

9:30

HEAD-LED GAZE SHIFTS BETWEEN NEARBY TARGETS ARE ACCURATE. R. L. Steinman, E. Collewijn, E. Kowler, C. Erkelens, E. Pelsig, & J. van der Steen. Dept. Psychol., Univ. Md.; Dept. Physiol. I., Erasmus Univ.-Rotterdam; & Dept. Psychol., Rutgers Univ., NJ

We made accurate measurements of binocular eye and head movements during conjugate and disjunctive gaze shifts between targets located within arm's reach. Head and eye rotations were recorded to 1° at 488 Hz with the Univ. Md. revolving field monitor; head translations to 1 mm with an acoustic ranging device. We found that:  
(1) Gaze shifts with the head free were very accurate, a surprising outcome because with nearby targets, translational movements of the eye (produced by head translations or rotations) displace the target on the retina and must be taken into account;  
(2) Gaze shifts with the head restrained often undershot the target, as if eye movements were programmed on the basis of planned (but unexecuted) head movements;  
(3) Saccade dynamics (peak gaze velocity, acceleration and deceleration) were faster with the head free than the head restrained. Differences between head-free and head-restrained gaze velocities were too large to be explained by the addition of the velocity of the eye/head and head/space. High peak gaze velocities were found when head movements were modest and when Ss tried to keep the head in place without aid of a biteboard, showing that artificial head restraints prevent normal saccadic performance. Our results show that gaze control is far more sophisticated than assumed in prior models based on rotational signals only. Gaze shifts are guided by a representation of target position in 3D space, which is updated continuously using signals coding head rotation, translation and target distance.

Support: AFOSR 88-0171 & 91-00555; MEDIGON 900-550-092

1735 — 9:45

SACCADE LATENCY CHANGES AFTER LESIONS OF THE FRONTAL EYE FIELDS AND POSTERIOR EYE FIELDS IN MONKEYS.

James C. Lynch, Junru Tian, and John S. Touliatos, Departments of Anatomy and Ophthalmology, University of Mississippi Medical Center, Jackson, MS.

The frontal eye fields (FEF) and the posterior eye fields (PEF) both contribute to the initiation and control of visually evoked saccadic eye movements. However, cortical lesions restricted to either area alone produce only modest saccade deficits. In order to study the possible interaction between the FEF and the PEF in the control of saccades, four rhesus monkeys were trained to perform horizontal and vertical saccades of 8° to 24° amplitude to small (0.2°) visual targets. Latency and accuracy were measured before and after bilateral FEF lesions and then after the addition of bilateral PEF lesions in 3 monkeys. Bilateral PEF lesions were followed by a unilateral FEF lesion in one monkey.

In 2 monkeys, mean saccade latencies for the 5 days following FEF lesions were not different from means for the 5 days before the lesions. In a third, in which the lesions included not only the anterior bank of the arcuate sulcus (as in the first two monkeys) but also both banks of the principal sulcus, latency increased by 60%. After combined FEF and PEF lesions, saccade latencies increased by 75% to 170% of the pre-lesion values. The monkey in which a unilateral arcuate bank lesion was combined with bilateral PEF lesions had normal latencies for saccades into the visual hemifield ipsilateral to the FEF lesion. Virtually no saccades were made into the contralateral hemifield for 7 days following the FEF lesion, and for the next 12 days, contralateral saccade latencies averaged 165% of ipsilateral saccade latencies. These results suggest that the FEF and the PEF normally work cooperatively to control visually guided saccades, and that either area alone can mediate relatively normal saccades. However, the loss of both areas produces significant saccadic impairment.

Supported by NIH EY04159

1736 — 10:00

COMPARISON OF LATENCIES OF SACCADES ELICITED DURING FIXATION AND SMOOTH PURSUIT. Richard C. Frecker, M.D., Ph.D., W. James MacLean, M.A.Sc., M. Eizenman, Ph.D., Ronald S. Collin, M.A., Visual Sciences Laboratory, Institute of Biomedical Engineering, University of Toronto, Toronto, Canada. M5S 1A4.

The literature suggests that when the oculomotor system is engaged in fixation prior to making a saccade, that saccadic latencies are longer than when the fixation is disengaged prior to the saccade being elicited (express saccades elicited by target blanking). We conducted the present investigation to examine the effects on saccadic latency of prior engagement in a smooth pursuit task.

Eight healthy subjects (4 male, 4 female) aged 24-49 were tested in a 2-dimensional, 1-kHz, corneal reflection eye tracker. The stimulus was a 1 mm green dot of constant, low intensity, presented on an oscilloscope placed 25 cm from the cornea. A total of 6,000 saccades were recorded, under three conditions: (normal) random target excursions (from fixation in the primary position), within a randomized 1-2 S time interval, of ±5°, to a new horizontal (H) or vertical (V) position (left/right; up/down); (blanked) the same, except that the visual target was extinguished for 100 ms immediately prior to the appearance of the new target; and, (pursuit) randomly elicited ±5° saccades to a new H or V position occurring from a H or V smoothly-pursued target moving at 3°/s.

A significant (p < .05) decrease in latency (50 ms) compared to normal was observed with target blanking. This is consistent with literature reports. A significant (p < .05) increase in latency compared to normal (20-35 ms) was seen for subjects engaged in prior pursuit. H and V saccades elicited during both H and V pursuit (4 conditions) revealed inter-individual differences, but group data showed a significant increase in saccadic latency for all conditions. Four subjects showed significant increases for all 4 conditions; the remaining 4 subjects showed significant increases for at least 2 of the 4 conditions. No subjects showed a decrease in latency for any condition. The decreased latency with target blanking suggests the possibility that overhead exists in fixation disengagement prior to generation of a saccade; our pursuit data may indicate a similar phenomenon related to pursuit disengagement.

1737 — 10:15

MOTION DETECTION DEFICITS REVEALED BY STEP RAMP TRACKING ERRORS IN MONKEYS WITH MAGNOCELLULAR LGN LESIONS. W.K. Page, W.M. King, W.H. Merigan, and J.H.R. Maunsell, University of Rochester Medical Center & Center for Visual Science, Rochester, NY 14642.

Cortical processing of retinal information utilizes two parallel pathways. These pathways are anatomically separated in the parvocellular and magnocellular layers of the primate lateral geniculate nucleus (LGN). Cortical areas MT and MST, which receive most of their retinal input via the magnocellular pathway, are involved in processing retinal image motion and provide inputs to the oculomotor system for ocular tracking. To test the contribution of the magnocellular pathway to ocular tracking of moving targets, we made unilateral ibotenic acid lesions of magnocellular layer 1 of the LGN in two macaque monkeys trained to pursue small target spots. Monkeys served as their own controls. Targets were viewed monocularly through the eye contralateral to the lesioned LGN, so that retinal images in the temporal field were processed by the lesioned LGN, but images in the nasal field were processed by the intact LGN. Eye movements were monitored using the search coil technique. Ocular tracking was tested several months after the lesions using step ramp target trajectories. Both animals detected and made accurate saccades to stationary targets stepped into either visual field, even when the target spot was extremely dim. The animals accurately pursued targets stepped onto their foveae and ramped temporally or nasally at speeds up to 20 deg/s. In contrast, smooth pursuit was absent until after the occurrence of a saccade for targets placed in the temporal field and ramped away from the fovea at 20 deg/s. Smooth pursuit was initiated, but eye velocity was deficient, for targets stepped into the temporal field and ramped toward the fovea. Pursuit of targets stepped into the nasal field was normal. Monkeys corrected for the direction of target motion in programming the amplitude of catch-up saccades in either field, but in the temporal field the correction was erroneous in magnitude. EY04045 (King), EY05911 (Maunsell) AFOSR890041 (Merigan)

1738 — 10:30

PLAIDS USED TO EVALUATE CORTICAL AND SUBCORTICAL INVOLVEMENT IN HUMAN OPTOKINETIC NYSTAGMUS (OKN)

L.R. Harris, T.L. Lewis and D. Maurer, York University, Toronto & McMaster University, Hamilton, Canada.

Young infants, unlike normal adults, show little or no OKN to monocular temporalward motion, a finding which suggests that OKN is mediated primarily by crossed subcortical pathways in early infancy (Lewis et al., 1989). Here we evaluate the subcortical pathways' contribution to adults' OKN by using a method similar to that used previously with cats (Harris & Smith, ARVO, 1990). Five normal adults viewed Type II plaids (Ferrara & Wilson, 1990) composed of two overlapping drifting gratings differing in orientation by 15 deg. The direction of coherent motion was 45 deg from the nearest component. Physiological evidence indicates that under monocular viewing, nasalward coherent motion optimizes any crossed subcortical contribution while temporalward coherent motion minimizes it. We recorded horizontal eye movement by infra-red reflection and asked subjects to report the perceived direction of motion.

During binocular viewing the direction of the slow phase of OKN fell between the direction of movement of the components and that of the coherent motion (16 ± 5 deg from the direction of the coherent motion). However, monocular viewing did not affect the direction of OKN, a finding which suggests little or no involvement of subcortical mechanisms in the generation of adult humans' OKN. The mean correlation between horizontal eye velocity and the size of the direction of verbal judgements was only 0.65 (0.91 for single gratings). This suggests that the pathways mediating OKN and those mediating the perception of plaids' movement may not be identical.

Supported by NIH EY03475 and NSERC (Canada) OGP0046271

1739 — 10:45

SPONTANEOUS NYSTAGMUS AND GAZE-HOLDING ABILITY IN MONKEYS FOLLOWING INTRAVITREAL PICROTOXIN INJECTIONS. Ronald J. Tusa & Michael Ariel. Depts of Neurology & Ophthalmology, Johns Hopkins Univ, Baltimore, MD 21205, Depts of Behavioral Neuroscience & Psychiatry, Univ of Pittsburgh, Pittsburgh, PA 15260.

GABA is a major inhibitory neurotransmitter in the primate retina, yet virtually nothing is known about the functional role of GABA in the monkey retina. We measured movements of both eyes using search coils in 3 rhesus monkeys following monocular intravitreal injections of a GABA antagonist (75-150 µL of 3.3 mM picrotoxin). Spontaneous nystagmus, optokinetic nystagmus (OKN), and smooth pursuit movements were measured.

Within 20 mins of the injection, a sustained conjugate spontaneous nystagmus developed in the dark, with the slow phase movement in the temporal-to-nasal direction with respect to the injected eye. The duration of the nystagmus and the peak slow phase velocity increased with the dose of picrotoxin (1/4- > 2 hrs, 15-43°/s). In a well-lit room, the nystagmus was completely suppressed, even during monocular viewing through the injected eye. When the lights were turned out, the nystagmus slowly increased to a steady-state value over a couple of minutes. Smooth pursuit eye movements to a small target moving in a triangular waveform (15-60°/s) and OKN to constant velocity drum rotations (5-180°/s), both monocularly and binocularly, were unchanged from pre-injection values. For OKN, both the initial pursuit and the velocity-storage components (OKAN) were measured.

This development of nystagmus in the dark after retinal GABA blockade is similar to that described in the turtle, rabbit and cat, with the slow phase in the temporal-to-nasal direction (Ariel et al., J Neurophys 60:1022-35, 1988). This pattern of nystagmus also resembles the nystagmus evoked by electrical stimulation of the nucleus of the optic tract (contralateral to the picrotoxin eye injection) (Schiff et al., Exp Brain Res 70:1-14, 1988; Mustari and Fuchs, J Neurophys 64:77-90, 1990). Intravitreal picrotoxin may increase spontaneous activity of a selective population of retinal inputs to the contralateral pretectum. Picrotoxin injections in other species also impair motion-dependent visual tracking and gaze-holding systems, which is thought to be mediated by disruption of direction-sensitive retinal ganglion cells. This was not found in the monkey. Supported by EY05978 & K02MH00815 (MA), NS-26988 & EY01765 (RJT).

1734 — 9:30

FREE-HEADED GAZE SHIFTS BETWEEN NEARBY TARGETS ARE ACCURATE. R. SEIBERMAN, R. Collewijn, E. Kozlark, G. Ekelund, E. Fiala, & J. Van der Stoep. Dept. Psychol., Univ. Md.; Dept. Physiol., Rutgers Univ.-Rutgers; & Dept. Psychol., Rutgers Univ., NJ

We made accurate measurements of binocular eye and head movements during conjugate and disjunctive gaze shifts between targets located within arm's reach. Head and eye rotations were recorded to 1° at 480 Hz with the Univ. Md. revolving field monitor; head translations to 1 mm with an acoustic ranging device. We found that:

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