered rare<sup>15</sup>. Dewel in 1946 was the first to point out that it is often difficult to make a diagnosis of a true abnormal frenum. Curran believed that many labial frena were resected unnecessarily due to improper diagnosis. As recently as 1977, Edwards found that the relationship between a clinically "abnormal" appearing maxillary midline frenum and a midline diastema showed a strong, but not absolute correlation. Moyers pointed out that a radiograph could be helpful in confirming a diagnosis. Although an abnormally large and marginally positioned labial frenum may result in a persistent midline diastema, there is agreement in the literature that rarely should any portion of even an "abnormal" labial frenum be removed prior to the eruption of the maxillary canines, since it has been observed that most diastemas close automatically with the final eruption of the remaining anterior teeth.

Although closing the diastema with an orthodontic appliance is relatively simple, successful retention has not always been achieved.

Many suspected causative factors for diastema relapse have been

investigated. These include: 1) superior labial frenum<sup>40,44</sup>, 2) interdental soft tissues<sup>22,23,24,30,44</sup>, 3) oxytalan<sup>30</sup>, and 4) transseptal fibers<sup>21,42,44</sup>. Other possible etiologies in relapse have been suggested but not investigated other than empirically. These include: 1) improper axial inclinations of the roots of central incisors, 2) tooth size discrepancies, 3) pernicious habits, 4) deleterious occlusal patterns, 5) actual anatomy of the teeth, and 6) muscular imbalances in the oral region.

From the results obtained in this examination of 22 patients, it was found that the mean diastema width at  $t_1$  of patients with a spade shaped osseous septum was significantly larger than patients with a normal V-shaped osseous septum but just barely so. No other significant differences between different types could be demonstrated.

This finding is probably of little, if any, importance and if the samples involved were larger, statistical significance between type 1 and 2 might not have been achieved. The computed value was very close to the critical value in this case.

Analysis of the data with the Friedman statistic revealed that significant relapse in diastema width after orthodontic space closure could not be demonstrated for any of the three types of patients. This statistical information could be misleading unless we consider the fact that any reopening of a midline space could be considered clinically significant to the patient or orthodontist even if it is not so large as to register statistically. Table IV shows a percentage breakdown of patients who demonstrated relapse. 40.9% of the total sample had some return of original spacing.

Further information was gained on relapse tendencies of the three different types by use of Chi Square analysis. The frequency of observed relapse from  $t_2$  to  $t_1$  did not reveal significant difference between the 3 different groups. This is not to be interpreted as meaning that the frequency of relapse is the same for each of the three types. It means that it was determined that the frequency of relapse noted for the three groups could have occurred by chance alone.

The findings of this investigation do not prove or disprove that the type of osseous septum associated with a diastema has an effect on relapse after orthodontic space closure.

Due to the lack of clinically useful studies concerning diastema relapse, additional investigation along the lines undertaken here could be useful if a much larger sample size were obtained. With a larger sample it might be possible to demonstrate a significant difference in frequency of relapse between the three different types of patients.

## SUMMARY AND CONCLUSIONS

This study consisted of an evaluation of pretreatment x-rays and study models of 22 patients obtained at three different time periods. The first set of models was taken before orthodontic treatment was instituted (time 1). The second set of models was taken after active orthodontic appliances were removed and retainers placed (time 2). The third model was taken as long after retention had been discontinued as was possible. The pretreatment radiograph was examined to determine the type of anatomy of the interosseous septum. The mean age for patients at the start of treatment was 12.8 years with a range of 11.0 years to 15.7 years. The mean age at the end of treatment was 15.4 years with a range of 13.2 years to 18.5 years. The mean time in retention was 2.2 years with a range from 0 years for one patient to 3.7 years. The mean time out of retention was 6.2 years with a range of 1.3 years to 13.9 years.

Measurement of the maxillary midline diastema was taken at the three different time periods with a John Bull millimeter micrometer accurate to 1/10 of a millimeter.

Standard error of the measure was determined by replicate measurements of maxillary diastemas on 12 randomly selected pairs of models taken at two different time periods. S.E.Meas. = .09 mm.

Measurement data on the three interosseous types of patients taken at time 1, time 2, and time 3 were subjected to the following kinds of statistical analysis:

- 1) ANOVA was used to test for significant difference in size of the diastema for type 1, 2, and 3 at time 1. The Scheffe test was used to make all possible comparisons between individual pairs of means.
- 2) Friedman analysis of variance by ranks, a nonparametric test, was used to tell if measurements at  $t_1$ ,  $t_2$ , and  $t_3$  were significantly different. It was again used but this time involving only  $t_2$  and  $t_3$ .
- 3) Another nonparametric test, Chi Square, was used to determine if there was a difference in frequency of relapse between the three different types from end of treatment ( $t_2$ ) until some time after retainers were removed ( $t_3$ ).

The conclusions which may be drawn from this study were:

- 1) After orthodontic closure of a maxillary midline diastema it can be expected that there will be some return of original spacing in a high enough percentage of cases to be a problem for the patient and orthodontist in terms of retention.
- 2) While there may be some reopening of a diastema in certain patients after orthodontic diastema closure, it is very unlikely that it would be large enough to be statistically significant when comparing measurement widths between  $t_2$  and  $t_3$ .
- 3) The findings in this investigation do not prove or disprove

that the type of osseous septum associated with a diastema has an effect on relapse after orthodontic space closure. It was determined that the frequency of relapse noted for the three types of patients could have occurred by chance alone.

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TABLE I

Diastema Measurements Over Time for Three Types of Patients

| Type | Patient | T <sub>1</sub> | T <sub>2</sub> | T <sub>3</sub> |
|------|---------|----------------|----------------|----------------|
| 1    | 1       | 1.2            | 0              | 0              |
|      | 2       | 1.3            | 0              | 0              |
|      | 3       | 1.2            | 0.1            | 0              |
|      | 4       | 1.2            | 0              | 0              |
|      | 5       | 2.3            | 0              | 0              |
|      | 6       | 1.1            | 0.3            | 0.5            |
| 2    | 7       | 2.4            | 0.1            | 0              |
|      | 8       | 2.4            | 0.6            | 0.3            |
|      | 9       | 3.3            | 0              | 0              |
|      | 10      | 2.3            | 0              | 0              |
|      | 11      | 1.2            | 0              | 0.4            |
|      | 12      | 1.7            | 0.4            | 0.8            |
| 3    | 13      | 1.0            | 0              | 0              |
|      | 14      | 1.1            | 0              | 0.6            |
|      | 15      | 2.0            | 0              | 0              |
|      | 16      | 2.6            | 0.3            | 0.5            |
|      | 17      | 1.8            | 0.1            | 0              |
|      | 18      | 1.3            | 0              | 0.9            |
|      | 19      | 1.5            | 0              | 1.0            |
|      | 20      | 1.5            | 0.3            | 0              |
|      | 21      | 1.6            | 0.4            | 0.8            |
|      | 22      | 2.0            | 0.6            | 1.1            |

TABLE II-A

Summary Table for ANOVA at Time 1

| Source | SS     | df | MS     | F       |
|--------|--------|----|--------|---------|
| Total  | 7.8636 | 21 |        |         |
| A      | 2.2230 | 2  | 1.1115 | 3.7439* |
| Error  | 5.6407 | 9  | 0.2969 |         |

TABLE II-B

Mean Diastema Widths at Time 1 for the Three Patient Types

| Type | N  | Mean   | Standard Error<br>of Mean |
|------|----|--------|---------------------------|
| 1    | 6  | 1.3833 | 0.2224                    |
| 2    | 6  | 2.2167 | 0.2224                    |
| 3    | 10 | 1.640  | 0.1723                    |

Overall Mean = 1.727

Number of Observations = 22

TABLE III

Ranks of Diastema Space Over 3 Time Intervals

| Patient | T <sub>1</sub> | T <sub>2</sub> | T <sub>3</sub> |
|---------|----------------|----------------|----------------|
| 1       | 3.0            | 1.5            | 1.5            |
| 2       | 3.0            | 1.5            | 1.5            |
| 3       | 3.0            | 2.0            | 1.0            |
| 4       | 3.0            | 1.5            | 1.5            |
| 5       | 3.0            | 1.5            | 1.5            |
| 6       | 3.0            | 1.0            | 2.0            |
| 7       | 3.0            | 2.0            | 1.0            |
| 8       | 3.0            | 2.0            | 1.0            |
| 9       | 3.0            | 1.5            | 1.5            |
| 10      | 3.0            | 1.5            | 1.5            |
| 11      | 3.0            | 1.0            | 2.0            |
| 12      | 3.0            | 1.0            | 2.0            |
| 13      | 3.0            | 1.5            | 1.5            |
| 14      | 3.0            | 1.0            | 2.0            |
| 15      | 3.0            | 1.5            | 1.5            |
| 16      | 3.0            | 1.0            | 2.0            |
| 17      | 3.0            | 2.0            | 1.0            |
| 18      | 3.0            | 1.0            | 2.0            |
| 19      | 3.0            | 1.0            | 2.0            |
| 20      | 3.0            | 2.0            | 1.0            |
| 21      | 3.0            | 1.0            | 2.0            |
| 22      | 3.0            | 1.0            | 2.0            |
|         | 66.0           | 31.0           | 35.0           |

TABLE IV  
Relapse in Type 1, 2, & 3 Patients

| Type | Number<br>Examined | Number with<br>Relapse | Number with<br>No Relapse | Percentage |
|------|--------------------|------------------------|---------------------------|------------|
| 1    | 6                  | 1                      | 5                         | 16.6       |
| 2    | 6                  | 2                      | 4                         | 33.3       |
| 3    | 10                 | 6                      | 4                         | 60.0       |
|      | 22                 | 9                      | 13                        | 40.9       |

TABLE V-A

General Format for Two by Three Table

|   |   |   |   |
|---|---|---|---|
| A | B | C | I |
| D | E | F | J |
| K | L | M | N |

Where in this specific case:

- A = Number of patients at  $t_1$  without diastema relapse  
 D = Number of patients at  $t_1$  with diastema relapse  
 B = Number of patients at  $t_2$  without diastema relapse  
 E = Number of patients at  $t_2$  with diastema relapse  
 C = Number of patients at  $t_3$  without diastema relapse  
 F = Number of patients at  $t_3$  with diastema relapse  
 I = Number of total patients without diastema relapse  
 J = Number of total patients with diastema relapse  
 K = Number of patients with Type 1 osseous septum  
 L = Number of patients with Type 2 osseous septum  
 M = Number of patients with Type 3 osseous septum  
 N = Total number of patients

TABLE V-B

Using the Values of Table IV in Table V-A

|   |   |    |    |
|---|---|----|----|
| 5 | 4 | 6  | 15 |
| 1 | 2 | 4  | 7  |
| 6 | 6 | 10 | 22 |



TABLE VI

Expected Chi Square Values for Contingency Table

|      |      |      |   |
|------|------|------|---|
| AC/N | AD/N | AE/N | A |
| BC/N | BD/N | BE/N | B |
| C    | D    | E    | N |

TABLE VII

Computation of Chi Square

| O | E    | O-E   | $(O-E)^2$ | $(O-E)^2/E$ |
|---|------|-------|-----------|-------------|
| 5 | 4.09 | 0.91  | .828      | .20         |
| 4 | 4.09 | -0.09 | .008      | .002        |
| 6 | 6.82 | -0.82 | .672      | .10         |
| 1 | 1.91 | -0.09 | .828      | .43         |
| 2 | 1.91 | -0.91 | .828      | .43         |
| 4 | 3.18 | 0.82  | .672      | .21         |

$$\chi^2_{(2)} = 1.37$$

# Appendix A

| Patient | Sex | Age at Start | Age at End | Time in Tx | Time in Retention | Time out of Retention | Classification |
|---------|-----|--------------|------------|------------|-------------------|-----------------------|----------------|
| 1       | F   | 15.67        | 18.5       | 2.83       | 3.0               | 3.42                  | I              |
| 2       | F   | 13.67        | 15.67      | 2.0        | 2.5               | 3.75                  | II             |
| 3       | F   | 12.42        | 14.75      | 2.33       | 2.83              | 4.17                  | II             |
| 4       | F   | 13.08        | 15.0       | 1.92       | 2.42              | 2.0                   | II             |
| 5       | F   | 12.17        | 14.17      | 2.0        | 3.08              | 2.92                  | E/E            |
| 6       | F   | 13.92        | 16.0       | 2.08       | 2.0               | 10.17                 | II             |
| 7       | F   | 13.08        | 16.25      | 3.17       | 2.17              | 3.92                  | II             |
| 8       | M   | 12.08        | 13.92      | 1.83       | 0.83              | 13.08                 | II             |
| 9       | F   | 12.58        | 15.0       | 2.42       | 2.83              | 8.08                  | E/E            |
| 10      | M   | 11.83        | 13.83      | 2.0        | 3.17              | 3.0                   | II             |
| 11      | M   | 14.83        | 18.0       | 3.17       | 2.17              | 2.08                  | II             |
| 12      | M   | 12.67        | 15.0       | 2.33       | 3.67              | 1.25                  | E/E            |
| 13      | F   | 11.25        | 14.58      | 3.33       | 2.83              | 4.17                  | II             |
| 14      | M   | 13.67        | 16.08      | 2.42       | 2.42              | 5.5                   | E/E            |
| 15      | M   | 14.92        | 17.08      | 2.17       | 1.42              | 7.25                  | II             |
| 16      | M   | 11.83        | 14.5       | 2.67       | 0.92              | 13.92                 | I              |
| 17      | F   | 11.25        | 13.25      | 2.0        | 3.58              | 10.67                 | II             |
| 18      | M   | 13.0         | 17.42      | 4.42       | 1.5               | 9.25                  | II             |
| 19      | F   | 11.0         | 13.67      | 2.67       | 2.75              | 3.58                  | II             |
| 20      | F   | 11.42        | 13.42      | 2.0        | 2.92              | 11.42                 | II             |
| 21      | F   | 12.58        | 15.08      | 2.5        | 0                 | 8.92                  | II             |
| 22      | M   | 13.08        | 17.25      | 4.17       | 0.33              | 3.75                  | II             |