Clinical EEG and Neuroscience

http://eeg.sagepub.com/

Lambda Waves and Occipital Generators

William O. Tatum, Reynold C. Ly, Monika Sluzewska-Niedzwiedz and Jerry J. Shih

Clin EEG Neurosci published online 31 March 2013

DOI: 10.1177/1550059413481029

Clinical EEG and Neuroscience
00(0) 1-6

DOI: 10.1177/1550059413481029
eeg.sagepub.com
SAGE

Lambda Waves and Occipital Generators

William O. Tatum[1], Reynold C. Ly[2], Monika Sluzewska-Niedzwiedz[3], and Jerry J. Shih[1]

Abstract

The objective of this study was to identify the relationship between lambda waves (LWs) and other occipital waveforms, in a retrospective analysis of electroencephalograms (EEGs) of clinic and hospitalized patients at a single center. The LWs were correlated with α rhythm, photic driving, and positive occipital sharp transients of sleep (POSTS). A computer-generated cursor quantified amplitude and duration of POSTS and LWs (3 waveforms and both hemispheres). Fisher exact test was used for significance ($P \leq .05$). A total of 116 patients were evaluated. Of 111 patients, with interpretable results, 74 (66.67%) had visual scanning during EEG, with 37 (50.0%) having LWs. The LWs (17.69 μV) were consistently smaller than POSTS (31.40 μV) despite similar morphology. Patients with an α rhythm of >8.5 Hz were strongly correlated with the presence of LWs ($P < .0001$), and those with LWs were strongly predictive of normal EEG ($P = .001$). Of the 37 patients, 27 (73.0%) with LWs had photic driving ($P = .0496$). No correlation was found between LWs and POSTS ($P = .45$). The presence of LWs and a low normal posterior dominant rhythm (PDR) suggests intact electrocerebral health. LWs and the photic driving response suggest similar generators but stimulus-specific networks. POSTS differ from LWs despite similar morphology, suggesting different network activation of occipital generators. LWs have clinical significance in excluding encephalopathy. Occipital generators are differentiated by state and stimulus-dependent network activation and not by location and morphology.

Keywords

EEG, occipital, generators, lambda waves, α rhythm, POSTS, photic driving

Received December 13, 2012; accepted February 10, 2013.

Introduction

Occipital generators are important for visual system function. EEG is an important tool to identify cortical dysfunction. The occipital lobe is thought to be the source for generating certain waveforms, including LWs, α rhythm, photic driving response, and POSTS.[1] LWs are surface-positive sharp transients that appear in the occipital region of the EEG during wakefulness, and are evoked by saccadic visual exploration.[2] The highest occurrence is in children between the age of 3 and 12 years, with a prevalence ranging between 2% and 88%.[3] They are "sharp" waves that appear in normal individuals, and therefore the morphological appearance could be misinterpreted as pathological epileptiform discharges, although they bear no relationship to epilepsy.[1-3] Other waveforms believed to be generated by the occipital lobe include POSTS, photic driving response, and α rhythm.[2-6] A similar morphology exists between LWs and POSTS, though their presence requires activation by triggers that are dissimilar.[4] Prior evaluations have compared the prevalence of some waveforms that seem to have the occipital lobe as their site of origin.[2,4] LWs probably have no clinical significance; however, a prior association was identified with the photic driving response,[5] suggesting a similarity between generators. In addition, if a relationship between LWs and a normal EEG is present[1,6], then the α rhythm should be concordant with the presence of LWs. Any cortical, or immediately subcortical, dysfunction can result in slowing the α rhythm, and clinically result in an encephalopathy.[6]

In this study, we sought to identify the relationship between common normal waveforms, that appear to have the occipital lobe as their primary generator, to address the neural basis of their origin.

Methods

We reviewed EEGs of routine consecutive reading sessions in 116 hospital patients, and outpatients, between August and

[1] Department of Neurology, Mayo Clinic, Jacksonville, FL, USA
[2] Mayo Graduate School, Mayo Clinic, Rochester, MN, USA
[3] Department of Neurology, Wroclaw Medical University, Wroclaw, Poland

Corresponding Author:
William O. Tatum, Mayo Clinic, Department of Neurology, 4500 San Pablo Road, Jacksonville, FL 32224, USA.
Email: tatum.william@mayo.edu

Table 1. The Reasons for Requesting an EEG in 111 Patients with Interpretable Results. Seizure-related Referrals Included Seizures (n = 21), Epilepsy (n = 4), and Epilptiform Discharges (n= 5).

Reasons for EEG in 111 patients	
Spells	37
Seizures/epilepsy/epileptiform discharges	30
Memory deficit/confusion	6
Encephalopathy/mental status changes/unresponsiveness	6
Dementia/cognitive problems	6
Syncope	5
Medical conditions[a]	4
Brain hemorrhage	3
Stroke	3
Fatigue	2
Multiple Sclerosis	2
Headaches	2
Tumor[b]	2
Dizziness/vertigo	1
Depression	1
Unknown	1

Abbreviation: EEG, electroencephalogram.
[a] Medical conditions include recurrent hepatitis C, cardiac arrest, renal failure, respiratory failure.
[b] Tumors include calcified extraxial mass in the anterior cranial fossa and glioblastoma multiforme.

October 2011. Approval from the institutional review board was obtained. All EEGs were read by electroencephalographers (WOT and JJS) certified by the American Board of Clinical Neurophysiology, to assess the presence of LWs, frequency of PDR, response to low, middle, and high frequencies during intermittent photic stimulation, and presence of POSTS. Demographic data included age, sex, date of performance, reason for patient referral, and the site where performed. Inclusion criterion was patients older than 18 years. Demographic information and clinical reason for referral were required for study entry. EEGs needed to include at least 20 minutes of recording with interpretable EEG, devoid of artifact occupying more than 50% of the record for more than 50% of the electrodes. Incomplete and technically limited recordings and continuous EEGs were excluded from this analysis. Routine digital EEGs were performed on the XLTEK system (Natus Medical Incorp, San Carlos, California) for 20 to 30 minutes. All EEGs were recorded using 21 or 23 electrodes placed in accordance with the International 10-20 System of electrode placement. Recording parameters included filter settings of 1 to 70 Hz with the 60-Hz notched filter, if necessary. Bipolar and reference montages were used for interpretation. The best PDR or sleep states achieved by the patient, intermittent photic stimulation, hyperventilation, and picture scanning were used routinely unless contraindicated. A software-generated cursor was used to measure the amplitude from peak to baseline and duration from onset to offset of principal deflections. Both LWs and POSTS were quantified with 3 representative waveforms and averaged between both hemispheres. Our quantitative analysis included the determination of the mean amplitude and duration of 111 LWs in 37 EEGs containing LWs, and 108 POSTS in 36 EEGS containing POSTS. LWs were identified only when clear surface-positive triangular-shaped waveforms were present in association with rapid eye movements and POSTS during sleep. The mean α rhythm frequency was assessed to the nearest 0.5 Hz. The PDR was listed as present in low (1-4 Hz), medium (5-12 Hz), and high (>12 Hz) rates of stimulation. Occipital waveforms were characterized and cross-correlated. Fischer exact test and the chi-square test were used for data analyses, and Bonferroni correction was used to address multiple variables.

Results

Demographics

A total of 116 patients (64 females) with a mean age of 55.9 years (range 18-89 years) were evaluated. Of all the EEGs, 73.3% were performed in the clinical neurophysiology laboratory and 26.7% in the hospital. The reasons for referral are listed in Table 1, with spells and seizure-related reasons for referral the most common. Table 2 illustrates the occipital waveform prevalence in interpretable EEGs of 111 routine hospital and clinic patients. Table 3 compares occipital waveforms with respect to the site where the EEG was performed.

EEG Interpretation

A total of 72 normal EEGs and 39 abnormal EEGs were present in the final report of interpretation for 111 patients. Five EEGs were excluded from the analysis. Most EEGs (64 of 72; 88.9%) performed in the clinic were normal. Hospital-based patients were more likely to have an abnormal recording ($P < .0001$). Diffuse slowing was the most common abnormality seen. Table 4 depicts the breakdown of the EEG findings in 39 abnormal recodings.

Occipital Generators

Lambda Waves. Of the 111 patients, 74 had visual scanning noted spontaneously or during active participation as part of our EEG laboratory routine. Cross-correlation is presented in Table 5, comparing patients with and without LW to POSTS, photic driving, α rhythm, and EEG status. Mean amplitude and duration of LWs were found to be 17.69 μV and 118.7 ms, respectively (Table 2). Consistent asymmetries with > 50% amplitude were not present for LWs. No correlation was found between LW occurrence and age, gender, or the site where the EEG was performed ($P =$ not significant (NS)). However, all patients (n = 36) had LWs during scanning eye movement and a PDR of 8.5 Hz or greater except for one with a PDR of 8 Hz ($P = .007$). An association between LWs during scanning eye movements and normal EEG was also highly significant ($P = 0.001$; Table 2).

α Rhythm. The mean α rhythm was 9.0 Hz in 111 patients (range: 8-11 Hz; Table 2). In all, 77 patients had 9 Hz or greater, 14 had 8 to 9 Hz, and 19 had 8 Hz and less. The α

Table 2. Occipital Waveform Prevalence in 111* Evaluable Routine Scalp EEGs.

	All patients	Hospital	Clinic	% All patients
Visual eye scanning	74 of 111	5	69	66.67% from all
LWs with scanning (mean amplitude; mean duration)	37 of 74 (17.69 μV; 118.7 ms)	1	36	50.00% from patients with scanning
Sleep	53 of 111	10	43	47.75% from all
POSTS (mean amplitude; mean duration)	36 of 53 (31.40 μV; 147.2 ms)	3	33	67.92% from patients with sleep
IPS	100 of 111			57.00% from patients with IPS
Photic driving	57 of 100	8	49	
Bandwidths				
Low (1-4 Hz)	29 of 57	6	23	
Medium (5-12 Hz)	47 of 57	6	41	
High (>12Hz)	30 of 57	7	23	
Mean PDR and range	9.0 Hz (2-11 Hz)	7.1 Hz (2-10.5 Hz)	9.6 Hz (6.0-11 Hz)	
PDR greater than 8.5 Hz	84 of 111	10	74	75.68% from all
PDR <8.5 Hz	27 of 111	17	10	24.32% from all
Normal EEG	72 of 111	8	64	64.86% from all
Abnormal EEG	39 of 111	19	20	35.14% from all

Abbreviations: PDR, posterior dominant rhythm; LWs, lambda waves; POSTS, positive sharp transients of sleep; EEG, electroencephalogram; IPS, intermittent photic stimulation.
Note: *1 patient did not have a posterior dominant rhythm due to excess sleep recorded in the EEG associated with an intracranial hemorrhage.

Table 3. Comparison of Occipital Waveforms Based upon EEG Impression and Location of Performance.

	Normal EEG	Abnormal EEG	*P* value comparing occipital waveform in normal EEG	Clinic	Hospital	*P* value comparing occipital waveform by location
α Rhythm > 8.5 Hz	70	14	<.0001	74	10	<.0001
α Rhythm < 8.5 Hz	2	25		10	17	
LWs + w/scanning	34	3	.001	36	1	.36
LWs − w/scanning	21	16		33	4	
Photic driving w/IPS	48	9	.0004	49	8	.20
No Photic Driving w/IPS	22	21		32	11	
POSTS with sleep	29	7	.11	33	3	.008
No POSTS with sleep	10	7		10	7	

Abbreviations: LWs, lambda waves; IPS, intermittent photic stimulation, POSTS, positive sharp transients of sleep; EEG, electroencephalogram.

rhythm amplitude averaged between 19 and 33 μV with a range of 5 to 80 μV. Table 2 illustrates the significance between occipital waveform prevalence and the final EEG reports. The presence of LWs predicted an α rhythm of >8.5 Hz, with a sensitivity of 90.9 (95% confidence interval: 0.57-0.995). When the α rhythm was <8.5 Hz, this had a negative predictive value of 97.2%. In our cohort of mixed hospital and clinic EEG, the odds ratio of a patient without LWs having an abnormal EEG with an α rhythm <8.5 Hz was 13.33.

Photic Driving Response. Of the 111 patients, 100 (90.1%) had intermittent photic stimulation performed. A photic driving response was present in 49 clinic patients and 8 inpatients, and driving response relative to the bandwidth of stimulation used are presented in Table 2. The middle bandwidth (range 5-12 Hz) was the most common stimulus frequency found to produce driving in 11 of the 47 patients, demonstrating an isolated range-specific response.

Of the 37 patients, 27 (73%) with LWs during scanning eye movements correlated with the presence of a photic driving response to at least one flash frequency ($P = .0496$; Table 3). No correlation was found between the presence of a photic driving response and the site where the EEG was performed ($P = .20$). Of the 57 patients, 52 (91.2%) had photic driving that correlated with an α rhythm of >8.5 Hz ($P = .001$), and the presence of a photic driving response correlated with a normal EEG ($P = .0004$; Table 3).

Positive Occipital Sharp Transients of Sleep. POSTS occur in late stage 1 sleep that was obtained in 53 (47.7%) of the 111 patients (Table 2). Thirty-three outpatients and 3 (67.9%) inpatients who slept demonstrated distinct POSTS. The mean amplitude and duration of POSTS were 31.40 μV and 147.2 ms. No correlation with age or gender was found ($P =$ NS). POSTS did not correlate with a normal EEG report when compared with an abnormal record ($P =$ NS). Similarly, POSTS did not correlate with the

Table 4. The Summary of the 39 Abnormal EEGs in 111 Patients With Interpretable Results.[a]

Diffuse slowing (includes intermixed θ/δ, diffuse slowing of the background, and 1 low-voltage recording)	25
Focal slowing (including 1 temporal intermittent rhythmic δ activity; 1 with left > right bitemporal focal slowing)	12
Epileptiform discharges (not including PLDs); focal (left and right temporal)-2 generalized (spike- and polyspike-and-waves)-3	5
Periodic patterns (PLDs and triphasic waves in 2)	3
Burst suppression	2
Electrographic seizures (right temporal and left hemispheric)	2
Technically limited (>50% of the background and >50% of the channels uninterpretable due to artifact)	5

Abbreviations: EEG, electroencephalogram; PLDs, Periodic Lateralized Discharges.
[a] Findings were counted more than once when significant features coexisted (ie, seizure with diffuse slowing).

Table 5. Comparison of Patients With and Without Lambda Waves.

	Patients with LW	Patients without LW	Patients with scanning
Number of Patients	37	37	74
Male (%)	11 (29.73%) of 37	18 (48.65%) of 37	29 (39.19%) of 74
Outpatient (%)	36 (97.30%) of 37	33 (89.19%) of 37	69 (93.24%) of 74
Sleep	22	16	38 (51.35%) of 74
POSTS	18 (81.82%)	11 (68.75%)	29 (76.31%) of 38
Epilepiform discharges	2	2	4
IPS	37	33	70 (94.59%) of 74
Driving	27 (72.97%)	16 (48.48%)	43 (61.43%) of 70
α rhythm (%)			
8.5-13 Hz symmetric	36 (97.30%)	27 (72.97%)	63 (85.13%) of 74
<8.5 Hz or focal slowing	1 (2.7%)	10 (27.03%)	11 (14.86%) of 74
Reason for referral			
Spells	16	13	29
Seizures	4	7	11
Epilepsy	2	1	3
Others	15	16	31
Abnormal EEGs	3	16	19 (25.68%) of 74
Normal EEGs	34	21	55 (74.32%) of 74

Abbreviations: LWs, lambda waves; POSTS, positive sharp transients of sleep, IPS, intermittent photic stimulation; EEG, electroencephalogram.

presence of LWs (P = NS). However, they did correlate with the site where the EEG was performed, and were more commonly seen in the outpatient setting ($P = .008$; Table 3).

The Bonferroni correction was applied, based on the multiple comparisons. Given there were 5 comparison tests for LWs, significance was determined to be $P < .01$. For others, it is $P < .013$ for 4 photic comparisons, $P < .02$ for 3 POSTS comparisons, and $P < .013$ for 4 α comparisons. All statistical P values retained significance during comparison using the Bonferoni correction, except for one involving LWs and photic driving.

Discussion

LWs were found in 50% of EEG recordings where visual scanning was performed. Other investigators have shown lower percentages of LWs, with only 1.7% noted in routine EEG in one study as opposed to 32.2% that had LWs when prolonged EEG was performed.[2] Therefore, the duration of evaluation is critical in terms of applying prevalence criteria. We found that the presence of LW predicts intact electrocerebral health on EEG despite frequency variance in the α rhythm. In our study, the presence of LWs predicted a normal EEG ($P = .001$; Table 3). Some authors suggest that EEGs with a PDR <8.5 Hz should be regarded as abnormal in a fully awake person.[7,8] We found a strong correlation between normal α rhythm and the occurrence of LWs generated by saccadic eye movement ($P = .007$), similar to prior reports.[5,9,10] Why LWs do not appear when the PDR is lower than 8 Hz is unclear. Either LW production is dependent on the ability to generate saccadic eye movements or their appearance may be limited due to dysfunction involving the visual cortex caused by a local or global cortical dysfunction. An association was found between the location where EEG was performed and the presence of an abnormal EEG. The majority (68 of 111; 61.3%) of patients did not have a lesion noted in the record. A third possibility is combined dysfunction impairing neural networks that activate LWs in parallel with visual system dysfunction limiting saccadic eye movements. Nevertheless, when a low-normal α rhythms exists (ie, 8-8.5 Hz), the presence of LWs suggests a low-normal α frequency as opposed to relative slowing of the PDR associated with encephalopathy. The odds ratio of 13.48 for our cohort of patients undergoing hospital and clinic EEGs, suggests that the presence of LWs can be useful in supporting a clinical diagnosis of electrocerebral health despite a low-normal α rhythm.

This may be particularly useful in evaluating elderly patients, where the α rhythm may slow down to the lower range of acceptability.

We found photic driving to be predictive of the appearance of LWs ($P = .0496$), in agreement with prior reports.[5] The association between LWs and the presence of a photic driving response suggests that a common generator may be present, but with different triggers of network activation. In a prior study, a relationship was found between LWs and photic driving.[5] In another, when comparing the lambda response of eye fixation-related potentials with the P100 component of pattern-reversal visual-evoked potentials estimated by a dipole tracing method, the resultant locations of dipoles at occipital sites were found to be very close to each other, implying a common neural generator in the visual cortex.[10] We postulate that this strong association reflects activation of similar stimulus-sensitive networks that influence different occipital generators, given the markedly different morphological appearance.

An association between photic driving and a normal α rhythm (>8.5 Hz) and normal EEG interpretation, suggest that the appearance of the addition of photic driving may also serve to support a normal background.

Several studies[4] have found an association between LWs and POSTS.[2] Despite similar morphologies, POSTS did not have an association with LWs in our study of routine EEG. However, prolonged EEG recordings were excluded from the analysis which might have had a higher yield. In one study of routine EEGs, only 8% had POSTS,[11] while in those with continuous EEGs, others noted its presence essentially in all tracings.[2] Therefore, our results differ from other studies, with greater sleep representation.[2] Nevertheless, the prevalence of POSTS in the adults of our study is comparable to similar studies.[11] Despite routinely performing a task to elicit scanning eye movement, there was only a moderate association between the presence of LWs and POSTS ($P =$ NS; Table 3). However, we did observe quantitative differences between LWs and POSTS despite the similar morphology. Our quantitative measurements are comparable to the metrics obtained in the prior studies with amplitudes of LWs that are below 50 μV and durations between 100 and 250 ms.[12] Therefore, while LWs are similar in morphology, they are smaller than POSTS both in amplitude and in duration. The POSTS are similar to LWs in their occipital location, frequency, and polarity, and therefore might be expected to have a similar generator. Despite the similarities in morphology, the lack of association with POSTS suggests that LWs have a state-sensitive occipital network activation or augmentation of similar generators.

LWs may be secondary to a functional activation of a common region in the brain, probably localized within the parieto-occipital region.[12] Schiller and Tohovnik propose 2 major targeting systems governing visually guided saccadic eye movements, including posterior parieto-occipital cortical influence on the superior colliculus, and the frontal and mesial eye fields that influence the occulomotor connections in the brainstem.[13] Several investigative tools including electrical brain stimulation that produces saccadic eye movements, pharmacological activation with bicuculline, and ablative techniques all suggest a central role of inhibition in generating target selection and saccadic eye movements.[13] Radhakrishnan et al reported a morphologically distinct waveform in the occipital regions, which in contrast to LWs was surface negative, briefer in duration, and sharper in contour. This was termed pattern-induced negative occipital potential, to distinguish it from the more commonly identified pattern-induced positive occipital potentials (LWs), suggesting that a differential area of activation in the occipital lobe during pattern (v picture) viewing was involved. Radhakrisnan et al postulated that the orientation of the activated areas to the scalp electrodes may also have accounted for the differences in the morphology,[14] though LWs may have a broad topography even during electrocorticography.[15] The robust features of LWs have been used to automate signal identification, using independent component analysis to develop signal processing techniques.[16]

Our study has the usual limitations of retrospective methodology; however, many EEG studies have been conducted in this fashion.[2,5,7,11] Our 116 EEGs contained predominantly normal EEG results. Given the comparison of several parameters, the possibility of a type 1 error exists. All statistical calculations using the Bonferoni correction retained their significance, except one involving the LW and photic driving comparison. Due to the retrospective nature of this study, larger prospective cohorts would help to further examine the association between LW and photic driving. Similarly, larger studies emphasizing abnormal EEG would be useful to confirm our findings given the larger sample size available for comparison. Although selection bias may have existed for waveform selection and identification, it remained consistent and in line with standard clinical practice. Our findings suggest that the concept of a single localized occipital generator for LWs is probably an oversimplification. Instead, we postulate that occipital waveforms on the scalp EEG require extensive bihemispheric interconnections for their expression. Quantitative metrics will help to further define the relationship between LWs and other common occipital generators observed in the scalp EEG.

Conclusion

The finding of LWs on EEG is a reflection of intact electrocerebral health. The presence of LWs generation similar to other occipital waveforms, probably involves networks that are not limited to the occipital lobe. These networks appear to involve state-sensitive and stimulus-specific activation for the expression of LWs. Elucidating intrinsic mechanisms for individual neural network initiation and activation may have clinical implications for normal aging, as well as abnormal function involving the occipital neocortex.

Acknowledgment

The authors thank Mrs Kelly Viola and Alison Dowdell from Academic and Research Support at the Mayo Clinic for their editorial assistance and Dr Jay Mandrekar and Prabin Thapa from the Mayo

Center of Translational Science for their review of the biostatistics of this study.

Declaration of Conflicting Interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The author(s) received no financial support for the research, authorship, and/or publication of this article.

References

1. Tatum WO 4th, , Husain AM, Benbadis SR, Kaplan PW. Normal human adult EEG and normal variants. *J Clin Neurophysiol*. 2006;23(3):194-207.
2. Alvarez V, Maeder-Ingvar M, Rossetti AO. Watching television: a previously unrecognized powerful trigger of lambda waves. *J Clin Neurophysiol*. 2011;28(4):400-403.
3. Chatrian GE. The lambda waves. In: Redmond A, ed. *Handbook of Electroencephalography and Clinical Neurophysiology*. Amsterdam, The Netherlands: Elsevier; 1976:123-149.
4. Egawa I, Yoshino K, Hishikawa Y. Positive occipital sharp transients in the human sleep EEG. *Folia Psychiatr Neurol Jpn*. 1983; 37(1):57-65.
5. Shih JJ, Thompson SW. Lambda waves: incidence and relationship to photic driving. *Brain Topogr*. 1998;10(4):265-272.
6. Niedermeyer E. Alpha rhythms as physiological and abnormal phenomena. *Int J Psychophysiol*. 1997;26(1-3):31-49.
7. Marcuse LV, Schneider M, Mortati KA, Donnelly KM, Arnedo V, Grant AC. Quantitative analysis of the EEG posterior-dominant rhythm in healthy adolescents. *Clin Neurophysiol*. 2008;119(8): 1778-1781.
8. Adrian E, Mathews B. The Berger rhythm: potential changes from the occipital lobes in man. *Brain*. 1934;57:355-385.
9. Vignaendra V, Matthews RL, Chatrian GE. Positive occipital sharp transients of sleep: relationships to nocturnal sleep cycle in man. *Electroencephalogr Clin Neurophysiol*. 1974;37(3): 239-246.
10. Kazai K, Yagi A. Comparison between the lambda response of eye-fixation-related potentials and the P100 component of pattern-reversal visual evoked potentials. *Cogn Affect Behav Neurosci*. 2003;3(1):46-56.
11. Rey V, Aybek S, Maeder-Ingvar M, Rossetti AO. Positive occipital sharp transients of sleep (POSTS): a reappraisal. *Clin Neurophysiol*. 2009;120(3):472-475.
12. Brigo F. Lambda waves. *American J Electroneurodiagnostic Technol*. 2011;51(2):105-113.
13. Schiller PH, Tehovnik EJ. Neural mechanisms underlying target selection with saccadic eye movements. *Prog Brain Res*. 2005; 149:157-171.
14. Radhakrishnan K, Sunku AJ, Donat JF, Klass DW. Pattern-induced negative occipital potentials (PINOP). *J Clin Neurophysiol*. 2007;24(3):277-280.
15. Perez-Borja C, Chatrian GE, Tyce FA, Rivers MH. Electrographic patterns of the occipital lobes in man: a topographic study based on use of implanted electrodes. *Electroencephalogr Clin Neurophysiol*. 1962;14:171-182.
16. Saatchi R. Single-trial lambda wave identification using a fuzzy inference system and predictive statistical diagnosis. *J Neural Eng*. 2004;1(1):21-31.