Evoked Potentials of the Lateral Geniculate Body during Saccadic Suppression of Vision in Cats

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Translated from *Byulleten' Eksperimental'noi Biologii i Meditsiny*, Vol. 119, № 2, pp. 155-159, February, 1995
Original article submitted April 11, 1994

Evoked potentials of the lateral geniculate body were examined in cats during gaze holding and horizontal saccades elicited by a short flash against a homogeneous background. Statistical analysis showed that the positive component of evoked potentials recorded during gaze holding from the lateral geniculate body contralateral to the direction of the saccade did not differ from their positive component during saccades, but that the differences between the corresponding negative components were significant ($p<0.01$). Under the same conditions, both the positive and negative components of evoked potentials from the lateral geniculate body ipsilateral to the saccade's direction were found to be significantly suppressed during saccades but not during gaze holding.

Key Words: *lateral geniculate body; evoked potentials; mechanisms of saccadic suppression; eye movements; cats*

The main mechanisms whereby vision is suppressed during eye movements are retinal and extraretinal (the efferent copy and proprioception from the eye muscles) influences that mediate the inhibition of visual perception, i.e., prevent, at the neurophysiological level, the transmission/processing of specific visual signals in the major structures of the visual analyzer [8,11-13].

It has been shown in many studies that when the visual field is nonhomogeneous, vision is suppressed through masking and blurring of the retinal image (i.e., by retinal mechanisms) [4], and this is reflected in the neuronal and evoked activity of the lateral geniculate body (LGB) [3,5].

On the other hand, relatively little is known about the role played by the LGB in suppressing vision when the visual field is homogeneous, so that the retinal mechanisms are inoperative and the suppression is produced by extraretinal mechanisms only [10]. In particular, published information relating the LGB activity to the direction of the saccade is scarce. The present study was designed to bridge this gap.

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MATERIALS AND METHODS

The study was carried out on 13 cats with electrodes of Nichrome wire 0.2 mm in diameter implanted under Nembutal anesthesia (35 mg/kg) into the LGB according to coordinates of a stereotaxic atlas [9]. The alert cats, with the head rigidly and painlessly fixed, were trained to perform voluntary centrifugal saccades. Evoked potentials and electro-oculograms were recorded with type UBF4-03 amplifiers (pass band 150 Hz, time constants 0.05 and 1.2 sec) and were stored and processed by an M6000 computer.

Photostimulation (diffuse, total, and binocular) was also controlled by the M6000 computer, which actuated a type FS-2 photostimulator (flash duration 50 μsec, flash intensity 0.3 J) during gaze holding and saccadic eye movements (SEM). Because a short flash and a homogeneous visual background (10-20 luxes) were used, the retinal image was not blurred so that only extraretinal mechanisms of visual suppression could be studied.

0007-4888/95/0002-0146$12.50

The procedure used is described in greater detail elsewhere [1]. The results were treated statistically using Student's t test.

RESULTS

In this study we examined and compared the main sensory components of evoked potentials (the first positive-negative complex) elicited in the major subcortical visual center, the LGB, during SEM and gaze holding.

Our previous studies demonstrated that in a homogeneous visual field, evoked potentials of the optic tract during SEM, vergences, and blinking do not differ from those during gaze holding [1,2]. This indicates, first, that the optic tract does not convey extraretinal influences and, second, that the afferent inputs to the LGB from the optic tract during SEM and gaze holding are virtually identical. Changes in evoked potentials may therefore be attributed to extraretinal influences only.

In setting about to study LGB evoked potentials during SEM, we expected these potentials to differ greatly from those recorded upon gaze holding because it seems logical to link the suppression of visual function during eye jerks with deteriorated conduction of visual signals through the LGB. Our assumption was borne out only partially, however. Thus, decreased evoked potentials were observed in only a third of the cases and for the most part only in the LGB ipsilateral to the direction of SEM, whereas suppression in the contralateral LGB was slight in the large majority of cases. As illustrated in Fig. 1, solitary evoked potentials recorded during gaze holding and SEM from the LGB contralateral to the direction of the saccade were very stable, although the negative component was decreased (cf. *a, 1* against *b, 1* and *a, 2* against *b, 2* in Fig. 1).

Comparison of the amplitude histograms to the peaks of the respective components on gaze holding and SEM (Fig. 1: *c, 1* against *d, 1* for the positive component and *c, 2* against *d, 2* for the negative component) clearly shows that the amplitudes of the positive component differ insignificantly ($p>0.01$), whereas those of the negative component are very variable ($p<0.01$). The latency histograms indicate that there are no significant changes (cf. *c, 3* against *d, 3* and *c, 4* against *d, 4* in Fig. 1). Reproducibility of the results can be judged from Table 1, which shows statistical data for amplitudes of the evoked potentials recorded at a given point in three out of many tests run at widely spaced intervals (in each test, evoked potentials during gaze holding and SEM were recorded sequentially). It also follows from Table 1 that the differences between the values of only the negative potential are statistically significant. Latencies to the peaks of both components (Table 2) during gaze holding and SEM changed relatively little, and the significant differences between the latencies (observed in half of the cases) are attributable precisely to their great stability and small scatter and to the error of the mean. For example, although the mean latencies to the peak of the positive potential at point 1 differed by only 2.2 msec (Table 2), this difference is significant. Possibly, changes in amplitudes of the negative wave during SEM are due to the fact that this wave reflects the moment at which the gaze becomes fixed when the eye stops moving.

TABLE 1. Amplitudes of Evoked Potential Components Recorded from the LGB Contralateral to the Direction of Saccade during Gaze Holding and SEM, μV ($M\pm m$)

Point number	Evoked potential component	Gaze holding	SEM
1	P	46.4±2.0	44.4±1.1
	N^*	11.6±2.7	−2.4±1.9
2	P	49.5±1.2	44.0±1.8
	N^*	17.4±1.3	−5.4±1.6
3	P	42.8±1.8	45.3±1.2
	N^*	14.8±1.5	−3.4±1.7

Note. Here and in Table 2: P and N are the positive and negative components and the asterisk denotes a significant difference at $p<0.01$.

As regards the evoked potentials of the LGB ipsilateral to the direction of the saccade, they were strongly depressed in a number of cases. On the electroencephalogram, solitary evoked potentials were not clearly marked and appeared appreciably transformed (split), and this is reflected in the averaged evoked potentials (Fig. 2: cf. *a, 1* against *b, 1* and *a, 2* against *b, 2*). Differences between evoked potential amplitudes during gaze holding and SEM are significant (Fig. 2: cf. *c, 1* against

TABLE 2. Latencies to Peaks of Evoked Potential Components Recorded from the LGB Contralateral to the Direction of Saccade during Gaze Holding and SEM, msec ($M\pm m$)

Point number	Evoked potential component	Gaze holding	SEM
1	P^*	52.7±0.3	54.9±0.5
	N	76.2±0.7	78.6±0.4
2	P	55.1±0.1	55.1±0.5
	N	75.1±1.7	74.6±0.8
3	P^*	52.5±0.2	55.2±0.5
	N^*	72.6±0.6	76.3±0.7

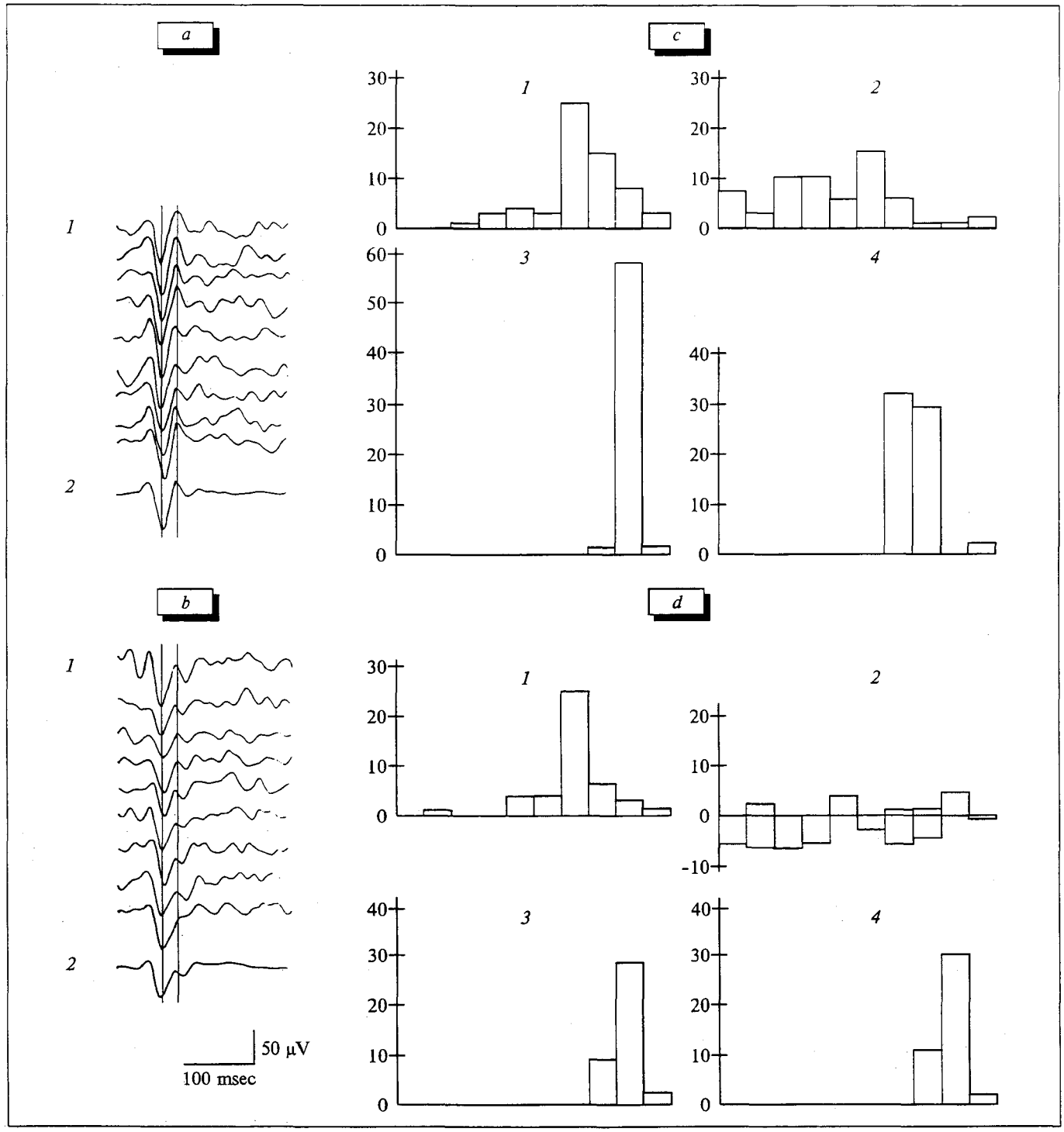

Fig. 1. Evoked potentials recorded from the LGB contralateral to the direction of saccade in response to a short diffuse flash during gaze holding (*a* and *c*) and SEM (*b* and *d*). *a* and *b*) evoked potentials: several solitary potentials (*1*) and an averaged potential (*2*). *c* and *d*) histograms of amplitudes (*1* and *2*) and latencies (*3* and *4*) to peaks of the positive and negative components, respectively, of evoked potentials (relative units). For SEM, the amplitudes and latencies were measured taking into account the vertical lines passing through the peaks of the evoked potential averaged during gaze holding. Origins of the curves coincide with the moment of stimulation. During SEM, stimuli were delivered at phase 10° and saccades at phase 20°. Number of averagings = 20; *n* is the number of measurements: $n=62$ (*c*) and $n=45$ (*d*). A homogeneous visual background. Light adaptation: 10 luxes. Positivity: bars facing downward.

d, 1 and *c, 2* against *d, 2*; $p<0.01$), whereas those between latencies are not (cf. *c, 3* against *d, 3* and *c, 4* against *d, 4* in Fig. 2; $p>0.01$). Suppression of evoked potentials in the LGB under conditions of a homogeneous background has not been previously reported. In studies where changes in evoked

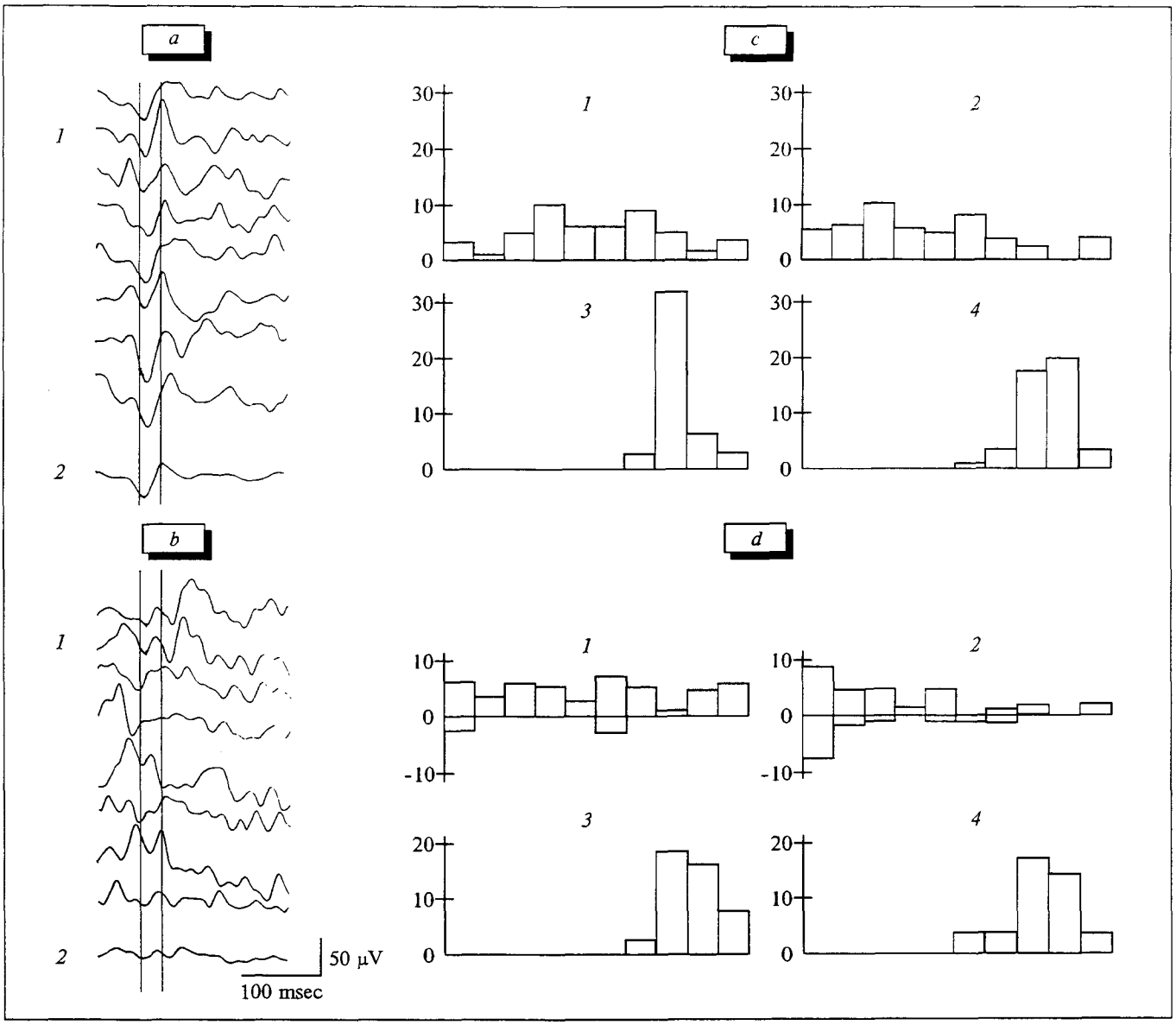

Fig. 2. Evoked potentials recorded from the LGB ipsilateral to the direction of saccade in response to a short diffuse flash during gaze holding and SEM. $n=46$ (*c*) and $n=42$ (*d*) Same designations as in Fig. 1.

responses of the LGB during SEM were examined [3,5,7], such changes may have been recorded on the side contralateral to the direction of SEM. (In these studies, the side of evoked potential recording was not specified.) Responses of LGB neurons were also found to remain unsuppressed in the dark [10]. These findings, as well as our data on evoked potentials of the LGB contralateral to the direction of SEM, support the view [3] that the LGB is not involved in oculomotor integration.

Our earlier studies showed that the contribution of proprioception to saccadic suppression is appreciably greater than that of the efferent copy [2]. Therefore, the cases where evoked potentials of the ipsilateral LGB were obviously reduced can probably be accounted for by the fact that the distension of the external ocular muscles elicits modulation (suppression in particular) of discharges by neurons of the LGB ipsilateral to the side of distension [6]. These neurons are distributed throughout the LGB layers, but quantitative data about such visual proprioceptive neurons are lacking. Nor is there any evaluative information on the number of proprioceptive inputs to the LGB from the lateral and medial rectus muscles. Presumably, suppression in the LGB ipsilateral to SEM is associated with unequal numbers of proprioceptive efferents ascending to the LGB from the medial and lateral rectus muscles (a proprioceptive mechanism) rather than with the power of the motor volley innervating these muscles (a mechanism of efferent copy). Proprioceptive signals may arrive here from the superior colliculi.

Thus, since, as this study showed, evoked potentials in both LGBs were suppressed during saccades insignificantly as compared to their suppression on gaze holding (which agrees with the reported findings), it may be stated with reasonable confidence that the LGB contralateral to the direction of SEM is virtually not implicated in visual suppression when the background is homogeneous as well as in darkness. Our findings provide first-time evidence that the ipsilateral LGB can convey extraretinal influences eliciting visual suppression.

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"Medium Molecules" as Nonspecific Regulators of Phagocytic Activity

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Translated *from Byulleten' Eksperimental'noi Biologii i Meditsiny*, Vol. 119, № 2, pp. 159-162, February, 1995
Original article submitted March 15, 1994

Three identical oligopeptide-containing fractions of so-called "medium molecules," isolated by sequential ultrafiltration and gel chromatography from the blood of 8 intact dogs and 15 dogs with an extensive thermal burn, were examined for their impact on phagocytic cells (neutrophil granulocytes and macrophages) in relation to the molecular-weight distribution of the molecules. The relatively high-polymer fractions of medium molecules, unlike oligomeric fractions, stimulated the phagocytic activity of these cells. Because of their increased polymerism, the fractions of medium molecules from dogs with thermal trauma stimulated phagocytosis to a greater extent than did those from intact animals.

Key Words: *medium molecules; molecular-weight distribution; phagocytic cells*

The early 1920s saw the discovery of a nonspecific phenomenon of accumulation of heterogeneous oligopeptides in body fluids in disease states [10]. Subsequently, these compounds became known under the general name of "medium molecules" (300-5000 D), whose elevated levels in the systemic circulation came to be regarded as a major factor in the development of a universal endogenous intoxication syndrome [6]. This view was based on the observed nonselective membrane-damaging effect of medium molecules (MM) [6]; one of the purported manifestations of this effect

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0007-4888/95/0002-0150$12.50