Saccade-related activity in the fastigial oculomotor region of the macaque monkey during spontaneous eye movements in light and darkness

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Abstract Saccade-related burst neurons were recorded in the caudal part of the fastigial nucleus (fastigial oculomotor region) during spontaneous eye movements and fast phases of optokinetic and vestibular nystagmus in light and darkness from three macaque monkeys. All neurons (n = 47) were spontaneously active and exhibited a burst of activity with each saccade and fast phase of nystagmus. Most neurons (n = 31) only exhibited a burst of activity, whereas those remaining also exhibited a pause in firing rate before or after the burst. Burst parameters varied considerably for similar saccades. For horizontal saccades all neurons, except for three, had a preferred direction with an earlier onset of burst activity to the contralateral side. For contralateral saccades the burst started on average 17.5 ms before saccade onset, whereas the average lead-time for ipsilateral saccades was only 6.5 ms. Three neurons were classified as isotropic with similar latencies and peak burst activity in all directions. None of the neurons had a preferred direction with an earlier onset of burst activity to the ipsilateral side. Burst duration increased with saccade amplitude, whereas peak burst activity was not correlated with amplitude. There was no relationship between peak burst activity and peak eye velocity. In the dark, neurons generally continued to burst with each saccade and fast phase of nystagmus. Burst for saccades in the dark was compared with burst for saccades of similar amplitude and direction in the light. Saccades in the dark had a longer duration and peak burst activity was reduced on average to 62% (range 36–105%). In three neurons a burst in the dark was no longer clearly distinguishable above the ongoing spontaneous activity.

Key words Saccades · Fastigial oculomotor region · Single unit activity · Light and darkness · Monkey

Introduction

Lesions of the cerebellum lead to a multitude of oculomotor disturbances (review, Leigh and Zee 1991). For saccades it is generally accepted that the cerebellum is not essential for their initiation. Even after large lesions saccades can be performed in all directions. However, saccadic inaccuracy results in saccadic dysmetria, often in the form of step-size error dysmetria. With this type of dysmetria, the saccade does not bring the eye on target and a corrective saccade is necessary. Step-size error dysmetria is found following lesions of the oculomotor vermis (lobulus VI, VII) and the underlying deep cerebellar nuclei (Optican and Robinson 1980; Leigh and Zee 1991).

Anatomical (Yamada and Noda 1987) and physiological (Noda and Fujikado 1987) studies have demonstrated that the oculomotor vermis in the monkey projects almost exclusively to the caudal fastigial nucleus, which has consequently been called the fastigial oculomotor region (FOR). Neurons in the FOR are modulated with eye movements in the form of either saccades (Ohtsuka and Noda 1991) or smooth-pursuit eye movements (Büttner et al. 1991).

A direction-specific involvement of the FOR in eye movement control has been shown by local microinjections of GABAergic substances. GABA is known to be a transmitter of Purkinje cells (PCs; Ito 1984), which provide the input from the oculomotor vermis to the FOR. Unilateral bicuculline injections (a GABA antag-
onist) into the FOR lead to hypermetric saccades to the contralateral side and hypometric saccades to the ipsilateral side (Sato and Noda 1992). Similarly, muscimol (a GABA agonist) leads to a marked reduction in the smooth pursuit-related component of the optokinetic nystagmus (OKN; “direct component”) towards the contralateral side (Kurzan et al. 1993).

The analysis of saccade-related activity in FOR during visually guided saccades also reveals a directional preference. Neurons exhibit a burst of activity with all saccades, but the vast majority of neurons show a contralateral preference with regard to the onset of the saccade burst (Ohtsuka and Noda 1991; Fuchs et al. 1993). It is also known that the burst (burst duration) increases with saccade duration. However, it is not quite clear exactly which saccadic parameters are encoded by saccade-related FOR neurons. Acceleration and deceleration signals have been suggested (Hepp et al. 1982; Fuchs et al. 1993). A specific involvement of converging visual information to the saccadic system has also been proposed (Ohtsuka and Noda 1992).

In view of these disparate conclusions, we decided to investigate this question further and compared saccade-related activity in the FOR during spontaneous eye movements in light and darkness. In contrast to visually guided saccades, which have a constant amplitude-duration relationship, the recording of saccades in light and darkness provides two samples with different amplitude-duration relationship and velocity profiles (Robinson 1981; Bon and Lucchetti 1988). It is known that, under identical conditions (e.g., light), saccade amplitude is directly related to saccade duration and saccade velocity, and that individual values fall within a relatively limited range, i.e., they are in the “main sequence.” Eye velocity is also directly related to eye acceleration (Bahill et al. 1981). Thus, it might be difficult to relate neuronal activity changes to one or the other eye movement parameters. With the two different samples of saccades in light and in darkness, more specific relations to saccadic parameters might be obtained.

Using quantitative criteria it will be shown that FOR neurons could modify saccadic profiles by directly influencing acceleration or deceleration of individual eye movements. This mode of action influences the saccade amplitude-duration relationship and is independent of visual tasks.

It is known that FOR saccade-related activity varies considerably, even for identical saccades (Fuchs et al. 1993). Thus, the activity of many identical or almost identical saccades has to be averaged for quantitative comparison. Since directional aspects were not the aim of our study and have been investigated elsewhere (Ohtsuka and Noda 1991), our analysis will concentrate on horizontal ipsi- and contraversive eye movements.

Materials and methods

Three monkeys (one Macaca mulatta and two M. fascicularis) were used for this study. Prior to the experiments, bolts were attached to the skull in order to secure the head during experiments. Horizontal and vertical eye positions were recorded by the scleral search coil method (Judge et al. 1980). Under aseptic conditions, all animals were implanted with recording chambers for single unit recordings. For calibrating horizontal eye movements, the vestibulo-ocular reflex (VOR) gain in the light at 0.2 Hz, ±40°/s was set equal to 1. For vertical eye movements the relationship between saccade amplitude and duration in the light was used (King et al. 1986). The midposition of the eye was determined by repeatedly attracting the monkey’s attention to look at defined fixation points. One monkey was trained to fixate visual targets at different horizontal and vertical positions. The calibration obtained with this method yielded very similar results.

Neural activity was recorded with varnished tungsten microelectrodes (for details see Boyle et al. 1985). The FOR was approached by vertical penetrations in the stereotactic plane. During the experiments the monkeys sat upright in a primate chair, with their heads fixed to the chair, which was on a servo-controlled vestibular turntable surrounded by an optokinetic cylinder (Toennies, Würzburg). Spontaneous eye movements were recorded in light and darkness. In addition, saccades were sampled during sinusoidal vestibular stimulation (0.2 Hz, ±40°/s) in the dark and light as well as during constant velocity optokinetic stimulation (60°/s) and optokinetic after-nystagmus (OKAN), i.e., the nystagmus which continues in the dark after optokinetic stimulation. The latter stimulation proved to be useful for generating a large number of mainly horizontal saccades of similar size in one direction in light and darkness.

Neuronal activity, horizontal and vertical eye position, light-on and -off signal, and all stimulus parameters were stored on a seven-channel FM magnetic tape (Teac XR 310). For inspection, data were plotted on an electrostatic multichannel printer (Gould) with a bandwidth of 0–3 kHz. A digital impulse rate meter was used to display instantaneous spike frequency (Fig. 1; Eckmiller and Petsch 1975). These frequency measurements were used for display only and not for quantitative analysis.

For quantitative analysis, an interactive computer program was used, in which horizontal and vertical eye position as well as neuronal activity were digitized at 2000 Hz, the latter having been transformed into pulses of 0.6 ms by a Schmitt trigger. The program prevented double-counting of one impulse. Data were displayed on a personal computer. Digitalized data were displayed on a personal computer. A computer program automatically identified the beginning and end of the horizontal and vertical saccade component. When necessary, the computer operator could correct the computer decision. The following parameters were used for further analysis: amplitude, direction, duration, peak velocity, time of peak velocity for each saccade component, eye position before and after each saccade and time of occurrence for individual neuronal events. Subsequently, saccades with similar features (direction, amplitude, duration, etc.) were grouped and the neuronal activity was related to certain features of the saccades (i.e., beginning and end of saccades). Neuronal activity of 15–150 saccades was averaged with a binwidth which could be varied between 3 and 10 ms. These data were displayed as time histograms. Another program was able to relate features of neuronal activity (i.e., peak burst activity, number of spikes per saccade) to individual saccadic parameters. These data were displayed as x–y plots. To compare sufficient numbers of horizontal saccades of similar amplitude and direction in light and darkness, spontaneous saccades were taken, the directions of which were within ±15° ±45° of the horizontal plane, depending on the number of saccades for a given neuron. Samples of similar mean amplitude were used for the comparison of contralateral and ipsilateral saccades.

Since all saccade-related FOR neurons were spontaneously active with an irregular discharge rate, onset of saccade-related burst activity was often difficult to determine for individual saccades. Latency of saccade-related burst activity was determined from the computer display of individual saccades by manually adjusting a cursor to the first interspike interval, which was shorter than those of the on-going discharge and which was followed by still shorter intervals. Similarly, the end of the saccadic burst