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Diagnosis of vestibular imbalance in the blink of an eye

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Abstract—*Background:* In a recent study, the authors found that blinks in healthy volunteers always triggered ocular torsion quick phases during dynamic roll movements of the head. On the basis of this observation, they hypothesized that blinks in patients with a vestibular tone imbalance would also trigger torsional quick phases. *Methods:* Using video-oculography with a fixation target, the authors recorded the ocular torsion position of the left eye of 37 participants while they made voluntary blinks once every 6 to 10 seconds. The participants were recruited from four groups: two age groups of healthy volunteers with a mean ± SD age of 32 ± 4 (n = 9) and 65 ± 11 y (n = 9); patients with a unilateral vestibular disorder in an acute state (n = 12, 53 ± 17 y); and those in a persisting state in which spontaneous nystagmus had already faded (n = 9, 65 ± 13 y). *Results:* In the control groups of healthy volunteers, blinks triggered no or only small quick phases on the order of 0.1 deg. In both patient groups blinks always triggered quick phases with significantly higher amplitudes of 1.85 ± 1.02 deg and were followed by exponentially decaying slow-phases with time constants on the order of 1 to 2 seconds. Patients in the persisting state clearly differed from patients in the acute state in that their torsional spontaneous nystagmus had already vanished due to vestibular compensation. But surprisingly, these two groups did not show a large difference in terms of the effect of blinks on ocular torsion. The authors always observed torsional quick phases with the upper pole of the eye beating away from the side of the lesion. *Conclusions:* Blinks are able to trigger torsional quick phases in patients with both acute and persisting vestibular disorders. The side of the impairment can be determined from the direction in which the eye is rotated after a blink. Thus, ocular torsion recordings during blinks can be used as a simple clinical test for a vestibular tone imbalance, particularly during a persisting failure in which spontaneous nystagmus has resolved and can therefore no longer be used for diagnosis.

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In a recent study on ocular torsion during prolonged angular head accelerations, we showed that blinks introduced characteristic torsional eye movements whenever a stimulus was applied that led to torsional nystagmus (figure 1B).[1] Each blink generated a torsional quick phase in the same direction as the acceleration-induced nystagmus quick phases, and it was followed by an exponential slow-phase-like drift back toward the ocular torsion position registered before blink onset. In that study the dynamic properties of both blink-induced eye movements and nystagmus proved similar in terms of the exponential time constant, but in terms of ocular torsion amplitude the effect of a blink was always significantly greater than nystagmus quick phases.

The spontaneous pathologic nystagmus that occurs in patients with acute unilateral vestibular failure closely resembles physiologic nystagmus generated in response to rotatory head accelerations, which stimulate the semicircular canals. We therefore examined patients with a vestibular tone imbalance due to a unilateral vestibular loss to determine if they show the same effect of blinks on torsional eye position with their head stationary as normal subjects during semicircular canal stimulation. If similarly marked effects were found in response to blinks in patients with both acute and persisting unilateral vestibular lesions, these characteristic torsional eye movements would provide a useful new test to unmask vestibular tone imbalances, even in persisting states, when central vestibular compensation mechanisms have caused spontaneous nystagmus to disappear.

To evaluate the sensitivity of this potential test, the differential effects of blinks were investigated in patients with acute unilateral vestibular neuritis or circumscribed brainstem ischemia that involved vestibular nuclei and in patients in a persisting state, when spontaneous nystagmus had faded and could no longer be used to identify the vestibular tone imbalance. Two groups of healthy subjects served as controls.

Methods. *Participants.* We recorded movements of the left eye of 37 participants (14 women) while they made voluntary blinks once every 6 to 10 seconds. The participants were recruited from four different groups: 1) healthy volunteers with a mean ± SD age of 32 ± 9 years (n = 9); 2) healthy volunteers with a mean age of 65 ± 11 years (n = 9); 3) patients with an acute unilateral vestibular neuritis or circumscribed brainstem ischemia that involved the vestibular nuclei (n = 12, 53 ± 17 y); and 4) patients with a persisting unilateral vestibular deficit due to a vestibular neuritis

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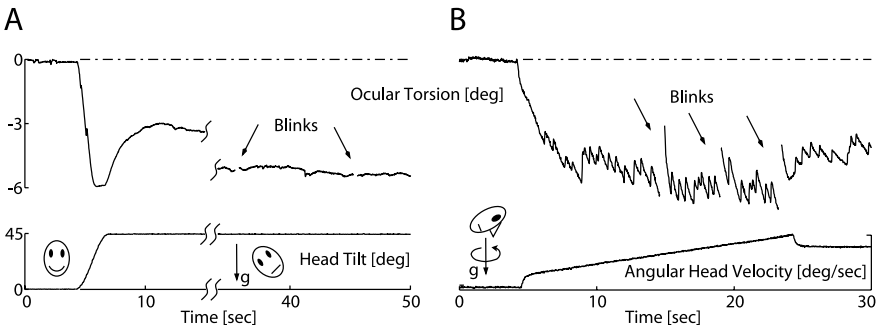

Figure 1. Ocular torsion recordings during stimulations in roll by static head tilt (A) and angular head acceleration in a nose-down position (B). The results of a control experiment with a healthy volunteer are shown in A and B. Ocular counter-roll was measured in a static condition more than 30 seconds after a 45 deg head tilt (A). With this otolith-only stimulus no resetting effect of blinks was observed. For direct comparison, the results of a previously published semicircular-canal-only stimulation are given in B (adapted and republished from elsewhere,[1] see figure 4 on page 2067, subject S.G.). The angular head acceleration caused nystagmus but also a considerable cyclotorsional deviation in B. Blinks always reset the eye toward the resting position.

or a unilateral eighth nerve section after an acoustic neurinoma, all in a compensated state in which spontaneous nystagmus and other signs like vertigo, nausea, and postural imbalance had already disappeared (n = 9, 65 ± 13 y; see the table for individual diagnosis). The classification of vestibular neuritis patients was based on the long-term course of their disease. In the acute phase, patients have perceivable signs like nystagmus, nausea, or falls. During the first 1 to 3 weeks after vertigo onset, compensation usually leads to a recovery from these symptoms.[2] Six months later normalization of caloric responses is achieved only in 20% of patients, and up to 50% still show canal paresis even after 5 or 10 years.[3] Patients who were examined less than 3 weeks after vertigo onset were therefore classified as acute and patients who were examined after more than 1 year and still showed a canal paresis were classified as persisting.

By coincidence, we were able to record a patient without any prior vestibular disorders before and after his first attack of presumably either Menière's or inner ear autoimmune disease. Although this patient had neither neuritis nor ischemia, his two data sets measured before and during the state of a vestibular imbalance were added to the groups of younger controls and acute patients, respectively. Another patient who did not recover from a vestibular neuritis was examined both immediately after neuritis onset and 2 years later. Her two data sets were assigned to the groups of patients with acute and persisting deficits. The participant's consent was obtained according to the Declaration of Helsinki (BMJ 1991; 302: 1194) after they had been briefed about the examination. The experiments were approved by the local ethics committee (approval numbers 87/96 and 212/96).

Clinical examinations. The diagnosis of the vestibularly impaired patients was based, among others, on two separate groups of standard clinical examinations, which we termed nystagmography and neuroorthoptics (see Results, table). These examinations were routinely performed by experienced clinical assistants and neuroorthopticians. The resulting diagnosis of a unilateral vestibular disorder was used as the preselection criterion for our experiment. In the group of examinations termed nystagmography, horizontal eye movements were recorded by means of electronystagmography to determine the velocity of the slow phases of horizontal spontaneous nystagmus (hSPN). Additionally, a binaural caloric test was performed with hot (44 °C) and cold (30 °C) water while the patients kept their eyes closed and their head 30° up from supine. From the slow phase velocities recorded during the four conditions (left/right ear and hot/cold water), the spontaneous nystagmus offset was subtracted and caloric asymmetry indices (CIX) were determined according to Jongkees' formula[4] (equation 1). The results of these tests, which are believed to primarily reveal horizontal semicircular canal dysfunction, are reported in the nystagmography section of the table.

In the group of examinations termed neuroorthoptics, the deviation of the subjective visual vertical (SVV) and tonic cyclotorsion were determined. The SVV was measured by having the patients sit in front of a full-field, static dotted pattern and instructing them to adjust a line to parallel their perception of verticality.[5-7] The tonic cyclotorsion was determined by laser scanning ophthalmoscopy.[6] The SVV values given in the table are means based on seven repetitions; the cyclotorsion values were calculated from the ex- or incyclotropia angles of the retinal meridian of the left and right eyes according to equation 2. Positive angles were used for excyclotropia and negative angles for incyclotropia. A nasally directed rotation of the upper part of the bulbus is defined as incyclotropia. The results of these tests, which are believed to reveal otolith dysfunction, are reported in the neuroorthoptics section of the table.

$$\text{CIX} = \frac{(\text{left cold} + \text{left hot}) - (\text{right cold} + \text{right hot})}{\text{left cold} + \text{left hot} + \text{right cold} + \text{right hot}} \times 100\%$$

$$\text{Cyclotorsion} = \frac{\text{right} - \text{left}}{2}$$

Experimental setup. The experimental equipment was chosen on the basis of its inexpensive and easy integration in any clinical environment. An off-the-shelf digital video camera recorder (Sony DCR-7000E PAL) was used to monocularly measure movements of the left eye. The camera was rigidly attached to a mobile forehead and chin rest and was adjusted so that the subjects were able to look directly into the camera lens (figure 2). The camera provided an infrared illumination to record the eyes in a room with dimmed light. Torsional eye movements were measured while the subjects fixated a luminous point at a distance of 1 m. The measurement of torsional eye movements during fixation did not require a calibration procedure. The ocular torsion was trigonometrically calculated from the Cartesian image coordinates of a pair of markers[1,8] that were artificially applied to the sclera by means of a dark cosmetic pigment (figure 3). The images were recorded on tape at a sampling rate of 50 Hz and were later analyzed off-line by a custom-made video-oculography software package.[1]

Examination of a patient before and during vestibular impairment. The patient we were able to measure before and after his first attack was a hospital employee who had participated in another video-oculography study 14 days before his first attack of unilateral vestibular failure. Because at that time the employee was considered healthy and did not report any history of vestibular disorders, this pre-attack recording (PRE) was included in the group of healthy volunteers (Group A). The second data set was included in the patient Group C. It was recorded 4 days after his first attack (PER). Because we had detected a sequence of unintentional blinks in the archived PRE recording by chance, it was possible to compare the effect of blinks on torsional eye movements before and during a vestibular impairment.

In addition, we were able to determine the absolute amount of static ocular torsion caused by the vestibular imbalance in this patient by comparing the iral structure recorded before and during the impairment. The static ocular torsion angle was estimated from cross-correlation of an iral landmark identified in both recordings (see figure 3). While the orientation of both the camera and the forehead and chin rest remained the same with respect to gravity, we did not control the head positioning between the two different sessions, because they were unrelated and the later vestibular failure could not be anticipated. However, it was possible to estimate the true head tilt difference from two lower-eyelid eyelashes, which we could identify in both the PRE and the PER video images. This difference was taken into consideration in the ocular torsion plot of figure 3.

Table *Blink test data and standard clinical examinations of patients with acute and persisting unilateral vestibular deficits*

Patient		Blink test			Neuroorthoptics			Nystagmography		
ID	Diagnosis	Amp, deg	tSPN, deg/s	Day	SVV, deg	Cyc, deg	Day	CIX, %	hSPN, deg/s	Day
Acute vestibular deficit (Group C)										
Ca62F	Neuritis R	−4.8	−1.4	1	+7.0	+18	1	+100	+4	1
Cb57M	Neuritis R	−3.1	−1.4	4	+4.3	+5	4	+100	+19	1
Cc38M	Neuritis L	+2.6	+1.4	2	−5.3	−10	2	−100	−16	2
Cd56M	Neuritis L	+3.4	+1.1	3	−6.1	−11	3	−100	−11	3
Ce32M	Neuritis L	+2.5	+0.2	3	−5.1	−15	3	−100	−13	3
Cf68F	Neuritis L	+2.0	0	5	−9.7	−10	5	−79	−8	5
Cg56F	Neuritis L	+0.9	+0.2	21	−4.2	−7	11	−38	−10	11
Ch56F	Neuritis L	+2.4	0	6	−2.0	−5	6	−3	0	6
Ch56F*		—	—	—	−6.1	−14	0	−20	−7	1
Cj38M	Menière L	+0.9	0	4	−5.1	−6	15	−71	−10	15
Ck81F	Ischemia R	−1.8	−0.2	9	+16.4	+11	3	—	—	—
Cm73M	Ischemia R	−1.2	−0.1	3	+1.6	+2	3	+19	+5	3
Cn23F	Ischemia L	+1.1	0	13	−0.5	+2	13	—	—	—
Persisting vestibular deficit (Group D)										
Da74M	Neurinoma L	+2.2	0	388	—	—	—	—	0	388
Db76M	Neuritis R	−1.5	0	2,240	+1.5	+2	2,240	+85	0	2,240
Dc59M	Neuritis L	+2.1	+0.1	504	+0.2	+3	504	−78	0	504
Dd75M	Neuritis L	+0.9	0	2,333	−9.3	−5	2,333	−23	0	1,927
De58M	Neuritis R	−1.3	0	1,061	+2.5	+1	1,061	+100	0	670
Df37M	Neuritis R	−1.0	0	676	−0.9	0	676	+44	0	676
Dg64F	Neuritis R	−1.1	0	711	+3.3	+1	711	+100	0	324
Dh71M	Neuritis R	−0.8	0	452	−0.6	+1	452	+51	0	455
Di74F	Neuritis L	+1.3	0	456	−4.0	0	456	−85	0	456

The columns Neuroorthoptics and Nystagmography contain the results of the routine examinations. The time from vertigo onset to examinations is given in the columns Day. The torsional spontaneous nystagmus intensity (tSPN) was determined from the same set of video recordings, i.e., on the same day as the blink test.

* For Subject Ch56F, who was tested in a compensated state with no SPN, a second recording from the acute phase was added to the table.

SVV = subjective visual vertical; Cyc = tonic cyclotorsion; CIX = caloric asymmetry index; hSPN = horizontal spontaneous nystagmus.

Control examination. In order to differentiate between the two possibilities of a semicircular canal or otolithic origin of the observed blink effect, an additional control examination with four healthy volunteers was performed. The subjects sat in a tiltable chair and were instructed to repeatedly blink while torsional eye movements were recorded with the described video-oculography setup (figure 1A). The chair was tilted around the roll axis to a static position of 45 deg in order to induce static ocular counterroll. The effect of blinks on this otolithic type of cyclotorsion is discussed in the context of a previous work,[1] in which the effect of blinks on purely semicircular canal induced ocular torsion was analyzed.

Data analysis. The angle of ocular torsion could be trigonometrically determined from the scleral markers. The angular difference between the ocular torsion position before and after a blink was used as an estimate of the amplitude of a blink-triggered quick phase. For each participant, mean values based on all detected blink amplitudes were calculated (see the table, Amp). Apart from that, the quick phases of spontaneous torsional nystagmus were also identified and for each patient their mean amplitude was multiplied with their mean frequency to obtain a measure for the intensity of the torsional spontaneous nystagmus (see the table, tSPN).

The signs of rotation quantities were defined according to the standard conventions of three-dimensional eye movement analysis.[9] A cyclo-rotation of the top pole of the bulbus toward the right ear is thus assigned a positive value. The significance of differences between the examined groups was calculated by *t*-tests, and the significance of correlations between variables with the Pearson's moment correlation. Amplitude differences or correlations were considered significant at a confidence level of 5%.

Results. A direct comparison of plotted torsional eye movement traces revealed a striking difference between normal and patient data. While blinks did not affect ocular torsion traces in healthy volunteers, they always triggered transient torsional eye movements with a characteristic trajectory in patients (figure 4).

During fixation, the blink-triggered pathologic eye movements consisted of a torsional quick phase—usually

Figure 2. Experimental setup for recording video images of an eye. The mobile video camera was positioned in front of an upright sitting patient. Participants were instructed to look for a duration of about 1 minute at a fixation point in a straight-ahead position and voluntarily blink once every 6 to 10 seconds.

hidden by the eyelid—which was followed by an exponential drift back to the initial position before blink onset. If the drift was not interrupted by another blink, its time constant was on the order of 1 to 2 seconds. The amplitudes of the quick phase were on average 2.2 ± 1.2 deg in acute and 1.3 ± 0.5 deg in chronic patients, but the difference between the two groups was not significant ($p > 0.05$). The small torsional deviations in both the groups of volunteers (Groups A and B) showed amplitudes of only 0.1 ± 0.1 deg (figure 5), which were smaller ($p < 0.0001$) than the marked pathologic quick phases observed in all patients irrespective of their acute or persisting state of a vestibular tone imbalance (figure 6). We have included plots of raw data in our report to demonstrate the fact that this pattern can be identified in each patient from the plotted pathologic ocular torsion traces. These blink perturbations can be easily discriminated from the rather unaffected ocular torsion trajectories of healthy volunteers (compare figures 5 and 6).

The quick-phase directions were always related to the side of the impairment. With respect to the beating direction, blink-induced torsional quick phases were similar to the pathologic tSPN quick phases: they always rotated the upper part of the bulbus away from the lesioned side. However, the blink-induced jumps in ocular torsion positions were greater than the spontaneous nystagmus quick phases ($p < 0.001$). This can be easily seen in the data of some patients in an acute state of a vestibular imbalance (see figure 6, plots Ca62F, Cb57M, Cc38M, Cd56M).

We also analyzed whether the amplitudes of blink-induced quick phases (see the table, Amp) correlated with the other clinically relevant variables from the table. Note that the different variables were obtained on different days and that the correlation analyses were only performed for patients in the acute phase; patients with a persisting deficit were not considered. We observed that the effect of blinks increased with increasing cyclotropia ($r = 0.68$, $p = 0.015$, figure 7), and that it decreased with the logarithm of time after the first attack ($r = -0.73$, $p = 0.007$). The latter effect was probably due to recovery. Another correlation could be found between the quick-phase amplitudes of blinks and torsional spontaneous nystagmus ($r = 0.77$, $p = 0.003$). Other variables like the horizontal spontaneous nystagmus ($r = 0.04$, $p = 0.9$) and the SVV ($r = 0.16$, $p = 0.6$) did not correlate with the blink-induced torsional quick-phase amplitudes.

The ocular torsion for Patient Cj38M recorded prior to his impairment showed no effect of blinks, whereas after his first attack blinks induced quick phases of 0.9 ± 0.4 deg. A comparison of the positions of the same iral landmark identified in the recordings before and after his first attack revealed an absolute static cyclotorsion in the range of −3 deg (see figure 3). In this patient, the blink-induced quick phases rotated the eye away from this static position of −3 deg toward the resting position of 0 deg, but without reaching the resting position. In contrast, blinks had no effect on static cyclotorsion during the otolith-only stimulation of the control experiment, in which ocular counter-roll was induced by a static head tilt around the roll axis.

Some of the patients in an acute state of vestibular imbalance showed a considerable amount of torsional spontaneous nystagmus (see Ca62F, Cc38M, Cd56M in figure 6). These patients also showed the most prominent effect of blinks with ocular torsion position jumps of 3.5 ± 0.9 deg, which were greater ($p < 0.001$) than those of all other patients. In other acute patients vestibular compensation was already in effect and spontaneous nystagmus had vanished. Patient Ch56F, for example, recovered well enough to leave the hospital on the day of our examination. Her routine examinations yielded almost normal values (see rows Ch56F and Ch56F in the table). Nevertheless, blinks still affected her torsional data. The most surprising effect, however, was observed in patients with a persisting vestibular tone imbalance. Even years after the onset of impairment, these patients' ocular torsion traces were still affected by blinks, although no spontaneous nystagmus was observed.

Discussion. In this study we observed marked quick-phase-like deviations of torsional eye positions after blinks in patients with a unilateral vestibular disorder, but not in healthy volunteers. Compared with the quick phases of spontaneous torsional nystagmus, the blink-triggered quick phases were strik-

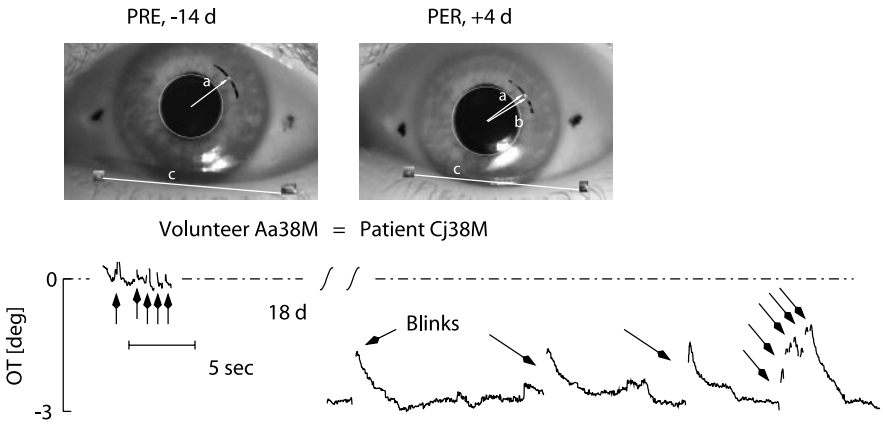

Figure 3. Video-oculography recordings of the left eye of a patient before (PRE) and after (PER) onset of unilateral vestibular failure. In the PRE recording, blinks had no effect on ocular torsion (OT, bottom left), but in the PER recording (bottom right), blinks always triggered torsional quick phases toward the resting position of the eye; they were followed by a drift back to the deviated static OT at −3 deg. The resting position at 0 deg was defined as the mean OT of the PRE recording. The absolute value of the PER torsional eye position was calculated from the displacement in polar coordinates of the maxima of the auto- and the cross-correlation functions of a high-contrast iris landmark (top), and the head tilt was calculated from the two lower-eyelid eyelashes connected by the line c. First, a pair of eyelashes was selected from an enlarged version of the PRE image and then the enlarged PER image was manually scanned for the lower eyelid eyelashes at the same relative distances from the corners of the eye (not shown). For better visibility, the used landmarks were highlighted by inversion. The iral landmark rotated around the center of the pupil from the initial PRE position (white arrow a in both images) to an offset PER position (white arrow b). The OT trajectories were calculated from the coordinates of the centers of intensity (see elsewhere[1] for details) of the dark artificial landmarks on the sclera. The numbers with the units d (days) on top of the images denote the time relative to vertigo onset; PER was therefore recorded 18 days after PRE. Note that the dark scleral landmarks are not identical in PRE and PER, because they only last for hours, not weeks.

ingly larger in amplitude and could thus be detected easily in the ocular torsion traces of patients. The fast torsional deviations were followed by similarly marked slow-phase-like drifts back to the initial position. This transient ocular torsion pattern could not only be observed in patients with spontaneous nystagmus but also in patients in whom signs of a unilateral vestibular deficit like spontaneous nystagmus had disappeared due to vestibular compensation. We therefore consider these characteristic blink-accompanying torsional eye movements to be a sensitive clinical sign not only of an acute but also a persisting state of a unilateral vestibular tone imbalance.

When the blink test is compared with other established vestibular tests one might first notice that it is basically different from the head impulse test, which reveals changes in the dynamic properties of the vestibular pathway or a vestibular gain reduction.[10] Secondly, it is also different from the rotational and the caloric irrigation tests, which both produce nystagmus by stimulating the vestibular periphery in order to reveal a peripheral vestibular gain deficit.[4] In contrast, the blink test unmasks the state of a vestibular offset or tone imbalance of either central or peripheral origin, without applying an external vestibular stimulus. In this aspect it is similar to pathologic spontaneous nystagmus; however, nystagmus is compensated for after about 1 week[11,12] and can then no longer be used for identifying the tone imbalance. Surprisingly, the effect of blinks on ocular torsion position is not compensated for, and it is still visible even after spontaneous nystagmus has vanished. Patients in a persisting state of a vestibular tone imbalance showed this effect even after years. The blink examination would constitute in conjunction with an absent spontaneous nystagmus a relevant clinical test of compensated vestibular tone imbalance. In this aspect it is similar to head shaking nystagmus, which can also be observed after nystagmus has faded. However, head-shaking nystagmus requires an additional test because it relies on a functioning central velocity-storage mechanism, which might also be impaired due to the vestibular loss.[13] In contrast, the proposed blink test does not require additional examination of the velocity-storage mechanism.

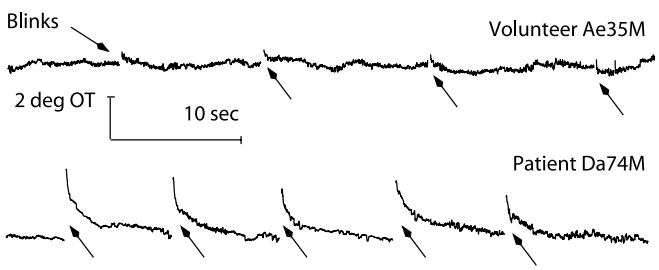

Figure 4. Ocular torsion (OT) recordings of a healthy volunteer (top) and a vestibular patient (bottom). While blinks had almost no effect on the OT of a healthy volunteer, they repeatably induced characteristic transient torsional eye movements in a patient with a surgically treated acoustic neurinoma.

Similar patterns of torsional eye movements after blinks have been observed before in healthy humans during rotations in roll[1,14] and galvanic vestibular stimulations.[1] However, this effect of blinks was not observed in the control experiment with static head tilts. In these aspects the blink-triggered torsional quick phases are similar to the torsional corrections which accompany horizontal and vertical saccades:

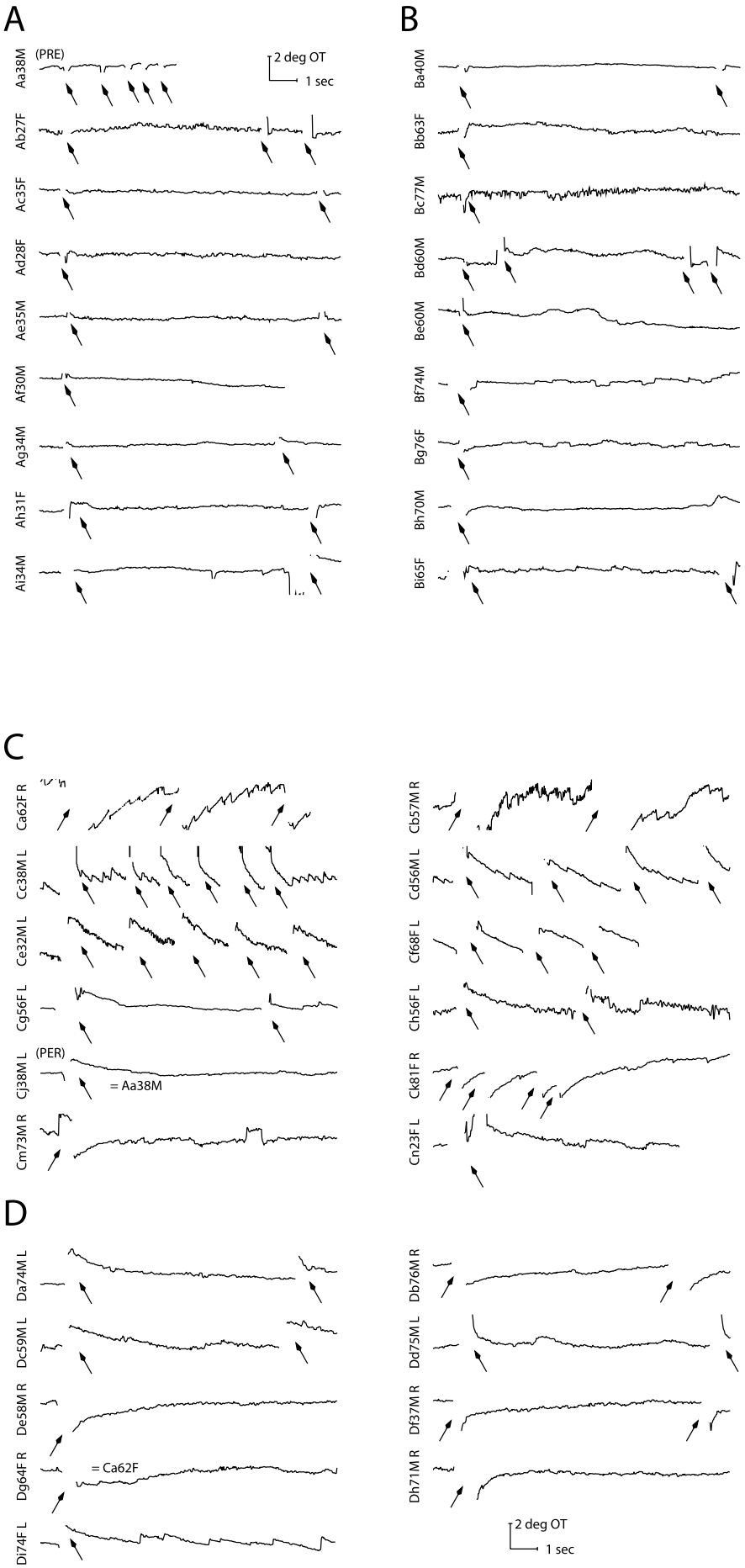

Figure 5. Ocular torsion (OT) recordings of healthy volunteers (Group A, age 32 ± 4 y; B, 65 ± 11 y) while repeatedly blinking. The onset of an arbitrary blink was used to align the OT traces in time at the abscissa value of 1 second. Given the same scale as in the plots of patient data (see figure 6), the effect of blinks on normal OT are almost invisible. Although blinks sometimes induced small OT deviations, these deviations were smaller in the volunteer group than in the patient group. Every participant was assigned a unique identifier, which included age and sex.

Figure 6. Ocular torsion (OT) recordings of patients with acute (C) and persisting (D) vestibular deficits while blinking. Blinks always triggered marked torsional eye movements consisting of quick phases with the upper pole of the eye beating away from the side of the lesion; they were followed by an exponential slow phase trajectory in the opposite direction. Only blinks are marked by arrows; nystagmus and saccades as well as other effects that may be confused with blinks are not marked. Some patients in C blinked more often than requested. The reduced blink interval creates the illusion of a linear instead of an exponential post-blink trajectory and thus the illusion of an increased time constant. Every participant was assigned a unique identifier which included age and sex and for better visibility of the directional sensitivity of the test, the patient identifier additionally includes a letter for the side of the impairment (L, left; R, right).

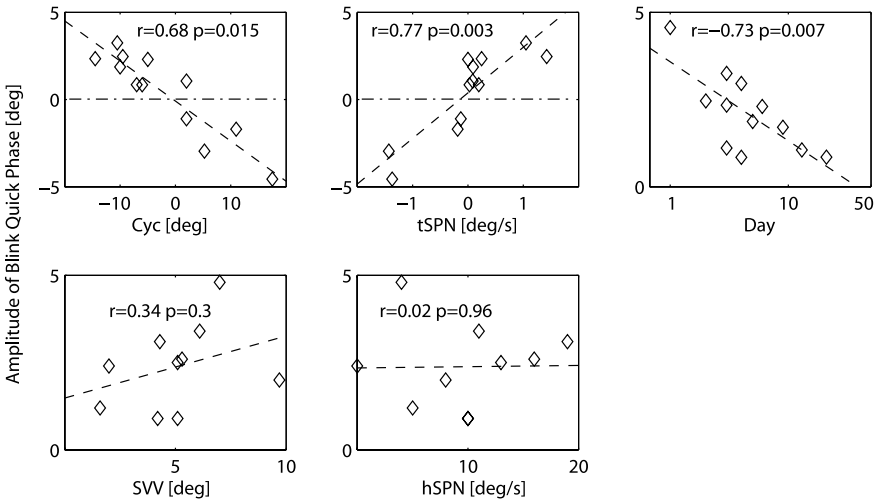

Figure 7. Correlations of blink-induced torsional quick phase amplitudes with other measured variables. The correlation coefficients (r) were calculated from the absolute (unsigned) values of the given variables. Only the data of patients in the acute state were included in the analysis. Cyc = tonic cyclotorsion; tSPN, hSPN = torsional and horizontal spontaneous nystagmus; Day = time from first attack; SVV = subjective visual vertical.

they can only be observed during dynamic stimulation conditions, such as optokinetic stimulation in roll,[15] but not during static head tilts in roll.[16] The observed time constant of the drift agrees well with the known time constant of 1 to 2 seconds for the torsional part of the oculo-motor neural integrator.[17,18]

Owing to its simplicity, the proposed blink test can be readily performed as an additional standard vestibular test in any clinical environment. The instructions for the patients are simple: they have to fixate a target in a straight-ahead position and repeatedly and voluntarily blink once every 6 to 10 seconds for about 1 minute. The pauses between blinks should not be shorter than 6 seconds due to the torsional integrator time constant.[17] An examiner can determine whether blinks had an effect on torsional eye movements from the plotted ocular torsion traces. Compared with the amplitude of torsional spontaneous nystagmus, the blink-induced quick phases are more prominent and easily detected. Therefore, the measurement equipment can consist of a standard portable video camera recorder as seen in figure 2. The video-oculography software package used in this study runs on any current personal computer and can be obtained either as an open source project from http://sourceforge.net/projects/xbinokel or by sending an e-mail to the corresponding author (E.S.). We observed a significant correlation between blink-triggered quick phase amplitudes and the amount of static ocular torsion. The measurement of static ocular torsion by laser scanning ophthalmoscopy has been proposed as a standard clinical test for vestibular imbalance.[6,19] The correlation with the values from fundus photographs implies that the proposed blink test could prove similarly reliable.

The results of this study also provide a cautionary note for clinicians who use laser scanning ophthalmoscopy for cyclotorsion measurements; under certain circumstances this method can give inaccurate findings. This is illustrated, for example, in the ocular torsion recording of figure 3. Here, the measured cyclotorsion value was dependent on whether the patient blinked before the fundus photograph was taken. If a photograph is taken before the eye has settled again to the static torted position, the torsion will certainly be underestimated, especially after a series of blinks. With this in mind, examiners must be cautious to take photographs no less than 6 seconds after a blink. Alternatively, one might consider the use of fundus videography and the analysis of fundus landmarks with image processing algorithms. The resulting landmark rotations might then be plotted over time in a way similar to that shown in figure 3. Thus a greater accuracy of fundus examinations would be gained by additionally including a simultaneous blink examination.

Various effects of blinks on eye position,[20-22] as well as on normal[23] and pathologic[24] saccades, and their ability to trigger saccades,[25] have been observed before. Blinks are also known to transiently affect ocular torsion during dynamic head rotations[14] and galvanic vestibular stimulation.[1]

During prolonged eyelid closures, for example, the eyeball rolls upward (Bell's phenomenon). However, it is not possible that this constitutes a source of interaction with the observed effect of blinks on ocular torsion, because Bell's phenomenon does not occur during short blinks like those applied in our study.[21] The eyeball is also retracted into the orbit during a blink by co-contraction of all extraocular muscles except the superior oblique muscle. When the eyes fixate a point straight ahead, this mechanism transiently induces a nasally and downward-directed eye movement[20,21,23] that is accompanied by an additional torsional component.[22] This movement, however, has only a short-term effect; it cannot explain the time constant of 1 to 2 seconds of the torsional drift that we observed in both healthy subjects during roll of the head[1] and in patients after a blink. Therefore, the observed blink-related torsional movements cannot be a consequence of a mechanical process, because this would cause drifts on the order of 0.1 second[17] rather than 1 to 2 seconds. The physiologic mechanism behind the observed effect of blinks can only have a neural origin. It could result from an inhibition of the omnipause neurons,[25] which cease

firing during a quick phase.[26] This additionally leads to a change in the dynamic properties of saccadic and vergence eye movements during blinks.[23] Their effect on pathologic saccades has been similarly attributed to the modulation of pause cells.[24]

After a unilateral vestibular impairment, static ocular torsion is observed among other symptoms, even after nystagmus compensation.[19] The question of which vestibular pathways—otoliths or semicircular canals—mainly cause this pathologic ocular torsion is part of an ongoing controversial discussion.[27-29] The answer could be very important for interpreting the observed pathologic resetting effect of blinks. In the past, static ocular torsion was primarily viewed as the result of impairment of utricular pathways,[6,7,30] mainly because a static head tilt in the torsional plane causes a static ocular counter-roll. In the control experiment with static head tilts, however, we did not observe that blinks have a resetting effect when ocular torsion was caused by an otolith-only stimulus (see figure 1A). Blinks affected torsional eye movements only when the stimulus was also able to induce torsional nystagmus, for example, during a purely semicircular canal stimulation[1] like that shown in figure 1B. This indicates that in addition to otolith impairment, semicircular canal imbalance may also contribute to the cyclotropia in unilateral vestibular patients. Because the ocular torsions originating from canal and otolith pathway activation have the same rotation directions,[31] they might be additive, thus leading to greater static pathologic torsions than would be expected from one source alone. This conclusion provides a cautionary note for researchers and clinicians who interpret the cyclotropia from fundus photographs as a test of otolith dysfunction.

Our study shows that blinks in patients with either acute or persisting vestibular tone imbalance induce marked torsional quick phases. This effect can be used as a clinical test for a unilateral vestibular disorder. The presence of blink-induced torsional quick phases in a compensated state, in which spontaneous nystagmus and other signs have resolved, not only constitutes a useful clinical sign for the diagnosis of persisting vestibular imbalances in particular but also contributes to the understanding that the semicircular canals and not only otolith pathways are part of the pathomechanism that leads to a static cyclotropia of the eyes. Video recordings can be performed at the patient's bedside by clinical assistants. Because the proposed test reliably reveals the state of a peripheral or central tone imbalance rather than a gain deficit in the vestibulo-ocular reflex pathways, its use can possibly be extended to other examinations, too, for example, the drug monitoring of unilateral intratympanic gentamicin therapy of Menière disease.

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