Slow build-up of optokinetic nystagmus associated with downbeat nystagmus

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Eye movement recordings in two patients with downbeat nystagmus demonstrated an unusual finding of severely impaired smooth pursuit and relatively unimpaired optokinetic nystagmus (OKN). OKN was characterized by a remarkable, slow build-up of slow-component velocity, similar to that found in afoveate animals. Optokinetic after-nystagmus (OKAN), or transient persistence of nystagmus after cessation of visual stimulation, typical of the optokinetic response of normal human subjects, was also preserved in these patients. These observations suggest that the normal contribution of smooth pursuit to the ocular motor response to rotation of the visual environment can be selectively removed by a lesion at the level of the craniocervical junction.

Key words: optokinetic nystagmus, downbeat nystagmus, smooth pursuit, electro-oculography, eye movement recordings

In the clinical examination of eye movements, smooth pursuit is induced by instructing the patient to track a small, smoothly moving target within the central visual field, whereas optokinetic nystagmus (OKN) is induced by moving patterns with features of high contrast, such as alternating black and white stripes, in the patient's central and peripheral fields. An important function of the smooth pursuit system is to stabilize the image of an object of regard on the fovea. The optokinetic system might have evolved phylogenetically as an aid to the vestibulo-ocular system in stabilizing retinal images during spontaneous movements of the head in the light.[1-3] During rotation of the head, the vestibulo-ocular response (VOR) rotates the eyes in the direction opposite to head movement. However, the velocity of these eye movements is less than that of the head movement, and the VOR alone cannot stabilize the retinal images. During head movements in the light, the optokinetic response acts synergistically with the VOR to increase the velocity of the compensatory eye movements to match the velocity of the head movement. Stabilization of the retinal images should lead to better maintenance of equilibrium and orientation in the environment.

In most patients, lesions in the cerebral hemispheres, brainstem, and cerebellum that affect smooth pursuit also impair OKN. This observation suggests that smooth pursuit and OKN might be outputs of the same ocular motor system subjected to different visual stimuli or, at least, that these two types of eye movements share some common, supranuclear pathways. We report studies of

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This work was supported by a grant from the National Eye Institute, NIH grant, 2 R01 EY 01853-03, and NIH grant NS 09823.

Submitted for publication Aug. 28, 1978.

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eye movements in two patients with downbeat nystagmus that document severely impaired smooth pursuit and relatively unimpaired OKN. OKN in these patients demonstrated an unusual, prolonged build-up of slow-component velocity (SCV), similar to that found in afoveate animals. These observations suggest that in humans smooth pursuit contributes to the eye movement response to stimulation of the entire visual field (OKN) and that this contribution can be selectively removed by discrete lesions at the level of the craniocervical junction.

Methods

Eye movement recordings. Eye movements were recorded with DC electro-oculography. Horizontal movements of each eye were recorded by electrodes at the inner and outer canthi, and vertical movements were recorded by electrodes placed above the eyebrow and below the lower eyelid. The bandwidth of the recording system was 0 to 100 Hz. The SCV of induced jerk nystagmus, velocity of smooth pursuit movements, and velocity, amplitude, and latency of saccades were calculated by digital computer techniques. Details of the recording and analysis systems have been described previously.[4-6]

Smooth pursuit movements and saccades were induced by instructing the patients to track a laser target moving in a sinusoidal pattern (frequency 0.2 Hz, amplitude 30°, maximum velocity 25°/sec) and in square-wave patterns of random frequency, direction, and amplitude. Optokinetic responses were obtained with patients seated within a cloth drum of 1 meter diameter whose interior consisted of alternating, 15° wide, white and black vertical stripes. Rotation of the drum produced stimulation of the entire visual field. The drum was rotated in steps of 30°/sec and in sinusoidal patterns (frequency 0.05 Hz, peak velocity 30°/sec). Drum velocity of 30°/sec was chosen because in our experience, less variability of SCV is present in individual normal subjects and patients at this velocity than at higher velocities. Patients were instructed to stare at the white stripes as they passed directly in front of them.

VORs were induced with the patients seated on a motorized chair within the optokinetic drum. Patients were rotated in darkness in sinusoidal patterns (0.05 Hz, 30°/sec). A relatively low frequency of 0.05 Hz was chosen for the following reasons. (1) The duration of half cycles was long enough (10 sec) for generation of a sufficient number of nystagmus cycles for reliable computerized curve-fitting. (2) Most patients with clinically significant abnormalities of the VOR in our laboratory have been identified at this frequency, as well as at higher frequencies. (3) The torque of the turntable (10 ft-lb) was not great enough to allow rotation at much higher frequencies. Suppression of the VOR by fixation was tested by instructing the patients to fixate a vertical stripe in front of them while the chair and drum were rotated simultaneously in a sinusoidal pattern (0.05 Hz, 30°/sec) in the light. Synergistic interactions of the VOR and OKN were studied by rotating patients sinusoidally (0.05 Hz, 30°/sec) within the stationary optokinetic drum in the light. During this test, the VOR and OKN both produce slow components of nystagmus in the direction opposite to that of chair rotation.

Patients

Patient 1. This was a 22-year-old woman who had been in good health until 15 months earlier when she noted diplopia and oscillopsia on lateral gaze. She had been clumsy since childhood but had not noted a recent change in her coordination. She had begun ballet lessons but found that she could not inhibit her sensation of rotation while spinning by fixating stationary objects. Her past history and family history were otherwise negative for neurologic disorders or congenital defects.

On examination, a downbeat jerk nystagmus was found in lateral and down gaze. Motor examination revealed slight spasticity of the left lower extremity, but it had normal bulk and strength. Deep tendon reflexes were more brisk in the left leg than in the right, and unsustained clonus was elicited in the left ankle. The plantar response was equivocal on the left and down-going on the right. The remainder of the neurologic examination, including the Romberg test and tandem gait, was unremarkable.

Results of skull and cervical spine X-rays, computerized axial tomography, and lumbar puncture were unremarkable. Pneumoencephalography revealed a type 1 Arnold-Chiari malformation, which was confirmed during a posterior craniotomy and decompression of the foramen magnum. Eight months postoperatively, downbeat nystagmus had disappeared, and the remainder of her ocular motor abnormalities, described below, had decreased moderately.

Patient 2. This was a 59-year-old woman who had slowly developed poor balance, intermittent dizziness, and vertical oscillopsia on lateral gaze 7 years earlier. She had also noted a sensation of

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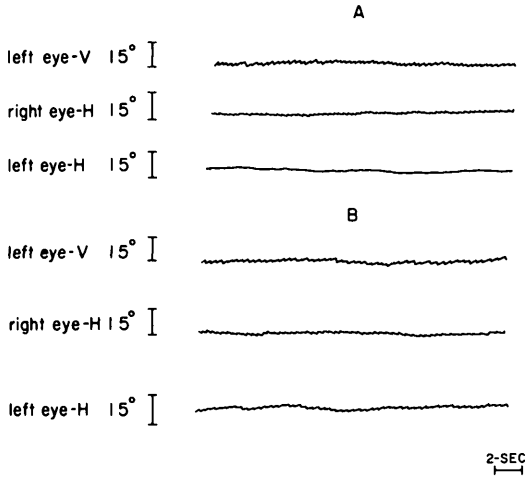

Fig. 1. Downbeat nystagmus. *H*, Horizontal; *V*, vertical. Deflections upward are up or to the right; deflections downward are down or to the left. **A**, Patient 2 in primary position of gaze. Note fast component to the right in the right eye. **B**, Patient 1 in gaze down and right 35°. Note fast component to the right in the right eye and to the left in the left eye.

spinning after sudden movements of the head lasting a few seconds. These symptoms had been stable. There was no past medical history of other neurologic disorders or congenital defects. Her family history was unremarkable.

On examination a downbeat jerk nystagmus was present in all directions of gaze. Motor examination and deep tendon reflexes were unremarkable. Her gait was wide-based and slightly unsteady, and she was unable to walk in tandem. During the Romberg test she fell backward and to the left. Slight dysmetria and dysdiadochokinesia were present in the left hand. The remainder of the neurologic examination was unremarkable.

Skull and cervical spine x-rays, computerized axial tomography, vertebral angiography, and lumbar puncture revealed no abnormalities. Pneumoencephalography revealed atrophy of the left inferior pons, upper medulla, and midline cerebellum. A diagnosis of olivopontocerebellar degeneration was made.

Results

Spontaneous nystagmus. Both patients had a vertical jerk nystagmus with the fast component downward in the lateral and downward positions of gaze (Fig. 1). Downbeat nystagmus was also present in the midposition and in upward gaze in Patient 2. The frequencies and amplitudes of the nystagmus were 2 to 3 Hz and 1.5° to 4.2°, respectively, in Patient 1 and 2 to 4 Hz and 1.7° to 5.0°, respectively, in Patient 2. The frequency and amplitude were greatest in the down and right and down and left positions of gaze in both patients. In most positions of gaze, a small horizontal component of the nystagmus was present to the right in the right eye and to the left in the left eye (Fig. 1). Similar disconjugate horizontal components have been found in most of the 15 other patients with downbeat nystagmus studied in our laboratory.

Patient 1 demonstrated horizontal rebound nystagmus, as described by Hood et al.[7] On lateral gaze, the SCV of the horizontal gaze–paretic nystagmus gradually decreased as lateral gaze was maintained. On return to center gaze, slow horizontal drifts of the eyes toward the previous direction of lateral gaze corrected by fast components in the opposite direction were present for a few seconds.

Smooth pursuit and saccades (Table I). Horizontal smooth pursuit was markedly impaired in both patients during attempts to track the sinusoidally moving target (0.2 Hz, 25°/sec). Smooth movements could not match the target velocity, and catch-up saccades were required to refixate the target (Fig. 2). The gains of the smooth pursuit (eye velocity/target velocity) were 0.33 to the right and 0.50 to the left in Patient 1 and 0.17 to the right and 0.22 to the left in Patient 2. The mean gain of normal subjects in our laboratory is 0.91 ± 0.10 (mean ± 1 S.D.).[5] Horizontal saccades in both patients had normal amplitude-velocity relationships and latencies. Comparison of target amplitude and saccade amplitude indicated slight hypometria in both patients, the mean accuracy (target amplitude/saccade amplitude) being 0.82 in Patient 1 and 0.83 in Patient 2. Mean accuracy of normal subjects in our laboratory is 0.88 ± 0.10.[8]

OKN (Tables I and II). After a step change in drum velocity from 0 to 30°/sec, the SCV and amplitude of the optokinetic response

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Table I

	Gain[A]					
	Pursuit	*OKN (constant)*[B]	*OKN (sinusoidal)*	*VOR*[C]	*VOR-fixation*[D]	*VVOR*[E]
Normal subjects[F]	0.91 ± 0.10	0.81 ± 0.09	0.75 ± 0.15	0.42 ± 0.11	—	0.92 ± 0.10
Patient 1:						
Right	0.33	0.52	0.49	0.82	0.50	0.99
Left	0.50	0.68	0.39	0.76	0.53	0.95
Patient 2:						
Right	0.17	0.84	0.12	1.22	0.79	1.10
Left	0.22	0.59	0.23	0.89	0.64	0.99

[A]Gain = peak SCV or pursuit velocity/peak stimulus velocity.
[B]In the patients, the peak SCV was measured after the prolonged build-up.
[C]VOR to rotation in the dark.
[D]In normal subjects, the VOR is completely inhibited by fixation during sinusoidal rotation (0.05 Hz, 30°/sec).
[E]VVOR to rotation in the light within a stationary optokinetic drum.
[F]Mean ± S.D.

Table II. Constant-velocity OKN[A]

	Full field		*Central 10°*	
	SCV (degrees/sec)[B]	*Duration of OKAN (sec)*	*SCV max (degrees/sec)*	*Duration of OKAN (sec)*
Patient 1:				
Right	15.7	15	4.5	0
Left	20.5	20	12.0	12
Patient 2:				
Right	25.3	23	3.0	0
Left	17.7	19	3.8	2

[A]Constant drum velocity of 30°/sec.
[B]Maximum SCV after prolonged build-up.

gradually increased over many seconds in both patients (Fig. 3 and 4). In Patient 1, SCV increased from 1.5° to a plateau of 15.7°/sec after 20 sec during drum rotation to the right and from 3.5° to 20.5°/sec over 11 sec during drum rotation to the left (Fig. 5). In Patient 2, SCV increased from 5.5° to a plateau of 25.3°/sec after 40 sec to the right, and from 2.5° to 17.7°/sec over 25 sec to the left (Fig. 5). OKN gains (maximum SCV/drum velocity) were 0.52 and 0.68 to the right and left, respectively, in Patient 1. In Patient 2, OKN gains were 0.84 and 0.59 to the right and left, respectively. Normal subjects have a mean gain of 0.81 ± 0.09.[9]

Both patients reported an illusion of self-rotation during optokinetic stimulation (circularvection) and for several seconds after stimulation was terminated after 60 sec by turning off the light (post-circularvection, PCV). Both patients had persistence of jerk nystagmus after stimulation was terminated by turning off the light. The SCV of this optokinetic after-nystagmus (OKAN) fell immediately to 70% to 80% of the maximum SCV of the OKN in the light during the first 1 to 2 sec after termination of stimulation and decreased gradually over the next many seconds. In Patient 1, after drum rotation at 30°/sec for 60 sec to the right, OKAN persisted for 15 sec, and after drum rotation to the left, it persisted for 20 sec. In Patient 2, OKAN persisted for 23 sec to the right and 19 sec to the left.

Constriction of the visual fields to the central 10° by a tube placed over the subjects' eyes markedly diminished the build-up of SCV and decreased the final OKN gain in both patients. OKAN was also decreased by constriction of the visual fields. OKN gain

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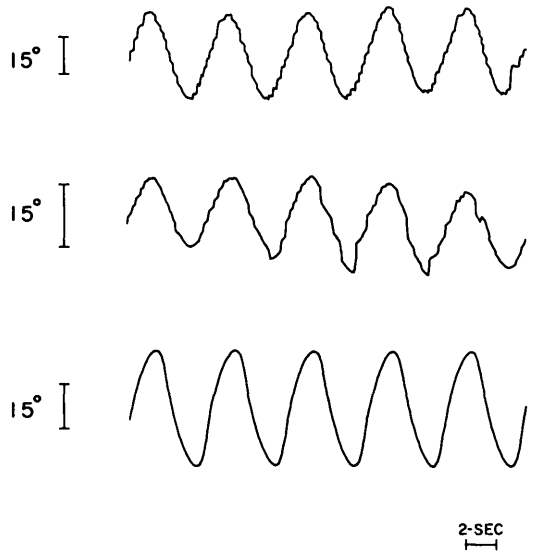

Fig. 2. Smooth pursuit. Deflections upward are to the right; deflections downward are to the left. *Top*, Patient 2; *middle*, Patient 1; *bottom*, target moving horizontally in sinusoidal pattern (0.2 Hz, 25°/sec). Note numerous catch-up saccades required to track the target.

(peak eye velocity/drum velocity) during sinusoidal drum rotation (0.05 Hz, 30°/sec) was reduced in both patients. OKN gains in Patient 1 were 0.49 during drum rotation to the right and 0.39 during rotation to the left. OKN gains were 0.12 to the right and 0.23 to the left in Patient 2 (Fig. 6). Normal subjects have a mean gain of 0.75 ± 0.15.[9]

VOR (Table I). The VOR was abnormally increased in both patients during rotation in the dark. In Patient 1, the VOR gains (eye velocity/chair velocity) were 0.82 during rotation to the right and 0.76 during rotation to the left. In Patient 2, the VOR gains were 1.22 to the right and 0.89 to the left. In normal subjects the VOR gain was 0.42 ± 0.11 during similar sinusoidal rotation (0.05 Hz, 30°/sec).[9] During simultaneous rotation of the chair and optokinetic drum (0.05 Hz, 30°/sec) in the light, the VOR is normally completely inhibited. However, the VOR could not be inhibited in either patient. In Patient 1, the gains of the VOR with fixation were 0.50 during rotation to the right and 0.53 during rotation to the left. In Patient 2, the VOR gains with fixation were 0.79 to the right and 0.64 to the left. During synergistic interaction of the VOR and OKN with rotation of the patients within the stationary drum in the light, both patients were able to increase their VOR gain, as do normal subjects. The gains of the visual-vestibulo-ocular response (VVOR) were 0.99 during rotation to the right and 0.95 during rotation to the left in Patient 1 and 1.10 to the right and 0.99 to the left in Patient 2. VVOR gain in normal subjects is 0.92 ± 0.10.[9]

Discussion

The downbeat nystagmus observed in our patients was characteristic of the vertical jerk nystagmus that has been described in other patients with lesions at the level of the craniocervical junction, such as Arnold-Chiari malformation, basilar impression, tumors, demyelinative disorders, and vascular disorders.[10, 11] The fast component of the nystagmus is predominantly downward but is often associated with horizontal components, and the frequency and amplitude of the nystagmus are often greatest in lateral positions of gaze rather than in down gaze. Downbeat nystagmus has been postulated to result from defects within ocular motor pathways in the medulla. However, both of our patients had ocular motor signs that were also characteristic of cerebellar system disorders, such as impaired smooth pursuit, increased VOR gain, inability to suppress the VOR by fixation, and rebound nystagmus.[7, 12–14] Similar ocular motor abnormalities have been described in other patients with downbeat nystagmus,[13, 15] raising the possibility that the vertical nystagmus may also arise from lesions within the cerebellum or its pathways.

A slow build-up of SCV of OKN, as observed in our patients, has not been described in normal human subjects. Gradual build-up of the optokinetic SCV with a long time-constant (over 15 sec) at constant drum speed of 30°/sec has been described in afoveate animals such as the rabbit.[16] Cohen et al.[17] have observed a rapid rise in SCV within the first few seconds after a step increase in drum velocity and a slower, gradual

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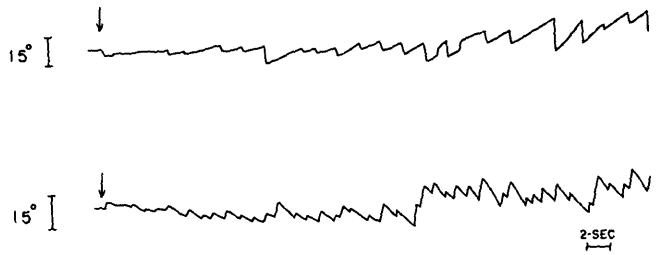

Fig. 3. Slow build-up of OKN, Patient 1. Deflections upward are to the right, and those downward are to the left. Lights were turned on at arrows. Drum rotating at 30°/sec to right *(top)* and to left *(bottom)*.

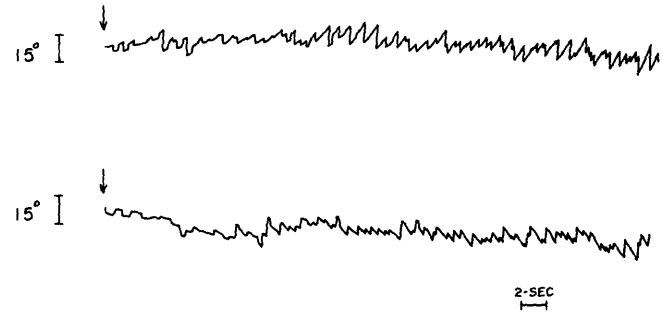

Fig. 4. Slow build-up of OKN. Patient 2. Deflections upward are to the right, and those downward are to the left. Lights were turned on at arrows. Drum rotating at 30°/sec to right *(top)* and to left *(bottom)*.

increase in SCV over the next 10 to 15 sec in normal rhesus monkeys. The time-constant of the gradual increase in SCV was about 3 sec at drum velocities of 60°/sec or less. Similar rapid and delayed increases in SCV have not been observed in normal human subjects in our laboratory or by Cohen (personal communication, June 1978). In humans the optokinetic SCV rises to a maximum within the first few beats of nystagmus after a step increase in drum velocity. We could not demonstrate that the slow increase in SCV in our patients was exponential. However, assuming an exponential function, the time-constant would be over 10 sec.

Zee et al.[18] have postulated that the initial, rapid rise in SCV in primates is due to response by the smooth pursuit system whereas the delayed rise is due to a phylogenetically older, separate optokinetic system. In the afoveate rabbit, an accessory optokinetic pathway that is anatomically separate from the retinogeniculocalcarine pathway has been described.[19] In primates the smooth

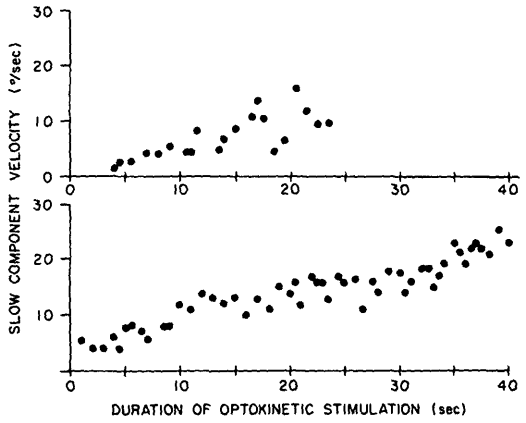

Fig. 5. SCV. *Top*, Patient 1; *bottom*, Patient 2. Note gradual increase in SCV during drum rotation at 30°/sec to the right.

pursuit contribution to OKN utilizes the retinogeniculocalcarine pathways, but other components of the optokinetic response may derive from separate pathways as in the rabbit. In humans, the smooth pursuit system can reach maximum velocity within 133 msec

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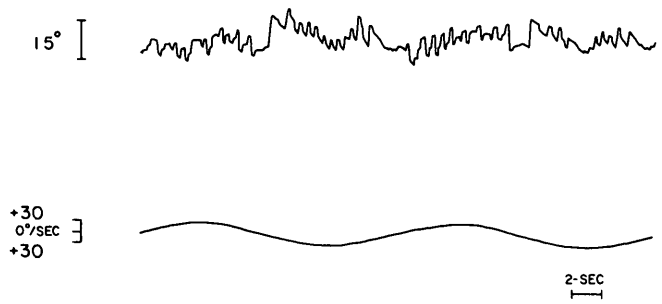

Fig. 6. Sinusoidal OKN, Patient 2. Deflections upward are to the right, and those downward are to the left. *Top,* Eye position; *bottom,* sinusoidal drum velocity (0.05 Hz, 30°/sec). Note poor nystagmus generated.

at target velocities of 5° to 20°/sec and has a gain of almost 1.[20] The smooth pursuit contribution to OKN may dominate the contribution of a separate optokinetic system with a much longer time-constant.

In most patients with supranuclear lesions of the cerebral hemispheres, cerebellum, and brainstem affecting smooth pursuit and OKN whom we have studied in our laboratory, both types of eye movements have been equally impaired, and no striking build-up of optokinetic SCV has been noted with the exception of the two patients described in this report. In these two patients smooth pursuit was markedly impaired, as indicated by the low gain of pursuit eye movements whereas the optokinetic gain after a prolonged build-up during constant velocity drum rotation was almost within the normal range of our laboratory findings. The optokinetic gain during sinusoidal drum rotation was markedly reduced, however. At a frequency of 0.05 Hz, each half-cycle in one direction was only 10 sec in duration. Sufficient time was not available for the slow build-up of SCV in the patients. The abnormally low gain during sinusoidal optokinetic stimulation probably represented the defective smooth pursuit in these patients. Ter Braak et al.[21] documented a slow build-up of OKN and preservation of OKAN in a patient with long-standing cortical blindness during optokinetic drum rotation in one direction. They postulated that subcortical pathways for OKN were partially spared in this patient. Zee et al.[13] also described slow build-up of SCV of OKN and preservation of OKAN in a patient with hereditary cerebellar ataxia. This patient had downbeat nystagmus.

Many authors have described persistence of nystagmus or OKAN and persistence of the illusion of self-motion or PCV after cessation of the optokinetic stimulus as characteristic of the normal optokinetic response.[17, 22, 23] In both of our patients these after-effects were dependent on stimulation of the peripheral visual fields. When stimulation was restricted to the central 10°, the build-up and after-effects were essentially abolished. In normal human subjects, optokinetic SCV[24] and after-effects[23] are also decreased by restriction of stimulation to the central visual fields.

Preservation of OKN and loss of smooth pursuit are not characteristic of patients with downbeat hystagmus in our experience. We have studied eye movements in 15 other patients with downbeat nystagmus. Smooth pursuit and OKN were equally impaired in those patients. Recent observations by Zee et al.[18] suggest that isolated lesions of the cerebellar flocculi can produce marked loss of smooth pursuit, but less severe reduction of OKN. Two rhesus monkeys with complete ablations of the flocculi demonstrated (1) downbeat nystagmus, (2) decreased smooth pursuit gain, (3) rebound nystagmus, (4) increased VOR gain to rotation in the dark, (5) impaired suppression of the VOR by fixation, (6) loss of the initial rapid rise in optokinetic SCV but preservation of the slower increase of SCV to nearly normal levels, and (7) preservation of OKAN. The ocular motor findings were precisely those found in our patients. Takemori and Suzuki[25] described downbeat

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nystagmus and moderately reduced optokinetic SCV in five rhesus monkeys with bilateral lesions of the flocculi. However, no observation on a possible build-up of SCV or OKAN was reported. These observations in the monkey and our observations in two patients suggest that the ocular motor response to rotation of the visual environment consist of contributions from the smooth pursuit system and from a separate optokinetic system and that the cerebellar flocculi are significantly more involved in the smooth pursuit response than in the optokinetic response. The optokinetic system can, perhaps, be studied in relative isolation from the smooth pursuit system only in animals and patients with lesions of the flocculi.

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