Functional Loss of the Horizontal Doll's Eye Reflex Following Unilateral Vestibular Lesions

Carol A. Foster, MD; Brian D. Foster, PhD; John Spindler, PhD; Jeffrey P. Harris, MD

The doll's eye reflex represents the vestibulo-ocular reflex (VOR) elicited by high-acceleration head rotation. After complete unilateral vestibular lesions, the ipsilateral, horizontal doll's eye reflex is replaced by a series of "catch-up" saccades. These cause permanent symptoms of blurred vision and dizziness during ipsilateral turns. We compared normal controls and patients with complete surgical lesions or canal paresis of up to 9 years duration via electronystagmography (ENG) to determine the usefulness of the doll's eye test as a diagnostic test for complete vestibular lesions. This test was found to be more sensitive in diagnosis of such lesions than head-shaking nystagmus, rotatory directional preponderance, and spontaneous nystagmus. It is also useful to document VOR function in patients in whom caloric irrigation is contraindicated.

INTRODUCTION

The doll's eye reflex (oculocephalogyric reflex, doll's head response) has been used for many decades in the clinical assessment of coma. This reflex is elicited by the doll's eye test.[1] The head of the comatose subject is briskly turned to either side, and the eyes are found to deviate conjugately in the opposite direction, giving the illusion that they are held by inertia. Within a few seconds the eyes then drift back toward midposition. This reflex is fully functional only in deep coma; in alert individuals it can be suppressed by voluntary eye movements, which can be prevented by having the subject visually fixate on a target while the maneuver is performed.[2] In the absence of this reflex (usually due to brainstem lesions or deep anesthesia), the eyes remain fixed in midposition during turns of the head. Unilateral absence of the reflex has been described due to lesions of the pons.[3,4]

The doll's eye reflex has been shown to be a manifestation of the vestibulo-ocular reflex (VOR).[5] The VOR is most often quantitated using caloric irrigation, although other methods such as the torsional swing chair, Bárány chair rotation, and position-step acceleration can also be used. The doll's eye test is essentially a high-acceleration, high-velocity position step; thus the doll's eye reflex is synonymous with the VOR under these conditions. A positive (intact) doll's eye test indicates that the high-acceleration VOR is functionally normal; when absent or impaired, it is a sign of VOR dysfunction.

Unilateral vestibular lesions are known to produce dramatic acute symptomatology, most of which resolves completely during the process known as compensation. Some authors have described abnormalities in ocular movements persisting beyond the normal compensation stage. Studies have shown a marked decrease in gain of the VOR on turns toward the lesioned ear,[6] a reduction in the time constant of the response,[7] directional preponderance and phase changes on sinusoidal oscillation,[8] the presence of head-shaking nystagmus,[9] and persistent spontaneous nystagmus.[10,11] The presence of head-shaking nystagmus and severely reduced VOR gain are found with high-acceleration provocational head movements. Thus it appears that the portion of the VOR represented by the doll's eye reflex is particularly disturbed by unilateral vestibular lesions.

Refixation or "catch-up" saccades have been noted on ipsilateral head rotation as long as 1 year after unilateral lesions.[12,13] Although these have been documented via magnetic search coil electrooculography, the only widely available method of detection has been direct visual observation. These saccades, however, are often difficult to appreciate visually.

In this study, the tracings that can be obtained via conventional ENG during performance of the doll's eye test have been studied to determine if catch-up saccades can be more effectively documented with this technique than by observation alone. We have also attempted to determine the permanence of the deficit by studying patients with lesions as long as 9 years postoperatively. To determine the influence of visual cues and eye closure on the doll's eye test, studies were performed both with visual fixation and with eyes closed. The usefulness of this test was assessed by

From the Division of Head and Neck Surgery, University of California San Diego, La Jolla, Calif.

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Send Reprint Requests to Jeffrey P. Harris, MD, 225 Dickinson St. (8895), San Diego, CA 92103.

comparing it with tests for head-shaking nystagmus, spontaneous nystagmus, and rotatory directional preponderance. Finally, we wished to determine if patients with unilateral complete vestibular lesions were clinically symptomatic during high-acceleration ipsilateral turns and, if so, how their symptoms were described.

METHODS AND MATERIALS

Subjects

Each of the six patients studied had no response to ice caloric stimulation unilaterally. Five of the subjects had complete unilateral surgical lesions, and one patient had a canal paresis of unknown etiology (Table I). The time since operation/loss of function ranged from 4 months to 9 years (mean 5.3 years). None of the subjects had a history suggestive of bilateral disease.

The 6 control subjects, 3 men and 3 women aged 19 to 45, had no history of balance disorder or ear disease.

Procedure

Each subject was seated 6 feet in front of a light bar. Eye movements were monitored via conventional ENG; electrodes were positioned mono-ocularly using horizontal channels on both eyes. Left eye velocity was recorded on the second channel. At the beginning of each session, the tracings were calibrated with two targets, each set 20 degrees from the midline. Paper speed was 50 mm per second during the doll's eye test, and 10 mm per second during the remainder of the session.

The doll's eye test was administered by a researcher standing behind the patient. The head of the patient was held firmly in the researcher's hands facing a target light; after a random delay it was then abruptly rotated horizontally, approximately 30 degrees to one side. The patient was instructed to fixate the target light throughout the maneuver. To allow the patient's head to be turned smoothly and comfortably, the fixation target was set at 10 degrees to the left of midline for turns toward the right, and a second target light (used by the examiner to determine the end of the turn) was set 20 degrees to the right of midline; the reverse procedure was followed for leftward turns. The test was repeated at least three times to the left, and three times to the right for each patient. The entire procedure was then repeated with the patient's eyes closed, with instructions to attempt to keep the eyes fixated on the imagined position of the target. Although changes in vertical VOR function could have been expected by vertical testing, this test was not performed vertically, due to the limitations of ENG in accurately recording vertical eye movements.

A test for head-shaking nystagmus was then performed, during which the head was oscillated manually at approximately 4 Hz for 10 seconds. The patient was instructed to keep the eyes closed and to perform mental arithmetic during recording. Rotatory directional preponderance was tested using a torsional swing chair. This was oscillated manually at 0.10 to 0.20 Hz and 90 degrees amplitude. Chair position was recorded by a potentiometer attached to its base. Recordings were made with the patient's eyes closed while performing mental arithmetic.

Each patient, with the exclusion of the labyrinthectomy patient, then underwent 0-degree caloric irrigation of the lesioned ear, in the sitting position with the head hyperextended, and leaning forward with the head dependent to confirm completeness of the lesion. At the conclusion of the tests, each patient was examined visually for the presence of catch-up saccades during head turns.

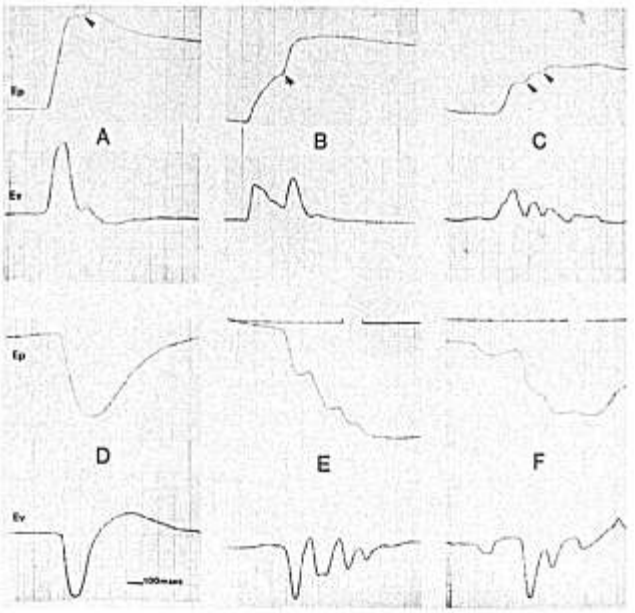

Fig. 1. Comparison of control and patient tracings (turns toward lesioned side). Top tracing is eye position (Ep); bottom is eye velocity (Ev). **A** through **C** are turns to the left; **D** through **F** are to the right. Upward deflection denotes rightward eye movement. **(A)** Control, normal tracing showing small corrective saccade at top of eye position trace (arrow) and corresponding small peak in velocity trace; **(B)** patient 8, 4 months after left vestibular neurectomy; **(C)** patient 3, 9 years after left acoustic neuroma; **(D)** control, normal tracing without saccades; **(E)** patient 5, right vestibular paresis; **(F)** patient 4, right labyrinthectomy. Note that the normal, smooth deflection of the eye position trace in the control subjects is broken up into step-like ripples by the presence of catch-up saccades (arrows) in the patient group.

Scoring

Each doll's eye test result was scored according to the criteria listed below. Copies of each test tracing, containing no identifying markings, were numerically coded and presented in random order to a researcher who had not previously viewed the tracings. The researcher assigned scores of A (abnormal), B (borderline), or N (normal) using these guidelines. Tracings which were disrupted by blinks were eliminated from scoring.

Normal (N). The velocity tracing consists of one large deflection, or one large deflection followed by one small corrective saccade (a 2- to 3-mm peak on the downslope or at the base of the complex). The large initial deflection must account for at least three fourths of the total eye position change achieved. The eye-position tracing shows a fairly smooth upslope which may have a small step near its summit if a corrective saccade occurred (Fig. 1-A, D).

Abnormal (A). One or more catch-up saccades are present. The velocity tracing contains more than one peak, with either two substantial peaks, or three or more peaks of

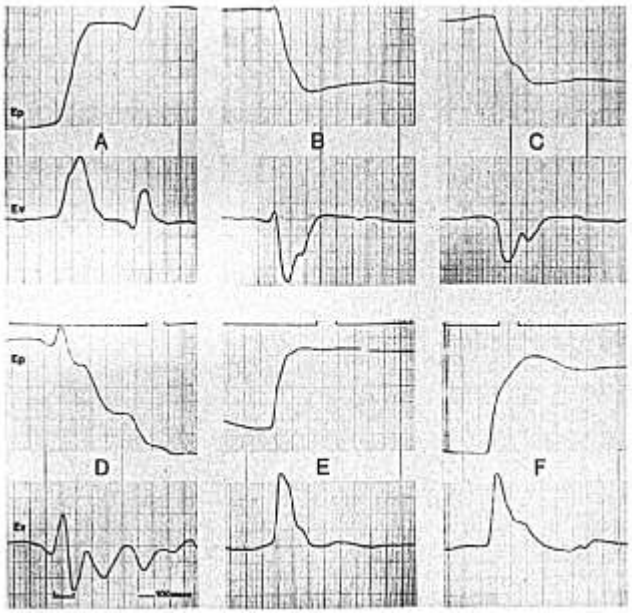

Fig. 2. Comparison of turns in patients, toward the lesioned side vs. the intact side. **(A)** Patient 7, left vestibular neurectomy turning toward the lesion, showing two large saccades and a corresponding deep stair-step deflection in the eye position curve; **(B)** and **(C)**, turns toward the intact side in this patient. **(C)** is borderline; the initial peak accounts for slightly less than three fourths of the eye position, but the complex does not meet the criteria for an abnormal score; **(D)** patient 6, right vestibular neurectomy turning toward the lesion showing multiple saccades preceded by a blink (bracket); **(E)** and **(F)**, turns toward the intact side, both normal.

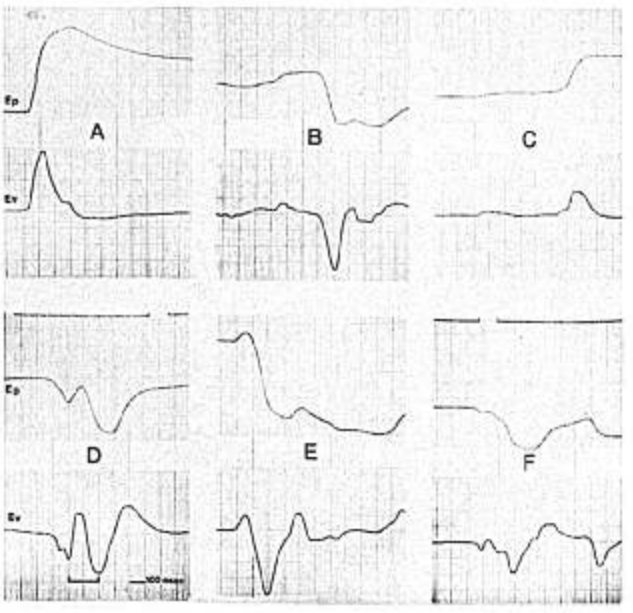

Fig. 3. Comparison of controls and patients during turns toward lesioned side, with eyes closed; same individuals seen in Figure 1. **(A)** Control, tracing is identical to 1-A; **(B)** the pair of saccades seen in 1-B is replaced by a single, large saccade in the direction of head movement, *i.e.*, a completely reversed response; **(C)** the triple-peak complex in 1-C is reduced to a single peak of low amplitude; **(D)** control, showing a large anticompensatory saccade disrupting the normal VOR response (bracket); **(E)** the pattern of four closely linked saccades in 1-E is replaced by a single deflection, followed late in the turn by a second saccade; **(F)** response consists of a single major deflection.

any size in which the first peak fails to account for at least three fourths of the total eye position achieved. The eye position tracing shows a rippled, wavy, or stair-step appearance (Fig. 1-B, C, E, F).

Borderline (B). Tracings which do not fit the normal pattern, but do not meet the criteria for an abnormal score (Fig. 2-C).

RESULTS

The results are presented in Table I. Using these scoring criteria, the doll's eye test was abnormal in the patient group on turns toward the lesioned side in 18 (100%) of 18 tests (Table I). In contrast, among the control group, 1 (3%) of 35 tests was borderline, and the remainder were normal (Fig. 1). The data were cross-tabulated and analyzed using the chi-squared distribution. There was a statistically significant difference when all patient turns were compared with all control turns ($P<.01$, $\chi^2 = 37.68$).

On turns toward the intact side in the patient group, occasional abnormalities were found (Fig. 2). Ten (56%) of 18 tests were normal; 4 (22%) of 18 were abnormal, and 4 (22%) of 18 were borderline. Turns toward the intact side in patients differed significantly from control turns ($P<.01$, $\chi^2 = 15.44$). There was a statistically significant difference between turns toward the lesion vs. turns toward the intact side within the patient group as a whole ($P<.01$, $\chi^2 = 23.71$).

Since the test was performed at least three times in each direction, results could be summated. On turns toward the lesioned side, every test in every patient was abnormal. In contrast, during turns toward the intact side, all patients had at least one normal tracing; however, 5 of the 6 patients also had an abnormal or borderline result on at least one of these turns. The specificity of the test in defining the presence of a lesion and its location was improved by using only the best tracing the subject was able to generate in each direction. By these criteria, all controls were normal; all patients were normal during turns toward the intact side; and all patients were abnormal on turns toward the lesioned side.

When the doll's eye test was performed with the patient's eyes closed, the tracings were distinctly different from those obtained in the light among both patients and controls. The normal VOR response in controls consisted equally of either a single compensatory deflection (Fig. 3-A), sometimes reduced in amplitude from that elicited in the light, or a deflection interrupted by one or two fast phases in the anticompensatory direction (in the direction of head movement) (Fig. 3-D). In the patient group, the distinctive

TABLE I.
Patient Description and Test Results.

Pt.	Age/Sex	Diag-nosis	Oper	Postop Years	Side of Lesion	Spont Nystag*	Head Shake	Dir Prepon	Doll's Eye Test	
									Ipsi	Contra
1	38 y/F	AN	VN	9	L	+ R	+ R	+ R	A A A	N B B
2	63 y/M	Men	Laby	5	R	–	+ L Reverses	–	A A†	N N N
3	67 y/M	CP	None	–	R	+ R	–	–	A A A	N B A
4	40 y/M	Men	VN	6	R	+ L	–	–	A A A A	N N A
5	61 y/F	Men	VN	6	L	+ R	+ R	–	A A†	N N B
6	36 y/F	Men	VN	4 mo	L	–	+ R Reverses	+ R	A A A A	N A A

*Direction indicates direction of fast phase of nystagmus.

†Not scored due to blink.

AN = acoustic neuroma; Men = Meniere's disease; CP = canal paresis; VN = vestibular neurectomy; Laby = labyrinthectomy; A = abnormal; B = borderline; N = normal; Oper = procedure performed; Spont Nystag = spontaneous nystagmus; Dir Prepon = directional preponderance; Ipsi = turns toward lesioned side; Contra = turns toward intact side.

stair-step appearance in the eye position tracing caused by corrective saccades disappeared in the absence of vision in all but one patient (Fig. 3-B, C, F). In one patient, two saccades appeared on two of three tests (Fig. 3-E). All turns toward the intact side followed the normal pattern. In contrast, only 39% of the ipsilateral turns appeared grossly normal; the remaining 61% of these turns showed either very reduced eye movements in response to the test (Fig. 3-C), or a completely anticompensatory response—a large fast phase in the direction of head movement with no visible VOR (Fig. 3-B). The presence of anticompensatory fast phases in both controls and patients made it difficult to develop a useful scoring method for this test.

Catch-up saccades were noticed on visual observation during ipsilateral head turns in all patients. However, several repetitions of turns were necessary in most cases to clearly identify these saccades, which were often of small amplitude.

Spontaneous horizontal nystagmus was identified during testing in 4 (67%) of 6 patients, although it was never present throughout the entire session. The direction of the nystagmus was toward the intact side in 3 of 4, and toward the lesion in 1 of 4. All of the patients were considered to be fully compensated, and none had complaints referable to this nystagmus. One of the six controls also demonstrated nystagmus, although his was vertical (up-beating) in direction and was also entirely asymptomatic.

Head-shaking nystagmus was present in 4 (67%) of 6 patients, and in all cases the initial nystagmus was in the expected direction—toward the intact side. In 2 of 4 patients showing a positive head shake, there was a reversal in direction of the nystagmus late in the response (Table I, patients 2 and 6). One control patient demonstrated a positive head-shake response consisting of a right-beating nystagmus which reversed to a left-beating nystagmus; no other tests were abnormal in this subject.

Directional preponderance toward the intact side on sinusoidal oscillation was present in 2 (33%) of 6 patients. No control subjects were positive.

Symptom Descriptions

All six patients demonstrated a doll's eye deficit, but only two spontaneously described symptoms before the start of testing. After testing, all patients described difficulty maintaining focus on the target during ipsilateral head turns, with blurring of the image. Two of the patients (Table I, patients 4 and 6) were aware of a persistent visual problem during ipsilateral head turns which was first noticed immediately after vestibular neurectomy. For a left-sided lesion, this was described as the tendency for the visual image to jump to the right in a blur whenever the head was turned suddenly to the left. The visual image remained still during slow or gradual head turns. Both patients noted a sensation of mild vertigo, which could be relieved by keeping the eyes closed during ipsilateral turns, when this visual slip occurred. Turns toward the intact side did not cause marked visual symptoms in any patient, other than a slight horizontal movement of the target image during some of the experimental turns.

DISCUSSION

Since both the VOR and the doll's eye reflex are mediated by the vestibular system, lesions of this system disrupt or eliminate both. Complete loss of vestibular input, as seen after bilateral labyrinthectomy, results in severe visual symptomatology. In the absence of the VOR, the eyes tend to turn en bloc with the head; as a result, visual targets cannot be normally fixated during rotation. Visual tracking mechanisms can restore fixation during low-velocity rotation, but such persons report visual illusions with rapid head movements (oscillopsia), often described as streaming, jerking, or drift of the environment in a direction opposite to the turn.[14] In such persons, some compensatory eye movements are provided by poten-

tiation of cervico-ocular reflexes, but these are not able to replace the absent VOR, and corrective catch-up saccades are often detectable.[15]

Until recently, most studies found few deficits after unilateral vestibular lesions, and compensation was believed to be an entirely innate, neurally mediated process. Our study confirms recent work which has shown that there is a significant deficit on high-acceleration ipsilateral turns after such lesions.[6,16] This portion of the VOR represents what has been known traditionally as the doll's eye reflex. After unilateral lesions, the horizontal portion of this reflex is severely disrupted on ipsilateral turns. Compensatory eye movements include a series of corrective catch-up saccades induced by retinal slip, instead of the smooth, gaze-holding motion of the VOR. In the absence of vestibular input from the lesioned ear, VOR-mediated compensatory movements can only be generated by the contralateral ear. It has long been known that contralateral (ampullofugal) input is less effective than ipsilateral (ampullopetal) input in generating eye movements during ipsilateral turns (Ewald's second law). In the case of high-acceleration rotation, the compensatory eye movements generated by contralateral inputs are insufficient to maintain visual fixation and, thus, the normal function of the reflex is lost. The symptoms of this deficit are unilateral and are identical to those described bilaterally in patients with complete absence of the VOR. Thus, although a small amount of VOR-mediated eye movement still occurs,[6] the doll's eye reflex can be considered to be functionally lost unilaterally. This results in a permanent disability.

In a normal person, the act of viewing an eccentric target while moving engenders a characteristic set of head and eye movements.[17] First, a saccade is made to fixate the target; then the head is rotated toward the target, while the VOR mediates eye movement in the opposite direction and at a rate which exactly compensates for the head. This allows the target to remain fixated throughout the motion. The ultimate result is that a target which is presented at the periphery of the visual field is accurately foveated and viewed with the eye in midposition.

Persons with unilateral lesions, such as those following vestibular neurectomy, lose this ability. When a target is presented in the visual field on the side of the lesion, they are able to make the initial saccade to acquire the target. However, if they begin to turn the head rapidly toward it, the eyes tend to travel with the head, and the target image seems to jump off in the opposite direction. This triggers a corrective saccade back to the target, but if head movement continues, the target is again lost, and additional catch-up saccades must occur to reacquire it. The retinal slip and the ensuing series of saccades mimic a nystagmus and engender a mild sensation of vertigo that is unpleasant to the patient. Failure to find a behavioral method to compensate for this deficit would result in permanent complaints of vertigo and visual blurring during rapid ipsilateral head turns. Fortunately, it appears that most patients adopt a behavioral strategy to reduce the symptoms to a comfortable level. In our group, this seemed to involve blinking or eye closure during rapid turns, although any method which reduces the acceleration of the head or eliminates turns toward the affected side would be effective.

Our study confirms other research[13] which documented the presence of catch-up saccades on ipsilateral turns up to 1 year after unilateral vestibular neurectomy. These saccades appear to be present in patients with complete unilateral lesions regardless of etiology, and to persist permanently. The appearance of this deficit was similar in patients whose lesions were as recent as 4 months, or as late as 9 years.

These saccades appear to be triggered by retinal slip. In tests on patients with eyes closed, the distinctive chains of linked catch-up saccades always disappeared and were replaced with either a single compensatory deflection, a compensatory deflection interrupted by one or two anticompensatory fast phases, or an anticompensatory deflection, *i.e.*, eye movement in the same direction as head movement. Anticompensatory eye movements occur in both normal and lesioned subjects during sinusoidal oscillation with the eyes closed, and we suspect that such movements are similar to those documented with the doll's eye test. Two of the patients, who demonstrated only anticompensatory eye movement when tested with eyes closed, may have developed an individual behavioral strategy to deal with their VOR deficit by using this common phenomenon, instead of using an inadequate VOR, the eyes are closed and then "thrown" ahead in anticipation of a new target, effectively eliminating retinal slip. These saccades occurred in spite of instructions to imagine fixating the target.

The doll's eye test in light was more useful, as a single test, than the presence of spontaneous nystagmus, head-shaking nystagmus, or rotational testing in diagnosing the presence of a unilateral lesion and the side involved. The test was diagnostic of the presence of a complete vestibular lesion in 100% of our patients. The other, more traditional, tests for vestibular lesions did not fare as well. Spontaneous nystagmus and head-shaking nystagmus were each found in 67% of the patients, but each also occurred in 1 of 6 controls. When it occurred, head-shaking nystagmus was diagnostic of the side of a lesion; in each case the nystagmus beat toward the intact side. Directional preponderance on sinusoidal oscillation was also diagnostic of the side of a lesion, but not sufficiently sensitive—only 33% of the lesions were detected.

When scored according to our criteria, the doll's

eye test was diagnostic of the side of a complete unilateral lesion. Turns toward the affected side always resulted in markedly abnormal tracings. In the patient group, abnormalities on contralateral turns were not infrequent, but these changes were not present on every such turn, and were always much less severe than those on the lesioned side (Fig. 2). Other studies have documented modest reductions in the gain and time constant of the VOR on contralateral turns which seem to be a consequence of a single unilateral lesion.[16] Our study would support these findings.

Documentation of test results by ENG was an improvement over visual inspection for catch-up saccades, in that it provided an objective, reproducible record. In addition, these saccades were very fast, small, and often difficult to identify by simple visual inspection in our group. ENG tracings, in contrast, could be enlarged and lengthened as needed to give a clear recording of these phenomena.

The absence of caloric response is a widely accepted indicator of severe VOR dysfunction. This test is a useful adjunct to calorics, as it is easier to perform, results in no discomfort to the patient, and can be performed even in the absence of an intact eardrum. Caloric testing remains a superior method to quantitate reductions in response, and allows assessment of low-frequency VOR responses not tested by this maneuver. To evaluate VOR function more completely, other standard ENG tests, including examination for positional nystagmus, and tests of smooth pursuit and saccadic function should be done. This test does not assess function in the vertical canals, and tests high-acceleration responses only. In laboratories in which more sophisticated testing equipment is available, low-frequency sinusoidal testing should be included; vertical canal responses may be accurately characterized with magnetic search coil techniques, and head-velocity recordings for calculation of VOR gain are of diagnostic benefit. However, when used as part of a routine ENG evaluation with widely available equipment, the doll's eye test is an extremely sensitive method to confirm complete unilateral vestibular loss.

CONCLUSION

When the doll's eye test was performed with visual fixation, catch-up saccades were found to be present during all ipsilateral turns in patients with complete unilateral lesions. These saccades were recorded up to 9 years following vestibular neurectomy, indicating that the doll's eye reflex is permanently disrupted in these patients. Documentation of these saccades by ENG provides a useful adjunct to calorics and other traditional tests of VOR function, and is a sensitive indicator of complete unilateral vestibular loss.

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