Temporal Variations of Visual Evoked Potentials in Structures of the Visual Analyzer in Different Phases of Saccadic Eye Movements in Cats

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Evoked potentials of the superior colliculus, pulvinar, lateral geniculate body, and striate cortex in response to a short flash of light delivered in a homogeneous visual field were studied in cats with a rigidly fixed head during gaze holding, before a saccade, and at different phases of saccadic eye movements to an angle of 20°. It is shown that proprioceptive afferent impulses from external ocular muscles participate along with the efferent copy in visual suppression during the saccade, and that the collicular and pulvinar structures are mainly responsible for the suppression.

Key Words: *visual evoked potentials; eye movements; saccadic suppression; efferent copy; proprioception from external ocular muscles*

Knowledge of the mechanisms by which visual (saccadic) suppression occurs is essential for understanding how the visual and oculomotor systems work. There have been shown to be both retinal and extraretinal components or mechanisms of visual suppression during eye movements. After the elimination of retinal components (such as masking or blurring of the image on the retina) through short-term stimulation of the eyes in a homogeneous visual field, it becomes possible to study the less powerful extraretinal mechanisms of suppression (the efferent copy and proprioception from external ocular muscles). Most investigators tend to ignore the role of proprioception in the act of visual suppression, concentrating on the efferent copy instead [9], especially because vision may be suppressed even before the eyes begin to move [14]. However, since visual suppression may also occur during passive eye movements [8], proprioception may well play a role in it [5]. Thus, the question of just how visual suppression is effected by extraretinal mechanisms remains unresolved. Some idea may be gained by following, as in the present study, variations of visual evoked potentials over time during different phases of saccadic eye movements, which was not done in any of the studies published by other workers.

MATERIALS AND METHODS

The study was carried out on 13 cats with electrodes of Nichrome wire 0.2 mm in diameter stereotaxically implanted [7] under Nembutal anesthesia (35 mg/kg) into brain structures for behavioral tests. These cats, with the head rigidly but painlessly fixed, were taught to perform voluntary centrifugal saccades to an angle of 20° after the central target (a light-emitting diode) was extinguished and the peripheral target (a similar diode) was lighted.

Evoked potentials (EP) and electro-oculograms were recorded with UBF4-03 amplifiers (pass band 150 Hz, time constants 0.05 and 1.2 sec) and the information obtained was stored and processed on an M6000 computer.

Photostimulation with a flash of light (diffuse, total, and binocular) during the swift oculomotor

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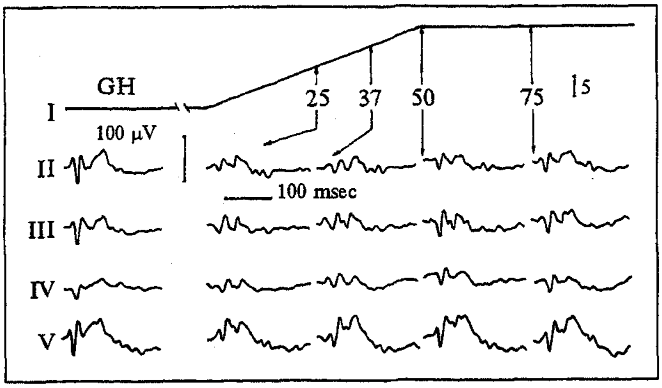

Fig. 1. EP (averaged for 25 stimulus presentations) from visual centers in response to a short diffuse flash during gaze holding, at different phases of the leading edge of a saccade to 20°, and after completion of the saccade. *I*) idealized electro-oculogram reflecting gaze holding (*GH*) and saccade; *II*) superior colliculus; *III*) lateral geniculate body; *IV*) pulvinar; *V*) visual (striate) cortex (all four structures being ipsilateral to the direction of the left-sided saccade). Numbers indicate delays (in msec) in flash delivery relative to the beginning of the saccade. Origins of the curves correspond to the moment of flash delivery (shown by arrows). During saccadic eye movements, the flash lamp was positioned at 10° (corresponding to a delay of 25 msec), 15° (37 msec), and 20° (50 and 75 msec). Homogeneous visual background; light adaptation 10 luxes. Positivity: downward.

act was also controlled by the M6000 computer, which actuated a photostimulator (flash duration 50 μsec, light intensity 0.3 J) during gaze holding (fixation) as well as before saccadic eye movements (SEM) and in different phases of these movements. Because a short flash and a homogeneous visual background (10-20 luxes) were used, the retinal image during SEM was not blurred so that only the extraretinal mechanisms of visual suppression could be studied. The procedure is described in detail in another article [1]. The results were treated statistically by Student's *t* test.

RESULTS

In this study, EP from the major visual centers - the lateral geniculate body (LGB), superior colliculus, pulvinar, and visual (striate) cortex - were examined.

As we showed previously, EP of the visual tract in the course of saccades differ little and insignificantly from those during gaze holding if the visual field is homogeneous [1]. This indicates that virtually identical visual signals arrive at their target structures during SEM and gaze holding; hence, the changes observed in EP may be attributed solely to extraretinal influences.

Figure 1 depicts temporal variations in the EP recorded from visual structures ipsilateral to the direction of SEM during gaze holding, at different phases of SEM to 20°, and after completion of the saccade. It can be seen that the shapes of EP from the structures under study undergo transformation. Thus, the EP waves become "split," indicating that the visual afferent input was modulated by an afferent input of another modality.

In addition, as shown in Fig. 1, the greatest change in EP as compared to their control values (i.e., during gaze holding) occurred at 25 msec after the start of SEM ($p<0.01$). Near the end of SEM (the delay of 50 msec in delivering the flash), EP in response to the stimulus clearly increased and were no longer split. However, EP were not fully restored even with the delay of 75 msec; their complete restoration was only observed somewhere near 100 msec or later after the SEM were completed. This temporal pattern of EP is similar to that reported for restoration of firing by superior collicular neurons in different saccadic phases in monkeys [12].

Figure 2, *b* shows temporal variations in the positive component of EP from structures ipsilateral to the direction of SEM before SEM, in different phases of SEM to 20°, and after the gaze was fixed. It can be seen that, qualitatively speaking, the visual structures all functioned in the same manner. The suppression curve is asymmetrical (U-shaped) and resembles the suppression curves plotted in psychophysical tests [14]. In these tests, the maximal suppression of visual sensitivity occurred 20-60 msec before the saccade - in contrast to the situation in neurophysiological tests, where it is observed after rather than before the start of a saccade. Suppression of the positive wave is maximal (minima on the plots) with delays of 12-15 msec in stimulus delivery (which correspond to phases 5-10°) before the beginning of SEM, after which all the curves rise to the level of 1.0, i.e., the positive wave becomes the same as it was during gaze holding. Complete wave restoration was most often observed 75-100 msec or even later after the gaze was fixed.

Suppression curves for the negative wave have the same shapes as for the positive (Fig. 3, *b*), but the suppression maximum is shallower. The suppression curves obtained for SEM to 10, 15, and 25° were similar to those shown for SEM to 20°.

Different patterns of suppression curves were observed for the LGB and visual cortex contralateral to the direction of SEM. Neither the positive nor the negative components of EP from the LGB were greatly suppressed (Fig. 2, *a* and Fig. 3, *a*). Both EP components were also less suppressed in the contralateral than in the ipsilateral visual cortex. In contrast, the degree of suppression in the superior colliculus and pulvinar contralateral to the direction of SEM was approximately the same as in the ipsilateral structures.

It should be noted that suppression of EP in the LGB and visual cortex ipsilateral to the saccade's direction only occurred in about one-third of the tests, whereas EP in the contralateral LGB and visual cortex were suppressed little or not at all in the large majority of tests.

Two important results emerge from this study: first, little or no suppression of EP close to the beginning of a saccade and their strong (maximal) suppression after its start; and, second, bilateral EP suppression in the superior colliculus and pulvinar, i.e., in the structures implicated in the "second visual system."

The first of these results may be accounted for by the addition of extraocular proprioception to the efferent copy. Indeed, after a saccade has started, the action of the efferent copy is no longer enhanced, having reached its maximum at the moment the saccade begins. This statement is supported by the following considerations. Regardless of how an oculomotor command (and hence its efferent copy) is organized in time and space, at the start of a saccade the command must provide a peak force for distending the ocular muscles in order to overcome the inertia of the eyeball. The acceleration of the latter's movement is also maximal at this time. The larger the saccade, the greater is the initial mechanical force [10]. This force then decreases to a value necessary to overcome the viscid mechanical milieu in which the eyeball rotates. That this is so is also indicated by the established leading edge of the saccade which has a constant velocity in its main part. The number of spikes in discharges of the cells that accomplish the saccade is highest at its beginning, after which their frequency rapidly falls off and then remains uniform [4]. Thus, the initial increase of firing by α-motoneurons at the beginning of a saccade is not followed by further enhancement of oculomotor discharges (and, hence, of the efferent copy), and if visual suppression were caused solely by the efferent copy, then the suppression should be maximal somewhere near the beginning of the saccade. It has been shown that as the saccade increases, so does the suppression, with a shift of its maximum in time to the right relative to the beginning of the saccade, the shift being the greater, the larger the saccade [13]. This assertion is validated by our finding that the suppression of EP reaches its maximum after the saccade has begun. Going back to the lack of concordance between psychophysical and neurophysiological data regarding the time when suppression begins, it is to be noted that the suppression of

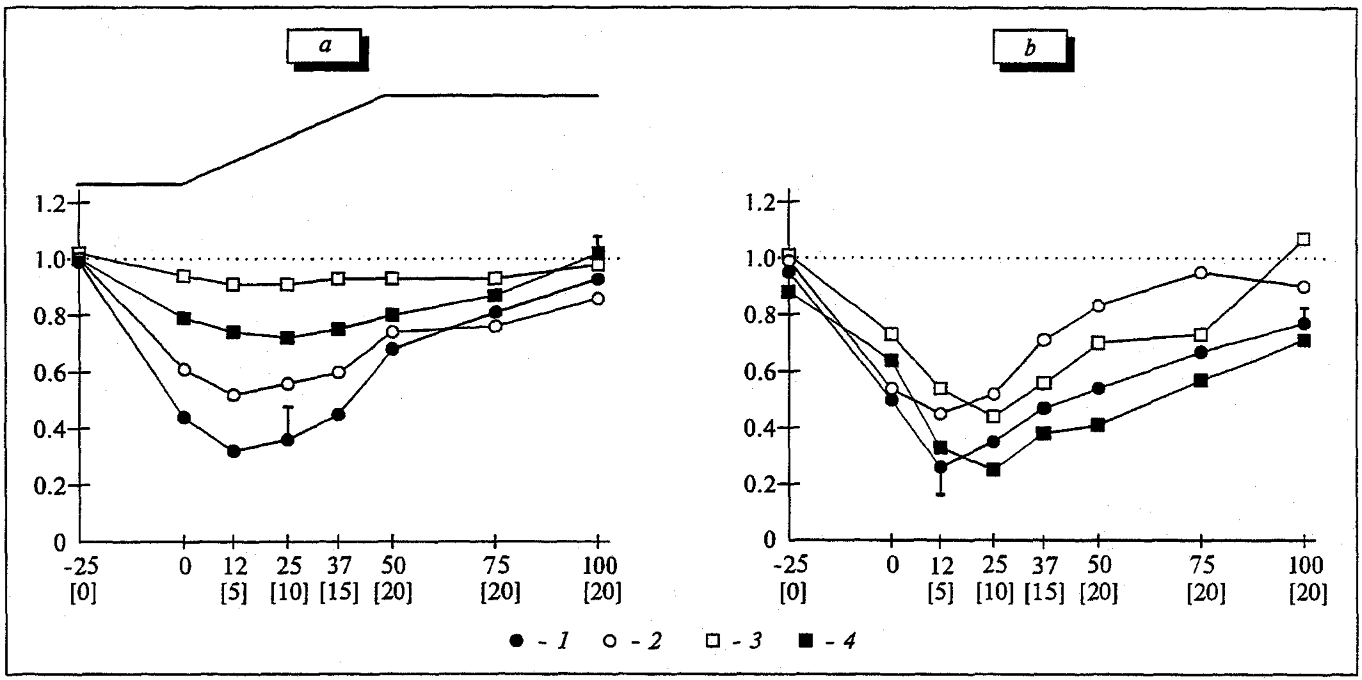

Fig. 2. Temporal variations in the positive component of EP from visual structures before, during, and after a saccade to 20°. *a* and *b*) sides contralateral and ipsilateral to direction of saccade, respectively. *1*) superior colliculus; *2*) pulvinar; *3*) lateral geniculate body; *4*) visual cortex (area 17). Abscissa: time intervals (in msec) between the beginning of a saccade and stimulation; figures in brackets denote the saccade phases (in degrees) when the stimulus was delivered. Ordinate: EP amplitudes before, during, and after saccades, expressed in relative (with respect to the EP amplitudes during gaze—holding) units. The two vertical lines in *a* and *b* indicate errors of the mean as calculated for all points of the curves. The topmost curve represents an idealized saccade.

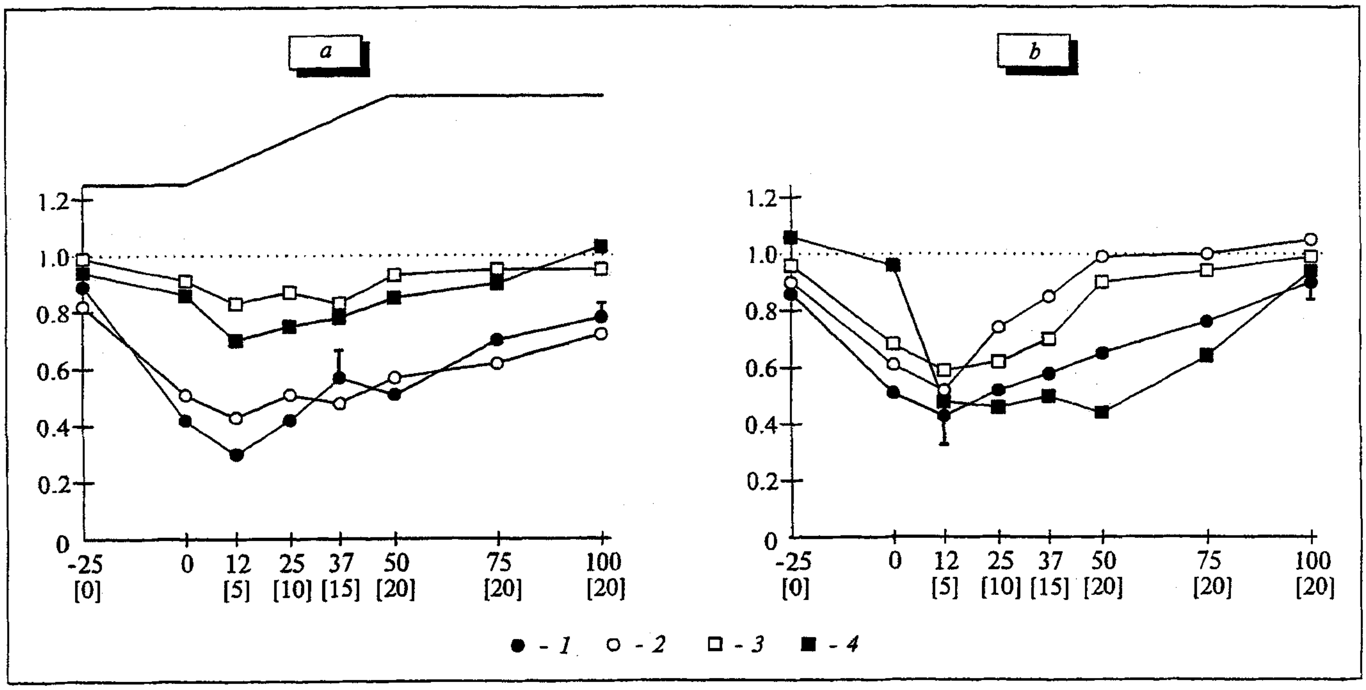

Fig. 3. Temporal variation in the negative component of evoked potentials from visual structures before, during, and after a saccade to 20°. Same designations as in Fig. 2. The topmost curve represents an idealized saccade.

visual function before SEM as identified from verbal reports of subjects may be accounted for by the masking of visual signals by extraretinal and, in the main, proprioceptive signals received by the subject at the stage of visual signal processing before the eyes begin to move.

The second result suggests that the tectothalamic pathways are involved in the organization of saccadic suppression to a greater extent than are the geniculate pathways. For example, both spontaneous and evoked neuronal firing in the superior colliculus and pulvinar were found to be suppressed during SEM in a homogeneous visual field [11,12], whereas no suppression was recorded for the LGB [3,6]. Distension of ocular muscles or passive shifting of the eyes modulates visual impulse discharges [2] - an indication of interactions between the visual and proprioceptive afferent signals passing via the colliculus-pulvinar pathways in the direction of cortical areas.

Taken together, the findings presented above point to the important role the proprioception of external ocular muscles plays in mediating visual suppression.

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