Rebound nystagmus: EOG analysis of a case with a floccular tumour

ATSUMI YAMAZAKI1 AND DAVID S. ZEE2
From the 1Kozawa Eye Hospital, 2-2-11, Goken-cho, Mito-shi, Ibaragi, Japan, and the 2Neurology Department, Johns Hopkins Hospital, Baltimore, Maryland, USA

SUMMARY Eye movements were recorded and quantitatively analysed in a patient with a tumour initially involving the cerebellar flocculus. Ocular motor abnormalities included (1) impaired smooth pursuit, (2) impaired cancellation of the vestibulo-ocular reflex when fixing an object rotating with the head, and (3) gaze paretic and rebound nystagmus. Comparable findings have been reported in monkeys with experimental floccular lesions. The rebound nystagmus (but not the other ocular motor abnormalities) disappeared when the tumour appeared to invade the brain stem in the region near the vestibular nuclei. This finding suggests that the floccular lesion unmasked a bias which created rebound nystagmus and that the bias probably arose in the vestibular nuclei.

Rebound nystagmus refers to a transient nystagmus that occurs when a patient returns the eye to the primary position after a prolonged attempt at eccentric gaze. Slow phases are in the direction of prior attempted eccentric gaze. It has been suggested that rebound nystagmus reflects a transient bias that has been created by the central nervous system in an attempt to hold eccentric gaze (Hood et al., 1973; Zee et al., 1976). Rebound nystagmus occurs even in normal individuals if, after a prolonged attempt at holding eccentric gaze, they return their eyes to the primary position in darkness (Zee et al., 1976). Darkness prevents the visual stabilisation systems from nulling the bias that causes rebound nystagmus. Therefore the bias of rebound nystagmus may be a normal compensatory phenomenon. In patients then rebound nystagmus appears in the light as well, because their visual stabilisation systems (that is, smooth pursuit) are impaired.

Hood et al. (1973) described rebound nystagmus as a useful sign of disorders of the cerebellum. However, it is not yet known where the bias of rebound nystagmus is created (Sharpe, 1974). Recently we observed a patient with a presumed floccular-nodular lesion who had gaze paretic and rebound nystagmus. However, the rebound but not the gaze paretic nystagmus disappeared when the tumour extended into the brain stem in the region of the root entry zone of the vestibular nerve, brachium pontis, and vestibular nuclei. We report a detailed oculographic analysis of this patient’s eye movements before and after several operations.

Methods

Electro-oculograms (EOG) of horizontal eye movements were recorded with a high-impedance direct current amplifier coupled to silver-silver chloride electrodes fixed at points lateral to both outer canthi. The eye position signal was also electronically differentiated to obtain eye velocity. Analogue signals were displayed on a rectilinear pen writer. The overall band width of the recording system was 30 Hz.

The ocular motor examination consisted of the following. (1) Fixation behaviour: fixation in the primary position, eccentric fixation, and fixation in primary position after a prolonged attempt at holding eccentric gaze (to elicit rebound nystagmus). (2) Smooth pursuit: pursuit gain (smooth eye velocity/target velocity) was measured as the eye passed through the primary position while the patient tracked a small target moving in a triangular wave fashion. (3) Vestibulo-ocular responses: vestibular nystagmus was elicited by rotating the patient en bloc in a Bárány chair in complete darkness. Mental arithmetic was used to maintain a constant mental state. Cancellation of the vestibulo-ocular reflex (VOR) was measured as the patient attempted to fixate a target rotating with her. Standardised hot and cold water caloric tests were performed with eyes open behind +30 lenses. The maximum...
slow phase velocity and duration of the induced nystagmus was calculated. Canal paresis was defined by the following formula: If (30°C left + 44°C left) — (30°C right + 44°C right) was greater than 40 seconds a canal paresis was indicated. (4) Saccades: saccadic eye movements were measured during tracking of a target jumping in a nonpredictable stepwise fashion.

Case report

A 17-year-old woman came to the Department of Ophthalmology with the complaint of oscillopsia of 1 week's duration. She also had a 10-month history of headaches. Neurological examination showed questionable papilloedema but apart from the abnormalities of eye movement no other neurological findings. On clinical ocular motor examination she showed striking gaze paretic and rebound nystagmus as well as abnormal smooth pursuit. Fig. 1 shows a horizontal EOG recording of her nystagmus. In the primary position fixation was steady. On assuming eccentric gaze in either horizontal direction she showed gaze paretic nystagmus with a slightly decreasing slow-phase velocity wave form (Holmes, 1917; Zee et al., 1976), which diminished as she maintained eccentric fixation. The initial slow-phase velocity of gaze paretic nystagmus during attempted rightward fixation was higher than that during attempted leftward fixation. On return to the primary position rebound nystagmus appeared.

Fig. 2 shows the patient tracking a target moving in a triangular wave fashion. Smooth pursuit movements were bilaterally impaired necessitating catch-up saccades. However, the gain (smooth eye velocity/target velocity) for rightward tracking was less (0.21) than that for leftward tracking (0.71).

Fig. 3 shows the patient attempting to cancel her vestibulo-ocular reflex using a small fixation target rotating with her head. Normally cancellation is complete and fixation is steadily maintained. However, the patient was unable completely to cancel her vestibulo-ocular reflex, so the eyes were taken off target by the vestibular slow phase requiring her to make saccades in order to reacquire the target. Cancellation during rightward head rotation was more severely affected (gain (eye velocity/head velocity) = 0.53) than during leftward head rotation (gain = 0.10).

Caloric testing showed normal responses in both ears. Saccadic eye movements showed normal accuracy and velocity-amplitude relationships.
Neuroradiological studies suggested a mass in the posterior fossa, and the patient underwent a suboccipital craniotomy. A choroid plexus papilloma was found in the fourth ventricle involving the right flocculus, nodulus, part of the right cerebellar hemisphere, and a part of the left flocculus as well. The tumour was partly removed together with a portion of the right flocculus and paraflocculus.

After this operation the pursuit deficit and gaze paretic and rebound nystagmus persisted (Fig. 4). However, the abnormalities were more marked, especially on rightward gaze.

Two months later, just before reoperation in an attempt to remove the remaining part of the tumour, the patient was again examined. Gaze paretic nystagmus and impaired pursuit were still present. However, she no longer had rebound nystagmus. At the second operation residual tumour was found in the region of the right flocculus and nodulus as well as involving the brain stem at the juncture of the right vestibular nerve and brachium pontis. As much tumour as possible was removed.

Fig. 5 shows an EOG recording 20 days after the second operation. In the primary position there was a small-amplitude jerk nystagmus with slow phases to the right. This increased on gaze left. In addition on rightward gaze there was prominent gaze paretic nystagmus. This combination of gaze paretic nystagmus to the right and vestibular nystagmus to the left is so-called Bruns nystagmus (Bruns, 1908; Cushing, 1921). On return to the primary position after prolonged eccentric gaze there was no change in the slow-phase velocity of the primary position jerk nystagmus. In other words rebound nystagmus was no longer present.

At this time caloric testing showed a definite canal paresis on the right side but a normal response on the left side. Fig. 6 shows an EOG recording of the cold water caloric-induced nystagmus. The maximum slow-phase velocity of nystagmus and the duration of responses showed a clear asymmetry, that is, a canal paresis on the right side. Saccadic eye movements were accurate and had a normal velocity-amplitude relationship.

One year after the second operation the patient was re-examined. Now there was no primary-
position jerk nystagmus, though on caloric testing
the right canal paresis persisted. Gaze paretic
nystagmus and the smooth pursuit deficit were still
present, and rebound nystagmus had reappeared.
There were no signs of tumour recurrence.

Discussion

Our findings suggest that many of our patient's
ocular motor abnormalities—gaze paretic and
rebound nystagmus, impaired smooth pursuit, and
cancellation of the vestibulo-ocular reflex—were
related to tumour involvement of the cerebellar
flocculus and possibly nodulus. In fact the constellati-
on of ocular motor abnormalities shown by our
patient has been reproduced in monkeys with
bilateral floccular ablations (Zee et al., 1978).
However, our patient's rebound nystagmus (but
not the other ocular motor findings) disappeared
when the brain stem and in particular the region of
the vestibular nuclei, eighth nerve, and brachium
pontis appeared to be involved. This finding sug-
ests that the bias that creates rebound nystagmus is
developed in the brain stem and not in the flocculus.
In fact, the floccular lesion, by causing gaze paretic
nystagmus and impairing smooth pursuit, acted as
the stimulus to create the need for the production
of a bias to counteract the centripetal drift of gaze
paretic nystagmus. This bias manifests itself as
rebound nystagmus. Of course other extra-floccular,
cerebellar structures might be essential for the
normal function of the brain stem network that
creates the bias of rebound nystagmus.

One might next ask by what mechanism the bias
of rebound nystagmus is created. Normally after a
saccade the eye is held in its new position by the
tonic output of a brain stem neural network that
provides the appropriate position coded informa-
tion for the ocular motor neurons to hold positions
of gaze. This gaze-holding neural network has been
perceived as a neural integrator, since functionally
it mathematically integrates velocity into position-
coded information (Robinson, 1974). There are
presumably 2 horizontal neural integrators (one for
rightward and the other for leftward movements)
that work reciprocally in a push-pull fashion. If the
neural integrators become defective, eccentric gaze
cannot be held, and the eye drifts back toward the
primary position with an exponentially decaying
velocity. In an attempt to maintain eccentric fixation
repetitive eccentric saccades are required, resulting
in the pattern of gaze paretic nystagmus. However,
if the defect is symmetrical, a null region exists near
the primary position of gaze where fixation is steady.

The centripetal drift of gaze paretic nystagmus
causes movement of images across the retina which
imparts visual acuity. What adaptive mechanisms
are available to the central nervous system to null
gaze paretic nystagmus so as to improve visual
acuity?

The smooth pursuit system could be used to
detect the movement of images on the retina caused
by centripetal drift and then promptly generate a
motor command in an attempt to track the moving
images. This tracking command would be oppositely
directed to the direction of eye drift and would in
effect null the gaze paretic nystagmus and permit
steady fixation. However, in our patient and in most
other patients with rebound nystagmus the smooth
pursuit system is impaired. Therefore other some-

what slower acting mechanisms must be called
upon in an attempt to hold the eyes steady.

Exactly how such mechanisms work or what
stimuli bring them into play are not known. The
central nervous system might detect the velocity of
the continuous centripetal drift of gaze paretic
nystagmus by using visual information about
retinal slip and/or extraretinal information about
eye motion. This stimulus could then be used to
produce an oppositely directed velocity bias in an
attempt to null the drift. Alternatively, the steady
state position of the eyes might be monitored and
after a prolonged attempt at holding eccentric gaze
a position bias could be created to shift the null
point of the 2 neural integrators towards the direc-
tion of attempted gaze holding. Exactly which of
these mechanisms are operative in producing
rebound nystagmus has not yet been established.

Finally, one must ask why our patient's rebound
nystagmus reappeared 1 year after her last opera-
tion. Coincident with the return of rebound nystag-

mus the primary-position jerk nystagmus, presum-
ably of vestibular origin, disappeared even though
the caloric responses still showed a canal paresis.
One can speculate that the central nervous system's
repair mechanisms that rebalanced the vestibular
nuclei to eliminate primary-position nystagmus also
restored the ability of the brain stem to generate
the bias for rebound nystagmus. In fact Haddad
et al. (1978) have shown that repair of nystagmus
after unilateral vestibular nerve lesions can be
accomplished without a vestibulocerebellum. How-
ever, the reappearance of rebound nystagmus may
have simply been coincidental and related to other
unknown mechanisms.

References

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