LATERAL RECTUS SAG AND RECURRENT ESOTROPIA IN CHILDREN

To the Editor: Clark and colleagues[1] have suggested some cases of acquired esotropia that recur after medial rectus recessions are caused by a lateral rectus path anomaly that they refer to as "muscle sag."[1] We find that, as illustrated in the article, it is not unusual to see (and feel) the lateral rectus muscle track toward the floor of the orbit as it courses posteriorly when tension is applied with a muscle hook. This is occasionally so dramatic that we feel compelled to check other landmarks—the inferior oblique insertion, inferotemporal vortex vein and inferior rectus muscle insertion—to assure that the inferior rectus muscle was not isolated by mistake. We see this phenomenon in patients with a variety of strabismus, perhaps most often in exotropia, simply because those are the patients in whom we most frequently have a hook under the lateral rectus muscle.

Because recurrent esotropia after surgery for acquired esotropia is likewise not rare, it is hard to know whether the 4 patients selected for also having "muscle sag" are not just a random association. As the authors point out, it would be helpful to know how often recurrent esotropia is associated with this lateral rectus finding and how often patients with this finding have esotropia. The response to myopexy is also not convincing with regard to the authors' proposed mechanism, because it was, in each case, accompanied by lateral rectus resection, which is by itself known to be effective treatment for recurrent esotropia. Myopexy was clearly not definitive treatment in the 1 patient, who initially had myopexy alone but subsequently needed lateral rectus resections when esotropia recurred. The initial response to myopexy in that patient might be taken as evidence that correcting an anomalous muscle path has some effect. Alternatively, consider that a myopexy that displaces the muscle from its natural course, anomalous or not, effectively increases the path length and is functionally similar to a resection (something that should be kept in mind when considering other articles regarding myopexy for muscle path anomalies as well).

While certainly a creative explanation, this article does not provide enough evidence to convince us that that this common lateral rectus phenomenon is related to acquired or recurrent esotropia in children.

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https://doi.org/10.1016/j.jaapos.2019.07.004
J AAPOS 2019;23:363.

1091-8531/$36.00

REPLY

We thank Drs. Archer, Christiansen, and Del Monte for critically reviewing our work and allowing us to elaborate on our findings. Fundamental to their criticism is the assertion that abnormalities in lateral rectus path, particularly inferior displacement, are relatively common findings at strabismus surgery and should not be considered to cause recurrent esotropia. Our imaging research, both the initial definition of normal extraocular muscle pulley locations[1] and subsequent analyses of hundreds of normal patients recruited as controls for other studies, has consistently demonstrated that extraocular muscle paths are highly stereotypical in people without strabismus. Contrary to the letter's assertion, the much higher prevalence of muscle path abnormalities in strabismic patients undergoing surgery supports—not contradicts—our hypothesis that muscle path disorders impair ocular motility and thus contribute to strabismus.

The "two-hit hypothesis" published by Knudson in 1971 provides an example illustrating how many medical conditions become manifest.[2] The basic premise is that many asymptomatic people have one hidden defect—genetic mutation, structural abnormality/variant, or trait—that does not cause pathology unless a second triggering condition exists. The classic Knudson research involved retinoblastoma, but the research relevant to strabismus involves the much more common accommodative esotropia. Most children who have moderate hyperopia are not strabismic.[3] The underlying hyperopia—the first "hit"—is by itself not sufficient to create esotropia. Other factors—analogous to the second "hit"—exist in children who actually manifest the condition: positive family history, reduced stereoacuity, and anisometropia.[4]

Abnormal lateral rectus paths, like uncorrected moderate hyperopia, also tend to imbalance ocular motility. From geometry, the abnormal force due to abnormal lateral rectus path is proportional to the magnitude of lateral rectus path displacement.[5] Analogous to a misaligned tire pulling a vehicle toward the roadside, muscle path displacements can be counteracted by applying

corrective forces until the abnormal forces become too great or a "second hit" occurs to overwhelm corrective mechanisms and manifest strabismus. Thus, the hypothesis that extraocular muscle pulleys support normal ocular motility by simplifying neurological control of binocular eye movements[6] is supported, not contradicted, by the frequent occurrence of extraocular muscle path abnormalities in patients who have abnormal motility.

The second major criticism offered by Archer and colleagues is that myopexy to restore normal lateral rectus path is ineffective without concurrent resection. This criticism misinterprets the effect of lateral rectus sag on muscle path length. In normal individuals, the lateral rectus path has minimal vertical displacement (approximately 1 mm)[1] along a straight path from orbital apex to insertion. A sagging lateral rectus, on the other hand, introduces a half tendon width or more vertical displacement to that path. Contrary to the offered criticism, a myopexy suture does not displace the lateral rectus "from its natural course" but rather restores its path into normal vertical alignment with both insertion and orbital apex. This change in lateral rectus path does not function "similar to a resection" but rather similar to a recession, since slack is introduced by straightening the vertically sagging lateral rectus path. It is not clear whether resection alone might reduce or increase lateral rectus sag by tightening the muscle, but our experience suggests the combination of resection and myopexy is more effective at normalizing lateral rectus function.

We agree with the criticism that findings in these 4 patients alone are not sufficient to prove a role for lateral rectus sag in childhood esotropia. This data, however, in combination with accumulating evidence for a causative role for lateral rectus sag in the great bulk of acquired esotropia in older adults,[7] further supports the vital role of normal extraocular muscle pulley locations in binocular alignment. Determining the pulley locations preoperatively is challenging in children, but given the high prevalence of abnormal muscle paths discovered serendipitously during surgery, further research in this area may prove valuable to improving surgical outcomes for many common types of strabismus.

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https://doi.org/10.1016/j.jaapos.2019.09.005
J AAPOS 2019;23:363-364.

1091-8531/$36.00