Clinical relevance of torsion to the ophthalmologist

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Ocular torsion is one of the most perplexing problems in strabismus diagnosis and management, partly because diagnosis may be difficult under certain circumstances, and complicated because treatment options are multifarious. The *perception* of torsion, or tilting in one's environment, does not always accompany ocular torsion, and vice versa, thereby further obfuscating the diagnosis. In this issue, Parsa has added an additional, clever approach to the diagnosis of ocular torsion.[1] His thorough review of the problem and new approach is worthy of a careful reading by those who manage strabismus. In the next paragraphs I will comment on diagnosis and management of ocular torsion, ending with some thoughts on the importance of this topic to the ophthalmologist who works with patients with ocular motility problems.

The strabismologist should be reminded that not every case of ocular torsion is caused by trochlear (4th) nerve palsies. Central nervous system aetiologies are common. Vestibular system and reticular system damage can cause torsional changes that significantly disrupt vision. Cerebellar damage, too, will also cause vague but troublesome torsional symptoms that are often intolerable to the patient.[2] On the other hand, a great number of cases of central torsion are asymptomatic, even when the degree of torsion is extreme. Lateral medullary infarcts are famous for causing substantial amounts of torsion, but beyond that, estimates suggest that over 90% of acute brainstem infarcts of any type will cause torsion,[3] and most of these cases are asymptomatic, diagnosed only when the patient's external visual cues are removed. The ocular tilt reaction, consisting of head tilt, skew deviation, and torsion, may cause no symptoms of torsion, at least in partial cases.

Which patients are most likely to require torsion management, and therefore most likely to benefit from diagnostic tests for torsion? While not always a consistent dichotomy, in general, central nervous system torsion, excluding trochlear nerve palsies, is asymptomatic, and peripheral torsion (ie, torsion due to extraocular muscle disease or palsy) is not. When torsion is disconjugate, as in bilateral superior oblique palsies, and/or when horizontal or vertical strabismus is also present, torsional fusion is hindered. Restrictive eye disease commonly causes symptoms of torsion. Macular translocation also leads to symptoms of torsion. Patients with these ocular lesions are the ones who may need some sort of procedure to fix their torsion, a subject that I will return to below.

Nevertheless, it should be noted that even patients with substantial amounts of peripheral torsion might be asymptomatic. Guyton pointed out that primary inferior oblique overaction—that is, overaction not caused by a weak superior oblique—may cause excyclotorsion in excess of that seen in superior oblique palsy.[4] Yet these patients are not symptomatic, and may fail to notice torsion when tested using the Maddox rod. In patients with unilateral superior oblique palsy, covering the fellow eye usually eliminates torsional symptoms in the paretic eye. Other examples include patients with Graves disease who experience significant torsional symptoms as when their inferior rectus muscle(s) are tight. Once these patients are straightened in the horizontal and vertical meridians, their torsional symptoms may disappear.[5] Also noteworthy is the fact that infants and young children with peripheral defects that cause large degrees of torsion, for example those with craniofacial anomalies and rotation of the orbits around a sagittal plane, seldom if ever complain of symptoms.

The diagnosis and severity of symptomatic torsion is most conveniently ascertained using the Maddox and double Maddox rod test, at least in adults. Torsion can also be diagnosed with Baggolini lenses, and the red–green Lancaster test, during which one eye is covered with a red lens and the other a green one. The examiner then maps out the patient's perception of a grid, comparing the left to the right eye. Torsion, or the degree of torsion, cannot always be based on a head tilt or the amount of head tilt.[6] The diagnosis can be based on a fundus examination. On the face of it, this would seem a far less quantitative and more inaccurate examination than those noted above, but there are times when a fundus examination and/or an analysis such as the one proposed by Parsa is superior to other tests.

A number of reasons account for this. First, anatomical variation exists between the fovea and the optic disc. When a horizontal line is drawn through the fovea to the optic disc, in the average person with no central nervous or peripheral extraocular muscle disease that could cause torsion, the line transects the disc approximately one-third from the top of the disc. That there is so much variation in this finding in 'normals' is testimony to the fact that a great many people have torsion that is suppressed or fused, and are never bothered by it. Or, central fusional mechanisms have a lot of 'play' and can bring together torsionally-disparate images which are many degrees off-set. In people with these normal variations diagnosed by fundus examination, it has been my impression that the finding is similar in both eyes. Here the fundus examination is most helpful. Next, and for elusive reasons, torsion in one eye may be measurable only in the fellow eye. In other words, a patient with, for example, a tight right inferior rectus muscle could measure no torsion in that eye, but torsion in the fellow eye, using conventional testing.[7] Fundus examination should disclose which eye has true torsion.

Lastly, for patients with new or questionable diagnoses that could cause torsion, and, of course, for the young patient who may not understand quantitative tests noted above, the fundus examination can be very important. A rough imaginary line can be drawn from fovea to disc, as described above, or a more quantitative approach to a photo can be used, as described by Parsa. Whether the technique described by Parsa will represent an advance in diagnosis of torsion, compared with standard retinal images, will depend on the development of quantitative retinal vessel analysis. The technique is promising but in need of further refinement.

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For those with symptomatic, 'peripheral' torsion, the surgeon should align the patient vertically and horizontally to learn whether torsion symptoms will disappear. This usually can be accomplished in the office with prisms, and in most cases, torsion symptoms are eliminated. But when they persist, the surgeon is faced with a requisite surgical plan. Harada–Ito procedures, wherein the anterior fibres of the superior oblique tendon are transposed circumferentially towards the lateral rectus, or horizontal transpositions of vertical rectus muscles, and other techniques all share a commonality; their effect is arguably less predictable than simple horizontal or vertical rectus muscle surgery. In these cases, more than any others, the use of adjustable sutures is important.

How clinically relevant is ocular torsion to the practising ophthalmologist? Its diagnosis can lead to clues that unmask complex central disorders, and that may elucidate peripheral diseases as well, but only occasionally. Parsa has shown us a new and interesting technique to diagnose ocular torsion for some diagnoses. How important is the surgical management of torsion? Here I am sure I part company with many of my colleagues. The ophthalmologist should know how to diagnose and manage torsion, and should seldom put a surgical plan to use. Torsion treatment can lead to unintended consequences, perhaps more so than any other strabismus surgical technique or practice. The ophthalmologist must learn to avoid torsion as an iatrogenic complication, perhaps one of the most important reasons for understanding this complex subject. Inadvertently inducing torsion symptoms when they did not previously exist can present a very difficult problem.

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REFERENCES

1. **Parsa CF,** Kumar AB. Cyclodeviation of the retinal vascular arcades: an accessory sign of ocular torsion. Published Online First: 12 May 2012. doi:10.1136/bjophthalmol-2011-300867.
2. **Brodsky MC,** Donahue SP, Vaphiades M, *et al*. Skew deviation revisited. *Surv Ophthalmol* 2006;**51**:105–28.
3. **Dieterich M**, Brandt T. Ocular torsion and perceived vertical in oculomotor, trochlear and abducens nerve palsies. *Brain* 1993;**116**:1095–104.
4. **Guyton DL.** Ocular torsion reveals the mechanisms of cyclovertical strabismus: the Weisenfeld lecture. *Invest Ophthalmol Vis Sci* 2008;**49**:847–57, 46.
5. **Ruttum M,** von Noorden GK. Adaptation to tilting of the visual environment in cyclotropia. *Am J Ophthalmol* 1983;**96**:229–37.
6. **Trobe JD.** Cyclodeviation in acquired vertical strabismus. *Arch Ophthalmol* 1984;**102**:717–20.
7. **Olivier P,** von Noorden GK. Excyclotropia of the nonparetic eye in unilateral superior oblique muscle paralysis. *Am J Ophthalmol* 1982;**93**:30–3.