The Etiology of Angle Class II Division I Malocclusion*

RICHARD A. SMITH, D.D.S., M.S.D.
Evanston, Illinois

WE have been taught that etiology must be the first consideration in caring for any case of malocclusion; for unless the factors which have caused the deformity can be determined and removed, successful treatment is not probable. It is important, therefore, that we consider the etiology of Angle Class II Division I malocclusion before going into the mechanics of its treatment.

Our textbooks lay out a very definite group of causative factors for the production of this malocclusion. One of the main offenders is mouth breathing, which may be the result of some nasal obstruction or adenoids, or may operate as a habit. "The oral deformities of the mouth breather are typical. They are exemplified in the Class II Division I case in which we find an undergrowth in the body of the mandible that establishes the mandibular dental arch in distal occlusion; a narrowing of the maxillary arch, and a protrusion of the upper incisor teeth."

In writing of mouth breathing, Strang states that the muscles which depress the mandible to open the mouth exert a backward pressure upon it. This pressure not only tends to displace the mandible distally and retard its growth but also takes away the force of inclined plane relationship by holding the teeth apart. The combination of this distal force and loss of functional pressure results in a distal locking of the inclined planes of the mandibular teeth with those of the maxillary. A continued backward pounding soon tips the lower molars mesially and retards normal growth in the mandible.

The buccinator muscles made tense by opening the mouth tend to cause a lingual pressure on the maxillary bicuspids and molars which do not receive sufficient support from the tongue when the mouth is open, so that the upper dental arch becomes quite narrow. The lip function is not normal, the lower lip being large and bulbous and the upper quite short and functionless. In my opinion, these abnormal lips play an important part in furthering the malocclusion. The large lower lip is usually forced up under the upper incisors during swallowing so that they are further protruded. The upper lip does not act to restrain the protrusion because of its size and lack of function. Thus the lips, instead of restraining the teeth in position, are, through malfunction, acting to further the malocclusion.

Other habits which are conducive to the development of this type of malocclusion are the various sucking habits, such as thumb or lip sucking. Abnormal swallowing habits are also factors.

Thumb sucking habits will, if practiced over a period of time, cause a protrusion of the upper anterior teeth and a lack of development in the lower

*Presented at the meeting of the Chicago Association of Orthodontists in Chicago, September 26, 1938.

arch, which may develop into a typical Angle Class II Division I malocclusion. The space which is made by the protruding teeth makes a place for the lower lip to rest, thus increasing the protrusion. When the protrusion becomes great enough it is easier to hold the lips apart than to close them, and in time the typical Class II Division I malocclusion may result.

There are two swallowing habits in Class II Division I cases which tend to increase the malformation. One is the habit of forcing the lower lip under the protruding upper anterior teeth when swallowing; the other is the habit of thrusting the tongue out between the anterior teeth.

The explanation given by Strang as to the mechanics of the development of Class II Division I malocclusion will be used as one typifying the logical reasoning of the orthodontic text book. Strang points out that the first permanent molar teeth erupt with the points of their cusps in occlusion instead of their inclined planes. Therefore, any force giving an abnormal backward pull on the mandible, such as mouth breathing, is sufficient to cause the distal locking of the cusps. When this has occurred the occlusal stress is not applied on the central fossae but on mesial sections of the lower first molars. This abnormal application of forces causes these teeth to tip mesially. A compensating adjustment takes place in the upper molars. This shift in the vertical alignment causes a continuous backward thrust on the mandibular teeth and bony support and a forward thrust on the maxillary teeth and their bony bases. This action causes a retardation in the forward growth of the body of the mandible and a stimulation to that of the maxillae. With an increase in the tipping of these molars there is a tendency for the bite to close. This tipping also encroaches on the space of the bicuspid teeth which results in their impaction and an exaggerated curve of Spee.

"Thus we see that the posterior relationship to skull anatomy of the mandibular dental units and the body of this bone, is due to the posterior thrust of the perverted forces of occlusion and the backward pull of muscles in abnormal and unbalanced action."

"The bony cells respond to these upset forces and shape the mandibular body to best resist the stresses brought to play upon it and the bone becomes, in form, shape, and position, ideally adapted to support teeth that are constantly receiving blows upon their occlusal surfaces, the direction of which is obliquely backward instead of in the true vertical plane or at approximately right angles to the normal plane of the denture."

You will note the text book gives local environmental causes as being the chief etiological factors in producing Class II Division I malocclusion and has set up a series of mechanical events to bring about its development. This theory of the etiology of Class II Division I malocclusion, logical as it may seem, is not shared by all investigators. Professor J. C. Brach of England has made a valuable contribution to the study of etiology. His findings are for the most part negative for he cannot accept factors as causes. He says: "It has still to be proved that there is any significant correlation between the presence of adenoids and the incidence of deformities of the jaws and palate." He further states that: "It is improbable that the tongue exercises any direct mechanical influence on the general form and size of the mandible or in molding the form of the growing palate." Another of Brach's conclusions is that: "It is probable that the bone conditions which underlie the great majority of mal-

occlusions are determined in the early years of life. Malocclusion of the deciduous teeth is common, and it must be recognized that variations in the relations of the jaws similar to those found in malocclusion occur in the fetus and in the new born child." He suggests "that the time has arrived to substitute for the provisional hypothesis of environmental influence in the wide sense—which appears to have inspired most of the investigations and most of the discussions that have taken place in the past—the other provisional hypothesis that irregularity and malocclusion are inherited conditions, and to let that inspire future discussions and future investigations."

Dr. Sim Wallace is also skeptical about adenoids being a causative factor, saying: "Taking all of the facts together, we are bound to admit that there is but very little evidence of a causal relationship between adenoids and the type of jaw deformity which used to be ascribed to the tension of the lips and cheeks on the dental arch as a result of mouth breathing."

Dr. C. C. Howard in his paper on "Inherent Growth and Its Influence on Malocclusion," says: "The growth anomalies of the jaws and dental arches remain unexplained." He showed in this same paper statistical evidence that only 159 out of 500 tonsil and adenoid cases were mouth breathers and that of the 159 mouth breathers, 94 exhibited normal jaws, arches, and occlusion, while only 22 exhibited Class II Division I malocclusion.

In his article on "Etiology of Class II Malocclusion" Hellman says "that when put to a test, not one of the recommended factors is found to bear any definite relationship to a particular form of malocclusion." He further states that "most of these factors of malocclusion accepted by modern orthodontists have come down to us by tradition, and are accepted on no other grounds but by the recommendations of some authority." He shows that mouth breathing and its cause, adenoid tissue, which is generally blamed for Class II manifestations, are equally divided between Class II and other classes. "The only instance in which there seems to be any positive correlation is sucking habits and Class II Division I." "In this particular group 60 per cent of those possessing this habit present Class II Division I cases." We may, therefore, be quite safe in saying that the habit of sucking is the only factor that stands in intimate and positive relationship with Class II cases and especially those in Division I." In questioning the mothers concerning the time at which the habit was started he found that in many cases the child sucked its fingers immediately upon delivery. Because of the close proximity of the hands to the mouth in utero, Hellman feels that this habit may be an acquisition that antedates birth.

Lewis also has shown in his work that pernicious habits cause deformities of the jaws. To deny that finger sucking and thumb sucking have a tendency to cause Class II Division I malocclusion is to deny the observation of clinical experience.

In considering Class II Division I malocclusion as a problem in growth, we must realize that not all children grow alike, or according to a standard of growth arrived at by a statistical method. There is quite a range of variation in which growth is considered normal. Any disturbance which affects bodily health may affect a period of bodily growth due at that time. Such a disturbance might at a critical time result in an underdeveloped mandible or short ramus. Growth is dependent to some extent on heredity. There is an inherited tendency to grow toward a certain type which gives us the family

resemblances. While the inheritance of the complete malocclusion is very improbable still in some cases facial patterns of offspring and parent would lead one to suspect that there had been a growth tendency toward a certain pattern. Hellman says: "Orthodontic treatment of Class II Division I and Class III cases influences the positions of the teeth, their occlusal relationships and the probable adjustments of the alveolar arches. Tendencies of growth peculiar to these types do not seem to be changed because they persist for long periods after culmination of orthodontic treatment. Inherited tendencies of facial growth are not altered by changes in the occlusion of the teeth."

Rogers has emphasized the possibility of a narrow maxillary arch interfering with the forward positioning of the mandible, and shows that by expansion in conjunction with muscle exercises enough growth force is liberated to bring the jaws into normal relationship. He also shows in his paper entitled "Dual Bite with Particular Reference to the Temporomandibular Articulation," that in some Class II Division I cases there is a considerable change in the mandibular joint which he shows by radiograph taken before and after treatment.

With such a decided variance of opinion on etiology of Class II Division I malocclusion, it would seem that all of our treatment should be undertaken with a great deal of uncertainty as to its final outcome. However, if the theories on which we have treated cases are entirely wrong, our results should, in most cases, be failures. We know that this is not true for we have seen many fine results with our present knowledge of etiology. It must be true, therefore, that the etiology on which we have based our treatment in the past cannot be entirely amiss.

While it is true that no one etiological factor may be responsible for the production of a Class II Division I malocclusion several factors, any one of which may be the initial one, may cause such a malocclusion. In many cases the initial factor may be difficult to discover for each of the factors are so closely allied that it is difficult to know which came first. There are many factors which enter into the etiology of Class II Division I malocclusion, which may leave when the malocclusion is treated. The initial factor in the production of Class II Division I might be local, such as mouth breathing, thumb sucking, lip sucking or a swallowing habit, or it might be a growth deficiency or an inherited tendency. But, regardless of what the initial factor may have been, I feel that the local factors as described by Strang all come into the picture and must be regarded as potent etiological factors in increasing the severity of the condition. We know that these factors must be eliminated if the case is to be successfully treated.

For a period of time after Simon's work was made known, there was a good deal of discussion as to just what was wrong in a Class II Division I malocclusion. We had been taught previously that the lower jaw was in distal relationship in Class II Division I, but now the theory was that in many of the Class II Division I malocclusions it was the upper arch that was too far forward. In answer to this opinion, Strang makes the following statement in his text: "Oppenheim, by scientific study of skulls in which the teeth were in Class II malocclusion, and Broadbent, by radiographic studies of children, have proved, beyond a doubt, that the mandibular denture is really in posterior relationship to the anatomy of the skull. Both of these investigators also

found some lack of forward growth in the maxillae—a rather surprising deduction, but a most reasonable one from the influence of perverted functional forces. The specimens studied also clearly showed that the posterior location of the mandible is not due to a distal position of the condyles in their mandibular fossae. Such location of the condyles would be an anatomical impossibility without infringement on the ear passages."

On the same subject, Hellman states: "In Class II Division I cases the position of the upper canine is not more forward but rather further back in relationship to the orbit than in the normal."

Dr. Strang's book has been quoted freely in the preparation of this paper to give the text book picture of the subject. In closing, I want to quote Dr. Strang once more. This time from his article, "A Discussion of the Angle Classification and Its Important Bearing on Treatment," read in New York City on May 2, 1938. This quotation will show you how strongly he feels concerning the importance of occlusal forces as a factor in modifying jaw form.

"I am fully cognizant of the fact that the pendulum is swinging far over to the conclusion that occlusal forces have little or no effect upon modifying the form of the basal structures. Many orthodontists feel very strongly that the form of a bone is determined by the degree of activity present in the centers of growth. To substantiate this contention we all have corrected cases that show little or no improvement in facial lines after treatment, although the teeth are occluding correctly as to their inclined plane adjustment. But it seems to me that these are cases in which the growth forces were lost to such a marked degree that their influence was negative at a critical period. A certain portion of bone that was to be laid down at a particular age period was then constructed. The opportunity for its being built was lost forever, then and there. Certainly no occlusal stress can ever be expected to influence something that is not, nor to stimulate into renewed activity a growth center whose work is finished. But in any bone that is still actually growing clinical evidence does apparently indicate that there is some influence exerted by occlusal stress."

BIBLIOGRAPHY

1. Strang, R. H. W. Text Book of Orthodontia. Philadelphia, pp. 117-125, 519-522. Lea and Febiger.
2. Brash, J. C. The Etiology of Irregularities and Malocclusion of the Teeth. pp. 6, 7, 151, 243, 246, 221. Dental Board of U. K. Lectures. 1929.
3. Howard, C. C. Inherent Growth and Its Influence on Malocclusion. p. 648, Vol. 19, J.A.D.A. 1932.
4. Hellman, Milo. Studies on the Etiology of Angle's Class II Malocclusal Manifestations. p. 147. Internat. J. of Orthodont. & Oral Surg. and Radiog. Vol. 8, 1922.
5. Hellman, Milo. Discussion of Dr. Stanton's paper. p. 187, Proceedings of The American Society of Orthodontists, 1927.
6. Roger, Alfred P. The Problems of the Dual Bite with Particular Reference to the Temporomandibular Articulation, p. 40, Vol. 21, Internat. J. of Orthodont. Oral Surg. and Radiog. 1935.
7. Franklin, Gerald. Etiology, Diagnosis and Treatment of Class II Division I Malocclusion. p. 1099, Vol. 22, Internat. J. of Orthodont., Oral Surg. and Radiog. 1936.
8. Strang, R. H. W. A Discussion of the Angle Classification and Its Important Bearing on Treatment. p. 182, Vol. VIII, Angle Orthodont. 1938.

636 Church Street.