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Authors' response

We thank Dr Karthickeyan for his interest in our article, and we would like to clarify some of his concerns with the methodology and conclusions.

In our study, as in previous studies, overbite was measured solely on lateral cephalograms, and the reliability and validity of the measurements have been confirmed.¹ In addition, the tracing and measurements were repeated, and the error of the method and the intraobserver reliability were determined. Although 4.5 mm of overbite is concordant with the reported average value for the definition of increased overbite (IO) in the literature,^{1,2} in our sample, the minimum overbite was 5 mm in all IO groups. There is no doubt that the selected cases for the IO groups had an increased overbite.

Patients with microdontia and macrodontia were not included in the study. Patients with stainless steel crowns or large restorations were also excluded from the sample, as mentioned in the Material and Methods section.

Dental casts were not used, and the effect of tooth size arch length discrepancies, Bolton discrepancies, and transversal dimensions was not evaluated. However, to our knowledge, there is no evidence-based information about their effect. Of course, Bolton discrepancies have an important influence on establishing good occlusion. However, their effect on overbite is questionable. Even in cases of severe tooth size deficiency (Bolton), an acceptable Class I molar relationship with a reasonable overjet and overbite was found.³

Dr Karthickeyan wrote that discrepancies in the transverse dimensions might be the root cause of IO; narrow arches may lead to crowding and possibly an increase in overbite as a consequence. By contrast, no evidence-based information exists, considering the relationship between crowding and deepbite. The effect of transversal discrepancy on crowding is questionable in such malocclusion. Walkow and Peck⁴ found that the posterior arch widths in the maxilla and the mandible of patients with Class II Division 2 deepbite are the same, but that the mean mandibular intercanine width is significantly smaller than that of the controls. The transverse underdevelopment in the mandible from canine to canine is probably responsible for mandibular

incisor compression and crowding. In another study, Peck et al⁵ claimed that mandibular incisors' mesiodistal diameters were found to be reduced in patients with Class II Division 2 deepbite, and this requires a smaller anterior arch perimeter, which might accommodate the reduced mandibular intercanine width to minimize the crowding potential of the incisors. Further studies are needed to establish statistical significance for the effects of transversal relationship, crowding, and Bolton discrepancy on overbite increase.

Dr Karthickeyan emphasized that when there is sagittal discrepancy between the maxilla and mandible as in case of Class II Division 1, lack of stable incisor contact causes extrusion of the incisors, which contributes to an increase in overbite gradually during the growth period. The various mechanisms that might increase overbite during the growth period were mentioned in discussion. However, the situation is not stable during growth. Overbite can be worsened or improved depending on the growth stage. In patients with Class II Division 1 malocclusion, overbite was reported to be increased in children between the ages of 7 and 14 years. Feldmann et al⁶ found a significant improvement in overbite from ages 12 to 23 years in untreated postadolescent patients with Class II Division 1 deepbite malocclusion. IO can be seen in different types of sagittal malocclusions. In each type of malocclusion, various factors interact during the growing period.

Our study was conducted on adult patients to evaluate the factors of IO by eliminating the effects of the growth process. The main concept was to draw attention to the factors that may affect the decision while selecting treatment options in adults with IO. We could not have concluded, as the author advises, "irrespective of the sagittal position of both jaws, overbites of various extent were observed," because we especially chose the cases with IO.

The results of our study could be of interest in view of the potential clinical implications in orthodontics in the selection of the proper treatment mechanism, such as molar extrusion, incisor intrusion, or the combination of them for stable results. Our results reveal that mandibular incisor intrusion seems to be the main treatment concept in Class II Division 1 malocclusion. We agree with Dr Karthickeyan that malocclusion should be clinically decided only after an overall evaluation of the patient. Treatment of the malocclusion must be personalized, addressing the individual skeletal, dental, and soft tissue components. If there are additional problems on the amount of maxillary incisor visibility in rest position and smile, upper lip length, and presence of gummy smile, they should also be resolved.

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