A Reexamination of End-Point and Rebound Nystagmus in Normals

Josephine Shallo-Hoffmann, Henning Schwarze, Huibert J. Simonsz,* and Hermann Mühlendyck

In order to detail the characteristics of end-point (EPN) and rebound nystagmus (RN), two series of experiments were performed with infrared oculography for measurement of horizontal eye movements. Experiment 1 consisted of EPN recordings during sustained lateral gaze (40° and 50°) in 20 normal subjects. Experiment 2 consisted of recordings of RN in 5 normal subjects. Nine of 20 subjects demonstrated a jerk EPN. EPN almost always appeared immediately and was sustained for 15–25 sec. In Experiment 2, RN occurred in 5 of the 5 subjects who demonstrated EPN. The mean amplitude of RN was always less than that of EPN, and decayed over a 5–10-sec time period. The experiment demonstrated that RN can be evoked in normals even when a fixation target, in a fully lit room, is present. Invest Ophthalmol Vis Sci 31:388–392, 1990

End-point nystagmus (EPN) is evoked by far lateral gaze occurring spontaneously[1] or as a result of fatigue.[2,3] The nystagmus has been described as jerk in form, the amplitude being greater in the abducted eye and the nystagmus either sustained or unsustained over the time that the eye is in eccentric gaze.[1,3]

Rebound nystagmus (RN) consists of a reversal of nystagmus that is evoked by a gaze shift to the primary position (PP) after a period of lateral gaze.[4–6] The direction of RN is always the reverse of the EPN. RN dampens after a short duration. It occurs in cerebellar lesions and in normals, allegedly only when no fixation target is present.[7,8] The duration and velocity of the rebound nystagmus is related directly to the time and duration of eccentric fixation,[7,8] and to the time and duration of the EPN.[9] We reexamined EPN and RN in a number of volunteers and found that RN may occur in normals when a fixation target is present.

Materials and Methods

Eye movements were recorded with a binocular infrared charged coupled device (CCD) eye tracker.[10,11] This table-mounted cameralike eye tracker illuminates the eyes with infrared light (950 nm). The eyes are imaged onto a linear CCD array, a line of 1728 photoelements. The positions of the pupil edges are detected from the intensity profile along this line (by detection of the dark pupil). Eye position is then calculated as the center point between these edges. The CCD arrays were scanned with 80 frames per sec. Because the eye tracker images the frontal plane of the eye, an error in linearity occurs from a gaze of 15° and beyond. This error can be described very well mathematically and was corrected with our software. Linearity tested up to 50° showed an error of 2% when the mathematical correction was used. Calibration error was of the same magnitude. The resolution was 1/10°.

A forehead rest and chin rest were used in both experiments. A bite board was added in Experiment 2.

Solid black dot targets 1 mm in diameter were placed at eye level and located 20°, 30°, 40°, and 50° right and left of the PP. Target distances ranged from 72.4 cm at PP (subtending a horizontal visual angle of 0.079°) to 112.6 cm at 50° eccentric gaze (subtending a horizontal visual angle of 0.051°). The targets were placed in the middle of discs of different color 1.2 cm in diameter (subtending a visual angle of 0.95° at the PP and 0.61° at 50° eccentric gaze) to enhance contrast of the fixation dot and to facilitate the instruction to the subject.

Subjects

Twenty normal unpaid volunteers (ages 19–28 yr, mean age 23.7 yr; 10 female) were recruited for Experiment 1, and 6 normal unpaid volunteers (age 22–27 yr, mean age 25 yr; 4 female) were recruited for Experiment 2 from the university population. The subjects were aware of the nature of the experiment and had given their informed consent. All subjects had normal binocular function, stereopsis, and visual acuity without correction. They had no history of nystagmus, squint, or vestibular disorders, either directly or in their immediate or extended families, and were not under medication or receiving sedatives or other psychoactive drugs.

The mean amplitude and frequency of EPN were calculated by a naive judge with the following procedure. The judge chose the three most typical 5-sec segments of the nystagmus for evaluation. From these three segments, the amplitudes of the quick and slow phases were measured for each wave of both eyes. For each wave, the fast phases were measured from top to bottom and the slow phase from trough to base. Half of the difference of the two phases was subtracted from the greater amplitude value to obtain the most objective assessment of the amplitude from which the mean was calculated. The mean amplitude of RN was calculated from the entire recording. The frequency of EPN and RN was measured in beats per second. The same judge carried out all measurements for the experiments reported here.

Procedure

Experiment 1: Each subject (n = 20) was asked to fixate a target located in the PP, and after 5 sec of recording time, to look at a target located either 20°, 30°, 40°, or 50° right and left of the PP for 45 sec. Recordings were made from all subjects at every gaze angle in both directions. The order of presentation of direction and target location was randomized in order to avoid anticipatory eye movements. Short pauses were made between every 50-sec recording to avoid subject fatigue.

Experiment 2: Targets located at 40° and 50°, right and left of the PP, were used. Each subject (n = 6) was instructed to fixate the target located in the PP, and after 5 sec of recording time, to look at a target (randomized order of presentation) in lateral gaze. After 20 sec, subjects were instructed to look back to the PP, and recordings were made for another 25 sec. Each subject underwent five recordings.

Head movements were measured to evaluate their contribution during high-resolution recordings. A patch with an artificial pupil was attached to the left eye and monitored in the same manner as described

above. Since the eye tracker requires the position of the pupil to calculate eye position, this simple control experiment was possible.

Another control experiment was performed to evaluate the possible influence of the colored discs on which each fixation target was placed. The question was whether the color or edge of the disc may have induced a nystagmoid eye movement; the eye may have been alternating from edge to edge of the disc. The procedure was the same as in Experiment 1. All targets were presented now on a plain, off-white, contour-free background. Three subjects, two of whom demonstrated and one who did not demonstrate EPN in Experiment 1, participated in this experiment.
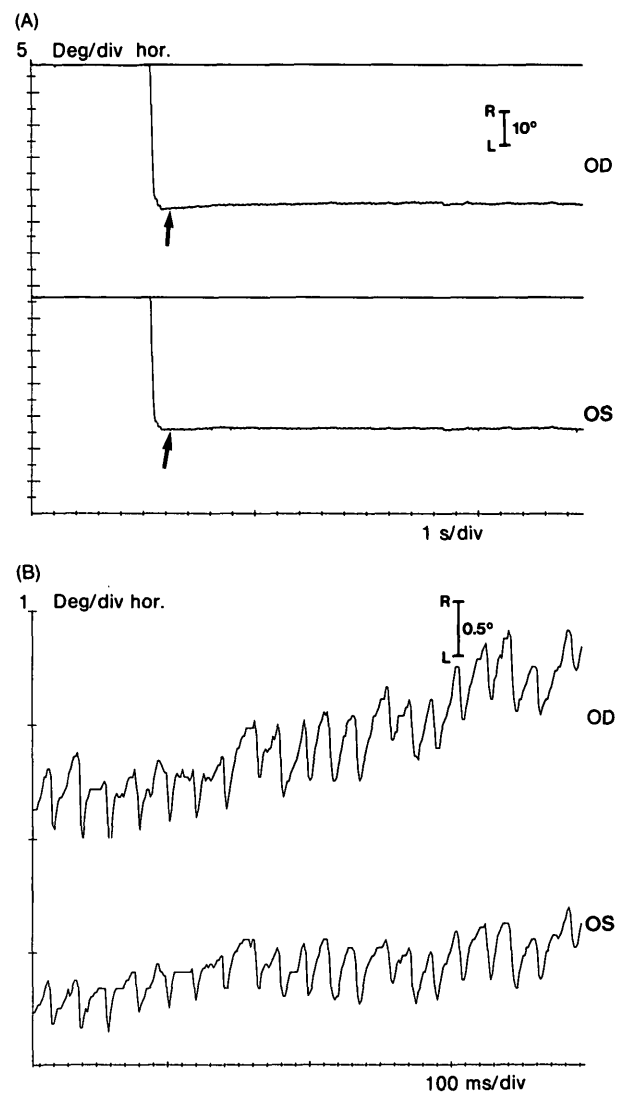

Fig. 1. Typical recording of EPN in Experiment 1. (A) The subject fixated in the PP for 5 sec, and then shifted gaze 50° to the left. Arrow marks onset of EPN. (B) High resolution recording of (A) shortly after the gaze shift. Nystagmus was 0.3°–0.5° in amplitude and was not visible on clinical examination.

Table 1. EPN in nine normal subjects
in Experiment 1

| | EPN | |
Gaze angle	Mean amplitude (degrees)	Mean frequency (Hz)
40° left	1.07 ± 0.39 [1.06 ± 0.48]	1.69 ± 0.60 [1.50 ± 0.83]
40° right	0.66 ± 0.14 [0.65 ± 0]	1.65 ± 0.45 [2.00 ± 0]
50° left	1.48 ± 0.56 [1.46 ± 0.55]	1.62 ± 0.90 [1.85 ± 0.93]
50° right	1.12 ± 0.48	1.61 ± 0.48

Mean scores were calculated from both eyes. Four of the nine subjects demonstrated EPN in only one gaze direction. Scores from these subjects are presented in brackets.

To compare the characteristics of RN evoked in darkness with that evoked in a lit room, rerecordings of RN from one subject in complete darkness (no target visible) and in a lit situation were performed.

A final control experiment concerned the reproducibility of EPN. Could EPN be evoked repeatedly in the same subject on different days? The procedure was the same as in Experiment 1. Five subjects that had participated in Experiment 1 were rerecorded in two to six additional testing sessions, each on a different day. Two of the five subjects demonstrated EPN in the first session; the other three did not.

Results

Experiment 1

Nine of the 20 subjects demonstrated EPN at gaze angles of 40° or 50°. The waveform was jerk, beating in the direction of gaze with a linear or slightly exponential slow phase. An example of EPN is presented in Figure 1.

Table 1 details the mean amplitudes and frequencies of EPN in each gaze angle. In general, the amplitude was larger in the abducted eye (mean increase, 0.38° ± 0.2°) than in the adducted eye. Four of the nine subjects demonstrated EPN in only one gaze direction: two at either 40° or 50°, and two at both 40° and 50°. The mean amplitudes and frequencies of these four subjects are reported in Table 1 in brackets.

EPN appeared immediately after onset of fixation except in one case, in which there was a short latency (5.5 sec), damping within 25 sec after onset. Two recordings from one subject showed EPN that was sustained for the entire 45 sec measurement time. Of the nine subjects, five demonstrated EPN in both directions and the remaining four in only one direction.
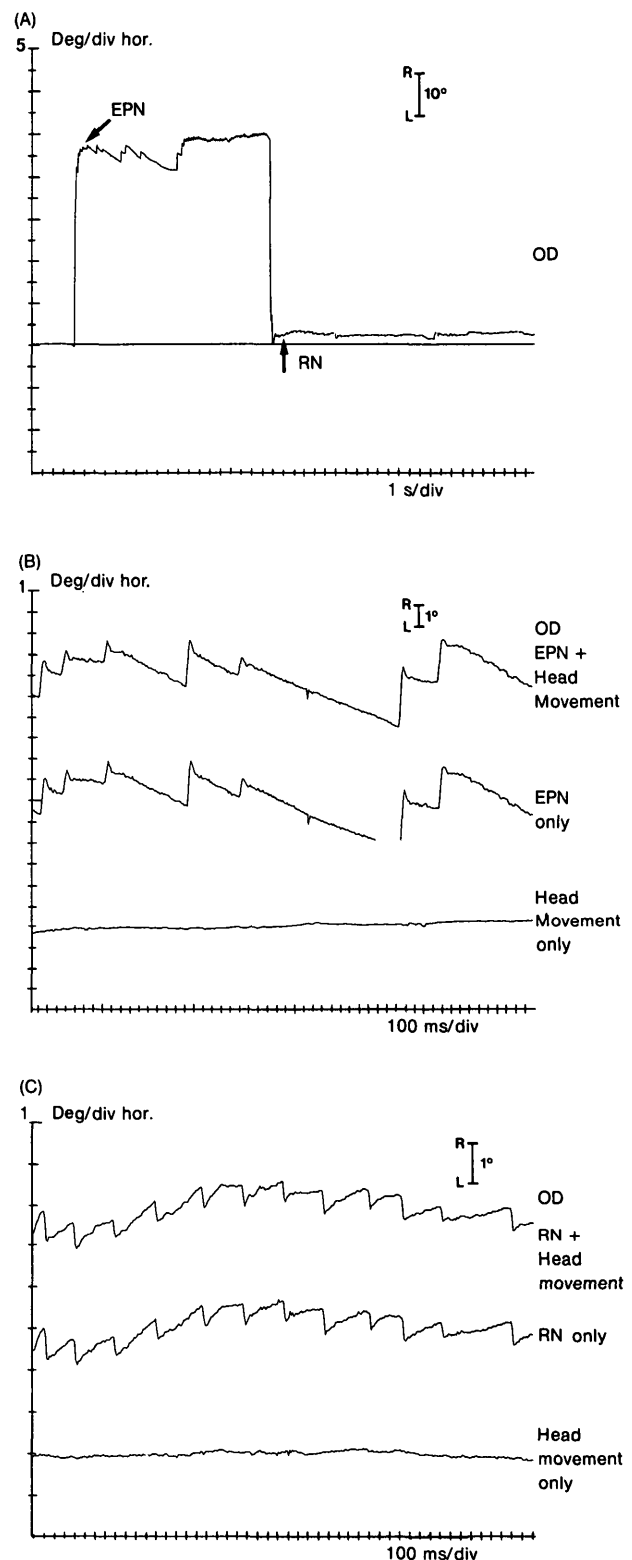

Fig. 2. Recordings of EPN and RN. (A) OD represents a 50-sec recording of the right eye. Head movement was recorded by fixing an artificial pupil over an eye patch covering the left eye. The eyes fixate a target in the PP for the first 5 sec, make a 50° shift to the right of the PP for about 20 sec (arrow indicates onset of EPN), and return to the PP for the last 25 sec of recording time (arrow indicates approximate onset of RN). (B) Higher resolution recording of EPN in (A). (C) Higher resolution recording of RN in (A).

Experiment 2

RN occurred in five of the six subjects. The one subject in whom RN did not appear also did not demonstrate EPN. In general, the amplitudes of EPN and RN were slightly larger in one eye as compared with the other (Fig. 2), without a preference for larger amplitudes occurring in previously abducted eyes.

In all cases the amplitude of RN was less than that of EPN (Table 2). RN tended to dampen after 5–10 sec.

Control for the Influence of the Target Surround

EPN was evoked in two of the three subjects when targets had off-white, contour-free surrounds. No significant differences in the amplitude (student t-test: t = 0.69; degrees of freedom [df] = 45) or frequency (t = 0.05; df = 45) of the nystagmus between the control and Experiment 1 were observed. EPN evoked with contour-free surrounds occurred in the same two subjects who demonstrated EPN in Experiment 1. The subject who did not demonstrate EPN in the Experiment 1 also did not show it here.

Comparison of RN in Light Versus Darkness

RN is far more robust in darkness: the amplitude is larger than that demonstrated in the light (Fig. 3).

Reproducibility of Evoking EPN

EPN was evoked in rerecording sessions in one of the two subjects who demonstrated EPN in the first recording session. Recordings from two of the three subjects who did not demonstrate EPN in the first session showed EPN in rerecording sessions.

Discussion

EPN was evoked in 9 of the 20 subjects. This confirms other reports[2,3,9] that EPN cannot always be evoked in normals. Except in two cases, in which EPN was sustained for the entire 45-sec recording time, the nystagmus damped within 15–25 sec.

In an earlier report,[3] EPN was found in 20° lateral

Table 2. Comparison of normal values for EPN and RN in Experiment 2

	EPN	RN
Amplitude (deg)	1.33 ± 0.45	0.663 ± 0.21
Frequency (Hz)	1.46 ± 0.64	1.680 ± 3.68
Duration (sec)	12.55 ± 5.16	6.090 ± 3.68

n = 5. All values were derived from recordings made at 50° left and right gaze. Duration of the nystagmus was determined at the point where the nystagmus completely damped.
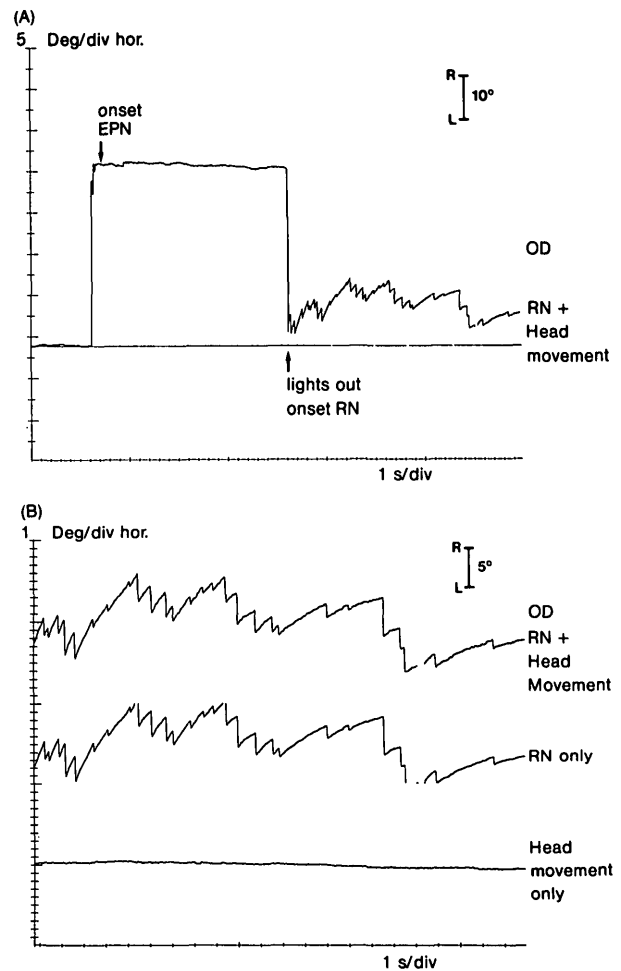

Fig. 3. RN in darkness. (A) The eyes were directed in the PP for the first 5 sec, followed by a gaze shift of 50° to the right (note the onset of EPN, arrow). After 20 sec of lateral gaze, the eyes were returned to the PP. At the same time, the room lights were turned off (note the onset of RN, arrow). Compare this recording with Figure 2A. (The scale is the same, and recordings are from the same subject, in Figs. 2 and 3.) (B) Higher-resolution recording of RN in darkness. The amplitude of RN in light is less than 1° as compared with 1°–4° in darkness. (Compare this recording with Fig. 2C.)

gaze in 1 of 12 subjects. We found EPN only in extreme lateral gaze (40° and over).

In this study, in a lit room, RN occurred only in cases in which EPN (five of six subjects) was induced. The amplitude was larger in one eye than in the other for both EPN and RN recordings. There was no preference (as in Experiment 1) for the abducted eye to show larger amplitudes in Experiment 2. RN dampened in 5–10 sec, and the duration of the effect correlated roughly with the duration of the EPN.

We found a robust RN in the dark with a less well defined waveform of high amplitude, as compared to RN in the light (compare Fig. 2 with Fig. 3), in the one subject in whom differences between RN in light and darkness were compared.

No artifacts due to the colored discs used were found. When the EPN test procedure was repeated in the same subject, EPN was evoked in all subjects but not in every recording session, suggesting that EPN can be repeatedly evoked in the same subjects.

We would agree with Gordon et al[8] that the cause of RN is a velocity bias of the neural integrator evolved during long eccentric gaze. The velocity bias is corrected normally by visual information (retinal slip), processed in the flocculus. As did Gordon and co-workers, we found the RN to fade quickly, a result interpreted by Gordon's group as a reduced time constant of the neural integrator. In their paradigm, retinal slip information was reduced by flashing the fixation light (flash duration 10 msec) every second. We are not absolutely sure, however, whether a flashing fixation light is completely unable to contribute to reducing the velocity bias. We chose a constant fixation target and a fully lit room to imitate clinical examination conditions and found, contrary to the conclusion of Gordon's group, that a small RN can be a normal finding in some individuals.

Key words: end-point nystagmus, rebound nystagmus, eccentric gaze maintenance, eye movements, gaze-evoked nystagmus

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