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Reply to Comment on: Masquerading superior oblique palsy

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Masquerading Superior Oblique Palsy

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Dear Editor,

We thank Dr. David Guyton for his thoughtful letter regarding our paper on masquerading superior oblique (SO) palsy. We agree with his contention that masquerading SO palsy is not a disorder of extraocular muscle strength. Our magnetic resonance imaging (MRI) evidence clearly excludes peripheral SO weakness as causative, as Dr. Guyton acknowledges. We concur that we have a problem insofar as we have not discovered the cause of masquerading SO palsy. Although we have recently shown elsewhere that sagging eye syndrome (SES) can also fulfill the 3-step test to mimic SO palsy[1], our masquerades did not have SES.

Dr. Guyton has noted that a defect in binocular fusion can lead to slowly progressive strabismus, offering the familiar example of sensory exotropia. Sensory exotropia utterly precludes binocular fusion since it occurs only with profound visual loss, which was not the case for our subjects. Binocular fusion has both motor and sensory aspects, with the former necessary but not sufficient for the latter. Unusually effective vertical fusional amplitudes of 3–27Δ were demonstrable in 16 of our 26 cases of masquerading SO palsy, indicating that absence of motor fusion cannot be the explanation. Nor did most of our masquerade subjects have primary binocular sensory defects. Of our 26 masquerades, stereopsis by Titmus testing was 40 arcsec in 8, was at least 60 arcsec in 11, and was measurable in 21 cases. Consequently, neither weak motor fusional vergence, nor defective sensory fusion, can be the primary cause of masquerading SO palsy.

Our recent magnetic resonance imaging (MRI) study of muscle contractility during normal vertical fusional vergence demonstrates that this is actuated by coordinated actions of multiple extraocular muscles, not primarily the obliques[2]. Enhanced vertical vergence

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compensating large hyperphoria is accomplished more by the inferior rectus than by the SO[3].

We agree with Dr. Guyton that another process must cause masquerading SO palsy. He proposes that defective fusional feedback somehow alters muscle lengths to mimic SO palsy, but does not detail a specific mechanism by which fusion would change particular muscle lengths, nor why such changes would cause hypertropia that varies with head tilting. We have shown by MRI that muscle lengths do increase in SES, but this appears involutional rather than due to fusional feedback[4]. Moreover, the enhanced vertical fusional vergence in the masquerades would seemingly be in the appropriate direction chronically to reduce, rather than increase, hyperphoria. Dr. Guyton's hypothetical non-zero vergence bias would have to exist external to muscle tension or visual vergence feedback loops.

We have offered the term "masquerading SO palsy" to counter the historically-based tendency of ophthalmologists to attribute cyclovertical strabismus to single muscle weakness, based solely on alignment measurements[5]. Such diagnostic bias has not been prevalent for concomitant horizontal strabismus such as esotropia and exotropia. Currently, nobody knows the cause of masquerading SO palsy. Solution of this mystery will require more precise clinical evaluations including orbital imaging of muscle status, and improvement in the basic science understanding of the central neural control and physiology of fusional vergence.

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