



INSIGHTS IN PRACTICE

Clinical Topics in Otoneurology

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THE FIXATION SUPPRESSION TEST

Kamran Barin, Ph.D., and Laurie R. Davis, M.N.S.

The fixation suppression test is part of the standard ENG examination. It tests the patient's ability to suppress vestibular nystagmus during fixation upon a visual target. The most commonly used test procedure is one described by Alpert (1974). For at least one right-beating and one left-beating caloric response, the patient's nystagmus is recorded with eyes closed until shortly after the peak of the response. At that time the examiner tells the patient to open the eyes and fixate upon a small stationary target for about 10 seconds. Then the examiner computes the average slow phase velocity of three nystagmus beats just before opening the eyes (SPV_{NoFix}) and the average slow phase velocity of three nystagmus beats during fixation (SPV_{Fix}), as shown in Figure 1.

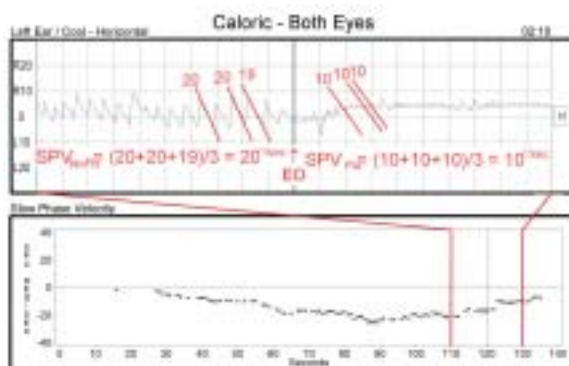


Figure 1. Calculating average slow phase velocity of nystagmus with eyes closed (SPV_{NoFix}) and during visual fixation (SPV_{Fix}). EO indicates the point at which the patient opened the eyes.

From these data, the examiner then calculates a *Fixation Index* (FI) by the formula,

$$FI = SPV_{Fix} / SPV_{NoFix} * 100.$$

FI is a measure of nystagmus intensity during visual fixation expressed as a percentage of nystagmus intensity just before opening the eyes. If nystagmus is completely suppressed by fixation, FI will be 0%. If nystagmus is incompletely suppressed by fixation, FI will be between 0% and 100%. If nystagmus is enhanced by fixation, FI will be greater than 100%. The normal range for FI is 60% or less, which means that, in 95% of normal individuals, visual fixation suppresses vestibular nystagmus by at least 40%.

FIXATION SUPPRESSION AND SMOOTH PURSUIT

There is strong experimental and clinical support for the idea that the *pursuit system* is responsible for suppressing vestibular nystagmus (Chambers and Gresty, 1982; Halmagyi and Gresty, 1979). The pursuit system is designed to keep the image of a small moving target on the fovea—the most sensitive part of retina. Movement of the target's image across the retina causes *retinal slip*. When the pursuit system detects retinal slip, it triggers an eye movement that tracks the target. This tracking eye movement minimizes retinal slip and tends to keep the target's image on the fovea. When a person with vestibular nystagmus fixates upon a stationary target, the slow phases of the nystagmus move the target's image across the retina, thus generating retinal slip. The pursuit system reacts by moving the eyes in the direction opposite the nystagmus slow phases, thus suppressing the nystagmus.

BIOGRAPHY

Kamran Barin, Ph.D., is Director of the Balance Disorders Clinic at the Ohio State University Medical Center and Assistant Professor, Department of Otolaryngology and Department of Speech and Hearing Sciences, The Ohio State University, Columbus, Ohio.

Laurie R. Davis, M.N.S., is a clinical audiologist at Mayo Clinic Scottsdale in Scottsdale, Arizona.

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Some investigators (e.g., Barnes, et al., 1978) have said that if the pursuit system is responsible for fixation suppression of vestibular nystagmus, then the fixation suppression test is really a test of the pursuit system, which means that the fixation suppression test is redundant, because the pursuit system is already being tested by two other ENG tests—the tracking test and the optokinetic test. On the other hand, Tomlinson and Robinson (1981) have said that a different system—the *vestibular cancellation system*—is primarily responsible for fixation suppression of vestibular nystagmus. They have said that the vestibular cancellation system reduces nystagmus intensity and places the image of the target near the fovea, and then the pursuit system eliminates any residual nystagmus. If so, then the fixation suppression test evaluates a different system and is therefore not redundant. It may reveal abnormalities not detected by the tracking and the optokinetic tests (and vice versa). Let us examine this issue by comparing the results of the fixation suppression test with those of the tracking and optokinetic tests.

NORMAL FIXATION SUPPRESSION WITH NORMAL TRACKING AND OPTOKINETIC RESPONSES

Figure 2 shows a patient with normal tracking, optokinetic responses, and fixation suppression.

In the tracking test (Figure 2A), the patient follows a small target oscillating back and forth in the horizontal plane. Frequencies of target motion range from 0.2 to 0.7 Hz with peak-to-peak amplitudes of 30° at all frequencies. Peak target velocities range from 20 to $70^\circ/\text{sec}$. The ratio of peak eye velocity to peak target velocity is determined for each target frequency and compared with age- and sex-matched normative values. The top panel shows a sample of target motion and superimposed eye motion at 0.2 Hz. The bottom panel shows tracking gains for rightward and leftward tracking at each target frequency. Tracking is within normal limits for both directions of target motion at all frequencies.

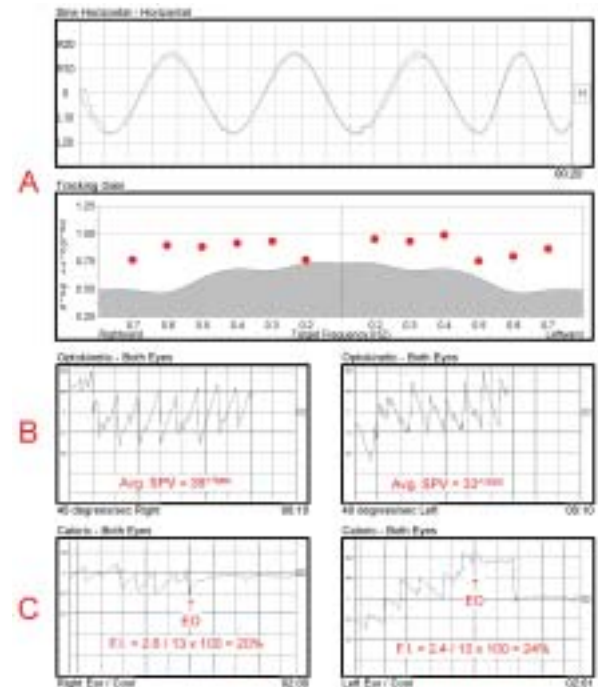


Figure 2. Test results from a patient with normal tracking (A), optokinetic responses (B), and fixation suppression (C).

In the optokinetic test (Figure 2B), the patient tracks a series of small targets moving first to the right and then to the left at a constant velocity of $40^\circ/\text{sec}$. Nystagmus responses are considered normal if slow phase velocities are at least $30^\circ/\text{sec}$ in both directions. In this patient, average slow phases velocities are $38^\circ/\text{sec}$ for rightward moving targets and $33^\circ/\text{sec}$ for leftward moving targets. Both values are within normal limits. (Note that the optokinetic test, despite its name, is not a test of the optokinetic system; it is a test of the pursuit system. A true test of the optokinetic system would require targets that subtend the full visual field.)

In the fixation suppression test (Figure 2C), FI is equal to 20% for nystagmus with rightward slow phases and 24% for nystagmus with leftward slow phases. Both values are within normal limits.





ABNORMAL FIXATION SUPPRESSION WITH ABNORMAL TRACKING AND OPTOKINETIC RESPONSES

Figure 3 shows a patient with abnormal tracking, optokinetic responses, and fixation suppression. In the

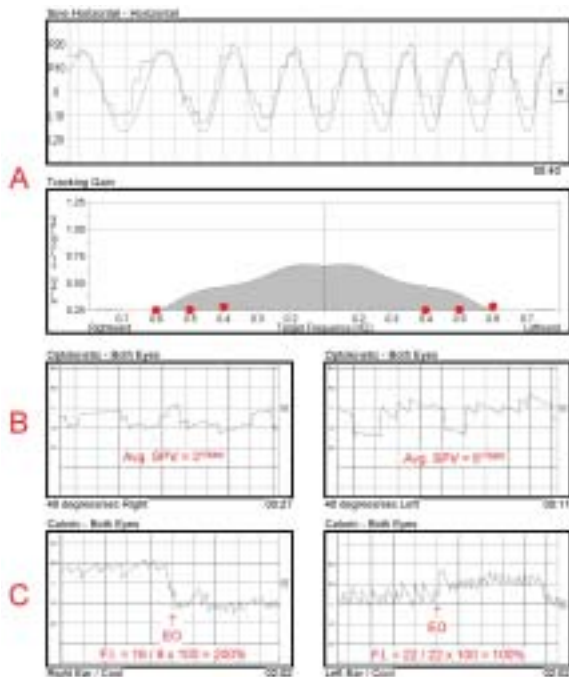


Figure 3. Test results from a patient with abnormal tracking (A), optokinetic responses (B), and fixation suppression (C).

tracking test (Figure 3A), the patient has saccadic pursuit and tracking gains are abnormally low for both directions of target motion. In the optokinetic test (Figure 3B), average slow phase velocity of optokinetic nystagmus is 3°/sec for rightward moving targets and 6°/sec for leftward moving targets. In the fixation suppression test (Figure 3C), FI is 200% for nystagmus with rightward slow phases and 100% for nystagmus with leftward slow phases. These abnormalities indicate a CNS lesion. They are seen in patients with a variety of neurological disorders involving the cerebellum, brainstem, or cerebral cortex.

ABNORMAL FIXATION SUPPRESSION WITH NORMAL TRACKING AND OPTOKINETIC RESPONSES

Figure 4 shows a patient with abnormal fixation suppression, but normal tracking and optokinetic

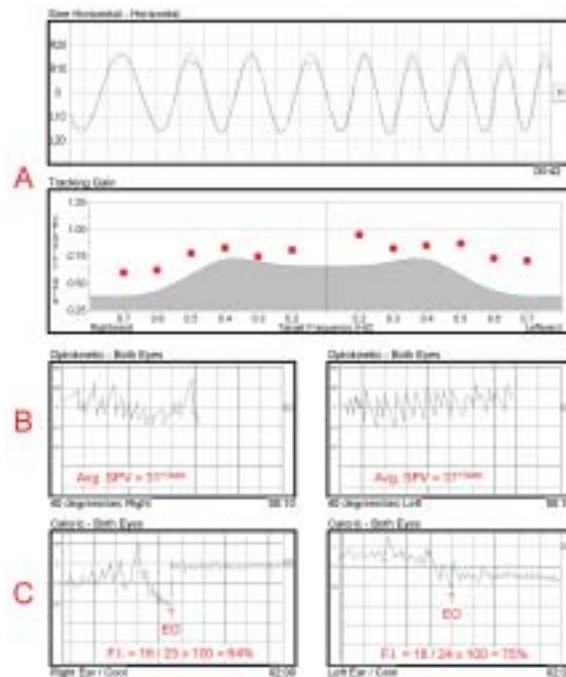


Figure 4. Test results from a patient with normal tracking (A) and optokinetic responses (B) and with abnormal fixation suppression (C).

responses. In the tracking test (Figure 4A), the patient follows the target smoothly and tracking gains are within normal limits at all target frequencies. In the optokinetic test (Figure 4B), average slow phase velocity of optokinetic nystagmus is 31°/sec for rightward moving targets and 37°/sec for leftward moving targets. In the fixation suppression test (Figure 4C), the patient fails to suppress the caloric nystagmus adequately. FI is approximately 64% for nystagmus with rightward slow phases and 75% for nystagmus with leftward slow phases.

This result is uncommon. It denotes a lesion in the central nervous system, most likely in the flocculus of the cerebellum.

Abnormal fixation suppression with normal pursuit supports the hypothesis of Tomlinson and Robinson (1981) that fixation suppression and pursuit are mediated by separate neural mechanisms, but there are other possible explanations. First, tracking and fixation suppression are under voluntary control, so perhaps the patient simply failed to fixate on the visual target during the fixation suppression test. Second, the stimuli used in the tracking and optokinetic tests have predictable trajectories, whereas the timing of nystagmus fast phases is less predictable. Predictable targets are easier to track than unpredictable targets (Leigh and Zee, 1991). Third, the velocity limit of the pursuit system is approximately 30-40°/sec for young healthy persons and closer to 20°/sec for persons over the age of 60. Therefore a patient could have normal pursuit and yet display abnormal fixation suppression if the intensity of to-be-suppressed nystagmus exceeds the patient's pursuit velocity limit. (It should be noted that this third explanation is implausible in the case shown here, since slow phase velocity of caloric nystagmus just before opening the eyes is only about 24-25°/sec.)

NORMAL FIXATION SUPPRESSION WITH ABNORMAL TRACKING AND OPTOKINETIC RESPONSES

Figure 5 shows a patient with abnormal tracking and optokinetic responses, but normal fixation suppression. In the tracking test (Figure 5A), the patient has saccadic pursuit and lower than normal tracking gains in both directions. The tracking defect is asymmetric, being much worse when the target moves to the right. In the optokinetic test (Figure 5B), average slow phase velocity of optokinetic nystagmus is 16°/sec for rightward moving targets and 27°/sec for leftward moving targets. In the fixation suppression test (Figure 5C), nystagmus is virtually eliminated during fixation and the FI is equal to 0% for both directions of nystagmus.

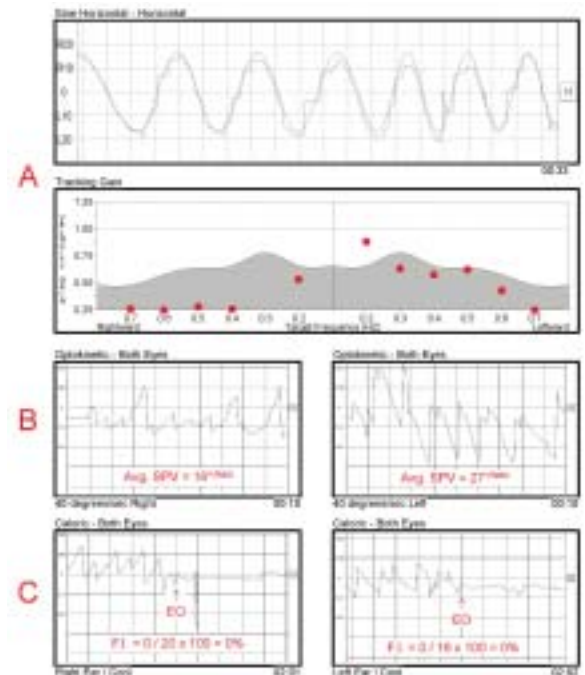


Figure 5. Test results from a patient with abnormal tracking (A) and optokinetic responses (B) and with normal fixation suppression (C).

This result is also uncommon. It also supports the hypothesis of separate pursuit and vestibular cancellation systems, because then it would be possible for a patient with defective pursuit to suppress vestibular nystagmus using the vestibular cancellation system. But again there might be another explanation. Weak caloric nystagmus does not pose much of a challenge to the pursuit system. Thus a patient who has a mild pursuit defect and weak caloric responses may be able to suppress caloric nystagmus, yet be unable to generate normal tracking and optokinetic responses.





CLINICAL IMPLICATIONS

Should we perform the fixation suppression test as part of the standard ENG examination? We think the answer is "yes." The procedure is simple and benign and does not take additional testing time. Two test results presented above (Figures 4 and 5) support the hypothesis that pursuit and fixation suppression are mediated by separate neural mechanisms. However, such results do not prove the hypothesis, since other possible explanations exist. Nevertheless it is a fact that the fixation suppression test sometimes detects abnormalities in patients who have normal tracking and optokinetic responses and *vice versa*.

The fixation suppression test has a major shortcoming—the difficulty of the test depends upon how strong caloric nystagmus happens to be when the patient opens the eyes. If the nystagmus is very weak, even a patient with defective pursuit (or cancellation) can suppress it. If it is very strong, even a person with normal pursuit (or cancellation) cannot suppress it. There are other shortcomings as well. Therefore the test must be conducted with care and the results interpreted with caution. We recommend that attention be paid to the following details:

1. Perform the test during all four caloric responses. Then you will have two opportunities to observe fixation suppression for each direction of nystagmus.
2. Closely watch the tracing and ask the patient to open the eyes immediately after the response reaches peak intensity, which usually occurs between 60 and 90 seconds after the onset of the irrigation.
3. When testing fixation suppression, make sure the patient is actually fixating on a small visual target. It is not enough for the patient simply to open the eyes. Some patients are reluctant to fixate, especially if distressed by the dizziness that is part of the caloric response.
4. When measuring nystagmus slow phase velocities, select beats from a 5 second time period just before opening the eyes and a 5 second time period just after

fixation. Avoid beats that occur within one second before and after opening the eyes, since these beats often contain artifact.

5. When interpreting the fixation suppression test, recognize that nystagmus intensities between 20 and 40°/sec are optimal for testing fixation suppression. Intensities above 40°/sec may overwhelm even a normal pursuit (or cancellation) system and yield false positive results, especially in elderly patients. Intensities below 20°/sec may not pose a sufficient challenge to the pursuit (or cancellation) system and yield false negative results.

REFERENCES

- Alpert, J.N., (1974). Failure of fixation suppression: A pathologic effect of vision on caloric nystagmus. *Neurology*. 24:891-896.
- Barnes, G.R., Benson, A.J., and Prior, A.R.J., (1978). Visual-vestibular interaction in the control of eye movement. *Aviat Space Environ Med*, 49:557-564.
- Chambers, B.R., and Gresty, M.A. (1983). The relationship between disordered pursuit and vestibulo-ocular reflex suppression. *J Neurol Neurosurg Psychiatr*, 46:61-66.
- Halmagyi, G.M., and Gresty, M.A. (1979). Clinical signs of visual-vestibular interaction. *J Neurol Neurosurg Psychiatr*, 42:934-939.
- Leigh, R.J., and Zee, D.S. (1991). *The Neurology of Eye Movements*. F.A. Davis Company, Philadelphia.
- Tomlinson, R.D., and Robinson, D.A. (1981). Is the vestibulo-ocular reflex cancelled by smooth pursuit? In Fuchs, A. and Becker W. (eds.): *Progress in Oculomotor Research*, New York, Elsevier/North-Holland, 533-539.



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