Disclosure: The author declares that in the past 12 months, neither he nor any member of his family has had a financial interest/arrangement or affiliation with any commercial entities or been paid directly by dentists and other health professionals within the field of dentistry or health care.

PART 1 OF A 2-PART SERIES

Permission To Intercept

An etiology-based approach to the interceptive treatment of preadolescent patients

BY DR. MICHAEL K. DELUKE

Short description

This course contrasts the current symptom-driven approach to interceptive orthodontic treatment with a new approach that focuses on diagnosing and treating the etiology of the malocclusion. The presented clinical case study demonstrates the results that can be achieved with an etiology-based approach to the interceptive treatment of preadolescent patients.

Synopsis

For more than 100 years, orthodontists have debated the value of interceptive treatment, especially as it pertains to the management of crowding in preadolescent patients who present in the early mixed dentition. The existing, antiquated paradigm is one that focuses on treating the *symptom*, but it is time to shift that paradigm to one that is more proactive and less

PACE ACADEMY of GENERAL DENTISTRY PROGRAM APPROVAL FOR CONTINUING EDUCATION

Farran Media is designated as an Approved PACE Program Provider by the Academy of General Dentistry. The formal continuing education programs of this program provider are accepted by the AGD for Fellowship/Mastership and membership maintenance credit. Approval does not imply acceptance by a state or provincial board of dentistry or AGD endorsement. The current term of approval extends from 1/1/2019 to 12/31/2023. Provider ID# 304396

AGD Code: 370

This print or PDF course is a written self-instructional article with adjunct images and is designated for 1.5 hours of CE credit by Farran Media. Participants will receive verification shortly after Farran Media receives the completed post-test. See instructions on p. 63.

ADA C·E·R·P® Continuing Education Recognition Program

Farran Media is an ADA CERP Recognized Provider. ADA CERP is a service of the American Dental Association to assist dental professionals in identifying quality providers of continuing dental education. ADA CERP does not approve or endorse individual courses or instructors nor does it imply acceptance of credit hours by boards of dentistry.

reactive—one that focuses on the diagnosis and treatment of the *etiology* of the malocclusion.

From an evolutionary perspective, dental crowding is a relatively new phenomenon that has largely emerged over the past 300 years. The dramatic increase in the prevalence and severity of dental crowding in preadolescent patients is the result not of increases in tooth size but the narrowing of the dental arches, secondary to environmental factors such as airway obstruction and a soft diet. As such, when we focus solely on addressing the crowding, we are incorrectly taking a symptom-based approach to treatment. Instead, we must first determine the underlying etiology of the malocclusion and then develop a treatment plan to address it. When we do, we can have a profound, life-changing impact on the physical and psychological health and well-being of our patients.

Learning objectives

At the end of this course, you should be able to:

1. Recognize the paradigm shift occurring in the diagnosis and treatment of malocclusion in preadolescent patients.
2. Describe the interconnectivity of the structures of the craniofacial complex as it pertains to the development of malocclusion.
3. Correctly identify the etiology of dental crowding in preadolescent patients.
4. Explain how airway obstruction can contribute to the development of malocclusion.
5. Recognize the clinical signs of airway obstruction in preadolescent patients.
6. Develop an etiology-based treatment plan for a preadolescent patient who presents with dental crowding in the early mixed dentition.

Introduction

Thomas Kuhn, an American philosopher of science and historian, stated that science does not evolve gradually toward the truth. Instead, it has a paradigm that remains constant before going through a shift when current theories can't explain some phenomenon and someone proposes a new theory. Kuhn defined a paradigm as a universally recognizable scientific achievement that, for a time, provides model problems and solutions to a community of practitioners. He argued that the masses will continue to follow the paradigm and do what they think is true while refusing to admit that what they're doing does not work. Further, it takes someone to come along and show that the old approach is no longer valid to shift the paradigm.[1]

The current approach to the interceptive treatment of crowding in the early mixed dentition (i.e., extracting teeth, rapid maxillary expansion, space maintenance or observation) is a paradigm that has remained constant for decades and continues to be followed, even though it neglects to address the etiology of the malocclusion and often yields less-than-ideal results.

As scientists and physicians of the oral cavity, it is incumbent upon us to break free from the existing symptom-based paradigm as it relates to the interceptive treatment of preadolescent patients. Further, we must shift the paradigm to one that focuses on diagnosing and proactively treating the etiology of the malocclusion.

Big teeth vs. small jaws

Dental crowding is one of the most common issues seen in our preadolescent patients. While crowding is technically defined as an inconsistency between tooth size and arch dimension, that simple definition says nothing about the etiology of the condition. In other words, is the crowding the result of teeth that are too big for the dental arch, or a dental arch that is too small to accommodate the teeth? Numerous studies have been published over the past 60 years in support of the latter.

In 1964, Mills concluded that the etiology of crowding was most often narrow arches, because little variation exists between crown diameters of persons with and without malalignment.[2] In 1981, McKeown reported that arch width and

crowding are strongly correlated and concluded that a narrow arch predisposes one to crowding of the teeth.[3]

In 1983, Howe, McNamara and O'Connor found no significant differences in tooth sizes between crowded and noncrowded groups, regardless of whether tooth size was compared individually or the mesiodistal sums of all the teeth in the arch were used.[4] Additionally, in 2018, Indriyanti and colleagues found the primary predisposing factors of crowding to be intercanine width, first primary intermolar width and alveolar arch width.[5]

It's all connected

The interconnectivity of structures of the craniofacial complex was recognized in the early 20th century. In an article published in 1928, Holtzman acknowledged the important roles the tongue and cheeks play in the development of normal arch width. Holtzman stated: "The normal action of the tongue and cheeks is in a great measure responsible for the shape of the arch. When the mouth is closed, the tongue lies in the floor of the mouth and the force of the atmospheric pressure sucks the dorsum of the tongue against the hard palate. This flattens the roof of the mouth. As the teeth erupt, the pressure of the sides of the tongue pushes them outward until the force is equaled by the inward pressure of the lips and cheeks."[6]

Research on craniofacial growth and development published in the 1960s further substantiated this concept. In 1962, Melvin Moss introduced the Functional Matrix Theory, which states that form follows function and, as such, the functional demands determine the final shape of osseous structures in the head and neck. This has also been referred to as "compensatory growth."[7]

In 1966, Enlow declared that the growth of the facial region is linked to that of other structural components and any alteration of one part of the craniofacial complex produces an equal alteration in another part, which in turn creates a functional imbalance.[8]

It is essential that orthodontists be aware of the environmental factors that contribute to this functional imbalance and subsequent compensatory growth in our preadolescent patients.

The role of the airway in the development of malocclusion

In the late 1970s and early 1980s, Harvold studied the impact of nasal passageway obstruction on the facial growth and development of rhesus monkeys.[9,10] Harvold found that monkeys who had silicone stuffed deep into their nasal cavities for six months developed narrower dental arches and more vertical facial growth than monkeys without obstruction. He then extended the trial period to two years and the results were even more profound, which led him to conclude that patients who are mouth breathers have more severe malocclusions, especially as it relates to transverse discrepancies and dental crowding.

While kids don't typically have silicone stuffed in their noses, many children do suffer from obstruction of the nasal passageways secondary to allergic rhinitis (AR). AR is the most common immune-mediated disorder in childhood, and it's estimated that AR affects up to 40% of children.[11] AR causes turbinate hypertrophy (TH), which can obstruct the nasal passageways; in fact, a 2019 study of 544 children between the ages of 3 and 10 found that the prevalence of TH in children with AR was 81%![12] If AR affects 40% of children and 81% of those affected have TH, we can assume that approximately one out of every three of our preadolescent patients is experiencing some degree of nasal obstruction from turbinate hypertrophy.

Furthermore, in school-age children, the prevalence of adenoid hypertrophy was reported to be 34.46%[13] and the prevalence of tonsillar hypertrophy was found to be 11%.[14] This hypertrophy of the lymphoid tissue often leads to upper airway obstruction with sleep-disordered breathing that ranges from mouth breathing, snoring and upper airway resistance syndrome to severe obstructive sleep apnea.[15]

When nasal breathing is obstructed, the nasal passageways and sinuses become underused and underdeveloped, leading to increased mouth breathing, further restriction of transverse development of the dental arches, and insufficient space for the tongue. The tongue is then forced inferiorly and posteriorly, where it can constrict the oropharyngeal airway and worsen the mouth breathing, especially when the patient is lying in a supine position.[16]

As the mouth breathing and snoring worsen, the transverse development of the arches continues to restrict. The modern soft diet and resulting weak tongue further exacerbate this negative cycle of aberrant facial growth (often referred to as "masticatory adaptation"),[17–23] exemplifying the interconnectivity described by Moss and Enlow many decades ago. We see this every day in our practices when young patients present with narrow arches and significant dental crowding.

The solution

Our traditional approach to the treatment of the aforementioned patient is one where treatment decisions are based upon the symptom (i.e., crowded teeth). Instead, we need to shift to a treatment approach that focuses on the etiology (i.e., narrow arches secondary to environmental factors). As such, our priority must be to address the environmental factors contributing to the malocclusion and normalize arch width, especially in the anterior and middle thirds of the arch.

Sample case

Let's look at this patient, 8 years and 10 months, who presented with the mother's chief complaint of "crowded teeth." The patient is Class I with a relatively normal OB/OJ and no posterior crossbite, yet she has moderate-severe anterior crowding and the maxillary canines are erupting toward the roots of the lateral incisors (Figs. 1–4). Our airway questions confirmed that she is a mouth breather and snorer, but the adenoids appear normal on the ceph and the palatine tonsils are not enlarged.

FIG. 1

FIG. 2

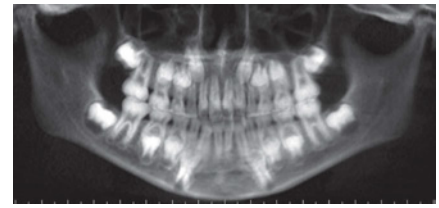
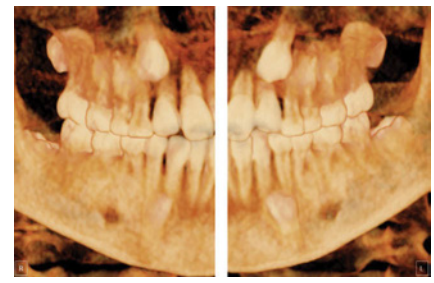


FIG. 3

FIG. 4



The existing paradigm says to base your treatment decisions on the management of the crowding/symptom. As a result, a common treatment plan would look something like this:

- Immediately send patient for extraction of all C's.

- Monitor the patient periodically until she is in late-mixed dentition.
- If she "grows well" and you think you can preserve enough arch length to accommodate the permanent teeth, insert space maintainers in the late mixed dentition in an effort to preserve the e-space and treat the case non-extraction.
- If she doesn't "grow well" and crowding becomes severe, extract four premolars.

Yet does preserving e-space or extracting premolars address the actual etiology? In other words, is the etiology of her crowding loss of arch length and/or large teeth? As you look past the teeth, you'll notice that she has narrow, V-shaped arches, large buccal corridors, a high/vaulted palate, a vertical growth pattern, insufficient space for the tongue, "tired eyes" with allergic shiners (indicative of atopy), erythematous gingival margins in the maxillary anterior sextant (or "chapped gums," as I like to say), and adherent plaque on the incisors—the latter two being symptoms of mouth breathing/snoring.

A low-dose CBCT is an invaluable diagnostic tool that allows us to obtain significantly more information than traditional 2D imaging. While it confirms that there is no obstruction of the nasopharynx (Fig. 5), we detect moderate turbinate hypertrophy in the nasal passageways (Fig. 6) and significant restriction of the oropharyngeal airway space (Fig. 7), providing us with a better understanding of the etiology of the presenting malocclusion.

Using this information, we can develop a treatment approach that addresses the etiology:

- Refer patient to the allergist for evaluation of turbinate hypertrophy and atopy.
- Place bite ramps and full braces with broad archwires to develop the arches, normalize arch width and create space for the tongue and the permanent teeth (Fig. 8).
- Retain with Essix C+.

Figs. 9–13 show the results that were achieved in just 14 months after only six adjustment visits (eight total visits including bond and debond). Notice the resolution of the crowding as well as the redirection of teeth #6 and #11 away from the roots of the permanent lateral incisors. Further, notice the improvement in both the interincisal angle and lip fullness relative to Rickett's E-plane. Amazing changes were also achieved in her facial aesthetics, tongue space, tired eyes, and allergic shiners (Fig. 14). Which child do you believe looks healthier?

FIG. 5

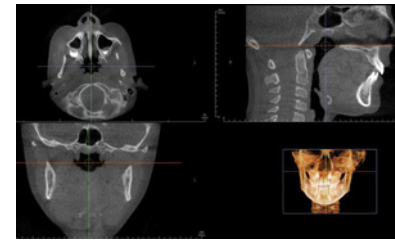


FIG. 6

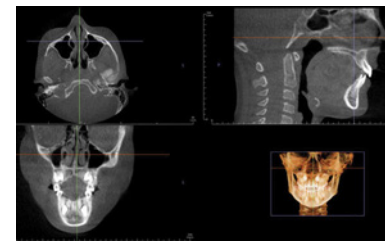


FIG. 7

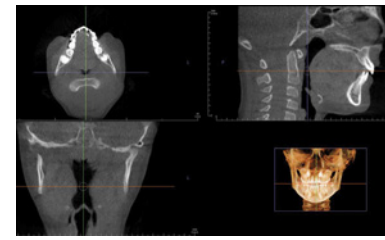


FIG. 8

FIG. 9

FIG. 10

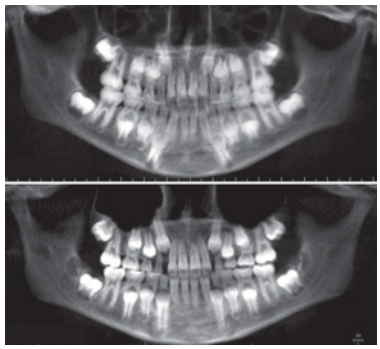


FIG. 11

FIG. 12

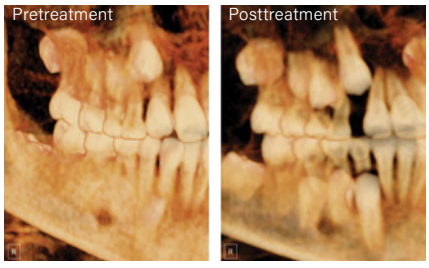


FIG. 13

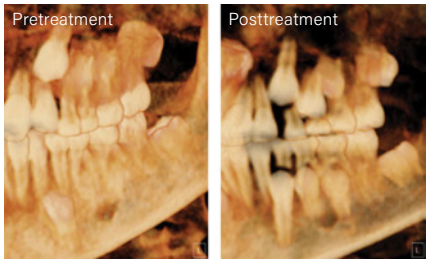


FIG. 14

The occlusal pictures (Figs. 15 and 16) demonstrate that the braces and wires achieved our objective of minimal expansion across the posterior teeth and significant expansion in the middle and anterior thirds of the arch, and the coronal CBCT slices (Figs. 17–21) show the magnitude of those changes. Furthermore, Fig. 18

FIGS. 15 AND 16

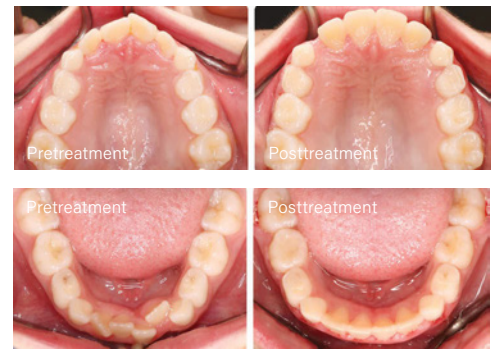


FIG. 17

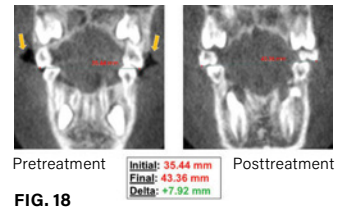


FIG. 18

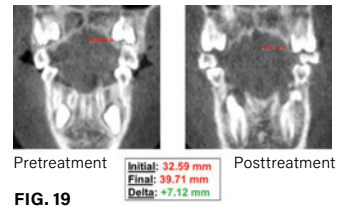


FIG. 19

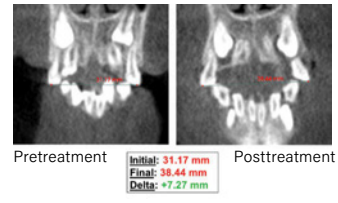


FIG. 20

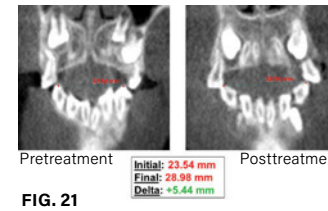
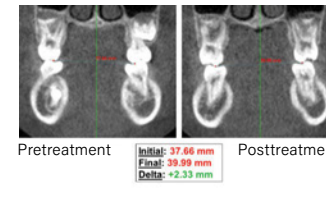


FIG. 21



demonstrates the reduction in the height of the palatal vault, the increase in tongue volume and the reduction of the buccal corridors.

Regarding the airway, the allergist diagnosed her with seasonal allergies and placed her on a steroid nasal spray and Zyrtec taken as needed. You can see the improvement in the nasal passageways in the posttreatment CBCT slice (Fig. 22) and the significant increases in total airway volume and minimum area (Fig. 23). The combination of managing the allergies and creating space for the tongue caused the mouth breathing and snoring to completely resolve. That's the power of treating the etiology versus managing the symptoms. We truly changed this little girl's life!

Lastly, the Phase I retention photos (Fig. 24), taken more than a year after the debond, demonstrate that the orthodontic results are beautifully stable when maintained with only Essix C+ retainers. Further, the mouth breathing and snoring have not returned.

In reviewing this case, it is important to consider the following questions: Which if any of the changes we made would have been achieved had we treated her in accordance with the existing paradigm described above? Would

FIG. 22

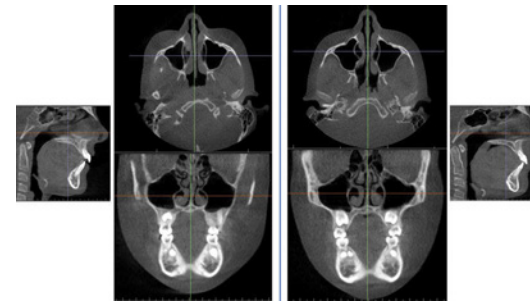


FIG. 23

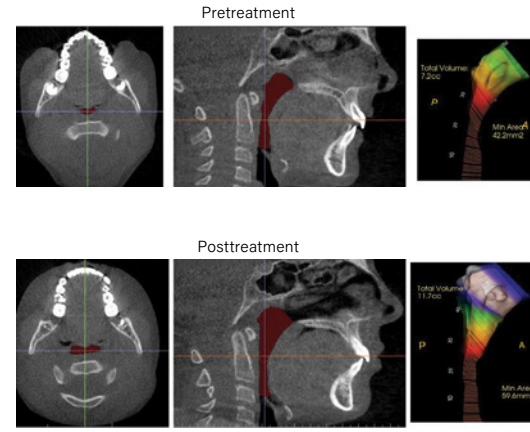
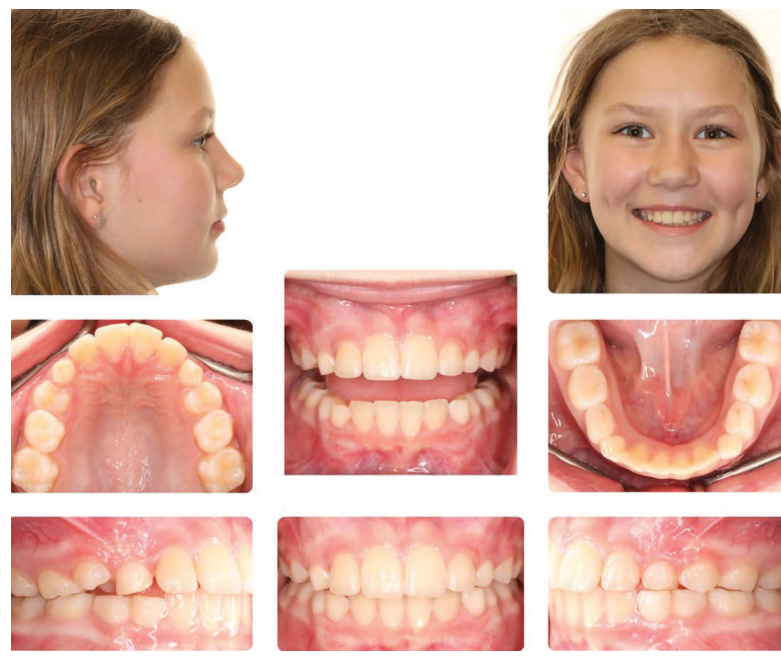


FIG. 24



she have stopped mouth breathing and snoring? Would her allergies have improved? Would her tongue space have increased? Would her canine eruption pattern have normalized? Would her soft-tissue profile have improved? Would her smile aesthetics have improved so significantly? Would her self-confidence have improved? I contend that the answer to all these questions is a resounding "no."

Patients, parents, team members and doctors love this approach, and incorporating it into your practice will allow you to change more lives and find more joy in the treatment of preadolescent patients than you imagined possible. It is time to embrace the new paradigm!

In the next course, we'll explore why slow expansion with braces and wires is a superior approach to RME in preadolescent patients, as well as present additional cases treated with this approach. **OT**

References

7. Kuhn T. The Structure of Scientific Revolutions (1962), p. 54.
8. Mills LF. "Arch Width, Arch Length, and Tooth Size in Young Males." Angle Orthod 1964; 34:124–129.
9. McKeown M. "The Diagnosis of Incipient Arch Crowding in Children." NZ Dent. J 1981; 77:93–96.
10. Howe RP, McNamara JA, O'Connor KA. "An Examination of Dental Crowding and Its Relationship to Tooth Size And Arch Dimension." Am J Orthod 1983; 83:363–373.
11. Indriyanti R, Efendi SH, Maskoen AM, Riyanti E. "Predisposing Factors Analysis of Mandibular Anterior Tooth Crowding in the Mixed Dentition Period by the Tooth Size and Dental Arch Width." Padjadjaran Journal of Dentistry. Nov 30, 2018:207–213.
12. Holtzman AC. International Journal of Orthodontia, Oral Surgery and Radiography, 1928; Vol 14, Issue 5, 390–392.
13. Moss ML. "The Functional Matrix." In: Vistas in Orthodontics. Karus B, Riedel R, editors. (Philadelphia: Lea & Febiger 1962: 85–98).
14. Enlow DN. "A Morphogenetic Analysis of Facial Growth." Am J Orthod 1966; 52:283–299.
15. Harvold EP, Vargervik K, Chierici G. "Primate Experiments on Oral Sensation and Dental Malocclusions." American Journal of Orthodontics 1973; 63:494–508.
16. Harvold EP, Tomer BS, Vargervik K, Chierici G. "Primate Experiments on Oral Respiration." American Journal of Orthodontics 1981; 79:359–372.
17. Ciprandi G, Tosca MA, Gallo F, Passali GC, Ameli F. "Turbinate Hypertrophy in Children With Allergic Rhinitis: Clinical Relevance." Acta Biomed 2020 Feb 17; 91(1-S):43–47.
18. Tsubamoto-Sano N, Ohtani J, Tanimoto K. "Influences of Mouth Breathing on Memory and Learning Ability in Growing Rats." Journal of Oral Science 2019; 61:119-–124.
19. Pereira L, Monyror J, Almeida FT, et al. "Prevalence of Adenoid Hypertrophy: A Systematic Review and Meta-Analysis." Sleep Med Rev 2018; 38:101–112.
20. Kara CO, Ergin H, Kocak G, et al. "Prevalence of Tonsillar Hypertrophy and Associated Oropharyngeal Symptoms in Primary School Children in Denizli, Turkey." Int J Pediatr Otorhinolaryngol 2002; 66(2):175–179.
21. Yoon A, Abdelwahab M, Bockow R, Vakili A, Lovell K, Chang I, Ganguly R, Liu SY, Kushida C, Hong C. "Impact of Rapid Palatal Expansion on the Size of Adenoids and Tonsils in Children." Sleep Med. 2022 Apr; 92:96–102.
22. D'Onofrio L. "Oral Dysfunction As a Cause of Malocclusion." Orthodontics and Craniofacial Research.
23. Proffit W, Fields H, Larson B, Sarver D. Contemporary Orthodontics, 6th Edition (St. Louis: Mosby/Elsevier 2007).
24. Evensen JP, Ogaard B. "Are Malocclusions More Prevalent and Severe Now? A Comparative Study of Medieval Skulls From Norway." Am J Orthod Dentofac Orthop 2007; 131:710–716.
25. Von Cramon-Taubadel N. "Global Human Mandibular Variation Reflects Differences in Agricultural and Hunter-Gatherer Subsistence Strategies." Proceedings of the National Academy of Sciences 2011; 108(49):19546–19551.
26. Pinhasi R et al. Incongruity Between Affinity Patterns Based on Mandibular and Lower Dental Dimensions Following the Transition to Agriculture in the Near East, Anatolia and Europe. PLoS One 2015; 10(2):e0117301.
27. Diamond J. The Third Chimpanzee: The Evolution and Future of the Human Animal (New York: HarperCollins, 1992).
28. Price WA. Nutrition and Physical Degeneration, 8th Edition (Lemon Grove, CA: Price Pottenger Nutrition Foundation, 2009).
29. Sim Wallace J. "Heredity with Special Reference to the Diminution in Size of the Human Jaw." Digest of Dental Record. Dec 1901, in Dental Digest 8, No.2 (Feb 1902):135–40.

Stay tuned for Part 2

In Part 2 of this series, Dr. Michael K. DeLuke will explain the drawbacks to some of the typical approaches many orthodontists would take to treat cases similar to the one in this article, and share additional case studies that illustrate a more effective way of treatment.

Dr. Michael K. DeLuke is a board-certified orthodontist who received his specialty training at the University of Connecticut. DeLuke practiced for 18 years at DeLuke Orthodontics before retiring from private practice to teach full time. He has served as a faculty member at several hospitals and orthodontic residencies, including as the cleft-craniofacial orthodontist at Albany Medical Center in New York.

DeLuke recently founded DeLuke Orthodontic Coaching (DOC), an ADA-CERP-recognized provider. He also created *The DOC Podcast* to help his colleagues attain the highest level of personal, professional and financial success. DeLuke is a fitness enthusiast and former boxer who still actively trains. He resides in Naples, Florida, with his wife and two teenage daughters.