ORTHO TRIBUNE
The World's Orthodontic Newspaper · U.S. Edition
AAO REVIEW 2013 — VOL. 8, NOS. 3/4 www.ortho-tribune.com

AAO brings it to Philly

Hot educational topics and a wide array of ortho offerings keeps annual meeting interesting

By Sierra Rendon, Managing Editor

Thousands of orthodontists and orthodontic team members traveled to Philadelphia in early May to take part in the various educational and social offerings at the American Association of Orthodontists 113th annual meeting.

The week's session offered an extensive list of clinical and practice management sessions, feature speaker sessions, oral research abstract presentations, table clinics and much more to fulfill attendees' educational needs.

▸ See AAO, page 10

AAO attendees head out of the Philadelphia Convention Center during a lunch break at the meeting in May. Photo/Sierra Rendon, Managing Editor

Diana P. Friedman, CEO, and Nicholas Wood, chairman, show off the Vespa that AAO attendees could win at the Sesame Communications booth.
Photo/Provided by Sesame Communications

Dr. Martin Van Vliet of Van Vliet Orthodontics takes the 'i-CAT FLX Challenge' at the i-CAT booth.
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Postretention relapse of mandibular anterior crowding

By Dennis J. Tartakow, DMD, MEd, EdD, PhD, Editor in Chief

Relapse of mandibular anterior crowding occurs in many well-treated cases, whether they had bicuspid extraction, non-extraction treatment, third molar removal, non-removal or agenesis. Why?

Is it related to (a) form and function, (b) tooth-mass issues, (c) occlusion, (d) temporomandibular relationship, (e) arch length discrepancy, (f) heredity, (g) orofacial musculature, (h) intra-oral forces, (i) extraoral forces or (j) oral habits? Or is it a combination of all the above?

The literature is filled with quality research studies attempting to discover answers to why relapse occurs, and despite decades of research, lower anterior crowding is still unpredictable (Freitas, K., Freitas, M., Henriques, J., Pinzan, A., & Janson, G., 2004). "The degree of postretention anterior crowding is both unpredictable and variable and no pretreatment variables either from clinical findings, casts, or cephalometric radiographs before or after treatment seem to be useful predictors" (Little, 1999, p.191).

For many years, the implication has been that nonextraction orthodontic cases might result in a higher percentage of postretention lower anterior crowding compared to extraction cases, but do they? Review of the literature regarding the effects that third molars have on lower anterior crowding presents both agreement and opposition for both sides of the problem, but the bulk of the evidence seems to indicate that third molars play an insignificant role in lower anterior crowding.

Parallel studies often show dissimilar deductions, and, more than likely, the problem is multifactorial. Bramante (1990) noted that in the 1930s Dr. Charles Tweed redirected orthodontic treatment procedures to extraction therapy with the removal of four first premolars as a more disciplined approach to effective orthodontic treatment. He added: "Fifty years later, we have found that extraction treatment and uprighting lower incisors does not prevent long-term postretention crowding and that flattened profiles are not always esthetically desirable" (p. 91).

Form and function certainly underlie growth and development in the craniofacial skeleton and the role that the biological environment plays. According to Carlson (1999), the "form-function" principle of craniofacial biology in general was attractive but primarily to account for the factors that may have influenced broad morphological variation and change associated with the evolution of the whole complex. Carlson added that the form-function principal is much less effective in explaining variations of craniofacial form, growth and treatment outcomes associated with causes of skeletal discrepancies and malocclusion.

So where does the discussion of lower incisor crowding end? Is the problem multifactorial, a product of improper orthodontic treatment modalities (or techniques), form vs. function, or does genetics play a large part in creating or solving the problem?

We can go on and on discussing virtues of the many possibilities and causative factors involved with postretention relapse of lower incisor crowding, but here we are in the 21st century and cannot provide an absolute answer to the riddle of postreatment stability. It is still an important objective but also still a scar of orthodontics.

Answers may never be absolutely identified, which, of course, begs consideration for some form of indefinite retention in almost all cases. However, we shouldn't feel totally alone with regard to our knowledge (or lack of knowledge) for an absolute answer to why relapse of postretention tooth movement occurs because, according to our medical colleagues at the Mayo Clinic (n.d.), even after years of research, physicians still have no cure for the common cold either.

'The literature is filled with quality research studies attempting to discover answers to why relapse occurs, and despite decades of research, lower anterior crowding is still unpredictable ...'

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Image courtesy of Dr. Earl Broker.

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Published by Tribune America

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Muscling in on the truth

A look at the causes of malocclusion

By Dr. Rohan Wijey, BOralH, Grad Dip Dent (Griffith)

Debate on the causes of malocclusion has been raging since the genesis of the orthodontic science, but has the answer already been found?

Various factions in the orthodontic tradition have declared the influence of both environmental and genetic determinants in malocclusion. Common consensus regards tooth position to be more environmentally influenced and skeletal development more genetically.

Genetic factors

Lauc et al (2003) claim that genetic factors are significant in malocclusion, citing a number of studies of twins. However, sibling genetic correlations are intrinsically fallacious in that they do not consider the influence of shared environments, which Garn et al (1979) have termed the "co-habitational effect."

Nonetheless, certain traits do seem to be characteristic among family members, and a possible explanation is that all animals seem to inherit certain muscular functions; Wiley (1962) describes the mating ritual of the three-spined stickleback, stating "the pattern and sequence of these movements is just as much a part of the genetic make-up of the fish as its body shape."

Epidemiological studies of malocclusion show it does not follow Mendelian laws of inheritance. Mew (1986) cites the example of sickle cell anemia, which provides near immunity to malaria. It has become endemic in populations where it is an asset for survival, and it displays a progressive familial and geographic spread.

Malocclusion, however, has shown no type of progressive spread, with every permutation found in every corner of the globe. An evolutionary change of this magnitude would also require millions of years, not one generation, and furthermore what genetic advantage has malocclusion provided for this supposed evolutionary change to materialize?

Certainly, there is a quality of irrationality to the genetic model for the etiology of malocclusion, but what is the answer?

Environmental factors

Evidence for environmental causes is formidable. Weiland et al (1997) compared skulls from 19th century Austrian males with their contemporaries, finding that change in diet ensured the latter displayed significantly higher malocclusion scores.

Corruccini and Lee (1984) reported that malocclusion was significantly worse in Chinese children born in the United Kingdom compared to their immigrant parents, raised in less developed areas. Because genetic factors remained unchanged, the malocclusion in the offspring was attributed to diet, premature deciduous tooth loss from caries and oral respiration.

Corruccini and Beecher (1981, 1983, 1984) have also shown that a soft diet significantly increases dental and skeletal malocclusions in rats, macaques and primates.

This is most likely because of less tonicity in muscles of mastication, resulting in compensatory overactivity in muscles of facial expression.

Perhaps most telling has been Harvold's series of experiments on primates in which induced oral respiration caused a range of malocclusions but all included increased face height, steeper mandibular place and larger gonial angle — in short, skeletal and dental discrepancies. Harvold's summation was that oral respiration was the trigger factor, but it is "deviant muscle recruitment" that directly causes maldevelopment.

The weight of the evidence, be it from the genetic or environmental school, seems to rest with muscle dysfunction being the cause of malocclusion.

Texture and nutritional value of diet has been shown to have an impact on tonicity of facial muscles, oral respiration causes "deviant muscle recruitment" and even from the genetic standpoint, the animal kingdom shows a marked tendency for muscle function (and dysfunction) to be inherited.

P.R. Begg's seminal 1954 manifesto asserted that a lack of grit in modern diets results in less interproximal wear and subsequently more crowding.

Although Begg believed that this environmental factor caused dental crowd-

▸ See MUSCLE, page 4

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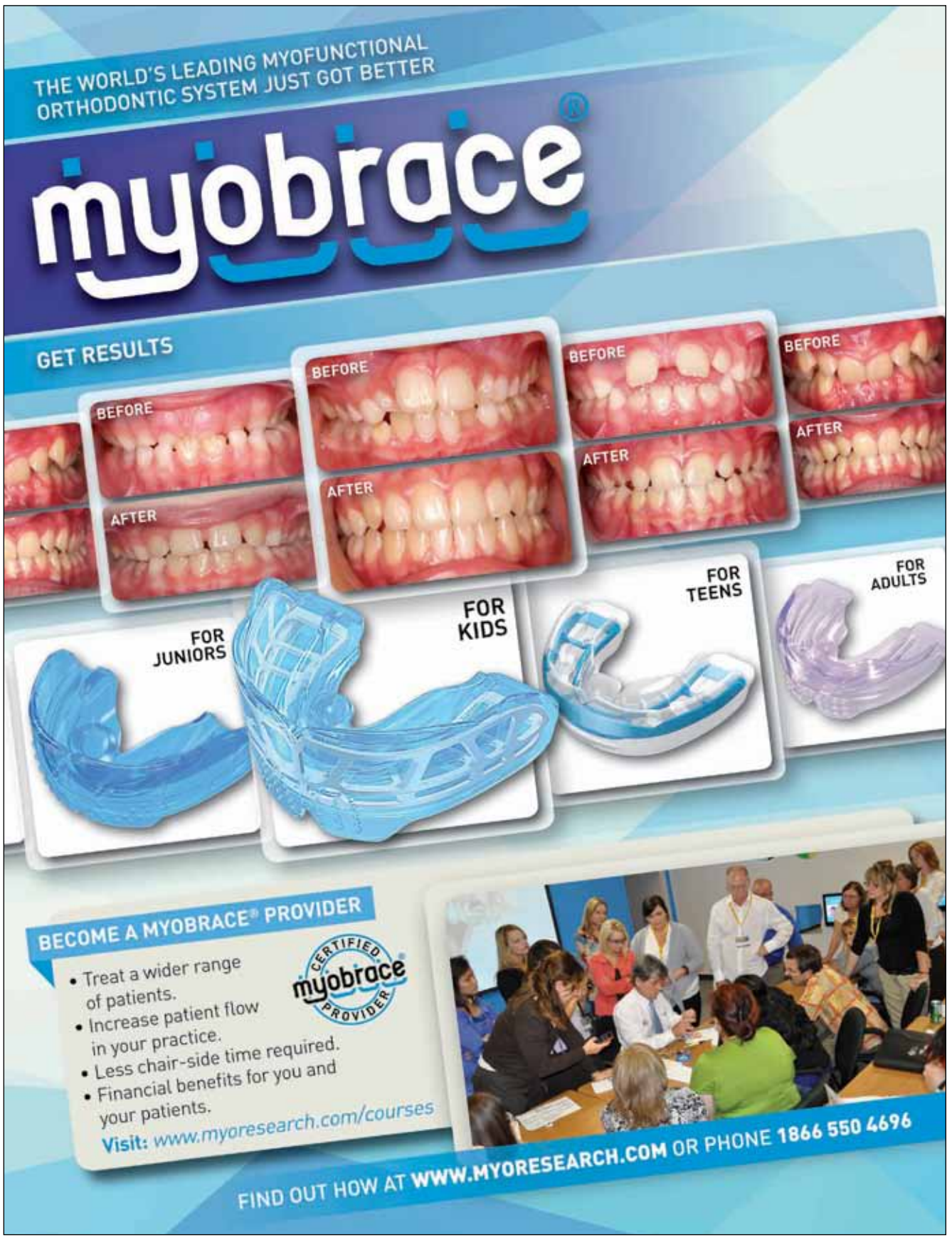

◂ MUSCLE, Page 3

ing, his theory was predicated on the belief that skeletal form is inherited and unmalleable.

Curruccini (1990), however, discredited this research and recognized that Begg's own figures render his theory redundant because both crowding and attrition increase with age.

Despite being roundly refuted, Begg's assertion still serves as the rationale and justification for orthodontists to shorten dental arches via extractions to this day.

Case study

This 13-year-old girl's profile (Figs. 1a–1d) shows a severely underdeveloped mandible, with a subsequent overbite. The strain of the mentalis muscle also betrays a "reverse swallow" with mentalis activity, which is the cause of this skeletal malocclusion.

After six months of myofunctional appliance use and myofunctional therapy, the release of muscle tension has allowed the mandible to translate anteriorly, with seemingly spontaneous lower dental alignment also a happy bonus (Figs. 2a–2d).

In spite of the evidence, the industry holds the concept of muscular causes of malocclusion at arm's length. Because when it is acknowledged, then the moral imperative for big changes will be inescapable.

That time is now.

Fig. 1a–1d: Before treatment, December 2009.

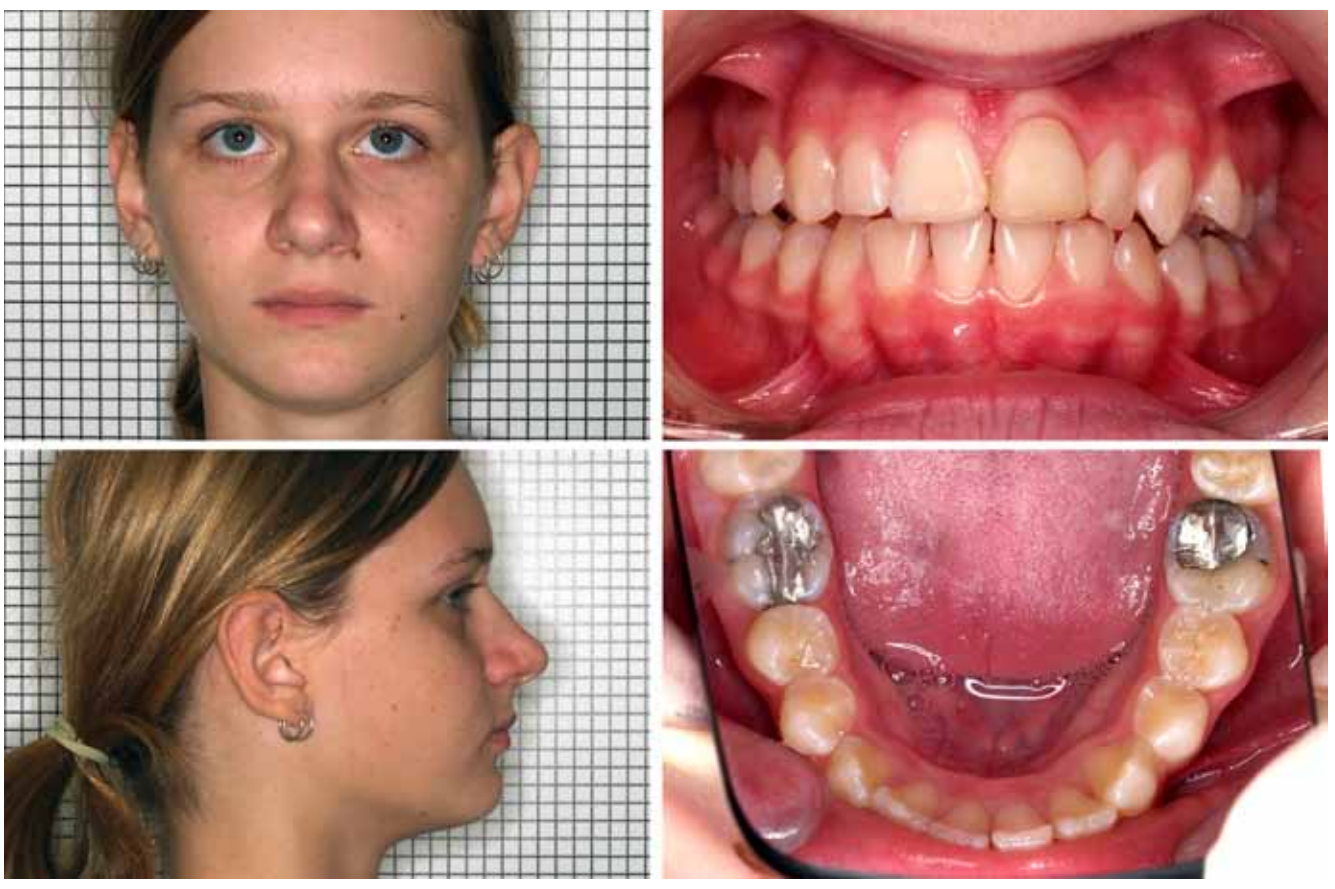

Fig. 2a–2d: May 2010.

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'In spite of the evidence, the industry holds the concept of muscular causes of malocclusion at arm's length. Because when it is acknowledged, then the moral imperative for big changes will be inescapable.'

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Boston's Yankee Dental Congress expands on team-development day

The bustling exhibit hall at the 2013 Yankee Dental Congress reflects the meeting's strong attendance figures — nearly 30,000. Photo/Provided by Yankee Dental Congress

28,000 expected at 2014 gathering

Organizers of the 2014 Yankee Dental Congress invite dental professionals to join 28,000 fellow professionals from Jan. 29 to Feb. 2 at the Boston Convention & Exhibition Center.

With a general theme centered on the concept of overall health starting with oral health, the YDC meeting offers the chance to explore and discover best practices, products and resources to improve your practice, increase your knowledge and better serve your patients.

Organizers describe Yankee Dental Congress 2014 as being the best opportunity in New England for every member of the dental practice to participate in a wide variety of programs.

YDC 2014 highlights include:

• *The Ritz-Carlton Leadership Center* — Back by popular demand, this program promotes performance and practice excellence by developing your leadership skills and creating the best possible service for your patients. Courses include "The Fire Within — Igniting Passion for Ritz-Carlton Performance Excellence," and "Legendary Service with a Smile," both presented by Jennifer Blackmon.

• *Hands-on cadaver programs* — This unique opportunity enables participants to attend hands-on courses using cadavers while exploring topics that cover areas such as anesthesia, crown lengthening and anatomy.

• *"Evolutionary Dentistry"* — Hear about the research and activities at the Manot Cave Dig in Israel and discuss the relationship of evolutionary biology to modern dental problems in a session led by Mark Hans, DDS, and Bruce Latimer, PhD.

• *The Pankey Institute: Update 2014* — Discover new techniques and innovative approaches in treatment planning that will have a valuable impact on your practice. The series includes three courses featuring speakers Greggory Di Lauri, DDS, Matthew Messina, DDS, and Kenneth Myers, DDS.

• *"Master the Skills of Marketing Your Practice in One Day"* — A one-day symposium designed to help expand your practice with the power of marketing. Courses include "High Energy Marketing to Explode Your New Patient Numbers," "Secrets of Social Media Success and Online Marketing," "Get Noticed, Get Booked, and Grow Your Practice" and "Best Practices for Leveraging Social Media to Engage Patients."

• *Dental Team Playbook: Strategies for Success (an expansion on the popular team-development day)* — Your entire dental team can benefit from this one-day program with courses tailored to dental assistants, hygienists and office personnel. Team members can learn from experts in their respective fields. Presenters include Lois Banta, Amy Kirsch, RDH, Shannon Pace Brinker, CDA, Diane Peterson, RDH, and Anastasia Turchetta, RDH.

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