Lambda potential in the rhesus monkey

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Averaged lambda potentials associated with saccadic eye movements were studied in five rhesus monkeys. The influence of field luminance and of saccade size on the latency/amplitude of potentials recorded transcortically from within primary visual cortex were analyzed. Decrease in the luminance of the stimulus field led to a decrease in the amplitude of the first prominent negative component (N_1) and to a lengthening of the late positive component (P_3). Saccade size had a different influence on the N_1 and P_3 components. N_1 latency from saccade onset changed only slightly with increase in saccade size. P_3 latency, on the other hand, increased significantly when measured from saccade onset, but showed no latency differences with saccade sizes when measured from saccade offset. These results support the conclusion drawn from human studies as well that the N_1 component is related to events at the onset of and during saccades (e.g., suppression), whereas P_3 is linked to saccade offset and reflects information pickup in a new fixation.

Brain potentials associated with visual scanning (lambda waves) have been extensively investigated in humans (Barlow & Ciganek, 1969; Becker, Hoehne, Iwase, & Kornhuber, 1972; Kurtzberg & Vaughan, 1973; Scott & Bickford, 1969; Yagi, 1979), but relatively few studies deal with lambda potentials in animals (Cohen & Feldman, 1971; Rhodes, Lanoir, Saier, & Naquet, 1962; Scott, Lichtenfeld, & Bickford, 1968). Scott et al. (1968) noted that the lambda potentials that appear in monkey and man are similar. They also pointed out that no response over the visual cortex accompanies eye movements performed in darkness in either species. However, questions concerning the functional significance of the different components of the lambda complex has received little attention in animal studies.

In humans, significant observations have been made about the relations among stimulus qualities, saccade characteristics, and the lambda potential. For example, Kurtzberg and Vaughan (1977) and Yagi (1979) showed that the averaged lambda potential ("lambda complex") is made up of a sequence of components, some of which are associated with the onset of the saccade, while others are associated with its offset. These two types of components (called "saccadic" and "fixational" by Kurtzberg & Vaughan) can be distinguished by investigating saccades of different sizes. Although the functional significance of the two classes of components has been interpreted within an extensive theoretical framework (Kurtzberg

& Vaughan, 1977; Yagi, 1979), there nevertheless still remains some disagreement concerning the origin and functions of certain of the components. Recording the lambda complex in primate visual cortex may help to resolve some of these issues.

The aim of the present study was to describe the influence of stimulus field and saccade size on the waveform of the averaged lambda complex recorded over the visual cortex in rhesus monkeys. As in the human studies, we examined the latency characteristics of the different components in relation to the onset and offset of saccades of different sizes, paying special attention to the most prominent negative and positive components (N_1 and P_3 , in Yagi's nomenclature). The behavior of the components was also assessed during saccades performed across visual fields (gratings) of different luminance/contrast values.

METHOD

The experiments were performed on five juvenile rhesus monkeys (*Macaca mulatta*). Under Nembutal anesthesia, platinum black electrode pairs were implanted in the primary visual cortex. The epidural part of the transcortical platinum pair was a 2-mm-diam coil that rested on the dura, and the subcortical reference probe, with a 1.5-mm platinized tip, extended through the center of the coil 5-6 mm into the cortex. The electrodes and the receptacles for immobilizing bars were secured to the skull with dental acrylic. The animals were allowed to recover for 14-21 days before recording was started.

During the experimental session, the monkey, with its head restrained, sat in a primate chair facing a tangent screen 70 cm in front of it. Eye movements were recorded by electrooculography (EOG) using dc amplifiers. Beckman nonpolarization skin electrodes were affixed with collodion at the outer canthus of each eye and above and below one eye. Brain activity was recorded with a Beckman Accutrace EEG machine and stored, along with EOG, on a magnetic tape (Analog-14, Philips). Surface electrodes were always connected to Grid 1, in which a negative change relative to the deep electrode leads to a negative potential in the record. Overall band-pass of the system was .16-1,250 Hz. Aver-

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aging was performed off-line either on a summing averager (Neurolog) or on a special-purpose computer (TPAi, KFKI). Eye movements were sorted according to the amplitude of horizontal saccades. Three categories of saccade size were used: saccades with an amplitude of 8-12 deg (mean 10 deg), 16-24 deg (mean 20 deg), and 26-33 deg (mean 30 deg). Eye movements were calibrated by measuring the amplitude of saccades made to a sequence of unexpected on and off LED fixation lamp flashes. Except when eliciting saccades for calibration purposes, the eye movements of the animals were spontaneous. The behavior of the animals was always monitored by one of the experimenters outside the viewing range of the animal.

There were three viewing conditions. Conditions 1 and 2 served to assess the effects of saccade size and field luminance on lambda potentials. In the first condition, 2-deg-wide black and white vertical stripes with an overall luminance of 15 cd/m², as measured by a Tektronix J16 photometer, were used. The same grating pattern was employed in Condition 2, its luminance attenuated by 1.5 log units, using a Kodak Wratten neutral density filter. Finally, in Condition 3, saccades were performed in total darkness. Four animals participated twice, and one animal once, in the experiment.

For two of the animals, the recording sites were determined histologically. In the remaining three, histological verification is not available since additional experiments are presently being conducted with these monkeys. However, the stereotaxic coordinates were the same as those in the two verified cases.

RESULTS

The Effect of Stimulus Luminance

Table 1 presents the mean latency values measured from saccade onset of the N₁ and P₃ components of lambda potentials accompanying saccades of different sizes. As shown there, decreasing the luminance value of the stimulus field (Condition 2) led to a shift in latency of both the prominent negative and positive components. Comparison of the N₁ latencies across all saccade sizes revealed a significant difference between Conditions 1 and 2, N₁ peaking slightly earlier when the gratings were viewed under high luminance. (Overall peak latencies were 60.7 vs. 72.2 msec, $p < .05$, Wilcoxon matched-pairs signed-ranks test.) Figure 1 illustrates the grand average waveforms of the five animals.

The influence of luminance was also evaluated within the three size categories. Although neither comparison reached significance in the Wilcoxon test, the

Figure 1. Grand mean lambda potentials for the five animals averaged across all saccade sizes in each condition. Condition 1: grating with high luminance. Condition 2: grating with low luminance. Condition 3: total darkness.

same tendency appeared in each category: the N₁ component peaked later in the low-luminance condition. This can be seen in Figure 2, which shows grand average waveforms, computed over the five animals, corresponding to the different saccade categories.

The amplitude of the N₁ component across all saccade sizes was significantly different between Conditions 1 and 2; in the high-luminance condition, it was significantly greater than it was in Condition 2. (Overall means 50.6 vs. 32.5 μ V, $p < .01$, Wilcoxon test.) This amplitude difference between the two conditions clearly appears both in the overall grand averages (Figure 1), and in the grand averages corresponding to saccade sizes (Figure 2).

The amplitude of the N₁ component was also compared between Conditions 1 and 2 in the three size categories (Table 2). Although the difference in amplitude reached significance only in the case of sac-

Table 1
Means and Standard Deviations for N₁ and P₃ Latencies (in Milliseconds) From Saccade Onset in Conditions 1 and 2

Component	Saccade Size/Saccade Duration					
	10 deg/52.22 msec		20 deg/70.67 msec		30 deg/84.17 msec	
	Mean	SD	Mean	SD	Mean	SD
Condition 1						
N ₁	57.78	10.05	66.55	17.53	68.11	21.50
P ₃	141.55	23.30	157.89	23.86	164.33	21.59
Condition 2						
N ₁	67.55	18.00	73.66	21.10	75.57	19.96
P ₃	163.77	18.41	176.44	19.18	189.22	19.31

Note—N = 9.

Figure 2. Grand average waveforms across all subjects according to saccade sizes. Solid line: Condition 1. Dotted line: Condition 2. Black arrow indicates saccade onset, white arrow, saccade offset.

cades of 10 deg ($p < .05$, Wilcoxon test), the two remaining categories also show a similar tendency with difference values falling just short of significance.

The latency of the prominent positive component (P_3) that appeared after saccade offset was also significantly shorter in the high- than in the low-luminance condition (overall means, 154.6 vs. 176.5 msec,

$p < .01$, Wilcoxon test). The same tendency could be observed in all three size categories, the difference between Conditions 1 and 2 not reaching the .05 significance level only in the 30-deg category.

The amplitude of P_3 across all saccade sizes did not differ significantly between Conditions 1 and 2, the overall means being 60.2 vs. 64.1 μV . Separate comparisons among categories revealed, however, that in the case of 10-deg saccades the amplitude of P_3 was significantly greater in the low- than in the high-luminance condition ($p < .05$, Wilcoxon test).

Lambda Potentials Accompanying Saccades in Darkness

Saccades performed in total darkness (Condition 3) were also accompanied by waveforms resembling the lambda complex. As shown in Figure 1, the grand average waveform shows a broad negative component appearing between 50-90 msec after saccade onset. The amplitude of this "endogenous" negativity was considerably smaller than that of the N_1 component recorded in Conditions 1 and 2 (its mean value was around 10-15 μV). It was followed by late components appearing a hundred or more milliseconds after saccade offset. The late components took the form of either a long-lasting positivity or a sequence of small positive/negative (alpha-like) waves.

The Effect of Saccade Size

The peak latencies of the two prominent components (N_1 and P_3) of the lambda potentials were measured in two ways: from saccade onset and from saccade offset. By this, we followed Yagi's (1979) approach, which was based on the following reasoning: saccade duration increases with saccade size; therefore, if a component is linked to the offset of the saccade, its latency, as measured from saccade onset, increases along with saccade size. When, however, its latency is measured from saccade offset, it does not change considerably with different saccade sizes. In contrast, components linked to saccade onset

Table 2
Means and Standard Deviations for Amplitudes (in Microvolts) of the N_1 and P_3 Components in Conditions 1 and 2

Component	Saccade Size					
	10 deg		20 deg		30 deg	
	Mean	SD	Mean	SD	Mean	SD
Condition 1						
N_1	45.88	20.09	49.51	23.87	56.37	23.94
P_3	56.03	21.08	60.60	24.75	64.01	22.45
Condition 2						
N_1	23.97	10.07	35.24	15.68	32.28	23.74
P_3	66.05	26.04	57.36	16.90	69.01	18.24

Note— $N = 9$.

Table 3
Means and Standard Deviations for N_1 and P_3 Latencies (in Milliseconds) From Saccade Offset in Conditions 1 and 2

Component	Saccade Size					
	10 deg		20 deg		30 deg	
	Mean	SD	Mean	SD	Mean	SD
Condition 1						
N_1	6.89	14.26	-4.22	21.60	-17.78	28.12
P_3	90.22	28.72	88.67	30.04	80.67	31.63
Condition 2						
N_1	13.88	18.02	-.67	27.26	-12.66	27.63
P_3	109.55	17.94	103.22	27.10	102.99	25.21

Note— $N = 9$.

show a different pattern, their latency measured from onset does not change much with saccades of different sizes.

Regression analysis was used to evaluate the relationship between latency/amplitude values and saccade size (Ostle, 1954). As shown in Table 1, the latency of N_1 from saccade *onset* shows only a slight increase with increase of saccade size in both Condition 1 and Condition 2. In neither case was regression analysis significant [$F(1,25) = 1.71$ and $.73$, respectively].

Table 3 presents the mean latencies of the N_1 and P_3 components measured from saccade *offset* in Conditions 1 and 2. The peak latencies of N_1 from saccade offset can be fitted by a linear regression line, the slope of which is steeper and the direction of

which is opposite to that of the regression line of N_1 onset latencies (see Figure 3A). Regression analysis revealed significant linear relationships [$F(1,25) = 5.85$ and 5.41 , respectively, $ps < .05$ in both conditions] in both Condition 1 and Condition 2. As the saccade duration increases, the N_1 component peaks increasingly earlier in comparison with the offset of the saccade.

The latency from saccade *onset* of the P_3 component shows a relatively steep increase with increasing saccade duration (Figure 3B). Regression analysis revealed significant linear relationships in both Condition 1 and Condition 2 [$F(1,25) = 4.57$ ($p < .05$) and 8.19 ($p < .01$), respectively]. When, however, P_3 latency was measured from saccade *offset*, no significant relationship emerged in regression analysis for peak latencies with different saccade sizes [$F(1,25) = .47$ and $.36$, respectively].

Regression analysis did not reveal significant relationships between amplitude values of the prominent peaks in lambda potentials and saccade size, although in both conditions there was a tendency toward increasing amplitudes with increasing saccade sizes (see Table 2). In Condition 1, the linear regression coefficient was $.52$ for N_1 and $.40$ for P_3 ; in Condition 2, it was $.72$ for N_1 and $.15$ for P_3 .

A small biphasic complex could often be observed between the prominent N_1 and P_3 peaks in the lambda potential (P_2 and N_2 in Yagi's nomenclature), especially in Condition 1. In Condition 1, the mean peak latencies of P_2 were 83.3 , 94.3 , and 97.2 msec in the three size categories; those of N_2 were 104.5 , 115.0 , and 119.4 msec. The linear regression coefficient was $.68$ for P_2 and 1.03 for N_2 ($p < .10$).

Figure 3. (A) Regression of N_1 latency values on saccade sizes in Condition 1 (C1) and Condition 2 (C2). Numbers in parentheses indicate the slope of linear regression. (B) Regression of P_3 latency values on saccade sizes in Condition 1 (C1) and Condition 2 (C2). Numbers in parentheses indicate the slope of linear regression. * $p < .05$. ** $p < .01$.

DISCUSSION

In the studies on lambda potentials in humans, there are controversies about the possible origin and functional role of the components of the potential appearing at the beginning of and during the saccades, respectively. The first prominent component in humans, a spike-like positivity (P_1), peaks simultaneously with saccade onset. Becker, Hoehne, Iwase, and Kornhuber (1972) suggested that this component reflected volume-conducted activity of extra-ocular muscles. In a topographical study, Kurtzberg and Vaughan (1977) found such spike potentials to be maximal over frontal and parietal scalp areas. They suggested, therefore, that the spike-like potentials "represent a phasic discharge which is related to activation of the ocular musculature and is concurrently transmitted to other parts of the nervous system which are directly concerned with the programming of saccades" (Kurtzberg & Vaughan, 1977, p. 327). Yagi also argued that, since the P_1 starts to develop 16 msec earlier than the onset of eye movement, it might be of neural origin. In our study with

monkeys, we did not find a comparable component in lambda potentials from the occipital lobe. This finding seems to support the notion that the P_1 component in humans originates from eye muscle activity reaching the scalp by volume conduction. However, the possibility that this component might be generated outside the striate cortex should be kept in mind, although a limited sampling of activity from the frontal and parietal cortices in three animals did not reveal any prominent spike-like positivity (Figure 4).

In psychophysical experiments, it has repeatedly been demonstrated (Woodworth, 1938; MacKay, 1970) that threshold elevation during saccades (suppression) is greater when the visual field is patterned. This observation has been substantiated by evoked potential measurements (Gross, Vaughan, & Valenstein, 1967). Based on these facts, it seemed reasonable to hypothesize that the N_1 component of the lambda potential which appears during the saccade might be related to retinal suppression, the strength of which in turn would depend on the luminance value of the patterned field. In an earlier study (Marton, Szirtes, & Donauer, in press), we found, with six humans, that the N_1 component of the lambda complex recorded from the scalp did not depend, in exactly the same way, on the level of luminance of the stimulus field as it did in the case of three monkeys with which the recordings were made from within the cortex. It therefore seemed necessary to analyze the dependence of the N_1 component of cortical lambda potential on the luminance of the stimulus field using a larger animal sample.

A more thorough analysis of the relationship between the N_1 component and stimulus luminance was also called for by Yagi's recent study (Yagi, 1979).

Figure 4. Grand average lambda potentials associated with 10-deg saccades in Condition 1 in three animals. From top to bottom: transcortical recordings from frontal, parietal, and occipital areas.

Based on his observations with humans, Yagi suggested that the N_1 component might originate from eye-muscle activity, a conclusion that stands in sharp contrast with our earlier suggestion about the functional role of the N_1 component of the lambda potential (Marton, Szirtes, & Donauer, in press). While such a possibility is not ruled out entirely by our use of transcortical recordings, such a technique nevertheless seems to make Yagi's suggestion that the N_1 could be a sign of volume-conducted eye-muscle activity improbable. Furthermore, the dependence of the N_1 component on stimulus characteristics also suggests a neural origin: saccades over high-luminance gratings were associated in the monkey visual cortex with lambda potentials whose N_1 component had a shorter peak latency and greater amplitude than those recorded during low-luminance conditions (see Figures 1 and 2). Therefore, these results lend support to our earlier suggestion that the N_1 component might be closely related to retinal suppression which would be mobilized at different rates by visual fields of different luminance value. To collect direct evidence for this proposition, it will be necessary to record neuronal activity and lambda potentials simultaneously.

A further interpretation of the N_1 component might be that it is an evoked potential component which reflects cortical activity generated by the onset of pattern movement across the retina. It has been reported that the onset of pattern movement can elicit lambda-like responses in cats (Ebersole & Galambos, 1973) and in humans (Clarke, 1973a, 1973b) independently of pattern offset or reversal. This also supports the suggestions that the N_1 component is related to image movement and associated inhibitory events (Kurtzberg & Vaughan, 1977).

The fact that a broader endogenous negativity can be observed during saccades performed in darkness (see Figure 1) raises the possibility that extraretinal processes may also exert their influence in the same time range in which the N_1 appears. Based on the results of single-unit recording and lesion experiments, other authors also have assumed simultaneous participation of retinal and extraretinal processes in suppression during saccades (Breitmeyer & Ganz, 1976; Bridgeman, Hendry, & Stark, 1975; Riggs, Merton, & Morton, 1974; Torda, 1980; Volkman, Schiek, & Riggs, 1978). We suggest, therefore, that the N_1 component accompanying saccades across a patterned field might be a combination of pattern-evoked and endogenous negativity, thus reflecting the interaction of retinal events and extraretinal processes.

This interpretation is also in harmony with the observed effect of saccade size on lambda components. Measuring the N_1 latency from saccade onset, we found that its value increased only slightly with increasing saccade duration, and regression analysis showed this change to be nonsignificant. However,

when N_1 latencies were measured from saccade offset, the values increased inversely with saccade size and regression analysis revealed a significant linear change. Taken all together, these data suggest that the N_1 component is related to processes linked to the beginning of the eye movement (such as suppression).

We found a significant linear relationship between saccade size and P_3 latency when measured from saccade onset both in Condition 1 and Condition 2, whereas P_3 latencies were not influenced by saccade size when measured from saccade offset (see also the differences in the steepness of the regression lines in Figure 3). These relationships suggest a close link between P_3 and visual events associated with saccade offset. Therefore, the P_3 component is viewed as a "fixational" component reflecting information pickup in the new fixation. A similar conclusion for humans was reached by Kurtzberg and Vaughan (1977) and by Yagi (1979). We also found in monkeys what had earlier been reported in human studies (Gaarder, Krauskopf, Graf, Kropfl, & Armington, 1964; Lesèvre & Rémond, 1973; Kurtzberg & Vaughan, 1977; Marton et al., in press), namely, that the latency of this positive component lengthens as the luminance of the stimulus field is decreased. Lengthening of peak latency with decreased stimulus intensity is a common finding in human evoked potential studies (e.g., Regan, 1972) and thus supports the contention that P_3 is linked to the processing of stimuli in the new fixation.

We would like to point out the similarity (except for the P_1 and a late "saccadic" positivity) between the lambda complexes recorded from the scalp in man and the lambda potentials recorded transcortically from within the visual cortex in monkey. Beside the similarity in wave shape, the lambda complex in the monkey is somewhat less complex, which suggests the possibility that not all of the human lambda components originate in striate cortex. This is also indicated by the topographic data of Kurtzberg & Vaughan (1977). Nevertheless, it is important to note that in both cases the *main* components of the lambda potential reacted similarly to change in luminance (Marton et al., in press). The different dependences of the N_1 and P_3 components on saccade size are also in accord with conclusions drawn from human studies, and suggest that further investigations of lambda potentials may help to understand the neural processes underlying a wide range of perceptual performances.

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