Re: Brodsky et al.: Intermittent exotropia and accommodative esotropia: distinct disorders or two ends of a spectrum? (Ophthalmology 2015;122:1543–6)

TO THE EDITOR: In their editorial, Brodsky and Jung describe neurophysiologic mechanisms for the development of intermittent exotropia. They state that “prolonged patching causes it (exotropia) to become manifest.”

Mohney et al investigated the treatment of intermittent exotropia with patching, but do not explain how interfering with binocular function would preserve “binocularity and a reduction in the frequency and/or magnitude of the exodeviation.” The inclusion of an untreated control group assists in determining that patching is not a useful treatment for intermittent exotropia.

Neither paper addresses aspects of the bony orbit in children and how this could be related to intermittent exotropia. The axes of infantile orbits are oriented more laterally than in the adult. Lines drawn from the middle of the orbital opening to the optic foramen make an angle of 115° to each other, whereas in the adult skull the angle is 80° to 90°. The position of the eyes at rest would, therefore, have an outward deviation. This tendency toward exodeviation diminishes as the facial bones grow and the inner walls of the orbits become parallel.

Variations in the anatomy of the bony orbit that occur with growth should be considered in evaluating the presence and therapy of intermittent exotropia.

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References


REPLY: In our randomized trial comparing part-time patching with observation for intermittent exotropia in children 12 to 35 months of age, we reported the incidence of deterioration of intermittent exotropia for each group over a 6-month period. We did not measure orbital parameters and we are therefore unable to comment on any association between intermittent exotropia and orbital anatomy. The potential effects of differences in orbital anatomy between patients, or potential effects of longitudinal changes in orbital anatomy, were minimized by our random allocation of treatment assignments in this study.

Regarding the proposed mechanism whereby part-time patching might reduce the rate of deterioration of intermittent exotropia, it is possible that patching may result in a lessening or elimination of suppression, and thus lead to the historically reported “preservation of binocularity and a reduction in the frequency and/or magnitude of the exodeviation.” Nevertheless, we did not find a difference in deterioration rates between children who were patched and those who were observed, leading to our conclusion that there was insufficient evidence to recommend part-time patching for the treatment of intermittent exotropia in children in this age group.

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Reference


REPLY: We thank Dr Lempert for his interest in our editorial. Although orbital anatomic derangements can cause exotropia in patients with craniosynostosis or hypertelorism, the natural history of intermittent exotropia is that it generally becomes more severe during the first few years of life (as the orbits become more parallel). This is the opposite of what would occur if the lateral orientation of the neonatal orbits engendered this exodeviation. Dr Lempert’s proposed mechanism would, therefore, require some anatomic evidence that the age-related degree of