

Anterior Open Bite: Causative Factors and Diagnosis



ABSTRACT

The real difficulty behind the treatment of open bite is the easy relapse, for it has a multifactorial nature. **Aim:** The aim of this study is to compare the reliability of two differential ways in the diagnosis of the open bites, looking out for the causative factors, aiding in more specific treatment plan and less relapse. **Materials and Methods:** A sample of 53 anterior open bite cases, all in the post-pubertal and early adulthood period (17–25 years), was assessed twice, clinically and cephalometrically. Due to clinical assessments, the whole sample was grouped as “*Morphogenetic*” and “*Functional*” groups. The same sample was also cephalometrically assessed and grouped as “*Skeletal*” and “*Dento–alveolar*” using *mandibular plane angle*, suspected that the *morphogenetic* group clinically matches the cephalometrically assessed *skeletal* group in number; also the *functional* group clinically matches the cephalometrically assessed *Dento–alveolar* group in number. Student’s t-test indicated a weak agreement between clinical judgment and cephalometric evaluation ($p \leq 0.001$). **Results:** Unexpectedly, the sample which assessed as skeletal hyper-divergent *cephalometrically*, half of it in fact was classified as functional cases *clinically*. This misdiagnosis may lead to inadequate treatment plan, in which relapse should be highly expected.

Conclusions: These findings highlighted that, it is not enough to depend on “cephalometric evaluation” alone to design the treatment plan for open bite cases. Clinical evaluation is also important to point out the real causative factors for designing an adequate treatment plan (i.e., rehabilitation of the soft tissue bad habits, when needed) to reduce the prevalence of relapse.

Asst Lect Anfal A Al-Ani (BDS, MSc)

Department of Orthodontics, Dentistry
College, Al-Mustansyria University

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Open bite develops as an interaction of many etiologic factors, both hereditary and environmental in nature.⁽¹⁾ Environmental factors include variations in dental eruption and alveolar growth, abnormal function of the tongue, disproportionate neuromuscular growth as its relation to malfunctions, and/or some other oral habits.^(2, 3)

Pure dental open bite, which caused by the tongue, anterior swallowing and other bad habits, has to be distinguished from open bites that involve the skeletal morphology as the maxilla or the mandible position (or both) in relation to the cranium.⁽⁴⁾ After the elimination of the causative factors, dental open bites are either self-correcting or respond readily to myofunctional treatment and mechanotherapy.^(5, 6)

Open bites associated with craniofacial malformations are much more difficult to treat and tend to relapse,⁽⁷⁾ but the early treatment of vertical dysplasia during the primary or the

mixed dentition period has been advocated to reduce the need of treatment in the permanent dentition, when surgery becomes a viable option.^(8,9)

A series of treatment approaches can be found in the literature regarding early treatment of open bite. These treatment modalities include mainly functional appliances, multi-bracket techniques, headgears, and bite blocks.^(10,11)

Orthodontists commonly agree with the sentiment “Treatment of open bite is difficult and relapse is easy” still the reasons behind this have not been sufficiently examined. The multifactorial nature of the etiology of open bite is largely responsible for the indecision surrounding its diagnosis and treatment.^(12,13)

Decades have passed since Sassouni⁽¹⁴⁾ suggested that the angle of the mandibular plane (MP) could be used as the criterion for classifying open bite cases as either “skeletal” or “dentoalveolar”. The MP angle is still commonly accepted and in use today as an identifying factor in vertical facial morphology, but its accuracy in classifying open bite seems to be not enough alone, for the high relapse incidence noticed after treatment.⁽¹⁵⁾

Conflicting systems of evaluation and classification often result in only partial and inadequate diagnosis, giving rise to problems during and after treatment.⁽¹⁵⁾

It is true that the MP angle increases in long-faced individuals; nevertheless, as stated by Fields and colleagues⁽¹⁶⁾ “not all long-faced patients have open bite, and not all open bite patients are long faced”.

The MP and gonial angles are genetically determined, but changes in them have been clinically and experimentally shown that it could also occur as a result of environmental and functional factors.⁽¹⁶⁾ Increased MP angle in such cases may be reasonably classified as hyper-divergent. Almost all “skeletal” cases show Hyper-divergency in the MP angle, but “skeletal” might be of other causative factors than “divergent” gonial angle.”Divergent” and “skeletal” are not the same causative terms. Unfortunately, this misdiagnosis is not infrequent and leads to much confusion.⁽¹⁷⁾

Cephalometric x-rays have the ability to identify normal and abnormal characteristics of the craniofacial structure. Cephalometry is routinely performed in orthodontic clinics because the procedure is simple and the results can be easily and quickly evaluated.⁽¹⁸⁾

As the usefulness of cephalometry, it cannot be disregarded in the evaluation of treatment results and the follow-up of growth and development, while still the information provided by cephalometry considered limited regarding to some clinical issues.^(19,20)

Because cephalometry is unable to provide adequate information about functional and environmental factors, these factors are naturally overlooked in cephalometric studies,⁽²¹⁾ and because functional factors are frequently implicated in the etiology of open bite, their neglecting is probably the most notable reason for the complications in diagnosing open bite and designing an appropriate treatment plan as well as for the high incidence of relapse after treatment.⁽²⁰⁾

The aim of the current study was to examine the problem of conflicting evaluations of open bite in group of post pubertal and early adulthood patients, using differential diagnoses “clinical” versus “cephalometric” assessment methods of open bite.

MATERIALS AND METHODS

Study Population

The study population consist of 53 young adults (19 males and 34 females), all were with anterior open bites, the mean over bite as clinically assessed was ($-4.15 \text{ mm} \pm 1.65$); range from (-1mm to -7.5mm) (Figure 1–A). Patients’ records were from those who attended to the

Orthodontic Department of Al-Dawoodi Dental Health Center from May/2005–May/2006 seeking for orthodontic treatment. All subjects were in their post pubertal and early adulthood period (17–25 years). The mean age of the subjects was (19.7 ± 1.3) years. All permanent teeth were present (the wisdom teeth were not included).



Figure (1): A- Anterior open bite from canine to canine, B- with continuous tongue thrust.

Records

Pretreatment records as lateral cephalograms, extraoral and intraoral photographs, and patient histories were evaluated and reassessed within three weeks intervals by the same orthodontic specialist. All the cases ($n= 53$) were classified independently, using clinical and cephalometric methods.

Clinical Records and Evaluations

Extraoral and intraoral photographs were recorded, as well as the patient history. One of the extraoral views was a true profile photo with a mirror handle pushed against the lower border of the mandible (Figures 2 and 3) to have the mandibular steeping with the horizon as recommended by Proffit and Fields,⁽⁷⁾ classifying the sample to “divergent” and “non-divergent” open bite cases.

Figures (2) and (3): Profile photos, true profile view with a mirror handle pushed against the lower border of the mandible.





The “environmental tongue thrust” and other oral bad habits (Figure 1–B) were evaluated by calculating the effective time of the soft tissue bad habits through half an hour (i.e., tongue thrust during talking, anterior swallowing and the low tongue position) without the patient attention, repeated for three independent intervals in different appointments for calculating the mean time, because it has been found that the effective soft tissue bad habit must be active at least 6 hours daily to have a permanent results on the dental arches.^(5–7) The clinical differential

diagnosis of the sample as “morphogenetic” and “functional” open bites was based on the criteria that:

- 1- If the functional characteristics effective time(mean) ≥ 7.5 minutes in $\frac{1}{2}$ an hour, collects 15 minutes or more/hour (6 hrs/day), then it was assessed clinically as “functional” open bite. A useful note can be added as the data collected, no doubt could be carried out about the bad habits collected time, as it was either very obvious (almost full time) or not existed.
- 2- If the cranio–mandibular morphology shows diverge gonial angle, increased anterior lower facial height and steeped mandible, then it is clinically “morphogenetic” type.

All the clinical criterion were Judged according to what had been recommended by Proffit and Fields⁽⁷⁾ (Figures 4 and 5).

Accordingly, open bite cases were clinically classified into functional and morphogenetic groups. The tabled clinical criterion (Figures 4 and 5) was conducted twice by the same orthodontic specialists at 3 weeks intervals.

Clinical Judgment Criterion		Criteria	Functional
<div style="border: 1px solid black; padding: 5px; width: fit-content; margin: 0 auto;"> Clinical Criterion Normal face </div> 	<i>Gonial angle</i>	<i>Normal</i>	
	<i>Lower facial height</i>	<i>Normal</i>	
Soft tissue factor		<i>Tongue posture</i>	<i>Thrusting</i>
		<i>lip competence</i>	<i>Competent lips</i>
Clinical Judgment Criterion		Criteria	Morphogenetic
<div style="text-align: center;">Hyperdiverg face</div> 	<i>Gonial angle</i>	<i>Increased</i>	
	<i>Lower facial height</i>	<i>Increased</i>	
Soft tissue factor		<i>Tongue posture</i>	<i>Thrusting < 6 h/day</i>
		<i>lip competence</i>	<i>Incompetent lips</i>

Figures (4) and (5): Clinical judgment criterion, with “functional” and “morphogenetic” grouped case.

Cephalometric Evaluation

The study population (n= 53) was evaluated according to Kalsen⁽¹⁷⁾ by measuring the MP Angle (MP= NS–GoGn = 32°–38° range normally) as: If (32° \leq MP < 38°) the angle was to be from normal to hypo–diverge, with no real skeletal factor effecting the open bite formation, and classified as “dento–alveolar” open bite, or if (MP \geq 38°) the angle was to be hyper–diverge, then the skeletal factor effecting the open bite formation, and classified as “skeletal” open bite. In these cases the soft tissue factor –if found– could be a sequence⁽¹⁷⁾ Each cephalograph was re–evaluated in three weeks intervals for the method reliability.

Method Error

The Student's t-test were calculated to determine the reliability of the clinical evaluation for the whole sample (n= 53). A score of 0.923 indicated reliability between the 1st and 2nd clinical evaluations ($p \leq 0.001$).

Intra-class correlation coefficients were calculated to determine the reliability of cephalometric evaluations, which was between 0.88 and 0.96 indicated almost perfect agreement.

Statistical Analysis

Student's t-tests were calculated to determine the rates of agreement between clinical and cephalometric evaluation (t-test : 0.374, $p \leq 0.001$).

The distribution of the cephalometric diagnostic *skeletal* and *dento-alveolar* open bites among the clinically assigned *morphogenetic* and *functionally* diagnostic open bites and vice versa, were shown in Table (1).

Table (1): The distribution of the sample due to clinical and cephalometric assessments.

Clinical Evaluation	Cephalometric Evaluation					
	Skeletal	%	Dento-alveolar	%	Total	%
Morphogenetic	16		1		17	32
Functional	16		20		36	68
Total	32	60	21	40	53	100

*Student's t-test: 0.374, $p \leq 0.001$.

RESULTS

Table (2) shows the clinical evaluation of the whole population grouped as "divergent" 31 (58.5%) and "non-divergent" 22 (41.5%). While Table (1) shows the distribution of open bite cases according to both clinical and cephalometric evaluation of the total sample (n= 53), clearing that "divergent" (n= 31) is not the same as "skeletal" (n= 32).

Table (2): The clinical distribution of the sample as "divergent" and "non-divergent".

Clinical Evaluation				Total
Divergent	%	Non-divergent	%	
31	58.5	22	41.5	53

Due to Table (1) clinical evaluation 17 (32%) were grouped as morphogenetic, and 36 (68%) as functional, while due to cephalometric evaluation 32 (60%) were grouped as skeletal, and 21(40%) as dento-alveolar.

Student's t-test of 0.374 indicated a weak level of agreement between clinical and the cephalometric methods ($p \leq 0.001$).

The distribution of the sample among the clinical and the cephalometric evaluation groups (Table 1) shows the actual distribution of "skeletal" cases (n= 16) among the "morphogenetic" group (n= 17) was similar to what could be expected (not significant, $p > 0.05$), while the actual distribution of "dento-alveolar" cases (n= 20) among the "functional" group (n= 36) varied significantly from expectation (significant, $p \leq 0.01$).

On the other hand the actual distribution of the "functional" cases (n= 20) among the

“dento–alveolar” group (n= 21) was similar to what could be expected (not significant, $p > 0.05$), but the actual distribution of “morphogenetic” cases (n= 16) among the “skeletal” group (n= 32) varied significantly from expectation (significant, $p \leq 0.01$).

DISCUSSION

For accurate diagnosis and precise treatment planning of each open bite case, an identification of the underlying cause(s) is essential. Due to Table (1) the open bite sample (n=53) were divided into “divergent” 31 (58.5%) and “non–divergent” 22 (41.5%) groups clinically, it seems as if the skeletal factor is the most causative one. “Divergent” as a term is very closely related to “skeletal” which used in table 2, but they are not the same terms. The skeletal increased lower facial height could be almost but not always caused by diverge gonial angle. This can lead to much confusion and misdiagnosing the open bite causative factors.⁽¹⁸⁾

The aim of this study is to identify the essential underlying cause(s) of open bite, by comparing the differential diagnosis of open bite cases using clinical evaluation and conventional cephalometric method.

Open bite cases with increased anterior lower facial height –as expected– must possess the “morphogenetic” group of the clinically evaluated method as well as the “skeletal” group of the conventional cephalometric method, while those with normal anterior lower facial height –as expectation– should cover the “functional” group in the clinically diagnostic method as well as the “dento–alveolar” group in the conventionally diagnostic cephalometric method, actually this is untrue regarding to Table (1), which revealed that the majority of open bite cases in this study were classified as “functional” (68%) by clinical evaluation. At the same time the majority of the overall cases in this study were classified independently as “skeletal” (60%) by cephalometric evaluation. However, the rate of the “morphogenetic” open bites was rather low (32%), which is not a surprising finding when the (dento–alveolar compensating mechanism) is taken into consideration.⁽²²⁾ Results of Student’s t–test showed weak agreement between clinical and cephalometric method in the classification of open bite.

Although “skeletal” cases were expected to be also evaluated as “morphogenetic” in nature, and “dento–alveolar” cases to be “functional” in nature, but as (Table 1) actually 16 of the skeletal cases (n= 32) were evaluated unexpectedly as functional, and only 20 of functional cases (n= 36) were surprisingly evaluated as dento–alveolar cases, while almost all cases diagnosed clinically as morphogenetic (n= 17) would be classified as skeletal (n= 16), but out of the 32 cases classified as skeletal only 16 were classified as morphogenetic. These findings indicate that morphogenetic open bites often show a skeletal hyper–divergent pattern, while skeletal hyper–divergency is not always morphogenetic in nature. This is going on with the finding of Arat and colleagues.⁽¹⁵⁾

It was suggested that, because of the effects of the dento–alveolar compensation mechanism, long–faced subjects demonstrate a narrow and elongated midsagittal projection of the basal and alveolar bone in the frontal part of the jaws.⁽²²⁾ Therefore, open bite rarely seen in long faced individuals. But in some instances, with mouth breathing, enlarged tonsils, oral habits, or postural relationships of tongue and lips, the dento–alveolar growth fails to compensate for the vertical discrepancy, thus leading to a functional/environmental open bite in persons with hyper–divergent facial patterns. Cephalometric analysis classify such cases as skeletal open bite, while in fact these cases are of functional origin.⁽²³⁾ These were represented by 16 cases in the current study. Orthodontic treatment of such cases represents a significant challenge. Rehabilitation of the soft tissue bad habits is recommended to reduce the prevalence of relapse.

Eliminating the causative factors during the early growth period will decrease the MP angle, while if these cases are not treated early, the hyper-divergance will become permanent.⁽²⁴⁾ Furthermore, the intensity and duration of the causative dysfunction or acquired habit, as well as the growth period of the person, have an effect on the way of open bite development.⁽¹⁴⁾

Functional factors (such as nasal restriction, abnormal functional pattern of the tongue, oral habits, abnormal swallowing patterns, and speech problems) often play a role in the etiology of open bite, but they are usually disregarded, beside the inability of cephalometric analysis to measure functional and compensatory mechanisms.⁽²⁵⁾

As a result, the use of cephalometric evaluation alone in the diagnosis of open bite results in inadequate classification, inaccurate diagnosis, poor prognosis and high incidence of relapse. The proper consideration of the functional factors, as well as the masticatory muscle functions should lead to greater treatment success and improve its stability.⁽²⁶⁾

CONCLUSIONS

- The current study showed a weak correlation between cephalometric and clinical classifications.
- Almost all cases which evaluated as “morphogenetic” clinically (n= 17) were evaluated as “skeletal” using cephalometry (n= 16), and almost all cases evaluated as “dento–alveolar” cases (n= 21) were evaluated as “functional” cases (n= 20) clinically. But unexpectedly, the clinically evaluated “functional” cases (n= 36) showed only 20 “dento–alveolar” cases, in addition to 16 “skeletal” cases when cephalometrically evaluated. Also half of the open bite cases which were cephalometrically evaluated as “skeletal” (n= 32) were clinically evaluated as functional (n= 16). The treatment of such cases represents a real challenge and rehabilitation for the soft tissue bad habits is recommended to reduce the prevalence of relapse.
- Cephalometric evaluation is an inadequate tool to be used alone for the differential diagnosis and classification of open bite cases for they are multifactorial in nature.

REFERENCES

1. Richardson A. A classification of open-bite. *Eur J Orthod.* 1981; 3: 289–296.
2. Ngan P, Fields HW. Open bite: A review of etiology and management. *Pediatr Dent.* 1997; 19: 91–98.
3. Speidel TM, Isaacson RJ, Worms FW. Tongue thrust therapy and anterior dental open bite: A review of new facial growth data. *Am J Orthod.* 1972; 62: 287–295.
4. Nashashibi IA. Variation of swallowing patterns with malocclusion. *J Pedod.* 1987; 11: 332–337.
5. Subtelny JD, Subtelny JD. Oral Habits—studies in form, function and therapy. *Angle Orthod.* 1973; 43: 349–383.
6. Warren JJ, Bishara SM. Duration of nutritive and nonnutritive sucking behaviors and their effects on the dental arches in the primary dentition. *Am J Orthod Dentofacial Orthop.* 2002; 121: 347–356.
7. Proffit WR, Fields HW. Contemporary Orthodontics. 3rd ed. The CV Mosby Co. St Louis. 2000; Pp: 350–352.
8. McNamara JAJr, Brudon WL. Orthodontics and Dentofacial Orthopedics. Ann Arbor, Mich: Needham Press; 2001; Pp: 112– 136.
9. Sankey WL, Buschang PH, English J, Owen AH. Early treatment of vertical skeletal dysplasia: the hyperdivergent phenotype. *Am J Orthod Dentofacial Orthop.* 2000; 118: 317–327.
10. Buschang P, Sankey W, English JD. Early treatment of hyperdivergent open bite malocclusions. *Semin Orthod.* 2002; 8: 130–140.
11. Basciftci FA, Karaman AI. Effects of a modified acrylic bonded rapid maxillary expansion appliance and vertical chin cap on dentofacial structures. *Angle Orthod.* 2002; 72: 61–71.
12. National Health Service (NHS) Centre for Reviews and Dissemination Report Number 4. Undertaking Systematic Reviews of Research on Effectiveness. 2nd ed. University of York: York Publishing Services; 2001. Available at: <http://www.york.ac.uk/inst/crd/crdrep.htm>. 2004.
13. Petreń S, Bondemark L, Söderfeldt B. A systematic review concerning early orthodontic treatment of unilateral posterior cross bite. *Angle Orthod.* 2003;73: 588–596.

14. Sassouni V. A classification of skeletal facial types. *Am J Orthod.* 1969; 55: 109–123.
15. Arat ZM, Akcam MO, Esenlik E, Arat FE. Inconsistencies in the differential diagnosis of open bite. *Angle Orthod.* 2008; 78: 415–420.
16. Fields HW, Proffit WR, Nixon WL, Phillips C, Stanek E. Facial pattern differences in long-faced children and adults. *Am J Orthod.* 1984; 85: 217–223.
17. Karlson AT. Association between facial height development and mandibular growth rotation in low and high MP–SN angle faces: a longitudinal study. *Angle Orthod.* 1997; 67: 103–110.
18. Cozza P, Mucedero M, Baccetti T, Franchi L. Early Orthodontic treatment of skeletal open-bite malocclusion: a systematic review. *Angle Orthod.* 2005; 75: 707–713.
19. Takada K, Sorihashi Y, Stephens CD, Itoh S. An inference modeling of human visual judgment of sagittal jaw-base relationships based on cephalometry: Part I. *Am J Orthod Dentofacial Orthop.* 2000; 117: 140–146.
20. Song HG, Pae EK. Changes in orofacial muscle activity in response to changes in respiratory resistance. *Am J Orthod Dentofacial Orthop.* 2001; 119: 436–442.
21. Landis JR, Koch GG. The measurement of observer agreement for categorical data. *Biometrics.* 1977; 33: 159–174.
22. Beckmann SH, Segner D. Changes in alveolar morphology during open bite treatment and prediction of treatment result. *Eur J Orthod.* 2002; 24: 391–406.
23. Ngan P, Fields HW. Open bite: A review of etiology and management. *Pediatr Dent.* 1997; 19: 91–98.
24. Lindsey CA, English JD. Orthodontic treatment and masticatory muscle exercises to correct a Class I open bite in an adult patient. *Am J Orthod Dentofacial Orthop.* 2003; 124: 91–98.
25. Arat ZM, Arman A. Treatment of a severe class III open bite. *Am J Orthod Dentofacial Orthop.* 2005; 127: 499–509.
26. Kondo E, Aoba TJ. Non-surgical and non-extraction treatment of skeletal class III open bite: Its long term stability. *Am J Orthod Dentofacial Orthop.* 2000; 117: 267–278.