Guest Editorial

Dental Anomaly Patterns (DAP)

A New Way to Look at Malocclusion

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Quick Quiz: (1) Why do children with missing permanent teeth show delayed formation of some of their other teeth? (2) Why do orthodontic patients with a palatally displaced canine almost always have enough dental arch space for their impacted tooth? (3) Why is maxillary canine/first-premolar tooth transposition so often accompanied by a missing maxillary lateral incisor? Answers below.

These interrelated conditions are examples of dental anomaly patterns (DAP): associated dental abnormalities that are observed together much more frequently than can be explained by chance alone. Dental anomalies such as missing teeth, teeth of atypical size or shape, certain ectopic tooth positions, and eruption abnormalities are an important part of the problem list for 10% to 20% of patients diagnosed with malocclusion. Orthodontists usually treat these anomalies without thinking much about their patterns of association or biological significance.

Based on previous studies, some of the measurable or visually discrete conditions that we may include as components of biologically related DAP are the following:

1. Absent teeth
2. Microform teeth (eg, peg-shaped lateral incisor)
3. Tooth-size reduction (generalized or localized)
4. Delay in tooth formation and eruption (generalized or localized)
5. Infraocclusion (most often of deciduous teeth)
6. Palatal displacement of canine
7. Maxillary canine-first premolar transposition (Mx.C.P1)
8. Mandibular lateral incisor-canine transposition (Mn.I2.C)
9. Distal angulation of unerupted mandibular second premolar

More associations no doubt will be discovered as the investigative spotlight shines brighter on the genetic patterning of dental anomalies, a fertile field for translational research.

Of all dental anomalies, tooth agenesis has been under the most extensive scientific scrutiny. The biological absence of one or more permanent teeth in an individual is the most frequently occurring discrete dental abnormality. This condition has been reported in the dentitions (including third molars) of about 25% of people studied worldwide. Associative, Mendelian, and molecular studies of patterns of tooth agenesis point to gene defects as the major factor underlying the occurrence of this anomaly.

An understanding of DAP in orthodontic diagnosis can help us modernize the century-old mechanical view of malocclusion. For example, take the frequently polarized discussions about the treatment of missing maxillary lateral incisors: prosthetic tooth replacement vs mesialized canine substitution. At meetings, we observe fine clinicians displaying treated cases of absent laterals, attempting to prove one point of view over the other: space opening or space closure. Rarely does anyone mention that patients with absence of one or both maxillary lateral incisors will likely exhibit other telltale signs of a biologically related DAP. In these patients, we may see significant reductions in the size of the teeth, delayed dental development and eruption, a greater chance of other missing teeth, and the presence of other dental abnormalities, such as palatal canines. These associated variations would surely affect the treatment decision significantly. Having small teeth signals the likelihood of adequate dental arch space, a factor that would weigh against a solution attempting heroic space closure since there is no associated arch-length deficiency. Furthermore, the tendency for these patients to present with other absent teeth would compound the argument against an aggressive space-closing treatment plan involving mesialized canine substitution.

My experience identifies four essentials in the diagnostic management of young orthodontic patients, especially for the nearly 20% who may show hints of DAP at their initial examination.

First, construct a panoral x-ray image for each new patient at about age 7 to 9 years. You would be surprised at the frequency of dental anomalies and developmental eccentricities you may find that will alert you to an aberrant dental pattern requiring close management and an interceptive eye. Generally, orthodontic patients with DAP are those who will need earlier treatment planning and often will have a longer treatment period than most patients.

Second, have close at hand a visually descriptive chart of the chronology of tooth development, especially one showing changes in tooth calcification and eruptive position annually from 7 to 12 years of age. Delayed tooth development may be detected by a quick dental-age assessment done by visually comparing the findings from a young patient's panoral radiograph and clinical examination with images from a reference chart. An ideal chart for this purpose was developed from the classic work of Isaac Schour and Maury Massler and was published by the American Dental Association.[1] It is reproduced today in several pedodontic textbooks.

Third, measure as many of the erupted permanent teeth as you can, accurately and with precision, to determine the patient's tooth-size profile: large, average, or small. Old-style vernier calipers, like the Boley gauge, produce unreliable measurements. Today, all of us should have a specially tipped dial caliper or a digital electronic caliper with a millimetric readout that can be used for direct or indirect odontometric measurement. Mesiodistal tooth-size dimensions should be compared with values from any of a number of published reference samples taken from your region's population.

Finally, take thorough family dental histories. In orthodontics, we have become lax about this. We should remember the familial nature of so many of the orthodontic problems we confront. It's remarkable how many parents will recall that they had a palatal canine or that their wisdom teeth never developed or that the patient's uncle has a retained anterior deciduous tooth or a very pointed small upper incisor. All of this information is important for the prediction of DAP in pretreatment children. There is a significant chance of expression of these autosomal dominant traits from one generation to the next.

I am not the only one excited about a DAP approach for better understanding and treatment of malocclusion. Associated dental anomalies are a focus of a number of biologically enlightened clinical orthodontists. Tiziano Baccetti, Daniela Garib, and Miriam Shalish are prominent among those who have contributed recently to our increased awareness of fundamental relationships among orthodontically significant dental abnormalities.

The DAP concept applied to clinical orthodontics should give you a feeling of new mastery the next time an 8-year-old with infraocclusion of deciduous molars and delayed tooth development sits in your consultation chair. While she is having a panoramic dental radiograph taken, you may confidently tell her parents that it is good they came in early, because such conditions are often accompanied by other dental variations, all very treatable. How comforting, how current, and how sensible—just what biologically based, modern orthodontics ought to be.

Answers

(1) Absent teeth and late dental development are biologically related. See: Garib DG, Zanella NLM, Peck S. Associated dental anomalies: case report. *J Appl Oral Sci.* 2005;13:431–436.

(2) Palatally displaced canine anomaly and generalized tooth-size reduction are biologically related. See: Langberg BJ, Peck S. Tooth-size reduction associated with occurrence of palatal displacement of canines. *Angle Orthod.* 2000;70:126–128.

(3) Maxillary canine-first premolar (Mx CP1) transposition and agenesis of maxillary lateral incisors are biologically related. See: Peck L, Peck S, Attia Y. Maxillary canine–first premolar transposition, associated dental anomalies and genetic basis. *Angle Orthod.* 1993;63:99–109.

REFERENCE

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