is not good enough for members of a learned, evidence-based calling. We need a rational theoretical basis, a healthy sense of skepticism, and a desire to be as right as we are successful.

Andreas Vesalius was a conservative scientist. Many of his pioneering dissections thus were designed to rediscover the truths of Galenic physiology, among which is the idea that blood passes from the right ventricle to the left by way of holes in the interventricular septum. Vesalius, however, could not find these holes and declared it a tribute to the glory of God that they could be so important, yet so small that he could not see them. Can it really be that the effects of functional appliances are a modern (since 1870) version of Vesalius’s holes—vitaly important, but so small that few can detect them?

A useful answer probably can be inferred from the contemporary literature; however, if we can keep enough canaries in the air, it probably will go unnoticed.

Lysle E. Johnston, Jr
Ann Arbor, Mich

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does not occur at a constant rate, especially in young children. Even children of the same chronologic age might not have equivalent skeletal maturity or growth potentials. Therefore, when studies such as these do not have skeletal age as a common factor, it is difficult to produce a conclusive statement regarding the amount of growth modification that might or might not occur.” We think that our decision to use “inconclusive” is appropriate for the review by Chen et al.1

With respect to the study by Pancherz and Fackel,2 the reported sentence (chosen sentence) was “the results of the study indicated that dentofacial orthopedics using the Herbst appliance had only a temporary impact on the existing skeletofacial growth pattern.” The part of that article that was omitted (neglected sentence) pertained to the Herbst sample’s timing of treatment, which was before the pubertal growth spurt. Enough evidence already exists (especially in the 4 randomized clinical trials3-6) to support the concept that the prepubertal use of functional appliances does not produce significant supplemental growth of the mandible.

On a different topic, we excluded the article by Wieslander7 from the review because the treatment protocols consisted of a combination of 2 appliances used separately (Herbst full-time plus headgear nighttime only), and thus this study was not considered. The addition of the second appliance (headgear, not a functional appliance) obviously could influence the overall treatment effects significantly. This does not mean that we disregarded the possibility that headgear can provide supplementary mandibular growth. McNamara et al8 noted a modest increase in mandibular growth in Class II patients treated with cervical face-bows compared with untreated Class II controls. Furthermore, the article by Burkhardt et al9 indicated that molar distalization can be associated with amounts of mandibular growth that are comparable to those achieved with Herbst appliances. This topic, however, was beyond the scope of our review because we focused on the effects of functional appliances as classically intended and not on every orthopedic treatment protocol for Class II correction.

Another aspect deserves attention. When Dr Johnston claims that, “for comparison”—or the outcomes of treatment with functional appliances—“untreated Class I or II subjects should be good enough,” substantial evidence in an opposite direction can be provided. As already indicated in our review, some relevant literature demonstrated that mandibular growth in Class II subjects differs significantly from that of subjects with normal occlusions.10-12 Ngan et al13 performed a longitudinal evaluation of growth changes in Class II Division 1 subjects and found that “mandibular growth showed differences between the two Classes of malocclusion. In the Class II sample, mandibular length and corpus length were found to be shorter . . . when compared with a Class I sample. This was particularly apparent during the pubertal growth period.” With these observations in mind, we can reinterpret the findings by Pancherz,14 when (as reported in Dr Johnston’s Commentary) he writes: “When the activator patients were compared with subjects exhibiting normal occlusion (Bolton standards), . . . mandibular growth appeared not to be affected by activator treatment.” This actually is a very desirable result for activator therapy: an amount of mandibular growth in the Class II subject that equals the amount of mandibular growth in a subject with ideal dentoskeletal relationships!

Moreover, we resigned ourselves with reluctance (especially the 4 Italians in our group) to the inclusion criterion of only articles in English for the final review. Important articles with local distribution were excluded. However, the issue of communication should be considered. English is the recognized and most-used language for scientific investigation and reporting. At any rate, and personally, in case we receive in our mail an elegant card written in Japanese that announces that we won the Eastern World Lottery, we would never claim the money because we would not be able to read the card.

Finally, we would like to thank Dr Johnston for his contribution to “a healthy sense of skepticism” that helps everyone to understand better. As he indicated, “working in some unknown way to some unspecified degree is not good enough for members of a learned, evidence-based calling.” The attention to treatment timing in relation to individual skeletal maturity when planning therapy for a Class II malocclusion patient is an issue that surely adds some knowledge and specification to our ability to achieve better therapeutic results. We do have a clue that, if the diameter of Vesalius’s holes had been larger than 2 mm, it would not have been difficult to see them.

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