Ocular Torsion: Rotations Around the "WHY" Axis

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Background: Traditional teaching holds that there is a partial compensatory countertorsion after head tilt because the intorters in the eye on the side of the head tilt and the extorters in the contralateral eye are stimulated. This teaching is inconsistent with a number of clinical observations. ***Methods:*** Review of existing literature, reanalysis of data from the investigator's previous experiments, and inductive and deductive reasoning were used to reconcile inconsistencies and present a theory on why torsional movements occur. ***Results:*** The inconsistencies can be reconciled if one considers that during the dynamic phase of head tilt, there is an alternating series of intorsional and extorsional movements of both eyes. Each eye has slow dynamic compensatory counterrolling phases that serve as torsional "doll's-head" movements to stabilize the image during head tilt. This counterrolling is partially eliminated by a series of anticompensatory torsional saccades in the direction of head tilt, which is in contrast to traditional teaching. ***Conclusion:*** Dynamic compensatory counterrolling occurs during head tilt. It is largely eliminated by anticompensatory torsional saccades in the opposite direction so that by the end of head tilt only minimal static countertorsion remains. The dynamic compensatory counterrolling motion is necessary to minimize peripheral visual movement during head tilt. The elimination of most of the counterrolling by the end of head tilt is necessary to preserve convergence and stereopsis. (J AAPOS 2004;8:1-12)

Traditional teaching dictates that if the head tilts, the two extraocular muscles which are intorters (the superior oblique [SO] and superior rectus [SR]) are stimulated in the eye on the side to which the head is tilted, and the two extraocular muscles which are extorters (the inferior oblique [IO] and inferior rectus [IR]) are inhibited. According to traditional teaching, for example, the LSO and LSR are stimulated in the left eye on left head tilt, and the LIO and LIR are inhibited. On head tilt in the opposite direction the converse occurs: The two superior muscles are inhibited, and the two inferior muscles are stimulated.[1,2] This results in a torsional rotation around the Y-axis, which is approximately coincident with the visual axis. In the normal individual, the vertical actions of the two stimulated muscles cancel each other out, and no vertical deviation occurs (Figure 1). In a patient with a unilateral LSO palsy, however, the left hypertropia (LHT) increases on left head tilt because the elevating action of the LSR is unopposed by the paretic LSO (Figure 2). This traditional teaching is the basis for the Bielschowsky head-tilt test[1,2] and the Park's Three-Step Test.[3] However, several important inconsistencies exist between some common clinical observations and what would be expected to occur if this traditional teach-

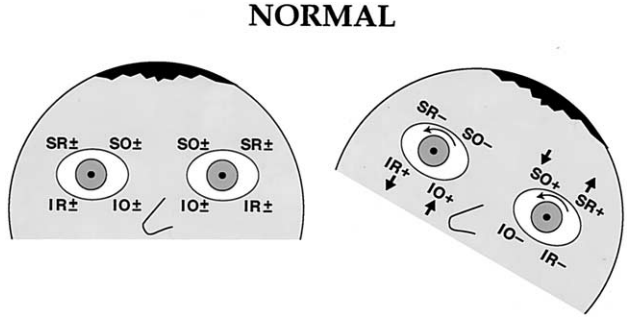

Fig 1. Depiction of traditional teaching. With the head erect, there is a normal resting tonus to all four of the vertical muscles. (Left) On left head tilt, the intorters in the left eye (the LSR and LSO) are stimulated, and the extorters (the LIR and LIO) are inhibited. (Right) In the right eye, the extorters (the RIR and RIO) are stimulated, and the intorters (the RSO and RSR) are inhibited. In each case the vertical actions of the adjacent oblique and rectus muscles are opposite and cancel each other, hence no hypertropia develops. Note that in this and subsequent figures, the location of the oblique muscles is depicted with respect to their anatomic location and not their field of action. Hence, the SO is shown as being superior nasal and the IO as being inferior nasal.

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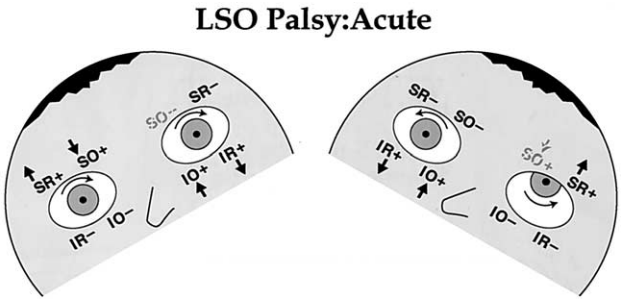

Fig 2. Depiction of traditional teaching for patient with LSO palsy. On left head tilt the intorters in the left eye (the LSO and LSR) are stimulated. The vertical action of the normal LSR is no longer balanced by the LSO because it is paretic, and the LHT increases. (Right) On right head tilt the paretic LSO is inhibited, as is the normal LSR, so the imbalance of the vertical forces is lessened and the LHT decreases.

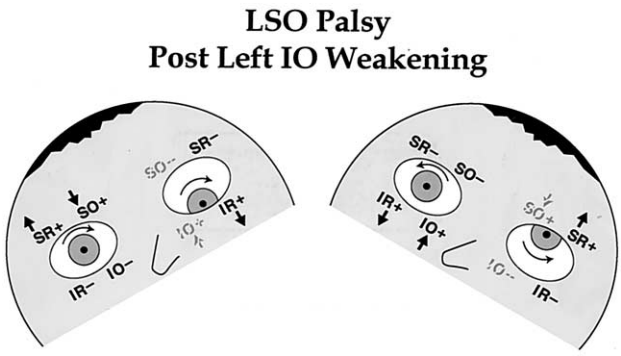

Fig 3. Theoretical depiction of traditional teaching for patient with LSO palsy after LIO weakening. On left head tilt the LSO and LSR are stimulated. As shown in Figure 2 the LHT increases. The fact that the LIO has been weakened has no impact on this because the LIO and LIR are inhibited. (Right) On right head tilt the LIO and LIR are stimulated. Because the LIO has been surgically weakened, the depressing force of the LIR is no longer balanced and the LHT decreases further (or a left hypotropia develops) (Left).

ing accurately described the extraocular muscle activity during head tilting.

INCONSISTENCIES BETWEEN THEORY AND OBSERVATION

Weakening of the ipsilateral IO is the most commonly performed surgical procedure to correct unilateral SO palsy. If the traditional teaching as described above were valid, this surgical procedure should result in a marked increase in the Bielschowsky head-tilt difference (defined as the difference in HT between right and left head tilt as measured in the dissociated state, eg, by the prism-and-alternate-cover test) for the following reason. Consider a patient with an LSO palsy after undergoing LIO weakening. On left head tilt, the LHT should still increase because the LSR is unopposed by the LSO, which is still paretic. The fact that the LIO has been surgically weakened should not influence this because the LIO is traditionally thought to be inhibited on left head tilt. However, on right head tilt a downward force should occur in the left eye because the depressing action of the LIR is now unopposed by the surgically weakened LIO. Depending on whether or not there remains a LHT in the primary position, the LHT should either decrease further, or a left hypotropia should develop (Figure 3). In fact, the opposite occurs: The Bielschowsky head-tilt difference typically decreases in patients with a unilateral SO palsy after ipsilateral inferior oblique weakening. In a previous study, I found that although ipsilateral IO weakening did not decrease the Bielschowsky head-tilt difference as much as some other surgical procedures, it did result in a mean decrease of 5.4 prism diopters (PD) in a series of patients with unilateral SO palsy.[4] Even more perplexing is an observation I have made in several patients who underwent ipsilateral SO tenectomy and IO extirpation to treat unilateral SO myokymia (unpublished personal observations). As early as 1 day after surgery, and thus before long-term vergence adaptations could occur, the patients had no HT on either right or left head tilt per the prism-and-alternate-cover test (Figure 4). Consider a patient who underwent such surgery in the left eye. Based on traditional teaching, the patient should manifest a large LHT with left head tilt, as well as a large left hypotropia with right head tilt, for the reasons outlined above. The effect of a surgical procedure on the Bielschowsky head-tilt difference may be important to some patients with certain occupational needs. For example, I have treated several patients for SO palsy whose occupation required them to work in a crawl space and frequently tilt their head to perform various types of overhead maintenance tasks. Similarly, I treated a professional violinist who had a left SO palsy. To read the music, he needed single binocular vision with his head tilted to the left.

A second inconsistency with traditional teaching can be found in observing the evolution of the Bielschowsky head-tilt difference over time in patients with acute unilateral SO palsy. Typically, such patients show little or no overaction of the antagonist IO initially; however, they often develop it months or years later. According to traditional teaching, as IO overaction develops, the Bielschowsky head-tilt difference should theoretically decrease for the reasons depicted in Figure 5. In a patient with a LSO palsy, the LHT will increase on left head tilt for the reasons outlined above. As the LIO becomes progressively more overacting, there should then be an increasing elevating force on right head tilt because the overacting LIO overpowers the normally acting LIR, thus decreasing the Bielschowsky head-tilt difference. In fact, the Bielschowsky head-tilt difference increases as overaction of the antagonist IO develops. In a series of 7 patients whom I examined shortly after the development of acute unilateral SO palsy, all had minimal or no overaction of the antagonist IO initially. They were all reexamined at

Fig 4. On postoperative day 1 after an 8-mm tenectomy of the LSO and extirpation of the LIO to treat LSO myokymia, this male patient is orthophoric on both right and left head tilt. *LIO* = left inferior oblique; *LSO* = left superior oblique (Video associated with this figure is available to subscribers at www.mosby.com/jaapos).

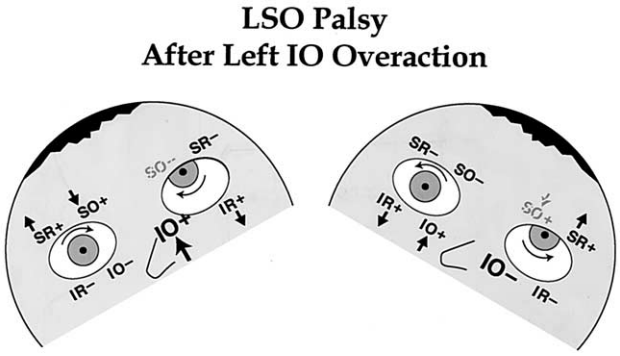

Fig 5. Depiction of traditional teaching for patient with LSO palsy after the development of LIO overaction. On left head tilt the LHT increases because of the reasons outlined in Figure 2. On right head tilt the LIO and LIR are stimulated. Because the LIO is overacting, its elevating force overpowers the LIR, resulting in an increase in the LHT. (Left) This increased LHT on both right and left tilt decreases the Bielschowsky head-tilt difference.

least 14 months later (mean, 23 ± 7.5), by which time they all had substantial overaction of the antagonist IO; none had undergone surgery in the interim. In all patients, the Bielschowsky head-tilt difference increased during that time interval (Table 1).

A third inconsistency in the traditional teaching can be found in reports of the usefulness of the three-step test in certain situations. Although it has frequently been described as being diagnostic for oblique muscle palsy, it is frequently not diagnostic in patients with vertical rectus palsy.[5-8] Yet, results of the Bielschowsky head-tilt test are typically markedly positive in patients with superior rectus contracture/overaction as pointed out by Jampolsky.[9-12]

A fourth inconsistency is evident when one compares the magnitude of the Bielschowsky head-tilt difference in patients with bilateral SO palsy with patients having unilateral SO palsy. One would expect that a bilateral SO palsy would result in a larger Bielschowsky head-tilt difference because the forces that cause it for each eye should be additive. In reality, bilateral SO palsy is typically associated with a much smaller Bielschowsky head-tilt difference.[13] Similarly, surgical weakening of both IOs, as is commonly done to treat primary inferior oblique overaction, does not result in a substantial Bielschowsky head-tilt difference after surgery. However, I observed a large Bielschowsky head-tilt difference in two patients immediately after the accidental severing of one IO stemming from trauma (unpublished personal observation). There appears to be something about bilaterality that is not taken into account by the traditional teaching.

TABLE 1 Data on patients with SO palsy over the course of development of IO overaction

Data	Acute (Mean ± SD)	Later (Mean ± SD)	Paired Student *t* Test
Primary-position HT (PD)	8.6 ± 3.2	14 ± 4.9	p = .0001
IO OA (0 to +4)	0.71 ± 0.49	3.4 ± 0.53	p = .0008
Bielschowsky HTD (PD)	8.4 ± 2.6	21.3 ± 4.4	p < .0001

IO OA = inferior oblique overaction.
HT = hypertropia; *HTD* = head-tilt difference;

A final inconsistency is evident if one carefully observes the torsional movements made by the eyes of a subject during head tilt. I have observed that by focusing one's attention on a marker, such as an iris crypt, one can see on left head tilt that the left eye does not make a simple intorsional movement, nor does it make a simple extorsional movement on right heat tilt (the converse holds for a right eye on right or left head tilt) as would be expected if the traditional teaching were valid. Instead, one can easily observe a series of cogwheel-like torsional movements (both intorsional and extorsional) on both left and right head tilt. To reconcile these obvious inconsistencies, we must first understand what really happens torsionally during head tilt. Table 2 summarizes the inconsistencies.

OCULAR TORSIONAL MOVEMENTS: WHAT ACTUALLY HAPPENS

Does ocular torsion occur? There is little doubt that human eyes have the ability to make torsional movements. The anatomy of the oblique muscles suggests it is possible. In fact, Balliet and Nakayama[14] trained normal volunteers

TABLE 2 Inconsistencies in traditional Teaching between theory and observation

Clinical Observation	Theoretical Finding Expected
1: Bielschowsky head-tilt difference increases after IO weakening for unilateral fourth-nerve palsy	Should decrease
2: Bielschowsky head-tilt difference increases as IO OA develops secondary to fourth-nerve palsy	Should decrease
3: Three-step test is inaccurate in patients with vertical rectus palsy	Should be accurate
4: Bielschowsky head-tilt difference is typically small with bilateral fourth-nerve palsy	Should be larger
5: Direct observation does not confirm traditional teaching	Should confirm

IO = inferior oblique; *IO OA* = inferior oblique overaction.

to tort their eyes more than 26° in an experimental setting. According to several sources, Hunter[15,16] deserves credit as being the first person to objectively observe torsional eye movements. In 1786, he described observing a rolling movement of his eye while watching it in a mirror when he tilted his head. In 1866, Javal[17] reported that he could no longer see clearly through his astigmatic spectacles when he tilted his head either to the right or left and concluded that ocular torsion must have occurred. Adler believed that ocular torsional movements were completely compensatory for head tilt, eg, the degrees of countertorsion were equal to the degrees of head tilt, thus keeping the eye in its original orientation (torsionally) with respect to the visual environment. He credits Nagel as formulating that belief.[18,19] However, most investigators who have actually studied and quantified ocular torsion found it is rather small and only partially compensates for head tilt.[20-29] Although the methodology used in these studies varied, the results of all found that the partial compensatory countertorsion present after head tilt was in the range of 10% to 30% of the size of the head tilt. Thus, for a 30-° tilt, the countertorsion would be between 3° and 9°. Jampel, however, is at odds with other investigators. In a series of articles written during many years, he has repeatedly insisted that no compensatory countertorsion exists in humans during the steady state after a head tilt has been completed.[30-32] The entire history of the study of torsional eye movements up until 1985 has been summarized nicely by Simonsz.[16]

So, if it appears that a partial compensatory countertorsion does occur, how then can one reconcile the abovementioned inconsistencies between theory and clinical observations? To answer this, we must explore the differences between the torsional change that may be present at the end of head tilt and the dynamic changes that occur during the active process of head tilt.

In 1973, Petrov and Zenkin[27] described the dynamics of torsional movements during head tilt as follows: As the head begins to tilt, each eye undergoes a slow compensatory counterrolling movement in the opposite direction of the head tilt. This movement is compensatory because it compensates for the movement of the visual environment across the retina because of the head tilt. During left head tilt, an observer would see this as extorsion of the right eye and intorsion of the left eye. In effect, this has the eyes torsionally lagging behind the head in what can be considered a "rotary doll's-head" response. This compensatory counterrolling is followed by a more rapid anticompensatory torsional rotation in the direction of the head tilt. This movement is anticompensatory because it is in the opposite direction and eliminates the effect of the previously described compensatory torsional rotation. During a left head tilt, an observer would see this as intorsion of the right eye and extorsion of the left eye. These cogwheel-like movements occur several times in sequence. As the head tilt is being completed, the final direction of the torsional movements is in the same direction as the head tilt (eg, intorsion of the right eye and extorsion of the left eye for a tilt left, which is the opposite direction as is traditionally taught). This final movement has the eyes almost, but not quite, catching up with the head torsionally. The difference between the number of degrees the head rotates and the number of degrees the eyes rotate represents a final compensatory countertorsional change. Subsequently other investigators have reported similar findings to those of Petrov and Zenkin.[28,29,33,34] These investigators all used some means of marking the position of the eye such as natural anatomic markings (eg, iris crypts), artificial markings placed on the eye, or scleral search coils. Some means was then employed to record the eye position using cinematography or oculography. The results of some of these studies showed that the ocular torsional movements appeared to precede or anticipate the head tilt depending on the experimental design (eg, whether or not the head tilt was voluntarily initiated by the subject); however, all revealed both intorsional and extorsional rotations of each eye when the head was tilted in either direction. Although Jampel[30-32] found similar intorsional and extorsional eye movements in his studies, he claimed that at the end of head tilt there was no final torsional change from the head-erect position.

In the 1980s, I studied the dynamics of torsion during head tilt using 16-mm cinematography (Figure 6).[28,29] With a marker placed on the subject's cornea, the rotation of the eye could be determined by comparing the relationship between the corneal marker with a reference line on the wall. The magnitude of the head tilt could be determined by comparing the relationship between markers on the forehead and the same reference marks on the wall. Using a frame-by-frame analysis of the films, a graph could be constructed comparing torsion of the eye with degrees of head tilt. A representative example of the torsional eye movements of the left eye of a normal subject on left head tilt from those previous studies has been reana-

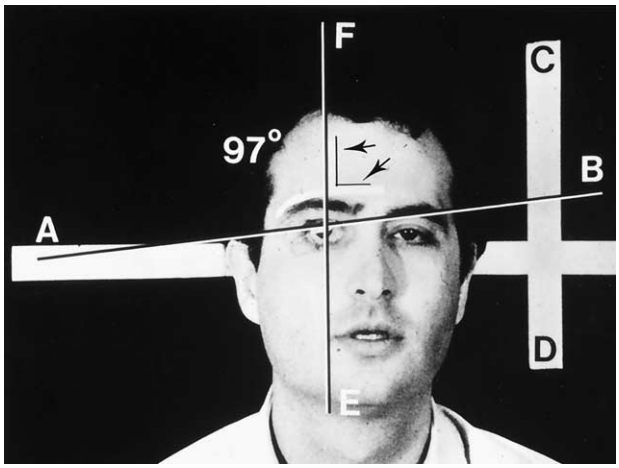

Fig 6. Test subject used for studying ocular torsion. A strip of egg membrane was placed on the cornea. Vertical and horizontal lines on the forehead (enhanced for reproduction purposes and identified by arrows) serve as a reference for head tilt. Vertical and horizontal lines on the wall serve as a stable reference for the environment. (Reprinted from the American Journal of Ophthalmology, Kushner BJ and Kraft S: Ocular torsional movements in normal humans. vol 95, p756, copyright 1983, with permission from Elsevier.) (Video associated with this figure is available to subscribers at www.mosby.com/jaapos).

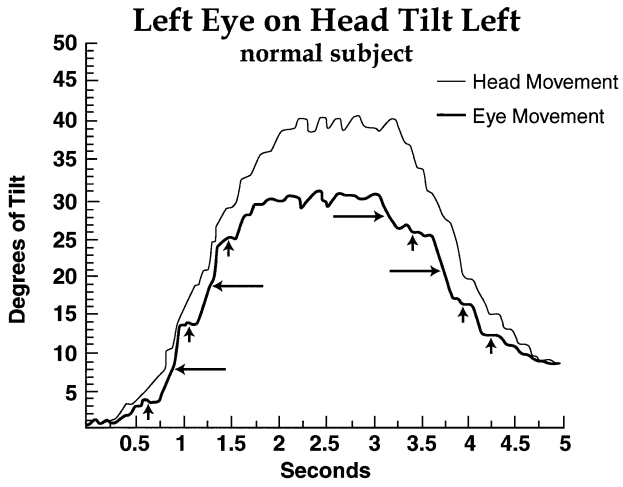

Fig 7. Graph of a 40° left head tilt for subject shown in Figure 6. The lighter line depicts the tilt of the head. The darker line depicts the rotation (torsion) of the eye referable to the environment. (Note: it is important to keep in mind that this is not the movement of the eye referable to the orbit). When the darker line is horizontal (small arrows), the eye is staying stable referable to the environment. Because the head is tilting, the eye in fact is making a counterrolling movement at the same speed, but in the opposite direction, as the head tilt. When the darker line is rising steeply (larger arrows) the eye is making a rapid anticompensatory torsional saccade resulting in the torsional position of the eye approximately matching the tilt of the head.

lyzed using digital computer analysis and is shown in Figure 7. It is important to note that the movement of the eye in Figure 7 represents movement referable to the environment and not the change in the orientation of the eye with respect to the orbit. As such, if the eye is staying stationary with respect to the environment, it will be represented by a horizontal segment in the graph (the eye is neither intorting nor extorting with respect to the environment). If, however, the head is tilting at that same time, the eye would by definition be counterrolling in the orbit in a direction opposite the head tilt (a rotary doll's-head response). The findings seen in Figure 7 are very similar to what Petrov and Zenkin[26] described. In the early phase of left head tilt, the left eye intorts slowly. The speed of this movement is approximately equal to the speed of the head tilt, thus keeping the eye (and retina) stable with respect to the environment. This is the previously described rotary doll's-head reflex. For a head tilt of approximately 40° at a velocity of 23°/second, this slow, compensatory counterrolling motion lasted approximately 0.186 seconds. It was followed by a rapid extorsional movement in the direction of the head tilt (eg, clockwise to an observer) at a speed of approximately 54°/second, which lasted approximately 0.2 seconds, thus rotating the eye approximately 11°. This resulted in the eye approximately catching up with the head torsionally. A series of three or four of these alternating intorsional and extorsional movements occurred depending on the speed and size of the head tilt. In the end, the final extorting movement was not sufficient to have the eye "catch up" with the head, thus leaving approximately 10° of final intorsion. On subsequent straightening of the head, similar but inverse movements occurred during the active phase of head tilt. Slow, compensatory counterrolling movements occurred, which kept the eye stable with the environment (extorsion of the left eye and intorsion of the right eye), were interspersed with faster anticompensatory torsional rotations in the same direction as the straightening head (intorsion of the left eye and extorsion of the right eye). During the steady state, after the head returned to the neutral position, the torsional alignment of both eyes returned to the original orientation that was present before the head tilt.[27-28]

Part of the reason that traditional teaching about ocular torsion fails to explain what happens clinically is because it does not address the difference between the torsional movements that occur during the dynamic state of head tilting and the static state existing after the tilt occurs. In this article, I refer to the above-described counterrolling movements that occur during tilt as "compensatory dynamic counterrolling." They always occur in the opposite direction of the head tilt and move in the direction described by traditional teaching. In Figure 7, they are represented by the small arrows. I also refer to the rapid movements that occur in the direction of the head tilt (the opposite direction from the compensatory dynamic counterrolling motion and contrary to traditional teaching) as "anticompensatory torsional saccades" because they occur in the opposite direction of the movements that compensate for the head tilt. In Figure 7, they are identified by large arrows. Although these movements are slower than

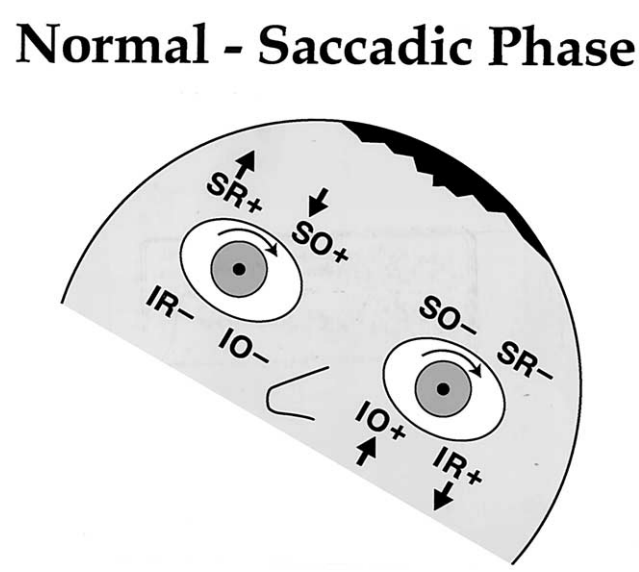

Fig 8. Depiction of muscle tonus in a normal subject on left head tilt during the normal anticompensatory saccadic phase. In the right eye, the RSR and RSO (intorters) are stimulated, and the LIR and LIO (extorters) are inhibited. In the left eye, the LIR and LIO (extorters) are stimulated, and the LSR and LSO (intorters) are inhibited. This results in a clockwise rotation of both eyes (as seen by the examiner) and is the opposite of the muscle tonus occurring during the dynamic counterrolling phase as seen in Figure 1.

typical horizontal or vertical saccades, I explain below why physiologically they are like saccades. They serve the purpose of having the eye partially catch up torsionally with the tilting head. Figure 8 depicts the relative tonus to the muscles during this normal anticompensatory saccadic phase. Finally, I refer to any final change in the torsional alignment of the eye that is present in the steady state end of head tilt as "static countertorsion." Although this is often referred to in the literature as "static counterroll," I believe that the juxtaposition of "static" and "counterroll" is an oxymoron that is confusing when describing a steady-state situation.

We know that the dynamic compensatory counterrolling and anticompensatory torsional saccades that occur during the process of head tilt are primarily mediated by the SO (which causes intorsion) and the IO (which causes extorsion). When I repeated the type of study represented in Figures 6 and 7 on patients with unilateral SO palsy combined with ipsilateral IO overaction, before and after ipsilateral IO weakening, I observed the following:[28] Consider the movements of the left eye of a patient with a LSO palsy. On left head tilt the dynamic compensatory counterrolling (intorsion) was slower and less effective in stabilizing the eye position with the visual environment compared with normal. Similarly, the anticompensatory torsional saccades (extorsion) were longer and faster (almost twice as fast) as seen in normal subjects. On right head tilt, the initial dynamic compensatory counterrolling (extorsion) did occur; however, the anticompensatory torsional saccades (intorsion) did not. After LIO weakening surgery, the dynamic compensatory counterroll (intorsion), which was absent before surgery, was present in a weakened form when compared with normal control subjects. This is what one might expect if the LSO was paretic instead of paralyzed. Similarly, the anticompensatory torsional saccades on left head tilt (extorsion) were slower than they were before surgery. All of these findings confirm the important role that the oblique muscles play in the dynamic aspects of torsion.

Interestingly, although anyone can observe these cogwheel-like movements by carefully observing a subject's eye during a slow head tilt, a different pattern is seen if one tries to make the same observations on one's own eyes while looking in a mirror and tilting one's head. One will then see their own eyes make the dynamic counterrolling movements in the opposite direction of head tilt (intorsion of the left eye on left head tilt, ie, a rotary doll's-head movement) followed by one slow anticompensatory rolling movement in the direction of the head tilt just as the head tilt is concluding (extorsion of left eye at the end of a left head tilt). One does not see the rapid anticompensatory torsional saccades, which are easily visible to an observer. Just as vision degrades during horizontal or vertical saccades, it also does so during these torsional movements. In this respect they are similar to saccades. Interestingly, in 1875, Donders did not accept the previous reports of torsional eye movements during head tilt because he was unable to observe the phenomenon when he studied his own eye in a mirror. According to Simonsz, Donders overlooked the obvious fact that vision degrades during the saccadic phase, thus preventing him from seeing these movements of his own eyes.[16]

Recall that the final torsional movement of the eye occurring at the end of a head tilt is in the opposite direction of what traditional teaching suggests. Consider the left eye on head tilt left. Traditional teaching dictates that the eye should be intorting. However, the final torsional movement one observes or records is an extorsional rotation, as though the eye is trying to "catch up" with the head. However, if the extorters in the left eye were in fact being stimulated at the end of a head tilt to the left, common observations using the Bielschowsky head-tilt test to diagnose a unilateral LSO palsy would be even more inexplicable. The answer to this seeming paradox may lie in an important observation. This final anticompensatory torsional movement can be observed by the subject using either after-images, Maddox rods, or by direct observation in a mirror.[28,35] Thus, it differs from the anticompensatory extorsional saccades in that vision is not degraded. Therefore, it is probably mediated differently and is not a saccade. We know from studies using electromyography,[36] as well as our observations with the Bielschowsky head-tilt test, that the final tonus of the cyclovertical muscles is characterized by increased innervation to the intorters and inhibition of the extorters in a

left eye on left head tilt. A possible mechanism that can account for all these observations is that this final extorsional anticompensatory rotation is caused by a subsequent relaxation of the intorters, which were responsible for the dynamic counterrolling during head tilt. However, they only relax partially, thus allowing for some anticompensatory extorsion to occur. Yet they maintain some tonus to produce the small partial static countertorsion (intorsion of left on head tilt left) that can be detected. Thus, there are two different types of anticompensatory torsional movements that occur during the active phase of head tilt. The initial anticompensatory torsional movements are saccades, which are mediated primarily by the IO in the eye in which extorsion is occurring and the SO in which intorsion is occurring. The final anticompensatory torsional movement is not a saccade and is caused by a partial relaxation of the oblique muscles, ie, relaxation of either the SO and IO, to cause extorsional or intorsional rotation, respectively.

INCONSISTENCIES BETWEEN THEORY AND OBSERVATION REVISITED

Three important factors are crucial to reconciling the inconsistencies between theory and observation:

1. One must realize that what is happening to the cyclovertical muscles at the end of head tilt does not reflect the dynamics that occurred during the course of the head tilt.
2. The observation by Carter and Jampolsky[11,12,37] that the IO muscle cannot raise the eye above the midline if the SR is not functioning is crucial to our understanding of this subject. They made this observation by evaluating attempted supraduction in the eye of a subject undergoing strabismus surgery under topical anesthesia while the SR muscle was temporarily detached from the globe. Their experimental observation should be essentially recreated during head tilt because the SR on the side of the head tilt is theoretically inhibited.
3. There is a difference between a muscle that is contractured and one that is truly overacting. In this context I am describing a muscle as being contractured if it is stiff and has a decreased elasticity to passive stretch but may actually be weak. It does not generate a greater-than-normal increase in force with increased stimulation. Contracture occurs as result of a muscle being chronically shortened, ie, not stretched out, or by a primary infiltrative process, eg, Grave's disease. In contrast, a muscle that is truly overacting may exhibit normal elasticity to passive stretch but generate a greater-than-normal increase in active force for a given amount of increase in its innervation. Muscles may become overacting in this manner by excessive repeated contraction (much as exercise will strengthen one's bicep) or by isometric stimulation as a result of fixation duress. Although contractured and overacting muscles are physiologically different, on versions they may look similar. A contractured IO and a truly overacting IO will both be characterized by an excessive elevation in adduction. By understanding these three principles, the previously described inconsistencies between traditional teaching and clinical observations can be reconciled.

As I had previously speculated, the reason IO weakening surgery decreases the Bielschowsky head-tilt difference in patients with unilateral SO palsy is because the IO plays an active role on head tilt toward the affected eye during the saccadic phase.[29] Consider a patient with an LSO palsy. The LIO is not simply inhibited on head tilt to the left as is traditionally taught. Before surgery, the LIO is stimulated during the anticompensatory torsional saccade on left head tilt. If it is overacting, it may then overpower the LIR and add to the degree of LHT present. After surgery, the normal LIR is unopposed during the anticompensatory torsional saccade (extorsion) on left head tilt, thus tending to decrease the degree of HT. This same mechanism can explain how a patient might have little or no HT on right or left head tilt after IO and SO weakening to treat SO myokymia.

Similar reasoning will explain why the Bielschowsky head-tilt difference increases in patients with unilateral SO palsy as ipsilateral IO overaction develops. In a patient with a LSO palsy, there is active recruitment of the LIO and LIR on left head tilt at the time of the anticompensatory saccades (extorsion). As the LIO starts to overact, it will tend to overpower the LIR during this phase and cause an increase in the LHT. This tends to balance the increased elevating effect that should come from the LIO on right head tilt as was seen in Figure 4. However, with these two effects canceling each other out, one might expect the result to be no change in the Bielschowsky difference rather than the increase that is typically observed. However, as Collins and Jampolsky have pointed out,[10,11,37]an overacting IO alone has almost no ability to elevate the eye above the primary position. Probably another factor is more important. I observed that the Bielschowsky head-tilt difference in patients with SO palsy correlates positively with the deviation in the primary position ($r = .70$, p & .001), and the deviation in the primary position is a function of the magnitude of IO overaction ($r = .77$, p & .001).[38] Thus, one would expect that as the primary position deviation increases concurrent with the development of IO overaction, the size of the Bielschowsky head-tilt difference should also increase.

It is now apparent why the Parks three-step test is often not diagnostic of cases of IR palsy.[5,7,8] In the presence of a paretic LIR, the Bielschowsky head-tilt test would require the LIO to elevate the eye and decrease the L Hypo on right head tilt. However, because an IO alone has a limited ability to elevate an eye, this elevation may not occur and the Bielschowsky head-tilt test may be inaccurate.[10,11,37] Also, the Parks three-step test may be inaccurate in patients with SR palsy for similar reasons. The test

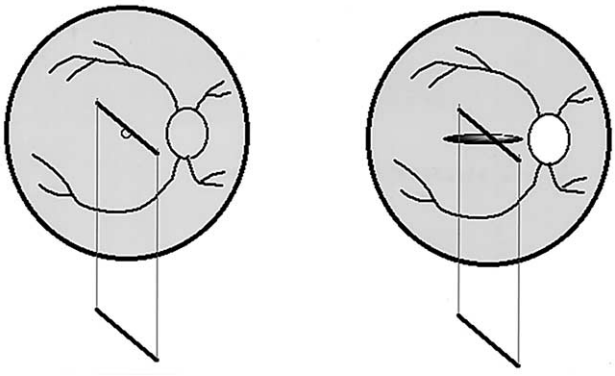

Fig 9. Diagram of a human retina with a foveal point. The torsional rotation of a horizontal line as the object of regard would still result in the entire fovea being stimulated by the line. (Left) Diagrammatic representation of a retina of with an elongated foveal line, similar to what is seen in rabbits. A similar torsional rotation of a horizontal line would result in a substantial amount of the fovea not being stimulated by the line, thus degrading vision. (Right)

assumes that if the SR is paretic, the ipsilateral SO will depress the eye with head tilt toward the affected side, thus increasing the hypotropia. If the SO acting alone has a limited to ability to depress the eye, one would expect the Bielschowsky head-tilt test to be unreliable in cases of SR palsy. To my knowledge, the SO has not been reported as being limited in this manner. However, because the SO mirrors the function of the IO, it seems reasonable to infer that such a limitation is likely. I have frequently observed that patients with complete absence of IR function stemming from trauma, iatrogenic muscle slippage, or third cranial nerve paralysis have essentially no ability to depress the affected eye despite normal SO function (unpublished personal observations). SR contracture/overaction presents a different situation. The SR is a powerful elevator. It can cause a markedly positive Bielschowsky heat-tilt test because an overacting SR has no difficulty overpowering a normal ipsilateral SO to cause an HT on head tilt to the side of the overacting SR. This occurs to a greater extent if the ipsilateral SO is paretic. In the SR overaction/contracture syndrome as described by Jampolsky,[9–12] the SR is better described as overacting than contractured. Forced ductions frequently do not reveal the abnormality of the SR, and the deviation is often greatest in the field of the SR as opposed to being greatest in the opposite field. In contrast, it is uncommon for the IR to become truly overacting as defined earlier, yet contracture is common in such conditions as Grave's disease, blowout fracture, etc. This is the reason why the Bielschowsky head-tilt test is usually not positive in cases of IR contracture. Because the inferior rectus muscle is not "overly strong," it does not overpower the ipsilateral IO on head tilt to the side opposite the affected muscle.[5,7,8]

Finally, we can now understand why the Bielschowsky head-tilt difference is smaller in patients with bilateral SO palsy than in those with unilateral palsy. Consider what happens on left head tilt in a patient with bilateral SO palsy. The left eye would tend to elevate because the LSR overpowers the LSO during the phase in which dynamic compensatory counterrolling occurs. However, during the anticompensatory saccadic phase on left head tilt (refer again to Figure 8), when the two eyes are making saccades in the direction of head tilt, the right eye would tend to elevate because the RSR and RSO are stimulated (intorsion) and the RSO is paretic. This would tend to decrease the LHT seen on left head tilt. The converse occurs on right head tilt, which tends to decrease the RHT that is expected to occur. In a similar manner, only minimal alternating hypertropias should occur after surgical weakening of both IO muscles when treating primary IO overaction for analogous reasons. Yet with true unilateral IO palsy (idiopathic or traumatic), the Bielschowsky head-tilt test should be positive, and it is. I realize that this theoretical construct implies that the final static measurement is a result of a complex interplay of the slow and fast phase torsional movements. Proof of this theoretical explanation would require additional studies of any vertical movements that may possibly occur during head tilt and is outside the scope of this article.

WHY ARE THERE ROTATIONS AROUND THE Y-AXIS?

The need for horizontal or vertical pursuit movements (as is seen in the doll's-head reflex) is very different than the need for torsional pursuit movements. For the former, they must be completely compensatory for head movement, or the object of regard will no longer remain on the fovea, eg, a 30° head turn must be accompanied by a 30° rotation of the eyes in the opposite direction. In contrast, for a head tilt centered on the visual axis, the object of regard will remain on the fovea even if no compensatory torsion occurs. However, the images falling on the peripheral retina will be in motion, thus causing peripheral visual movement and degradation of peripheral vision. This movement is called "retinal slip" and is dependent on the speed of the head tilt. The dynamic counterrolling that occurs during head tilt would serve to minimize retinal slip and hence decrease peripheral visual degradation. According to Simonsz,[16] Hunter inferred in 1786 that dynamic counterrolling served the purpose of maintaining vision during head tilt. It has been shown that dynamic counterrolling decreases when subjects are tested in the dark and increases when the visual target becomes more complex or when the speed of head tilt increases.[33] In some ways, the torsional movements that occur during head tilt can be thought of as a torsional optokinetic nystagmus. The dynamic compensatory counterroll is analogous to the slow phase of the optokinetic reflex, and the anticompensatory torsional saccades are analogous to the fast phase. However, optokinetic nystagmus requires visual input. The dynamic torsional movements that occur during head tilt are dimin-

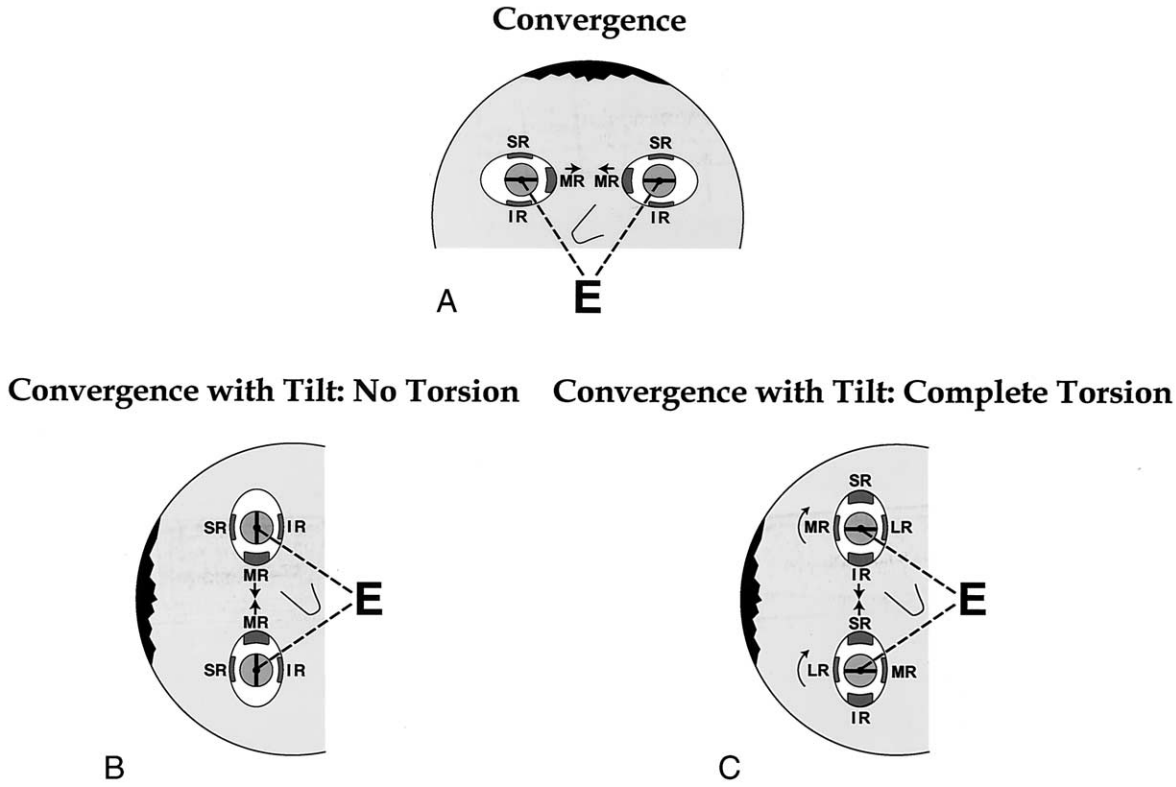

Fig 10. (A) Convergence is normally meditated by the medial rectus muscles. (B) If the head were tilted 90° and there was no compensatory torsion, convergence would still be mediated by the medial rectus muscles. (C) If static countertorsion were completely compensatory for head tilt (eg. 90° static countertorsion for a 90° head tilt), the orientation of the extraocular muscles would be altered. Then the IR of the higher eye would be opposite the SR of the lower eye. A convergence movement would need to be mediated by those two muscles. This theoretical construct assumes no influence of the orbital pulleys. *IR* = inferior rectus; *SR* = superior rectus.

ished in the dark, but they still occur. In that respect the analogy is incomplete.

Tweed[39] observed that when a subject's head is voluntarily tilted while watching a moving target, the eye torted in anticipation and in advance of the subsequent tilt of the head. Thus, the eye reached its final position before the head tilt was completed, after which the eye remained stable in space.[39] This all supports the idea that the oculomotor control system is extremely efficient in stabilizing the retinal image during head tilt and that dynamic counterrolling plays a major role in that regard. Interestingly, some lateral-eyed animals (such as rabbits) have an elongated fovea rather than a point-shaped fovea.[40,41] These animals have planes of sight rather than lines of sight, as is the case with primates. It would stand to reason that a torsional rotation of the object of regard by animals with an elongated fovea would have a more adverse effect on visual acuity than it would in animals with a foveal point (Figure 9). As one would expect, static countertorsion in rabbits is much greater than in humans, approaching 50% the magnitude of the head movement. Counterroll (and counterpitch) help keep the planes of sight near the horizon plane, the better to watch for approaching danger.[40,41]

WHY IS DYNAMIC COUNTERTORSION NOT COMPLETELY COMPENSATORY FOR HEAD TILT?

Now that we have seen how dynamic counterrolling helps to minimize retinal slip and peripheral visual movement during the active phase of head tilt, a final question remains. As long as dynamic counterrolling does occur, why is it not completely compensatory for head tilt? Why do the anticompensatory torsional saccades occur to counteract the dynamic counterroll? In lateral-eyed animals, there is no problem (and some benefit) if static countertorsion occurs to a large degree as shown in Figure 9. However, in animals with frontally placed eyes that share a binocular field (such as primates), substantial static countertorsion would have serious adverse effects on binocular cooperation. For example, complete compensatory countertorsion would seriously limit one's ability to converge as seen in Figure 10. Normally, the medial rectus muscles mediate convergence. For viewing at one third of a meter, one needs to converge approximately 15 PD to 18 PD depending on one's interpupillary distance. Normal horizontal fusional amplitudes can easily accomplish that. However, if complete compensatory countertorsion occurred, the relative locations of the rectus

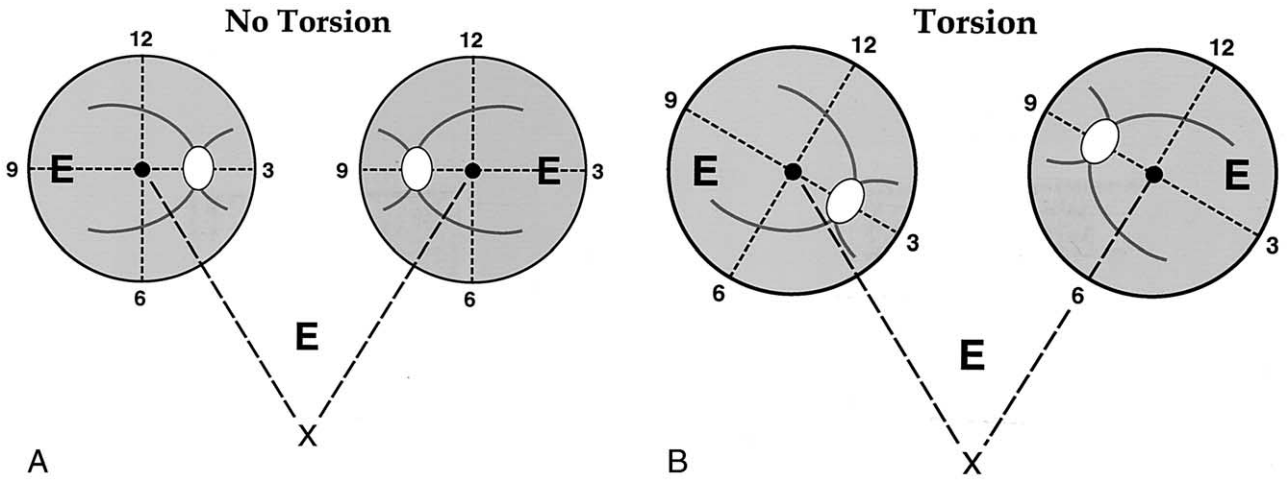

Fig 11. (A) The eyes are converged fixating on point "X." Point "E" is proximal to point "X" and falls on the temporal retina of each eye along the horizontal meridian. (B) If substantial torsion occurred, point "E" would then fall on the superior temporal retina of one eye and the inferior temporal retina of the other, thus creating a vertical disparity.[38] (Video associated with this figure is available to subscribers at www.mosby.com/jaapos).

muscles would shift accordingly. As seen in Figure 10 a, 90° head tilt would require convergence to be mediated by the SR of one eye and the IR in the other, which in effect would be a vertical vergence depending on the influence of the orbital pulleys. Most people do not have 18 PD of vertical vergence amplitudes, which are much weaker than horizontal amplitudes. Hence, viewing a near target when the head is tilted would be problematic. Similarly, if convergence occurred first, and then the eyes torted around the naso-occipital axis, a vertical deviation would be created as described by Misslisch et al.[41] As they indicated, however, this vertical deviation could be prevented if the axis of torsion shifted to stay in line with the visual axis rather than the naso-occipital axis (bear in mind that with the eyes converged, the visual axis is slightly oblique to the naso-occipital axis). However, another problem would still be present that would adversely affect stereopsis. As seen in Figure 11, substantial torsion would result in a vertical disparity between the two eyes for objects proximal or distal to the object of regard. The "X" in Figure 11A, which is closer to the subject than the object of regard, falls on the temporal retina of each eye. This horizontal disparity permits stereopsis. If torsion occurs as seen in Figure 11B, this same "X" would fall on the superior temporal retina of one eye and the inferior temporal retina of the other. This would result in a breakdown of the stereoscopic mechanism, which has limited ability to factor in a vertical disparity. It is probably for this reason that static countertorsion is so small in most animals with stereoscopic vision.[41] It is noteworthy that humans can still have stereoscopic vision despite small amounts of vertical disparity.[42] It appears the amount of vertical disparity that would occur from the small static countertorsion seen in humans is just within the limits that the stereoscopic system can tolerate.

Thus, it appears that ocular torsion is a primitive reflex that serves a different purpose in lateral-eyed animals than it does in frontal-eyed animals. In the former, static countertorsion is more completely compensatory for head tilt. This is necessary to optimize acuity and is possible because of the lack of binocular vision (refer again to Figure 9). In frontal-eyed animals this reflex is still present but in a different form. It serves to stabilize the image on the retina during head tilt, but it must be largely eliminated after a head tilt is complete to allow for convergence and stereopsis.

OTHER THEORIES

Brodsky[43] recently set forth a hypothesis that the evolution of frontal binocular vision "has exapted the human oblique muscles for stereoscopic detection of slant in the pitch plane, and nonstereoscopic detection of tilt in the roll plane."[43] I consider his theory to be wholly compatible with, and complementary to, the ideas set forth in this article.

Finally, a word must be said about the discrepancy between Jampel's[32] work and the findings of so many others. In some respects, Jampel's findings are in keeping with those of other investigators. Jampel feels that the main purpose of torsional movements is to "protect the retinas by dampening [sic] the effect of the head movement on this sensitive tissue."[32] To this degree, he is in agreement with most other investigators. However, the main area of disagreement relates to the issue of there being any final static countertorsion. Jampel contends that although many investigators thought they were observing static countertorsion, they in fact were observing false torsion. This artifact can mimic torsion when an angle on a curved surface is viewed obliquely; it has been described previously in detail.[28,44] However, the findings of some previous studies simply cannot be attributed to false torsion.[43] I am not certain why Jampel has not observed static countertorsion when so many other investigators have. I assume that because static countertorsion (if it occurs) is

quite small, it may be absent in certain testing situations. In Jampel's experiments, much of the visual environment rotated with the subject during tilt. If torsional movements serve to stabilize the visual world on the retina, perhaps the rotation of the visual environment prevented the occurrence of torsional movements. Also, his experiments were conducted at close range. It is known that convergence minimizes torsional movements.[41] All of these factors may have influenced Jampel's results.[43]

In conclusion, it appears that torsional movements do occur in humans during head tilt. In understanding them it is important to differentiate the movements that occur during the active phase of head tilt and during the static state after head tilt. During head tilt, there are alternating phases of a dynamic counterrolling serve to keep the eye stable with the visual environment. However, this countertorsion must be eliminated to a large degree at the end of head tilt, or there would be adverse effects on convergence and stereopsis. This transformation is accomplished by a series of anticompensatory torsional movements in the direction of head tilt. These largely eliminate the countertorsion that occurred during the dynamic counterrolling phase. They only correct that amount of countertorsion necessary to still permit binocular vision and stereopsis. As Misslisch et al[41] so elegantly stated, when "there is a conflict between phylogenetically old gravity driven reflexes [countertorsion] and newer vergence mechanisms that serve stereopsis . . . vergence dominates. However the old reflexes have not been eliminated."[41] The system is phenomenally complex yet elegantly functional.

Susana Gamio, MD (Buenos Aires, Argentina), suggested the term "anticompensatory" for describing the torsional movements that occur in the direction of head tile, and she pointed out to me the similarity between the dynamic torsional movements that occur during head tilt and the optokinetic reflex. I thank her.

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An Eye on the Arts – The Arts on the Eye

French marine scientist Roger Chesselier wears a patch over his right eye. A story he likes to tell on meeting Israeli colleagues dates from the time he was working on the Calypso with Jacques Cousteau's scientific team off the coast of Beirut.

Lebanon was at war with Israel, and Gen. Moshe Dayan's heroic achievements were widely known.

Chesselier was a good swimmer and, wishing to try out Beirut's famed Mediterranean coast, hired a cabin, changed into a pair of swimming trunks, and took off for a long swim in the blue sea. He hadn't gone far when he spotted a guard boat speeding toward him. The people on board, gesturing wildly, were shouting at him in a language he didn't understand. It was soon made clear what they were up to, as he was forcibly, bodily, hauled out the water. The language they spoke turned out to be Arabic, and they didn't know French, so our scientist had to restrain his annoyance until the boat reached land.

More trouble was in store for him there, as he got off the boat and found himself before a group of men in uniform. The one who spoke French was the chief of police. In his hand was Chesselier's French passport, which Chesselier had left in the cabin together with his clothes.

"Whose passport is this? It's a false passport! You're Moshe Dayan! You've come to spy on us! You are an Israeli spy!" And so it went, on and on. It was entirely up to Chesselier, and not at all easy, to prove that he wasn't.

"Imagine what a mythical figure Moshe Dayan must have been in the eyes of the Lebanese at the time, that they thought him capable of swimming from Tel Aviv to Beirut to spy on them," the French scientist concluded, filled with wonder.

The story has been widely repeated, and told also to Dayan's daughter, Yael.

"Yet people miss the most important point of my story." Chesselier exclaims in his voluble French, "Didn't they know that my patch is over the right eye, while Dayan's was over his left?"

—Emma Kimor (from *Jerusalem Post, Online Edition*)