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The Influence of Age on Optokinetic Nystagmus*

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Summary. The influence of age on optokinetic nystagmus (OKN) was studied in 63 healthy subjects, who were divided into three groups according to their age, group I (20–39 years), II (40–59 years) and III (60–82 years). It was found that on average maximal OKN velocity decreases considerably with age, from 114°/s in group I to 93°/s in group II and 73°/s in group III.

Two mechanisms participate in the generation of OKN, the so-called 'fast' component and 'velocity storage' component. The 'fast' component leads to immediate changes in slow phase nystagmus velocity and is related to smooth pursuit eye movements. The 'velocity storage' component causes more gradual velocity changes and expresses itself during optokinetic afternystagmus (OKAN). To study the relative contribution of these two components, maximal smooth pursuit *and* OKAN velocity were determined in addition to the maximal OKN velocity for the same individuals. It was found that both smooth pursuit and OKAN performance decrease with age. Consequently the maximal OKN velocity, which depends on both factors, is even more affected than smooth pursuit eye movements.

Key words: Optokinetic nystagmus (OKN) – Age – Smooth pursuit eye movement – Optokinetic afternystagmus (OKAN)

Introduction

Optokinetic nystagmus (OKN) is the oculomotor response to large moving visual fields. It consists of a slow compensatory phase and a fast resetting one. The investigation of OKN is a routine clinical test and a sensitive indicator of lesions in the cortex, cerebellum and brainstem (Jung and Kornhuber 1964; Dichgans et al. 1974).

It has been known since Ter Braak (1936), and supported by more recent experimental studies in animals (Cohen et al. 1977; Büttner et al. 1983) that two components participate in the generation of OKN slow phase velocity. One is called the 'fast' or 'direct' component. It leads both to a fast rise in slow phase nystagmus velocity after the sudden presentation of an optokinetic stimulus, and a fast drop at the end of stimulation (Fig. 1). It has been related to smooth pursuit mechanisms (Robinson 1981) and neuronal activity changes in the flocculus (Waespe and Henn 1981; Büttner and Waespe 1984). The other, so called 'indirect' or 'velocity storage' component leads to a more gradual increase during continuous stimulation and a slow decrease in the dark after the end of stimulation, the latter being called optokinetic afternystagmus (OKAN) (Fig. 1). The 'velocity storage' component can be correlated with neuronal activity changes in the vestibular nuclei (Waespe and Henn 1977a, b). The combination of the two components leads to the slow phase velocity profile shown in Fig. 1.

From human studies it is well known that smooth pursuit performance decreases considerably with age (Sharpe and Sylvester 1978; Spooner et al. 1980). This influence of age has to be considered when the pathology of smooth pursuit eye movements is evaluated. If smooth pursuit mechanisms participate in the generation of OKN, then OKN performance should also decrease with age. However, this decrease could be relatively small if the 'velocity storage' component is not affected by age. An influence of age on OKN was shown by Spooner et al. (1980), but no attempt was made to evaluate the contribution of the two components to the OKN response. Furthermore only small stimulus velocities (30°/s) were applied, at which OKN pathology is not easy to detect (Jung and Kornhuber 1964). We therefore studied maximal OKN, smooth pursuit and OKAN velocities in normal subjects of different ages in order to determine the contribution of each component to the OKN response.

Methods

Results were obtained from 63 healthy subjects (29 females, 34 males), in the age range from 22 to 82 years. All had, if necessary corrected, eye sight of at least 20/40 and normal visual fields. None were taking drugs known to affect eye movements (Brandt and Büchele 1983) and none had spontaneous nystagmus of more than 2°/s in the dark. According to their age they were divided into three groups: Group I: 20–39 years (n = 27, average 26.9 years), group II: 40–59 years (n = *15, average 49.4 years), group III: 60–82 years* (n = 21, average 68.5 years).

DC eye position was recorded with conventional electrooculography (EOG). Skin electrodes (Fa. Hellige) were placed bitemporally for horizontal eye position recording and above and below one eye for reecording of vertical eye position. During recordings the head was stabilized by two head rests. To minimize adaptation changes of EOG-sensitivity (Gonshor and Malcolm 1971) subjects were kept for 20–30 min at constant background illumination prior to actual recordings. For calibration subjects looked at small lights placed in

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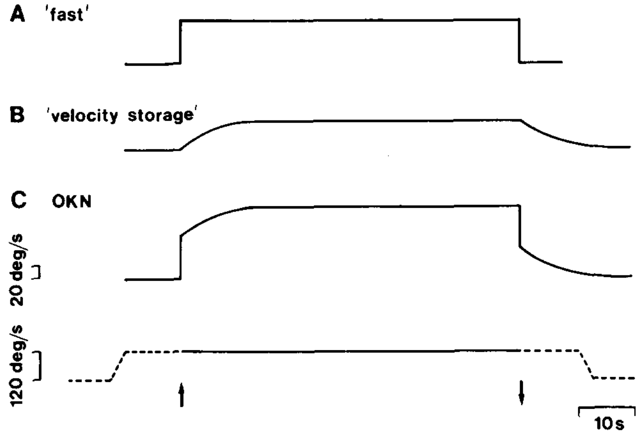

Fig. 1. Schematic drawing of the velocity profile for the 'fast' (**A**) and 'velocity storage' (**B**) component of OKN, and OKN slow phase (**C**) in response to sudden presentation and termination of a high, constant velocity optokinetic stimulus for 60 s. Light-on at *upward arrow* and light-off at *downward arrow*. The 'fast' component (**A**) is characterized by immediate changes in eye velocity, whereas the changes for the 'velocity storage' component (**B**) are more gradual. Both components add during high velocity OKN (**C**)

front and at 10° intervals up to ± 40° horizontally and ± 20° vertically. During recording sessions the calibration was repeatedly controlled.

All subjects were exposed to the same stimulus protocol.

1. For smooth pursuit eye movements subjects tracked a pendulum (frequency 0.4 Hz) swinging in front of them. The maximal stimulus amplitude was 91.5°, corresponding to a maximal velocity of 115°/s. Over a period of 25 s, amplitude (and hence velocity) decreased to 80° and 100°/s respectively.

2. To produce OKN in initial experiments subjects were asked to pay attention to, but not track, moving stripes generated by a shadow projector on a large curved screen extending over 120° horizontally and 80° vertically. With this stimulus it was possible to obtain high OKN velocities of more than 120°/s. However, particularly for older subjects it was difficult to follow the optokinetic pattern for prolonged periods of time. Therefore, all results described below were obtained while subjects, sitting on a vestibular chair, were rotated at constant velocity in the illuminated room. Stimulus velocities were applied in a staircase-like fashion. Each step (acceleration 2.5°/s^2) increased velocity by 10°/s. Velocity plateaus lasted 20–30 s, maximal stimulus velocity was 180°/s. Control experiments in the dark showed that acceleration of 2.5°/s^2 was close to vestibular nystagmus threshold for most subjects. If nystagmus beats occurred, they never outlasted the end of acceleration by more than 5–10 s, OKN measurements were only obtained 10 s after the end of each acceleration. After this time it was assumed that nystagmus is solely determined by the visual input (Robinson 1981). For determination of the intial 'fast' OKN and OKAN response, subjects were rotated in the dark at constant velocity of 120–150°/s (depending on the individual performance, but always above the maximal OKN velocity determined previously), illumination was suddenly switched on for 50–60 s and afterwards turned off, then OKAN was measured in the dark for 20–40 s. Stimulus sequences were applied for left and right nystagmus.

All eye position recordings were DC up to 100 Hz. The signals were amplified with conventional methods. Horizontal and vertical eye position and stimulus parameters were written out on paper charts with a jet ink writer (Siemens Mingograph, cut-off frequency above 1000 Hz) generally at a speed of 1.5 cm/s. Calibration of the eye position signal was in most instances 20°/cm. All measurements were taken from these paper charts. Maximal smooth pursuit eye velocity was calculated from the amplitude of the largest sinusoidal eye movement, which was smooth. No corrections were introduced for smooth pursuit eye movements, which were not perfect sinusoidally (Fig. 2B), and for small catch-up saccades (Fig. 2A); OKN slow phase velocity was determined from the slope of individual eye movement traces. The average of 3–5 measurements was taken to determine OKN velocity at a given stimulus velocity. Slow phase velocity of the initial fast rise (after 'light-on') and initial OKAN (after 'ligh-off') was determined from the first (fast initial rise) or second (OKAN) slow phase after change of illumination. All nystagmus values shown are the average of left and right stimulation values. In no instance were left and right differences more than 10%. All nystagmus velocity measurements showed a high correlation with the number of nystagmus beats/s.

Results

Maximal OKN Velocity

For slow phase nystagmus velocities up to 40–50°/s, responses closely followed stimulus velocity for all age groups (Fig. 3). At higher velocities differences between age groups became obvious (Figs. 2, 3). Group I (age 20–39 years) followed with a high gain (nystagmus velocity/stimulus velocity) of more than

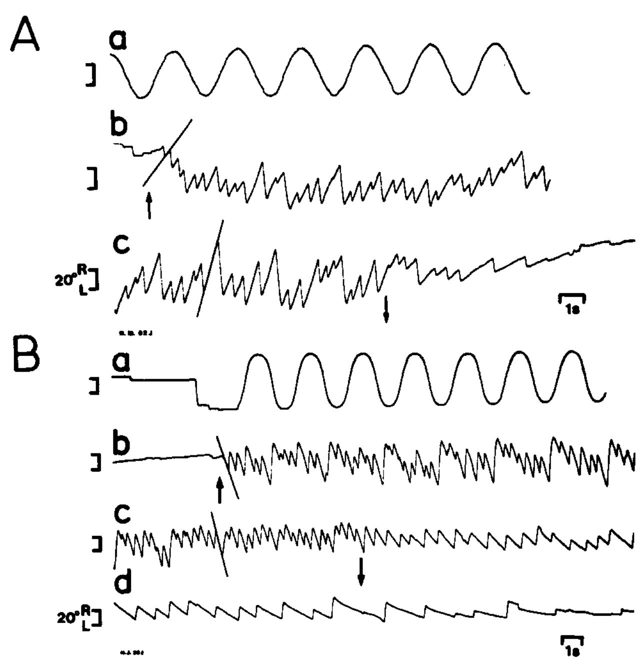

Fig. 2. Original record of horizontal eye position during smooth pursuit (*a*), OKN and OKAN (*b, c, d*) of an older (**A**, group III, age 62) and a young (**B**, group I, age 22) subject. Upward deflection indicates eye movement to the right. Maximal smooth pursuit velocity was 56°/s in **A***a* and 94°/s in **B***a*. *b, c* and *d* are continuous records of OKN. The optokinetic stimulus (130°/s) was presented suddenly by switching the light on (*upward arrow*). *Downward arrow* indicates light-off, after which nystagmus continues as OKAN. Black lines underline different slope of OKN slow phases. Initial slow phase velocity was 31°/s (**A***b*) and 79°/s (**B***b*). Nystagmus velocity rose to 78°/s in **A***c* and 110°/s in **B***c*. Note that OKAN lasted much longer in **A** than in **B**

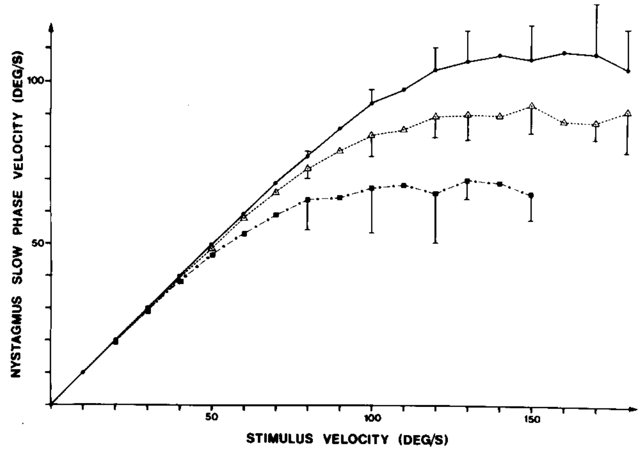

Fig. 3. OKN slow phase velocity for age group I (●——●) 20–39 years), II (△----△ 40–59 years) and III (■–·–·–■ 60–82 years) at different stimulus velocities, with average values shown. Vertical bars indicate standard deviation (not shown in all instances). With increasing age maximal OKN velocity decreases

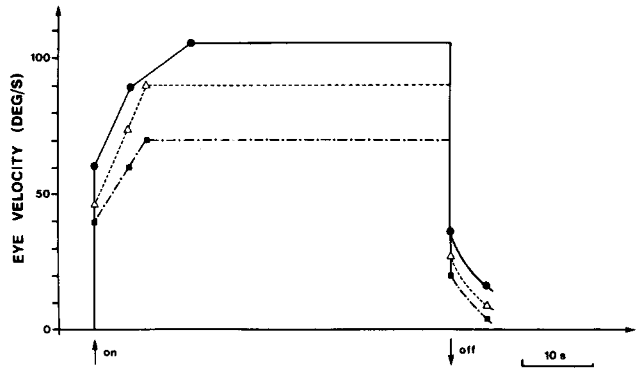

Fig. 4. Slow phase nystagmus velocity profile for different age groups in response to sudden presentation (light-on, *upward arrow*) and extinction (light-off, *downward arrow*) of a high velocity optokinetic stimulus. Each symbol represents averages of 15–26 subjects. The following parameters decrease with age: the fast initial rise (after light-on), maximal OKN and OKAN velocity. Symbols: ●——● 20–39 years (I); △----△ 40–59 years (II); ■–·–·–■ 60–82 years (III)

0.95 to 90–100°/s stimulus velocity. At higher stimulus velocity responses tended to saturate and reached a maximal response of 114°/s (average, range 96–134°/s) at stimulus velocities of 140–170°/s. For group II (age 40–59 years) a decrease in gain compared with group I was already evident at 80°/s stimulus velocity. Responses saturated above 120°/s without further increase at higher stimulus velocities. Maximal nystagmus velocity was on average 93°/s (range 72–114°/s). Group III (age 60–82 years) subjects had a maximal nystagmus velocity of only 73°/s (average, range 47–100°/s). These velocities could be generally maintained up to stimulus velocities of 150°/s (Fig. 3).

Thus, high stimulus velocities revealed rather large response differences between groups. The difference between group I and III was more than 40°/s on average. It should be noted that none of the group I subjects had a maximal nystagmus velocity of less than 96°/s.

Fast Initial Rise

The fast initial rise of slow phase nystagmus velocity was determined after sudden presentation of the visual surround at constant velocity rotation of 120–150°/s. The actual stimulus velocity for each individual was 20–40°/s above the maximal OKN velocity determined previously. This initial rise was also strongly influenced by age (Figs. 2, 4). Whereas group I reached on average a value of 61°/s (range 31–112°/s), this value was only 46°/s (range 29–63°/s) for group II and even less for group III (average 40°/s, range 8–60°/s). After this initial rise it took on average 7 s (group III) to 14 s (group I) before maximal OKN velocities were reached (Fig. 4).

Optokinetic Afternystagmus (OKAN)

When the light was suddenly switched off after 50–60 s of constant high velocity stimulation nystagmus velocity fell rapidly by 50–70°/s to initial OKAN values of 37°/s (average, range 0–62°/s) for group I, 27°/s (range 7–50°/s) for group II and 20°/s (range 7–35°/s) for group III. Afterwards OKAN decreased more exponentially. Duration of OKAN was not systematically investigated. Generally subjects were kept for 20–40 s

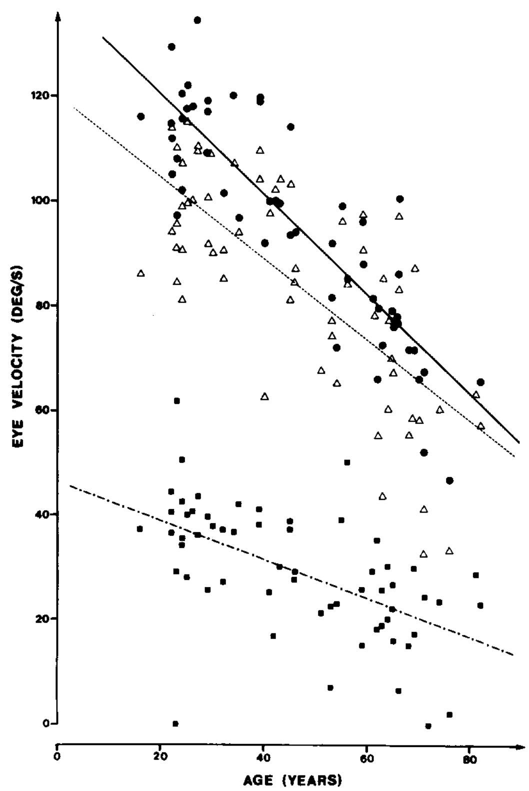

Fig. 5. Influence of age on maximal OKN (●——), smooth pursuit △---) and OKAN (■–·–) velocity. Smooth pursuit (a measure for the 'fast' OKN component) and OKAN (a measure for the 'velocity storage' component) performance decrease with age. As a result the decrease of OKN is even more pronounced than that of smooth pursuit, since both parameters participate in the generation of OKN. Shown are the linear regression lines. Group I: $y = -0.96 \times +140$; $r = -0.87$, group II: $y = -0.77 \times +120$; $r = -0.70$, group III: $y = -0.38 \times +47$, $r = -0.57$

in the dark. During this time about 50% of group I and II subjects had not completed their OKAN I (i.e. after nystagmus beating in the same direction as the preceding OKN). From the remaining subjects about one-fourth showed nystagmus reversal (OKAN II) (Brandt et al. 1974; Büttner et al. 1976; Waespe et al. 1978). For group III most subjects completed their OKAN I in 20–30 s, and OKAN II was seen in about 30% of group III subjects. Thus, there was a clear tendency that the duration of OKAN I also decreased with age.

Smooth Pursuit Eye Movements

In this study smooth pursuit performance was found to decrease with age (Figs. 3, 5), confirming previous reports (Sharpe and Sylvester 1978; Spooner et al. 1980). The maximal smooth pursuit velocity decreased from 99°/s (average, range 81–115°/s) for group I to 86°/s (range 62–104°/s) for group II and to 63°/s (range 33–97°/s) for group III. Maximal stimulus velocity was 115°/s. Thus, potentially some younger subjects might have attained even higher smooth pursuit velocities (Lisberger et al. 1977). Since our smooth pursuit stimulus (a pendulum) was not well defined quantitatively, no attempt was made to determine actual gain of smooth pursuit eye movements. It should be noted that maximal smooth pursuit velocity was generally higher than the fast initial rise slow phase nystagmus velocity. The differences were on average for group I 39°/s, group II 40°/s and group III 23°/s.

Discussion

The results clearly demonstrate that OKN performance decreases considerably with age. This is not restricted to ages above 60 but rather a continuous process already significantly affecting middle aged subjects (group II) (Figs. 3, 4, 5). As a rule the maximal OKN velocity decreases every year by 1°/s (Fig. 5). It has been described above that the size of the maximal OKN response is determined by the 'fast' and the 'velocity storage' component. Figure 5 shows that performance for both components, i.e. smooth pursuit ('fast') and OKAN ('velocity storage'), decreases with age. It is to be expected that maximal OKN velocities are higher than maximal smooth pursuit velocities, due to the additional 'velocity storage' influence during OKN (Fig. 5). Figure 5 also shows that the OKN velocity decrease is even more pronounced with age than smooth pursuit; reflecting that both of its components are affected. Furthermore it stresses, that OKN in humans is mainly determined by the 'fast' and less by the 'velocity storage' component. For older subjects (group III) the 'fast' component dominates the OKN response, almost entirely.

Thus for clinical testing it has to be taken into account that the influence of age is even more pronounced for OKN than for smooth pursuit. Clinically, OKN pathology is generally assumed if slow phase velocity is less than 60°/s (Jung and Kornhuber 1964). Our results show that with consideration of the age factor more sensitive measurements in establishing OKN pathology can be made. This applies particularly for younger subjects (group I). In our sample the lowest individual nystagmus velocity in this group was 96°/s. With two standard deviations (95% limits) the lower value was 95°/s. Thus in this age group nystagmus velocities of less than 95°/s are pathological. However, it should be stressed that our subjects were highly motivated by the investigators and that OKN was measured during constant velocity rotation of the subjects, which provides an optimum of full field stimulation. Thus, these velocities may not be applicable to the routine clinical conditions. So for clinical purposes the normal range of OKN velocities for each age group should be determined individually for each laboratory.

As described earlier neurophysiological and lesion studies support the hypothesis that the 'fast' component of OKN and smooth pursuit eye movements share the same premotor structures (Dichgans et al. 1978; Robinson 1981). In our experiments it was found that maximal smooth pursuit velocity was generally 20–40°/s higher than the fast initial rise of OKN. One contributing factor may be that the maximal 'initial fast rise' velocity was not reached during the first slow phase nystagmus beat. The main cause of this difference probably reflects the fact that 'active' pursuit (tracking the pendulum) leads to higher gain values than 'passive' pursuit (exposure to the moving environment) (Barnes and Hill 1984).

Most of our knowledge about the mechanisms underlying the generation of OKN stems from animal experiments. In the monkey, both the 'fast' and the 'velocity storage' component are well developed, which allows maximal OKN velocities up to 200°/s (Cohen et al. 1977). In animals with poor (cat) or no foveal vision (rabbit, rat) OKN depends mainly on the 'velocity storage' mechanism (Collewijn 1969; Keller and Precht 1978). Man, in contrast, relies predominantly on the 'fast' component for OKN generation. These species differences have to be considered when reports on OKN are evaluated. It is further known, that the 'velocity storage' mechanism takes a long time (5–10 s time constant) to 'charge-up' (Robinson 1981; Waespe et al. 1981; Boyle et al. 1985). Thus, during routine bedside investigation of OKN with a striped cylinder only the 'fast' component, i.e. smooth pursuit mechanisms are investigated. This is reflected in clinical findings that particularly with brainstem and cerebellar lesions smooth pursuit and OKN are affected in parallel (Dichgans et al. 1978), since basically the same neuronal mechanisms are responsible for their generation (Waespe and Henn 1981; Büttner and Waespe 1984).

In conclusion, the same premotor structures participate in the generation of smooth pursuit eye movements and the 'fast' component of OKN. This partly explains why both OKN and smooth pursuit performance decrease with age. However, the decrease of OKN is relatively more pronounced, since, in addition, the 'velocity storage' component also declines with age.

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