

THE ORTHODONTIC/ TMD PATIENT: REVIEW OF THE LITERATURE AND AN INTRODUCTION TO STRUCTURED CLINICAL DECISION MAKING

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Introduction

Any knowledge of the treatment of temporomandibular disorders (TMD) is based on subjective opinion contained in the dental literature and is confused. The introduction of new terminology which lacks both scientific and clinical validity only adds to the confusion.

The quip: "What happens to a patient depends on what floor the elevator in the professional building stops at." was used by Greene¹ to describe the predicament of the TMD patient. This paper attempts to consider the clinical situation of the patient with TMD alighting at the orthodontist's office; and also the patient who develops TMD during orthodontic treatment. It also provides a guide to clinical decision making based on current scientific knowledge.

An integral component of good clinical practice is sound decision making. A decision tree (a diagrammatic aid to decision making based on interpretation of the literature) constitutes the means of making a best guess, and as such should be subject to periodic review and modification as new knowledge arises. Because much of the orthodontic related TMD literature to date comprises case reports and, as

stated above, subjective opinion, care should be exercised when interpreting case reports as appropriate control information is often missing, and spurious associations between treatment and outcome may be made. Reynders² has recently surveyed the literature (1966-1988) which addresses associations between TMD and Orthodontics. Of a total of 91 articles, 55 were "Viewpoint" articles (such as that used in *The American Journal of Orthodontics and Dentofacial Orthopedics*), 30 were case reports and six were sample studies. Thus, it would seem that, until 1988 at least, there was very little scientifically sound information in a subject which attracts much interest and debate. The importance of this topic to contemporary orthodontic practice is reflected in the devotion to TMD of the January 1992 issue (Volume 101) of the *American Journal of Orthodontics and Dentofacial Orthopedics*. It is further emphasised in the Editorial and a response to the Editorial in the June 1993 issue of the same Journal.^{3,4}

Two questions to be considered are: (1) does orthodontic treatment cause TMD? and (2) does orthodontic treatment cure TMD? As orthodontists it is obviously attractive to answer "No" to the first question, and "Yes" to the second. The first question has raised much interest recently, particularly in the wake of a much publicised Michigan orthodontic-TMD lawsuit.⁵

Does Orthodontic Treatment Cause TMD?

For some time now various writers⁶⁻¹¹ have claimed that premolar extractions and some forms of treatment mechanics, particularly incisor retraction trapping the mandible in a retruded position, cause TMD. Much criticism of these treatment modalities is directed to "traditional" university-trained orthodontists with the insinuation that the so-called "functional orthodontists" (FOs) are able to offer a "TMD kind" treatment.

Recent evidence supports the view that there is no relation between premolar extractions and TMD dysfunction.¹²⁻¹⁴ Dibbets and van der Weele¹² concluded that where dysfunction did occur in extraction cases, it related to the growth pattern, rather than the extractions; that is, the selection for extraction also appears to select for TMD.

The study of O'Reilly *et al*¹⁴ investigated the relation between the use of Class II elastics, premolar extractions and TMD. They used an experimental design with both experimental (orthodontic treatment, extractions, Class II elastics) and control (untreated - the experimental subjects nearest age siblings) groups. The only statistically significant finding was mild pain on palpation lateral to the TMJ capsule - reported in 40 per cent of the patients at the 8 to 10 month period during orthodontic therapy. However, there were no other discernible differences between control and experimental groups during treatment and following appliance removal. These results support the view that orthodontic treatment with Class II elastics does not cause TMD. These authors postulated that the increase in reported pain at the 8 to 10 month period may have been due to the fact that this period coincided with closure of extraction spaces and that the pain was due to soreness because of tooth movement.

Studies evaluating condylar position by using tomograms fail to demonstrate any difference in condylar position in patients who had premolar extractions when compared with those treated non-extraction¹⁵ and the untreated controls.¹⁶⁻¹⁸ In fact, the main feature of these studies is the large variation in condylar morphology and position between individuals. Furthermore, Blaschke and Blaschke¹⁹ have noted that, although the means of measurements of joint position indicated the condyle to be positioned in the centre of the fossa in centric occlusion, the amount of standard deviation suggests a large dispersion of individual positions around the mean. Dixon, in a recent review²⁰, concluded that not only is any technique of temporomandibular joint imaging inappropriate for assessing condylar position, but also that condyle-fossa eccentricity, particularly posterior condylar displacement, is not an indicator of dysfunction. Support for this claim derives from earlier studies such as that by Katzberg *et al*²¹ indicating that severe internal derangement of the joints is not related to retrusion of the condyle either alone, or in comparison with normal subjects. Furthermore, depending on the evaluation method used, patients who are seen to have normal arthrograms may demonstrate tomographic evidence of condylar

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repositioning.²² Thus, the recommendation of Witzig and Spahl²³ that radiographic assessment of condyle-fossa relations be made must be rejected. Recently, Luecke and Johnston²⁴ used a technique of regional cephalometric superimposition to evaluate dental and skeletal changes in patients who had upper incisor retraction following premolar extractions. Of the patients who had been treated for Class II Division 1 malocclusions, 70% showed a net forward displacement of the mandibular basal bone. However, regardless of the direction of basal displacement, condylar remodelling acted to stabilise the relative position of landmarks thereby supporting the view that serial radiographs are of little use in assessing changes in condylar position in growing patients.

The question of whether or not the Class II Division 2 characteristic feature of retroclined maxillary incisors has consequences for the temporomandibular joints because the mandible is trapped by the incisors and forced backwards is controversial. Demisch *et al* (1992)²⁵ recorded the relation between the retruded and intercuspal mandibular positions in a group of such patients. They found that the anteroposterior distance between the retruded contact position and the intercuspal position was large before the start of treatment and did not change with upper incisor proclination. This result suggests that anterior mandibular positioning does not occur as a result of "freeing up" of the occlusion. In addition, a relatively large distance between the retruded contact position and the intercuspal position would appear to speak against a posteriorly trapped mandible.

In regard to Class III traction, it has been proposed that mechanics exerting a distal force on (for example) the condyles may be harmful to the temporomandibular joint.¹¹ There is, as yet, no evidence to support this proposition. The only sample data concerning TMD and Class III mechanics (including chin cups) indicate that these treatment modalities do not differ from other treatments in relation to the signs and symptoms of TMD, and appear to have no relation to TMD many years after treatment completion.¹²

Thus, in view of the above studies, and other sample reports,²⁶⁻³¹ it would seem that orthodontic treatment, by

itself, does not cause TMD.

Does Orthodontic Treatment Cure TMD?

As the premise that TMD is caused by orthodontic treatment has been rejected, the question of whether or not orthodontic treatment can effect a cure for TMD must be approached with caution. In fact, to claim that orthodontic treatment can provide a cure despite a lack of scientific data could only be interpreted as "self serving". Although it would seem improbable that while not causing TMD, orthodontic treatment can cure TMD, it cannot be ruled out.

It has been suggested that TMD may be resolved by means of orthodontic therapy in the case of the "trapped" mandible that occurs in the Class II Division 2 case.²³ To ascribe such a primary role to the dentition is in direct conflict with the concept of a dentoalveolar compensatory mechanism³² whereby the dental arches are depicted as flexible ribbons conforming to varying jaw relations.

Sample studies have reported decreases in TMD with orthodontic treatment³³⁻³⁴ suggesting that it has a curative role. However, the studies laying claim to such an effect were inadequately designed to test whether or not orthodontic treatment itself gives relief of TMD. These studies failed to acknowledge that TMD is actually a number of disease entities (for example, myofascial pain, disc displacements, arthralgia and arthritis) with multifactorial aetiology. It should also be kept in mind that signs and symptoms may not only be part of disease, but also part of "normality". It is not sufficient to demonstrate a decrease in TMD signs and symptoms as a result of orthodontic treatment. To correlate orthodontic treatment with TMD one needs to show a decrease over and above the placebo and "hands on" effects: that is, appropriate controls are required. In addition, subjective and objective TMD symptoms come and go in an unpredictable fashion; a feature borne out once again in the recent study of Hirata *et al*.²⁷ Symptoms, therefore, are not necessarily steady indicators of a particular underlying disease.

Temporomandibular joint sounds have been used as a sign of TMD. Reports to date indicate that clinical

temporomandibular joint sounds do not develop as a consequence of orthodontic treatment,³⁵ and furthermore, there does not appear to be a correlation with functional occlusion.³⁶ In view of temporomandibular joint sounds being only one sign of TMD, the fact that they are intermittent, and occur in approximately 10 to 35 per cent of the general population in the absence of any evidence of disease progression,³⁷⁻³⁹ suggests that temporomandibular joint sounds alone do not necessarily indicate degenerative pathology, and should not serve as a reason to undertake orthodontic therapy. In addition, it seems probable that all joints have discrete sounds, with the only limitation to their detection being the sensitivity of the recording apparatus.⁴⁰

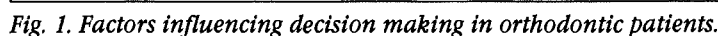
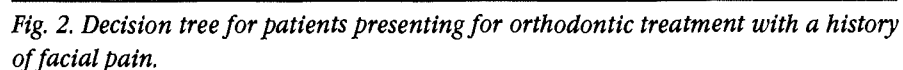
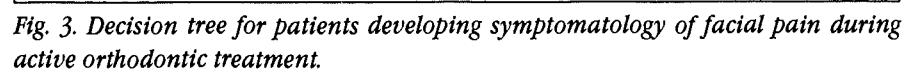
As orthodontists it is tempting to take a mechanistic approach to TMD, ignoring other factors such as the psychological, which are now thought to play a significant role.⁴¹⁻⁴³ Some studies have concluded that signs and symptoms of TMD are correlated with certain malocclusions. These include: Class II molar relations,⁴⁴ Class III malocclusions and crossbites associated with lateral excursions;⁴⁵ other studies have been unable to demonstrate such associations.^{30,46} On the basis of the lack of consistency of statistical associations in studies relating TMD and malocclusion, one must concur with Greene¹ who, in 1982, stated: "A person presenting with symptoms of this disorder in combination with a structural malocclusion should not be advised to undergo orthodontic treatment for either short-term relief from symptoms or as a preventive long-term therapy."

The Orthodontic Patient with Concurrent TMD

As discussed above, signs and symptoms of TMD come and go in an unpredictable manner. Therefore, it is more than likely that a number of patients who commence orthodontic treatment free from any recognisable form of TMD will go on to develop signs and symptoms of TMD during treatment. It has been observed that symptom frequencies may be age-dependent, thereby increasing the likelihood of TMD occurrence at some state during treatment. However, the lack of an association between TMD and orthodontic treatment in no way makes a full and proper examination redundant.

With regard to the changing perceptions about the relation between orthodontic treatment and TMD, and countering the un-scientific approach of groups such as the American Association of Functional Orthodontists (AAFO), a comment by Vig³⁰ sums up the state of TMD: "The eradication of firmly held beliefs, particularly if appealing and shared by many, is far more difficult than refutation of scientific theories which in turn leads to the advancement of knowledge. This is another way of saying that paradigms are more resistant to change than scientific theories."

Patients who exhibit generalised musculoskeletal symptoms require evaluation by a rheumatologist. The orthodontist should be alert to the



possibility of fibromyalgia (particularly in young females), connective tissue disease and polyarthritic conditions such as: rheumatoid arthritis, juvenile rheumatoid arthritis, Lyme disease, and other relatively rare systemic conditions.³¹ Such conditions have serious implications for the long-term and overall well-being of the patient, and, in the presence of generalised musculoskeletal pathology, orthodontic considerations should take a secondary role to management by the rheumatologist. In the case of localised facial pain a diagnosis needs to be made to exclude the non-TMD patient. Conditions falling within the category of TMD may be subdivided according to disk interference disorders, TMJ arthropathies and masticatory muscular disorders. Monitoring of the condition prior to initiation of orthodontic therapy is essential in order to determine whether or not the disorder is progressive; or if the signs reflect a previous episode of disease. In cases where an anterior open bite is evident, particular attention should be given to evaluation for osteoarthritic conditions; even in the absence of symptoms.³² The tomogram provides a useful initial screening method for detection of bony pathology.²⁰

Factors influencing clinical treatment decisions are shown in Fig. 1, while Figs. 2 and 3 show decision trees for orthodontic patients displaying symptomatology of facial pain. These are based on the above conclusions that: (1) orthodontic treatment does not cause TMD, and (2) orthodontic treatment does not cure TMD. When treatment is to be initiated in the patient with a history of TMD, that patient should have a clear understanding of the disease process and appreciate that the treatment - that is, correction of a malocclusion - will not necessarily cure the disorder.

In treatment planning, symptoms are regarded as more important than signs which may be regarded as normal or as the remnants of a previous condition long since resolved. When a history of facial pain exists prior to commencement of orthodontic therapy, a number of value judgements should be made by the patient and orthodontist in deciding whether to instigate or delay active orthodontic treatment. The extent of the treatment may range from a simple single arch procedure to a comprehensive procedure involving combined

Orthodontics and orthognathic surgery. In most cases, orthodontic treatment may be classified as elective; however, there are situations in which timed intervention is desirable in order to prevent progression of further pathology. These situations may involve a variety of considerations unrelated to TMD: for example, maxillary canine impactions where the radiological appearance suggests that the roots of the lateral incisors are in danger of resorption. Economic considerations also have an influence on the decision-making process; however, the temptation to allow these to dictate treatment should be avoided. One such example is the cleft lip and palate patient who may be eligible for some financial support, but with an upper age limit imposed. Thus, in delaying treatment, a balance between the risk to health and some future financial hardship must be struck. Although some orthodontic treatment is undertaken for reasons of function, aesthetics is usually the prime reason for seeking treatment.^{33,34} Aesthetic concerns are of little importance in short delays in the initiation of treatment although they may require consideration in relation to the psychosocial health of the patient^{35,36} when a long postponement is envisaged.

Conclusion

Orthodontic treatment and its association with TMD has been, still is, and will continue to be, a matter of debate and contention. Much of what has been written in the past is of an anecdotal nature and only relatively recently there has been a concerted effort to distinguish between such material and that derived from "sample" studies. A review of the scientific literature overwhelmingly supports the fact that, in general, there is no direct relation between TMD and orthodontic therapy, from either a causative or curative standpoint. The decision trees presented here reflect this view.

References

1. Greene CS. Orthodontics and the temporomandibular joint. *Angle Orthod* 1982; 52: 166-72.
2. Reynders RM. Orthodontics and temporomandibular disorders: A review of the literature (1966-1988). *Am J Orthod & Dentofac Orthop* 1990; 97: 463-71.
3. Graber TM. Editorial. Hardcore,

softcore or fringe. *Am J Orthod Dentofac Orthop* 1993; 103: 556-9.

4. Greene CS. Response to Graber editorial. *Am J Orthod Dentofac Orthop* 1993; 103: 16A-17A.
5. Pollack B. Cases of note: Michigan jury awards \$850,000 in ortho case: a tempest in a teapot. *Am J Orthod Dentofac Orthop* 1988; 94: 358-9.
6. Bowbeer GRN. Saving the face and the TMJ - Part 2. *Funct Orthod* 1986; 3(2): 9-24.
7. Broadbent JM. Second molar removal, third molar replacement. *Funct Orthod* 1986; 3(2): 37-9.
8. Levy PH. Orthodontics and TMJ. Disorders. *Am J Orthod Dentofac Orthop* 1991; 99(1): 21A-23A.
9. Spahl TJ. Problems faced by fixed and functional schools of thought in pursuit of orthodontic excellence. *Funct Orthod* 1988; 5(2): 28-34.
10. Witzig JW. An interview - AAFO's man of the year 1984. *Funct Orthod* 1984; 1(4): 9-19.
11. Wyatt WE. Preventing adverse effects on the temporomandibular joint through orthodontic treatment. *Am J Orthod Dentofac Orthop* 1987; 91: 493-9.
12. Dibbets JMH, van der Weele LT. Extraction, orthodontic treatment and craniomandibular dysfunction. *Am J Orthod Dentofac Orthop* 1991; 99: 210-9.
13. Kremenak CR, Kinser DD, Harman HA, Menard CC and Jakobsen JR. Orthodontic risk factors for temporomandibular disorders (TMD). I: Premolar extractions. *Am J Orthod Dentofac Orthop* 1992; 101: 13-20.
14. O'Reilly MT, Rinchuse DJ and Close J. Class II elastics and extractions and temporomandibular disorders: A longitudinal prospective study. *Am J Orthod Dentofac Orthop* 1993; 103: 459-63.
15. Gianelly AA, Anderson CK and Boffa J. Longitudinal evaluation of condylar position in extraction and non-extraction treatment. *Am J Orthod Dentofac Orthop* 1991; 100: 416-20.
16. Gianelly AA, Cozzani M and Boffa J. Condylar position and maxillary first premolar extraction. *Am J Orthod Dentofac Orthop* 1991; 99: 473-6.

17. Gianelly AA, Hughes HM, Wohlgenuth P and Gildea G. Condylar position and extraction treatment. *Am J Orthod Dentofac Orthop* 1988; 93: 201-5.
18. Kunding KK, Austin BP, Christensen LV, Donegan SJ and Ferguson DJ. An evaluation of temporomandibular joints and jaw muscles after orthodontic treatment involving premolar extractions. *Am J Orthod Dentofac Orthop* 1991; 100: 110-5.
19. Blaschke DD and Blaschke TJ. Normal TMJ bony relationships in centric occlusion. *J Dent Res* 1981; 60: 98-104.
20. Dixon DC. Diagnostic imaging of the temporomandibular joint. *Dent Clin North Am* 1991; 35: 53-74.
21. Katzberg RW, Keith DA, Ten Eick WR and Guralnick WC. Internal derangements of the temporomandibular joint: An assessment of condylar position in centric occlusion. *J Pros Dent* 1983; 49: 250-4.
22. Brand JW, Whinery JG, Anderson QN and Keenan KM. Condylar position as a predictor of temporomandibular joint internal derangement. *Oral Surg Oral Med Oral Path* 1989; 67: 469-76.
23. Witzig JW and Spahl TJ. The clinical management of basic maxillofacial orthopedic appliances, volume III: temporomandibular joint. Mosby Year Book, St. Louis 1991.
24. Luecke PE III and Johnston LE Jr. The effect of maxillary first premolar extraction and incisor retraction on mandibular position: Testing the central dogma of "functional orthodontics". *Am J Orthod Dentofac Orthop* 1992; 101: 4-12.
25. Demisch A, Ingervall B and Thuer U. Mandibular displacement in Angle Class II, Division 2 malocclusion. *Am J Orthod Dentofac Orthop* 1992; 102: 509-18.
26. Dahl BL, Krogstad BS, Øgaard B and Eckersberg T. Signs and symptoms of craniomandibular disorders in two groups of 19-year-old individuals, one treated orthodontically and the other not. *Acta Odontol Scand* 1988; 46: 89-93.
27. Hirata RH, Heft MW, Hernandez B and King G. Longitudinal study of signs of temporomandibular disorders (TMD) in orthodontically treated and non-treated groups. *Am J Orthod Dentofac Orthop* 1992; 101: 35-40.
28. Keremenak CR, Kinser DD, Melcher TJ, Wright GR, Harrison SD, Ziaja RR, Harmanb HA, Ordahl JN, Demro JG, Menard CC, Doleski KA and Jakobsen JR. Orthodontics as a risk factor for temporomandibular disorders (TMD). II *Am J Orthod Dentofac Orthop* 1992; 101: 21-27.
29. Rendell JK, Norton LA and Gay T. Orthodontic treatment and temporomandibular joint disorders. *Am J Orthod Dentofac Orthop* 1992; 101: 84-87.
30. Sadowsky C and BeGole EA. Long-term status of temporomandibular joint function and functional occlusion after orthodontic treatment. *Am J Orthod* 1980; 78: 201-212.
31. Sadowsky C and Polson AM. Temporomandibular disorders and functional occlusion after orthodontic treatment: Results of two long term studies. *Am J Orthod* 1984; 86: 386-390.
32. Solow B. The dento-alveolar compensatory mechanism: background and clinical implications. *Br J Orthod* 1980; 7: 145-61.
33. Janson M and Hasund A. Functional problems in orthodontic patients out of retention. *Eur J Orthod* 1981; 3: 173-179.
34. Egermark I, Thilander B. Craniomandibular disorders with special reference to orthodontic treatment: An evaluation from childhood to adulthood. *Am J Orthod Dentofac Orthop* 1992; 101: 28-34.
35. Sadowsky C, Theisen TA and Sakois EI. Orthodontic treatment and temporomandibular joint sounds - a longitudinal study. *Am J Orthod Dentofac Orthop* 1991; 99: 441-447.
36. Runge ME, Sadowsky C, Sakois EI and BeGole EA. The relationship between temporomandibular joint sounds and malocclusion. *Am J Orthod Dentofac Orthop* 1989; 96: 36-42.
37. Dworkin SF, Huggins KH, Le Resche L, Von Korff M, Howard J, Truelove E and Sommers E. Epidemiology of signs and symptoms in temporomandibular disorders: Clinical signs in cases and controls. *J Am Dent Assoc* 1990; 120: 273-81.
38. Sadowsky C. The risk of orthodontic treatment for producing temporomandibular disorders: A literature overview. *Am J Orthod Dentofac Orthop* 1992; 101: 79-83.
39. Stockstill JW and Mohl ND. Evaluation of temporomandibular joint sounds. Diagnostic analysis and clinical implications. *Dent Clin North Am* 1991; 35: 75-88.
40. Sutton DI, Sadowsky PL, Bernreuter WK, McCutcheon MJ and Lakshminarayanan AV. Temporomandibular joint sounds and condyle/disk relations on magnetic resonance images. *Am J Orthod Dentofac Orthop* 1992; 101: 70-8.
41. Green CS, Olson RE and Laskin DM. Psychological factors in etiology, progression and treatment of MPD syndrome. *J Am Dent Assoc* 1982; 105: 443-8.
42. Grzesiak RC. Psychological considerations in temporomandibular dysfunction. A biopsychosocial view of symptom formation. *Dent Clin North Am* 1991; 35: 209-26.
43. Marbach JJ and Lipton JA. Biopsychosocial factors of the temporomandibular pain dysfunction syndrome. Relevance to restorative dentistry. *Dent Clin North Am* 1987; 31: 473-486.
44. Riolo M, Brandt D and TenHave DR. Associations between characteristics and signs and symptoms of TMJ dysfunction in children and young adults. *Am J Orthod Dentofac Orthop* 1987; 92: 467-77.
45. Mohlin B and Thilander B. The importance of the relationship between malocclusion and mandibular dysfunction and some clinical applications in adults. *Eur J Orthod* 1984; 6: 192-204.
46. Pullinger AG and Seligman DA. Overbite and overjet characteristics of refined diagnostic groups of temporomandibular disorder patients. *Am J Orthod Dentofac Orthop* 1991; 100: 401-15.
47. Dibbets JMH and van der Weele, LT. Orthodontic treatment in relation to symptoms attributed to dysfunction of the temporomandibular joint. *Am J Orthod Dentofac Orthop* 1987; 91: 193-9.
48. Barbat LD. Orthodontic TMJ. Litigation in the 1990s: An ounce of

prevention is worth a pound of cure.
Am J Orthod Dentofac Orthop 1992;
101: 97-98.

49. Greene CS. Orthodontics and temporomandibular disorders. Dent Clin North Am 1988; 32: 529-38.
50. Vig PS. Reflections on the rationality of orthodontics: Toward a new paradigm. In: Vig PS, Ribbons KA, eds. Science and clinical judgement in orthodontics, craniofacial growth series, monograph 19, Center for Human Growth and Development, The University of Michigan, Ann Arbor, Michigan 1986: 15-59.
51. McCarty DJ. Arthritis and allied conditions. A textbook of rheumatology. 11th edition, Lea and Febiger, Philadelphia, 1989.
52. Seligman DA and Pullinger AG. The role of intercuspal occlusal disorders in temporomandibular disorders: A review. J Craniomand Disord Facial Pain 1991; 5: 96-106.
53. Albino JE, Cunat JJ, Fox RN, Lewis EA, Slakter MJ and Tedesco LA. Variables discriminating individuals who seek orthodontic treatment. J Dent Res 1981; 60: 1661-1667.
54. Jenny J. A social perspective on need and demand for orthodontic treatment. Int Dent J 1975; 25: 248-256.
55. Berscheid E. An overview of the psychological effects of physical attractiveness. In: Lucker GW, Ribbons KA, McNamara JA, eds. Psychological aspects of facial form, monograph No.11, Craniofacial Growth Series, Ann Arbor, University of Michigan, 1980: 1-23.
56. Striker G, Clifford E, Cohen LK, Giddon DB, Meskin LH and Evans CA. Psychosocial aspects of craniofacial disfigurement: A "state of the art" assessment conducted by the Craniofacial Anomalies Program Branch, The National Institute of Dental Research. Am J Orthod 1979; 76: 410-22.

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DIFFERENTIAL DIAGNOSIS IN ORTHODONTICS

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"The origin of all science is in the desire to know causes; and the origin of all false science and imposture is to accept false causes rather than none."

If Orthodontics is to progress by becoming more scientific, then a greater understanding of the causes of malocclusion has to be its objective. At this point, specific causes of malocclusions and malocclusion traits are often difficult to describe and, many times, at best, we can only propose aetiologic factors that may contribute to the variance of malocclusion. Nevertheless, our philosophy should be to continue to strive to identify causes because a known cause implies prophylaxis and rational therapeutics and *not* treatment that is empirical and symptomatic.

The concept of the aetiologic basis of malocclusion has received little attention in the orthodontic literature. The much quoted R.B. Dockerell² reference: "Classifying the Aetiology of Malocclusion" (Dental Record, February 1952) is singular in discussing conceptually the genesis of a malocclusion. Dockerell's fundamental

flow chart is often cited and is reproduced below for the reader to stimulate further thought.

Apart from our preoccupation with treatment mechanics, there is the pervading tendency to categorise malocclusion causes as essentially genetic or hereditary and it is these attitudes that have stifled the development of our differential diagnostic thinking. More and more findings from genetic research^{3,7} as well as research in primates^{8,9} are suggesting that the variance of malocclusion is the result of environmental factors as much as, if not more than, that of genetic influence. This appears to open up new possibilities for differential diagnosis and therapeutic modifiability; however, this should not, as a consequence, further reinforce the attitude that all malocclusions of genetic origin are necessarily more intractable.¹⁰

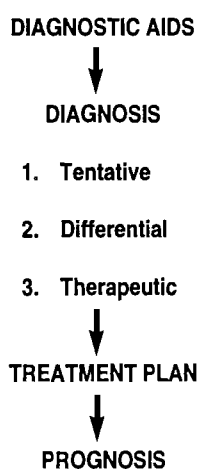
The meaning of differential diagnosis should be stated succinctly. A malocclusion or dental anomaly presenting apparently the same in two patients may be either analogous or homologous:¹¹ analogous means that only the outward occlusal factors are the same and homologous means that all characteristics and influencing factors of the malocclusions are in common. For example, analogous anterior openbites have exactly the same dimension in both patients; however, because of the aetiologic background to each case, one

CAUSES	act at TIMES	on TISSUES	producing RESULT
some predisposing	prenatal	which in turn affect	malocclusion
some exciting	or	other tissues so that	and / or
	postnatal	eventually one of	malfunction
such as:		these is affected	
hereditary		primarily and others	
nutrition		secondarily:	
virus and		muscle of face and jaws	
bacterial diseases			
trauma		bone of the jaws	
physical agents		teeth	
habits		soft parts excluding	
tumours		muscle	
etc.		temporomandibular joint	

may have an excellent prognosis for correction and, for the other, therapy may be poor. Considering differential diagnosis from another perspective, there can be many aetiological and associated factors which bring about a Class II canine relation; identifying the aetiological background will help decide which of the eight options¹² should be used in treatment objectives, namely:

1. distalise the upper canine
2. mesialise the lower canine
3. orthopaedics (move maxilla distally or retard forward growth)
4. differential growth of mandible (maximise mandibular expression of growth)
5. occlusal plane tipping or mandibular closure
6. extraction
7. orthognathic surgery
8. combinations of above

Unequivocally, the end purpose of all diagnosis and treatment planning is to determine a prognosis which indicates the worth of treatment.



Bibliography

1. Moyers RE. Handbook of orthodontics. 4th Ed. 1988. Year Book Medical Publishers.
2. Dockerell RB. Classifying aetiology of malocclusion. Dent Rec Feb 1952; 25-31.
3. Corruccini RS, Townsend GC, Richards LC, Brown T. Genetic and environmental determinants of dental occlusal variation in twins of different nationalities. Hum Biol Jun 1990; 62:3, 353-67.
4. Corruccini RS. An epidemiologic transition in dental occlusion in world populations. Am J Orthod Dentofac Orthop 1984; 86: 419-26.
5. Harris EF and Smith RJ. A study of occlusion and arch widths in families. Am J Orthod Dentofac Orthop 1980; 78: 155-163.
6. Lundstrom A and McWilliam JS. A comparison of vertical and horizontal cephalometric variables with regard to heritability. Eur J Orthod 9: 104-8.
7. Corruccini RS and Potter RH. Genetic analysis of occlusal variation in twins. Am J Orthod Dentofac Orthop 1980; 78: 140-54.
8. Harvold EP, Chierici G and Vargevik K. Experiments on the development of dental malocclusions. Am J Orthod Dentofac 1972; 61: 38-54.
9. Harvold EP, Tomer BS, Vargevik K and Chierici G. Primate experiments on oral respiration. Am J Orthod Dentofac 1981; 79: 359-372.
10. Lindstrom A. Biology of occlusal development. Cranial facial growth series, Centre for Human Growth, The University of Michigan, Ann Arbor, Michigan.
11. Ackerman JL and Proffit WR. The characteristics of malocclusion: a modern approach to classification and diagnosis. Am J Orthod Dentofac 1969; 56: 443-54.
12. Watson WG. Anticipation and midcourse changes for the Class II malocclusion. Am J Orthod Dentofac 1979; 75: 90-92.

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COMPUTERISED CEPHALOMETRICS

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The discussion of computerised cephalometrics may be divided into 3 parts: namely, the benefits and limitations of computerised cephalometrics; practice considerations; and the software packages available.

Benefits and Limitations of Computerised Cephalometrics

The reasons for using computer cephalometrics in orthodontic practice vary from practitioner to practitioner. The advantages and disadvantages may be summarised as follows:

Advantages

1. A more systematic and structured approach to the examination and analysis of radiographs; particularly for practitioners who do not routinely measure radiographs.
2. It is quick to measure and undertake a number of different analyses, often including a customised analysis, so that a given radiograph may be assessed numerically from a number of different perspectives.
3. Measurements for a given patient may be easily compared to population standards.

Treatment planning, particularly of surgical cases, may be facilitated as computers allow the effects of different treatment alternatives to be rapidly compared.

5. Tracings may be superimposed quickly and easily on a number of different reference planes and registration points so that the radiographic changes during treatment may be assessed more readily.
6. The formation of databases of pre- and post-treatment records so that the effects of treatment on different groups of patients may be evaluated objectively.
7. Patients gain the impression that the practitioner is up-to-date with current technology.
8. The tracings of patients' cephalograms (for example) may be included through word processing systems, into correspondence to referring dentists.

Disadvantages

1. Those problems usually associated with manual cephalometric analysis such as landmark identification and measurement errors.
2. The numbers produced may be misleading. Subtleties in form and structure apparent on a radiograph - which is at best a 2-dimensional representation of a complex 3-dimensional structure - may be lost once a tracing has been further reduced to a series of angles and linear measurements.
3. Once certain associated landmarks have been entered, certain software packages draw in a pre-determined anatomy for certain structures such as the pituitary fossa, nasal bone and condylar head regions. This leads to further inaccuracies when assessing tracings of radiographs.
4. The cost of acquiring the required hardware and software as well as the learning time for practitioner and staff. This is particularly the case if one wishes to combine cephalometric details with a word processor; or to form a database which may involve the learning of an additional program.
5. Population standards do not necessarily represent those of the patients being treated.
6. Programs are generally US-based which may mean compromised software support and delayed software upgrades.

Practice Considerations

A dedicated computer is virtually mandatory; and either some local area networking between computers or an additional printer is necessary in order to print tracings and analyses produced from the information that is entered. The physical size of an additional computer, screen and keyboard, digitiser, printer and possibly video equipment (the "footprint") is not inconsiderable and may be prohibitive.

Considerable time is required to allow the person entering data to become familiar with the concepts of computer cephalometrics and to learn the procedures involved. Previous experience or an enthusiasm for computers is an advantage.

Software Alternatives

Cephalometric software packages may be divided into those that have associated video imaging components and those that do not. IBM programs may be divided further into those which are Windows-compatible and those which are not.

Two non-video, Windows-based programs are RMO Joe (Jiffy Orthodontic Evaluation) (Rocky Mountain Orthodontics, (02) 570 9500 / 008 226 304) and Trace-X (John Mamutil, (02) 686 1200). These programs are well suited to the static analysis of radiographs, and to superimposing serial radiographs and forming databases.

The Orthodontic Treatment Planner (distributed by Data Software, Adelaide (08) 364 4074) is a Windows-based program suited to both radiograph examination and video image manipulation. Prescription Planner (Rx Data Design 0011 1 615 396 2110) and Dentofacial Planner (from Toronto (Rick Walker, 0011 1 416 360 0494) are programs that have similar radiograph and video capabilities (but different features); but in a non-Windows environment.

Quickceph in conjunction with Quickceph Image (Dr Gunter Blasio, Orthodontic Processing, 0011 1 619 437 4484, fax 0011 1 619 437 4485) allows comprehensive interpretation of radiographs with video capabilities on the Apple Macintosh platform.

An alternative, for those not considering an in-house system would be the RMODS radiograph evaluation service offered by RMO Australia ((02) 570 9500 / 008 226 304).

Conclusions

Before obtaining a cephalometric program, careful consideration should be given to what is hoped to be achieved by incorporating any such program into the practice; as well as how its utilisation will change the day to day regime of both practitioner and staff. Before acquiring any program it is worth while ascertaining both from the supplier and a current user, what the program is capable of and what training, maintenance and support is offered.

Certainly, the benefits of using computers in orthodontic practice as a clinical adjunct are worth considering in much the same way as history has shown that computerising the business aspects of orthodontic practice to be most beneficial. Which program a practitioner selects will ultimately depend on practice requirements and personal taste.

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THE USE OF THE HAND AND WRIST RADIOGRAPH IN SKELETAL AGE ASSESSMENT; AND WHY SKELETAL AGE ASSESSMENT IS IMPORTANT

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The initiation of active orthodontic treatment is often routinely determined by chronological age and the stage of dental development of the patient; however, neither of these factors is a reliable indicator of the skeletal development necessary to estimate height at maturity (EMH). The expected height is a vital factor in many decisions regarding the extraction of teeth. It might be better – and would permit a more objective diagnostic evaluation – if chronological age was not even considered as a basis on which to formulate a treatment plan.

In *The American Journal of Orthodontics and Dentofacial Orthopedics*, Vol. 69, No.6, 611-619, 1976, I reported the close relation between the ages at which peak growth velocities occur in stature and facial dimensions; and the attainment of ossification events in the skeleton of the hand and wrist. Information of this nature is useful in orthodontic diagnosis and for planning appropriate treatment where it is necessary to estimate whether growth is accelerating or decelerating.

The hand-wrist radiograph provides this, more reliable, information and permits recognition of early, average and late maturers. It may be used in clinical Orthodontics as a means of: growth prediction; as an estimation of the likely duration of retention; and the timing and efficiency of orthodontic treatment. For example: the timing and utilisation of extra-oral traction forces; the use of functional appliances; whether treatment is extraction or non-extraction; and the timing of orthognathic surgery are all based strongly on consideration of the growth of the cranio-facial complex.

Awareness of skeletal age is of significant importance in the management of a Class III malocclusion and the initiation of orthognathic surgery. Similarly, in the planning of a Class II malocclusion, the decision to extract two upper teeth or to institute distal movement with headgear often depends on the stage of the patient's growth.

Conclusion

Growth diagnosis is essential to a good treatment plan; and when treating adolescents it is important to obtain a hand and wrist radiograph as a routine part of diagnostic records.

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