Research report

Visually induced motion perception and visual control of postural sway in congenital nystagmus

Thomas Eggert *, Andreas Straube, Karin Schroeder

Department of Neurology, Klinikum Grosshadern, Ludwig-Maximilians University, Marchioninistr. 15, 81377 Munich, Germany

Received 20 November 1996; received in revised form 16 January 1997; accepted 17 January 1997

Abstract

In congenital nystagmus (CN) the threshold for detecting motion of visual objects is increased. To determine whether this increase is due to a deterioration of visual motion signals or whether visual-vestibular interactions (which are necessary to judge object-motion in space) are also involved we examined how CN patients use visual motion signals to evaluate self-motion in perceptual and behavioral tasks. Using an optokinetic drum we measured the minimum optokinetic acceleration necessary to induced motion perception of the visual environment in CN patients. This threshold was significantly elevated in the CN patients compared with normals (20.1 deg/s² to 3.25 deg/s²). We further addressed the question whether the elevation of this threshold is due to a deficiency in evaluating visual motion in general or to a specific modification affecting the percept of visual object-motion with respect to the inertial reference only. We thus measured the latency of visually induced self-motion perception, which was found to be very similar or even slightly smaller (1.7 ± 0.7 s) compared with normals (2.2 ± 1.7 s). Moreover, subjects with CN were found to use vision quite efficiently for the visual stabilization of posture (Romberg quotient 2.0 ± 1.16), even if they did not reach the level of normals (Romberg quotient 3.7 ± 1.1). The results indicate that CN affects the estimate of object-motion in a specific and much more severe way than the estimate of self-motion. The minimal effect of CN on self-motion perception can be explained by the low pass characteristics of the optokinetic input to self-motion perception. The specific deficiency in detecting object-motion indicates that adaptation to CN occurs on the level of visual–vestibular interactions for the perception of visual object-motion and not on the level of visual motion signals. © 1997 Elsevier Science B.V.

Keywords: Congenital nystagmus; Visually induced self-motion perception; Visual control of body sway; Efference copy signal

1. Introduction

Congenital nystagmus (CN) is an eye movement disorder that generally begins at birth or in early infancy. Normally the direction of nystagmus is horizontal, its waveform is variable. Hallmarks of CN are increased nystagmus velocity during attempted fixation, the inversion of optokinetic nystagmus, and the dependence of the so-called static neutral zone (SNZ), i.e. the eye position in which the eyes have minimal nystagmus velocity, on stimulus velocity [9]. The nystagmus can cause reduced visual acuity, if retinal slip velocity exceeds 1.6–4.0 deg/s [3]. Motion perception is also impaired in patients with CN [1,5]. Surprisingly, most of these patients do not complain about perceiving illusory movement of the visual environment (oscillopsia) or postural instability, even though they prefer to gaze in the direction of minimal nystagmus velocity (neutral zone).

Several mechanisms have been proposed to explain the absence of oscillopsia: (I) elevation of the visual-motion detection threshold, (II) an efference copy signal or alternatively an extra retinal eye position signal...
to suppress the motion of the retinal image induced by the involuntary nystagmus [2,7,10] and (III) the possibility of a perceptual adaptive process [1].

To determine the mechanisms involved, we investigated the trade-off in the perceptual interpretation of optokinetic motion cues between object- and self-motion. This trade-off can be clearly observed in purely optokinetic stimulation, where visual ganzfeld motion can cause, in addition to the perception of object-motion in the direction of stimulus velocity, also a sensation of self-motion in the opposite direction [4]. With optokinetic stimulation the threshold of motion detection is either a threshold of self-motion or a threshold of object-motion, depending on the acceleration of the stimulus. With high optokinetic accelerations the detection threshold of object movement (drum in space) is reached before the perception of self-motion (here: circularvection) occurs. In contrast, when normal subjects are exposed to an optokinetic velocity ramp at small accelerations of an optokinetic drum, they perceive an illusionary circular self-motion before object movement. This effect is well known [11,16]. A similar one can be observed in linearvection, e.g. linear self-motion is perceived when watching from a stationary train the slow departure of the neighboring train [8].

We measured the minimal drum acceleration necessary to provoke patients with CN to perceive object-motion earlier than self-motion. It should be pointed out that the initial occurrence of self-motion perception in pure optokinetic stimulation does not indicate a special mode of circularvection with very low latency. Melcher and Henn [11] showed that for optokinetic accelerations below 5 deg/s², the latency of circularvection increases with decreasing drum acceleration. At 0.1 deg/s² they found latencies around 10 s. The initial occurrence of self-motion perception at low drum acceleration seems to be due to an even stronger increase of the latency of object-motion perception.

Moreover, we wanted to investigate whether the differences found between normals and CN patients in this experiment were primarily related to a deterioration of visual motion signals or whether they were, on a higher level of processing, due to anomalies of the visual-vestibular interactions for motion perception. For this purpose we compared the latency of the onset of circularvection in subjects with CN and in normal subjects.

In addition to its use for the perception of self-motion and object-motion, the visual motion signal is also used for visual control of posture, which in contrast, does not rely on perceptual thresholds. This opens another possibility for addressing the question of whether the elevation of visual-motion detection thresholds in CN [5] reflects a problem of the retinal motion signal and of the velocity transformation from retinal to head coordinates or, alternatively, a problem of the visual-vestibular interaction for motion perception. We investigated to what extent vision improves the control of postural sway in subjects with CN. Parts of this investigation were presented at the XI Congress of Posture and Gait Research in Portland, Oregon, 1992 [15].

2. Methods

2.1. Subjects

Fifteen subjects (age: 18–72 years) with CN, all verified by DC-EOG recordings and neuro-ophthalmologic examination, and 18 age-matched normal controls (age: 20–65 years) participated in the study. All were inexperienced in psychophysical experiments on motion perception. All CN patients showed a dependence of nystagmus amplitude on eye position. The direction of the nystagmus reversed at the SNZ, i.e. it beat to the right for eye positions right of the SNZ and vice versa. The eye movements of these subjects are analysed in more detail in another study [9]. They had no symptoms other than those of CN. All subjects gave their informed consent to participate in the experiments.

2.2. Experiment 1: visually induced self-motion perception (circularvection)

(a) The minimum optokinetic acceleration necessary to provoke the perception of object-motion prior to circularvection was measured in normal subjects and in patients with CN. A rotating drum-chair system (diameter 1.8 m) was used. The inner wall of the drum was covered with black and white vertical stripes (7.5 deg). Subjects sat in an upright position, aligned with the earth vertical z-axis, which was also the rotating axis of the drum. The chair always remained fixed in space. Drum illumination stayed on throughout the experiment. At a random time, the drum velocity started to increase from zero with constant acceleration. The subjects had to indicate whether the first noticeable rotation appeared to be an object-motion or a self-motion, as well as the direction of the rotation. From trial to trial the acceleration of the drum was always changed by steps of 0.1 deg/s². Starting at 0.1 deg/s² the acceleration was increased until the subject perceived object-motion prior to self-motion. In subsequent trials the acceleration was then decreased, beginning at this value until the subject again perceived self-motion prior to object-motion. The drum acceleration of the last trial before this transition to the initial response pattern, i.e. self-motion prior to object-motion, was taken as threshold acceleration. The hysteresis found with this method ranged between 0.4 and 2 deg/s². For each trial the direction (right or left) of drum accelerations was randomly chosen.
(b) In 12 of the normal subjects and in seven of the patients with CN we measured the latency of circularvection. At a viewing distance of 50 cm, subjects fixated a chair-fixed LED which was placed straight ahead or at 40 deg eccentricity to the left or right side. The drum was accelerated in the dark to a constant velocity of 40 deg eccentricity to the left or right side. The light was then turned on and the subjects were asked to press a button as soon as they perceived self-motion. Latency was measured as the delay between the onset of the light and the moment the button was pressed. For each trial the direction of drum rotation was randomly chosen. The mean latency was calculated out of eight trials for each gaze direction, four trials on each direction of drum rotation. Eye position was monitored by binocular horizontal DC-EOG recordings. The amplitude (and the direction) of the nystagmus was judged by qualitative inspection of these recordings.

2.3. Experiment 2: visual stabilization of posture

The postural sway of subjects was quantified by means of a force-measuring piezoelectric platform (Kistler, type 9281B). The subjects stood freely on a slab of foam rubber (10 cm thick, density 10 g/dm³) covered by a second rigid foot support in order to enhance the particular sensorial weight of the visual system [13]. At a viewing distance of 1 m, a semi-cylindrical, highly structured laser beam projected on the screen provided the fixation target. All measurements were made under daylight illumination (60–70 lux). Then, 25-s segments of sway were analyzed and the recordings were made with sample intervals of 25 ms. On the basis of measured forces on the platform, the trajectory of the subjects center of gravity was computed. The total length of this trajectory, projected on the support plane, was computed and divided by the total recording time in order to quantify the overall stability of posture. We will call this measure ‘sway path’ (m/min). The ratio of the sway path with eyes closed to the one with eyes open is known as the ‘Romberg quotient’ and expresses the visual stabilization of posture.

Subjects were tested under the following conditions:
1. eyes closed
2. fixation straight ahead
3. fixation at 40 deg lateral eccentricity to the side of minimum nystagmus amplitude
4. attempting fixation at 40 deg lateral eccentricity to the opposite side as in 3

Subjects from the control group were also tested with the fixation target placed laterally (40 deg) to the left and right. While data from these conditions in subjects with CN were sorted corresponding to the side of the SNZ, in normals they were pooled together.

2.4. Statistical analysis

To test for the effect of gaze and direction of drum rotation on the latency of circularvection in subjects with CN we used a two-way analysis of variance in a repeated measures design. The differences of group mean values between subjects with CN and normals have been tested by the non-parametric Mann-Whitney U-test. This analysis was applied for the optokinetic threshold acceleration and the latency of onset of circularvection in Section 2.2 and the Romberg quotient and the sway path values in Section 2.3. The effect of gaze on the Romberg quotient was tested using the nonparametric Wilcoxon-matched-pair test. All statistical analysis was performed by means of a commercial software package [14].

3. Results

3.1. Experiment 1

(a) In all 18 normal subjects and in 11 of the patients with CN we measured the minimum acceleration necessary to perceive object-motion earlier than self-motion. In normal subjects this acceleration was on average 3.25 deg/s² (±1.3 deg/s²). In contrast, in patients with CN much higher accelerations (20.1 ± 11.76 deg/s²) were necessary, as shown in Fig. 1. This difference between the groups was highly significant (Mann-Whitney U-test: P < 0.001). In other words, CN patients tend to interpret full-field optokinetic stimulation as self-motion even at high visual accelerations.

(b) The amplitude (and the direction) of the nystagmus of the patients differed between the three tested eye positions (straight ahead, 40° on the side of maximum nystagmus amplitude, and 40° to the other side). However, the two-way analysis of variance did not show a significant effect of either eye-position (\(F(2, 12) = 0.24; \ P = 0.79\)) or the direction of drum rotation (\(F(1, 6) = 0.07; \ P = 0.80\)) on the mean latency of the onset of circularvection in CN patients. Fig. 2 shows the group means of latency in CN patients and in normals and their standard deviations, pooled over both directions of drum rotation. The mean value of the latency in patients was slightly smaller than in normals for lateral gaze as well as for central gaze. The mean latencies over all gaze directions were 1.7 ± 0.7 s (\(n = 42\)) in subjects with CN and 2.2 ± 1.7 s (\(n = 72\)) in the controls. The difference between these overall means, judged by the non-parametric Mann-Whitney U-test, reached a \(P\) level below 5%.
3.2. Experiment 2

In a test of the stability of the subject’s posture while standing, the following sway path values were found (Table 1):

Standing on the foam rubber subjects with CN clearly showed a visual stabilization of posture. When they attempted fixation in the zone of maximal nystagmus amplitude with eyes open, they were able to reduce the sway path compared with that with eyes closed on average by a factor of $2.0 \pm 1.16$ as shown in Fig. 3. Improvement of postural stability was slightly better when they fixated to the direction of minimum nystagmus amplitude (Romberg quotient $2.5 \pm 1.16$; Wilcoxon-matched-pair test; $P < 0.05$). However, visual stabilization of posture did not reach the level of the controls, which showed a Romberg quotient of $3.7 \pm 1.1$ (Mann-Whitney $U$-test; $P < 0.01$). Otherwise, with closed eyes, there was no significant difference between the sway values of CN patients and the controls (Mann-Whitney $U$-test; $P = 0.85$), indicating a normal contribution of the remaining sensory afferences (vestibular and somatosensory system) as well as a normal efferent postural control in CN patients.

Table 1

<table>
<thead>
<tr>
<th>Visual condition</th>
<th>Congenital nystagmus (m/min)</th>
<th>Controls (m/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Eyes closed</td>
<td>$2.87 \pm 0.86$</td>
<td>$2.88 \pm 0.79$</td>
</tr>
<tr>
<td>Eyes open straight ahead</td>
<td>$1.31 \pm 0.5$</td>
<td>$0.84 \pm 0.33$</td>
</tr>
<tr>
<td>Lateral gaze (40 deg)</td>
<td>$1.32 \pm 0.52$</td>
<td>$0.99 \pm 0.27$</td>
</tr>
<tr>
<td>(minimum nystagmus amplitude)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lateral gaze (40 deg)</td>
<td>$1.71 \pm 0.92$</td>
<td>$0.99 \pm 0.27$</td>
</tr>
<tr>
<td>(maximum nystagmus amplitude)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Fig. 1. The individual and the group means of the minimum acceleration of the drum wall necessary to induce the percept of object-motion for (A) controls and (B) CN patients. Only one of the CN patients perceived an object-motion at a threshold in the range of the controls.

Fig. 2. Latency of circularvection (mean ± S.D.) induced by an optokinetic velocity step of 40 deg/s. The left two bars show the data from the conditions with the fixation spot presented straight ahead. The right three bars correspond to the conditions with the fixation spot at lateral eccentricities of ±40 deg (pooled for the controls and sorted by the side of maximum and minimum nystagmus amplitude for the CN patients).

4. Discussion

The main results of our study are as follows:

1. The minimal drum acceleration necessary to elicit perception of object-motion prior to self-motion is significantly higher in patients with CN than in normals.

2. When stimulated with an optokinetic velocity step, the latency of the onset of circularvection tends to be slightly smaller in CN patients than in normals.

3. CN patients show a preserved visual stabilization of posture but to less extent than normals.

In Experiment 1 the threshold for discrimination between object- and self-motion was 3.25 deg/s² in the control group. This value for controls corresponds well with the results of Melcher and Henn [11]. The slightly higher acceleration threshold of 5 deg/s² found by these authors is perhaps due to the smaller drum (diameter 1.40 m) used in their experiments. Our finding, that this threshold is increased in subjects with CN, is closely related to the elevation of the motion thresholds in CN patients.
that can be observed using a sinusoidally moving dot target in darkness. With such a stimulus moving at low frequencies (0.25–1.0 Hz), Bedell [1] found a logarithmic threshold elevation of 0.7–1.0 (500–1000%) increasing towards higher frequency. Because the threshold for object-motion with constant acceleration of optokinetic drum is reached after some seconds [11], most of the energy of the stimulus used in our experiment fell within this frequency band. Bedell's result fits well to the threshold elevation of about 600% we found in CN patients with a full field stimulus (Experiment 1). This similarity suggests that the threshold elevation found with these very different stimulations might be due to similar mechanisms.

First, an elevated threshold for visual motion may result directly from the additional retinal motion induced by nystagmus, because the threshold for detecting a change in velocity is an approximately constant fraction of the velocity itself. Such a mechanism is proposed [1] to explain that an elevation of the object-motion threshold can be induced in normal subjects just by superimposing a nystagmus-like movement onto the target motion. In addition there is a second mechanism involved in CN which leads to a suppression of oscillopsia. Oscillopsia is present in similar eye movements in acquired nystagmus as well as in normals when they are exposed to comparable retinal velocities. In contrast to the first mechanism, which reflects just the normal dependency of the differential threshold on stimulus intensity, this second mechanism seems to be an anomalous modification in the system that is adaptive to the particular situation of CN.

Our second finding that the latency of the onset of circularvection in CN is very similar to that in normal subjects, indicates that visual induced self-motion perception is not affected in such a strong way as object-motion perception. The tendency towards shorter latencies in subjects with CN could be related to a slight speed up of the optokinetic input to the percept of self-motion. On the other hand, a decrease in the latency of circularvection is not necessarily related to such a modification on the level of visual-vestibular interaction for self-motion perception. Alternatively, it may also be explained by a natural dependency of circularvection on the amplitude of the optokinetic velocity step. Decreasing latency with smaller velocity steps is one of the features of the model of Mergner et al. [12]. According to their model a gain reduction of the visual motion signal in CN would also lead to a slightly reduced latency.

Even if we did not measure the mean value of absolute retinal velocity during optokinetic stimulation, the gaze dependency of CN was so strong in our subjects that it certainly differed between the different gaze directions. Nevertheless, there was no significant influence of the gaze position, nystagmus velocity respectively, on the latency of circularvection. This suggests that the slightly shorter latencies of visually induced self-motion perception in CN are not directly linked to the retinal velocity. More insight to the question of whether the elevation of the object-motion threshold in CN is due to changes of the visual motion signals can be gained from Section 3.2.

On average, subjects with CN showed preserved visual stabilization of posture but to a less extent than normals as indicated by the Romberg quotients of about 2.25 compared to 3.7. This raises the question of whether both the increase of the object-motion threshold of about 600% and the deficiency of visual control of posture in CN may be explained exclusively by a higher noise level (induced by nystagmus) of a visual motion signal, which is common to the estimation of object-motion and the control of posture. Our experimental results are not compatible with such a hypothesis as it is shown in more detail in the Appendix A; here we will give only a general idea of the argumentation. We estimate the ratio of the noise power of the visual motion signal between subjects with $\tilde{\sigma}_2^2$ and normal subjects $\sigma_2^2$ from, (a) the elevation of the object-motion threshold, and (b) from the measured Romberg quotients. For this estimation we suppose that the multisensory integration of visual and inertial velocity signals used in the control of posture is optimal (see Eq. (3) in Appendix A). But even if this may not be
perfectly true, the rough estimation shows that both estimates of this ratio $(\tilde{\sigma}_v^2/\tilde{\sigma}_x^2)$ differ by more than a factor of ten. Consequently the initial assumption that both, the deficiency in visual stabilization of posture and the elevation of the object-motion threshold, can be explained by a modification of a common visual motion signal, does not seem to be acceptable. Visual stabilization in subjects with CN is much more efficient than one would expect in this case. It is important to note that it is not necessary for this argumentation to assume that the visual–vestibular interaction for the control of posture is the same as for self-motion perception. We only assume that the visual motion signal used for posture control and for motion perception is the same. The result suggests that the increased object-motion threshold in CN is not exclusively due to a deterioration of the visual motion signal. Most probably, changes of the processing on the level of visual–vestibular interactions are also involved. On this higher level a dissociation of the mechanisms between motion perception and posture control is likely, because requirements for precision and dynamics of internal velocity estimates may be very different between perceptual processes and more reflectory motor control.

4.1. Predictions of existing models

The finding that in CN the percept of object-motion is much more affected than the percept of self-motion and the control of posture can be understood from the low-pass characteristics of the visual input to self-motion perception as modeled by Zacharias and Young [17] and Mergner et al. [12]. Both of these models use a visual velocity input which is expressed in head-fixed coordinates. In CN the extraretinal signals necessary to transform retinal velocity signals to head coordinates are not precise enough to compensate perfectly for the nystagmus [2]. Consequently, the direct effect of CN is an increased noise level of this head referenced visual-motion signal. We computed the responses of the models to a pure visual stimulation with the vestibular velocity input set to zero. We used a sinusoidal movement of the visual environment with an amplitude of 8 deg, which is still in the range of nystagmus amplitudes. The gain of the two models was evaluated within a frequency range between 0.015 and 4 Hz, corresponding to velocities between 0.75 and 200 deg/s$^2$. The result (Fig. 4) shows that both of the models have a response characteristic very similar to a first-order low-pass with a time constant of 5 s. Thus, we can predict that at a frequency of 4 Hz the effect of nystagmus on self-motion perception will be negligible (about 1%). Fig. 5 shows the primary disturbance of the optokinetic signal and its low-pass filtered transfer to the internal estimate of self-motion.

To proceed from this basic prediction to an expectation about the immediate effect of CN on the percept of object-motion, we assume a type of trade-off between the percept of self-motion, the percept of object-motion, and the internal estimate of head referenced visual motion. For the external velocities there is the simple constraint that the sum of the head referenced visual velocity and the head velocity with respect to space yields the visual velocity in space. We suppose that in its normal working range, a system that estimates these external velocities accounts for this constraint. This is illustrated in the shaded area of Fig. 5. Even though the real relation between the percept of object-motion and that of self-motion perception is certainly not as simple as a linear summation, any such trade-off between both, splitting the head referenced visual motion signal in two parts, will lead to the following consequence. Since the transfer of the optokinetic signal to the percept of self-motion has a low pass characteristic, the transfer function to the percept of object-motion will be a high pass filter. This transfer function results from the direct and indirect transfer of the optokinetic input to the percept of object-motion as illustrated in Fig. 5. The finding that the percept of object-motion is much more affected in CN than that of self-motion fits very well to this prediction, because the high frequency disturbance of the optokinetic signal induced by CN will be transferred with a high gain to the object-motion signal. Oscillopsia induced by this disturbance may be avoided by threshold elevation for the object-motion signal as indicated in the right part of Fig. 5.

In conclusion the results suggest that both, the elevation of the object-motion thresholds and the reduced visual contribution to the control of posture in CN, cannot be explained solely by a deterioration of the visual motion signal. We have to assume that there is an additional adaptive modification involved in CN which occurs on the level of visual–vestibular interaction for the perception of object motion.
Fig. 5. Diagram of the signal flow illustrating the low-pass characteristic of the optokinetic influence on the percept of self-motion (head in space). Shaded area: hypothetical trade-off between the three internal velocity estimates. The optokinetic velocity signal transfers twice to the estimate of drum velocity in space: (I) directly, when it is added as a signal of drum velocity with respect to the head, and (II) indirectly, when it is also subtracted via the optokinetic low pass and via the estimate of head velocity in space. The resulting high pass characteristic of the optokinetic input to the velocity estimate of drum in space (1 – ‘Low-pass’ = ‘High-pass’) explains why CN has a more severe effect on the perception of object motion than on the perception of self-motion.

Acknowledgements

We thank Prof H. Mittelstaedt for helpful suggestions concerning the quantitative estimation of noise in multisensory integration and J. Benson for her friendly help in the preparation of the manuscript.

Appendix A

Assuming that the differences observed between normal subjects and subjects with CN can be explained exclusively by a higher noise level of the visual motion signal, we will estimate the ratio of the noise power of this signal between subjects with CN (\( \sigma_v^2 \)) and normal subjects (\( \sigma_v^2 \)) from, (a) the elevation of the object-motion threshold and (b) from the measured Romberg quotients.

(a) As discussed above, the threshold for detecting the motion of a dot target in darkness increases in CN by about the same factor as the visual acceleration threshold measured in Section 3.1. This increase is about 600%. If the threshold for detection of object-motion is proportional to the standard deviation of the noise in the underlying visual velocity signal a reasonable estimation for the noise ratio is given by

\[
\frac{\tilde{\sigma}_v^2}{\sigma_v^2} = \left( \frac{\tilde{\sigma}_v}{\sigma_v} \right)^2 = 6^2 = 36
\]

(b) The sway path value computed is the mean of the absolute velocity of the body (Section 2). Consequently, the square of the sway path value is roughly proportional to the total power of the body velocity. The body velocity should be driven by a command that is the negative of an internal estimate of the actual body velocity, in order to minimize the postural sway. Consequently also the noise power \( \sigma_{vi}^2 \) of this internal estimate is proportional to the sway path value. With the subject’s eyes open, the noise \( \sigma_{vi}^2 \) will depend on both: the noise \( \sigma_v^2 \) of the visual velocity input and the noise \( \sigma_i^2 \) of the velocity input gained from inertial force sensors. According to the theory of statistical estimation [6], the lower limit of the noise power of the multisensory velocity estimate can be expressed by the noise of the two unimodal velocity estimates as follows:

\[
\sigma_{vi}^2 \geq \frac{\sigma_v^2 \cdot \sigma_i^2}{\sigma_v^2 + \sigma_i^2}
\]

This limit is reached when inertial and visual noise are independent and the multisensory integration works optimally. This case we consider here. With the subject’s eyes closed, the noise of the internal estimate depends exclusively on \( \sigma_i^2 \), i.e. the noise of the inertial inputs. Now we can express the square of the Romberg quotient \( R^2 \) in terms of the unimodal noise powers:
Using this equation, we can estimate the ratio \( \frac{\sigma_v^2}{\sigma_{\dot{v}}^2} \) on the basis of the Romberg quotients of normals \((R = 3.7)\) and of subjects with CN \((\bar{R} = 2.25)\):

\[
\frac{\sigma_v^2}{\sigma_{\dot{v}}^2} = \left( \frac{R^2 - 1}{R^2 - 1} \right) = 3.1
\]

This value is much smaller than the estimate of 36 as gained from the elevation of the object-motion threshold in Eq. (1).

References