

Brainstem Auditory Evoked Potential in Patients with Posterior Circulation Ischemic Stroke

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Abstract

Background Approximately 20% of ischemic strokes affect the posterior circulation of brain structures. Mortality and risk of recurrent stroke are high with the possibility of misdiagnosis. As a result, an effective mode of examination is required for early diagnosis.

Objective To investigate the clinical diagnostic utility of brainstem auditory evoked potentials (BAEPs) in patients with posterior circulation ischemic stroke (PCIS).

Methods Twenty patients with PCIS aged 40 to 70 years were studied, along with 20 age-matched healthy controls. Medical history was taken, as well as a clinical neurological examination and BAEPs.

Results Waves IV and V latencies, and III-V inter-peak latencies were significantly prolonged and wave V amplitude was significantly reduced in patients with PCIS. Fifteen patients with PCIS show retrocochlear (central) dysfunction abnormality. Wave V latency and amplitude showed the highest estimated specificity and sensitivity with 80 % and 90%, respectively. Wave V latency negatively correlated with a duration from onset of stroke to neurophysiological examination.

Conclusion BAEPs are a useful tool as a biomarker for the clinical evaluation of PCIS. Wave V latency and amplitude is the most specific and sensitive among BAEPs parameter in the diagnosis of PCIS. Topographic distribution of the lost BAEP waves (according to diffusion-weighted MRI findings) may be closely linked to lost generators of individual waves, which suggests regional diagnostic validity of BAEPs. Finally, an early post-stroke BAEPs examination is better in detecting the abnormality and can provide reference values for further evaluation.

Keywords Posterior circulation ischemic stroke, brainstem auditory evoked potentials

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List of abbreviations: AUC = Area under the curve, BAEPs = Brainstem auditory evoked potentials, IPL = Inter-peak latencies, PCIS = Posterior circulation ischemic stroke, ROC = Receiver operating characteristic

Introduction

The posterior cerebral circulation provides only about one-third of the total flow perfusing the brain ⁽¹⁾. This circulation supplies blood to the posterior portion of the brain that includes the occipital lobe, most of the anterior and posterior

portions of the brainstem, thalamus, hippocampus, and all of the cerebellum ^(2,3).

Stroke is a clinical syndrome caused by vascular diseases with a high incidence rate and quite complicated etiological causes ⁽⁴⁾. Approximately 20% of the strokes involve the posterior cerebral circulation ⁽⁵⁾.

The clinical course of posterior circulation ischemic stroke (PCIS) is difficult to predict and the clinical manifestations tend to disappear quickly making it difficult to detect positive

symptoms. There may be progressive stroke-induced brain damage during the subacute stage, which further aggravates the neurological outcome⁽⁶⁾.

As a result of the lack of objective techniques for diagnosing the disease, the diagnosis is primarily dependent on the neurologist's clinical experience and is thus prone to misdiagnosis. Moreover, even imaging studies such as skull computerized tomography (CT) and magnetic resonance imaging (MRI) may not discover any responsible foci⁽⁷⁾. Therefore, precise monitoring and evaluation of brain injury are paramount for establishing effective treatment strategies and prognosis prediction. Evoked potentials are commonly used to predict the outcome of individuals suffering from sudden severe strokes. These evoked potentials were examined at various points following the commencement of the stroke^(8,9). Physicians frequently seek early prognosis forecasts to improve treatment techniques.

Brainstem auditory evoked potentials (BAEPs) are a sensitive objective indicator of brainstem injury that can objectively reflect peripheral auditory sensitivity and brainstem conduction capability. Clinically, wave I-V is the most consistent and stable of the seven waves. The integrity and normal function of the auditory nerve and brainstem pathway is required for normal BAEPs⁽¹⁰⁾.

Wave I represents the extracranial section of the auditory nerve; wave III represents the activity of the medial superior olive nucleus or cochlear nuclear power, and wave V principally represents the inferior pontine segment and the central nucleus of the inferior colliculus of the midbrain^(11,12).

The origin of BAEP waves I-V corresponds to the blood supply area of the vertebral-basilar artery (posterior circulation) system⁽¹³⁾. Thus, detection of latencies and inter-peak latencies (IPL) can reflect brainstem ischemia and changes in blood flow perfusion in brainstem nuclei, as well as more early subclinical abnormalities in patients with PCIS⁽¹⁴⁾. Furthermore, publications imply that BAEPs

have a unique diagnostic value and can provide objective proof for PCIS diagnosis^(15,16).

The objectives of this work are to study BAEPs in PCIS, explore the clinical diagnostic value of BAEPs in the evaluation of patients with PCIS, look for the site of abnormality within the BAEPs pathway, and evaluate the sensitivity and specificity of different waves of BAEPs in PCIS.

Methods

This is a prospective case-control study that was conducted at the Neurophysiology Unit of Al-Imamein Al-Kadhimein Medical City from May 2019 to November 2020. The Iraqi Board of Medical Specialization approved the study (order no. 931: date: 1/3/2020). All subjects provided written consent for participation. All participants were informed about the technique and aim of the study and written informed consent was obtained from them.

Twenty patients of either gender (12 males and 8 females) were studied and chosen from those attending the Department of Neurology with a diagnosis of PCIS according to clinical history, examination, and diffusion-weighted MRI. Their age ranges between 40 and 70 years (58.25 ± 7.95 years). The duration from stroke onset to neurophysiological study was in the range of 2 to 30 days.

Another 20 healthy and symptoms-free normal persons (10 males and 10 females), aged 40 to 70 years (58.9 ± 5.72 years) served as the control group.

A senior Neurologist performed a thorough medical and neurological history and assessment. All patients who met the following criteria were included in the study: in the case history, age ranges from 40 to 70 years, neurological deficits signs and symptoms must be able to be located in a specific posterior circulation distribution region, abrupt start, peaks within a few minutes, and usually subsides within 24 hours, at least 2-3 occurrences of signs and symptoms such as dizziness, vomiting, nystagmus, ataxia, perioral numbness, trouble swallowing, and sudden deafness, as well as indications of an ischemic

lesion in the territorial circulatory system detected by diffusion-weighted MRI.

The study excluded any patient with diabetes mellitus, a history of significant hearing loss, a history of a previous stroke, craniocerebral trauma or hemorrhage, or an intracranial tumor.

Keypoint electromyography (EMG) machine (Medtronic functional Diagnostic A/S -DK-2740 Skovlunde Denmark) was used in this study. Monoaural stimulation was done by conventional audiometric earphones to deliver an electric square wave "click with a rate of 10/sec". The preferred stimulus intensity for waveform recognition was 60 dB above the click hearing threshold. The non-stimulated ear was masked by white noise at 60 dB. Two replication was done for each ear and the averages of 2000 responses (number of sweeps per replication) were obtained from each ear after auditory stimulation. The filter band-pass used in this study was LF 100– HF 3000 Hz⁽¹⁷⁾.

The recording was done by surface electrodes and all electrodes have an impedance of less than 5 k Ω that is placed at the vertex (CZ) (reference electrode) and on the nasal root (ground electrode) and each ear mastoid (A1 and A2) (active electrodes) to record the auditory waveforms. The channel derivations include ipsilateral ear to vertex and contralateral ear to vertex⁽¹⁸⁾.

The peak latencies of waves I, III, and V, as well as the IPL of waves I-III, III-V, and I-V, and the amplitudes of waves I and V to determine the V/I amplitude ratio were investigated.

Statistical package for social sciences (SPSS) software, version 25, was used for all statistical analyses (IBM Corporation, USA). A normality test (Shapiro Wilk test) was performed on continuous data, and it was discovered that the data was normally distributed. An independent student t-test was used to evaluate quantitative data that were reported as mean

standard deviation (SD). The chi-square test was used to assess categorical variables that were expressed as counts and percentages.

The receiver operating characteristic (ROC) curve was used to assess the diagnostic significance of wave IV, V, and ILP-IIIIV delay, as well as wave V amplitude, in the context of patients with stroke and control discrimination. The two-tailed Pearson's correlation analysis was used to examine the relationships between age and disease duration and other conduction characteristics. A statistically significant level of statistics was accepted for all tests when $p < 0.05$.

Results

Table 1 demonstrates no significant difference between the patients and control groups regarding age and sex. Stroke onset ranges from 2 days to 30 days. According to the diffusion-weighted MRI, brain stem ischemia is present in 11 (55%), occipital lobe ischemia in 6 (30%), and cerebellar ischemia in 3 (15%) of patients.

No significant difference was noticed in the mean values of all BAEPs parameters between the right and left ear and between males and females of the control group. Also, no significant difference was noticed in the mean values of all BAEPs parameters between the right and left ear and between males and females of the patients. Accordingly, these data were pooled together and tabulated as one group for further comparison.

According to the cutoff values for the abnormality of wave IV, which is 4.5 msec, of wave V, which is 5.5 msec, and of III-V IPL, which is 1.8 msec, 10 (50%) patients showed a central abnormality, 5 (25%) showed central and peripheral abnormality at the same time, 1 (5%) showed peripheral abnormality and 4 (20%) showed no abnormality (Figure 1).

Table 1. Age and sex difference in the study population

Variables		Patients N=20	Controls N=20	P-value
Age (years)	Mean±SD	58.0±8.1	55.2±7.31	0.267
	Range	40-70	42-63	
Sex	Males	12 (60%)	10 (50%)	0.408
	Female	8 (40%)	10 (50%)	
The onset of stroke (days)	Mean±SD	9.0±9.48		
	Range	3-30		
DW-MRI	Brainstem ischemia	11 (55%)		
	Occipital lobe ischemia	6 (30%)		
	Cerebellar ischemia	3 (15%)		

DW-MRI = diffusion-weight Magnetic resonance imaging

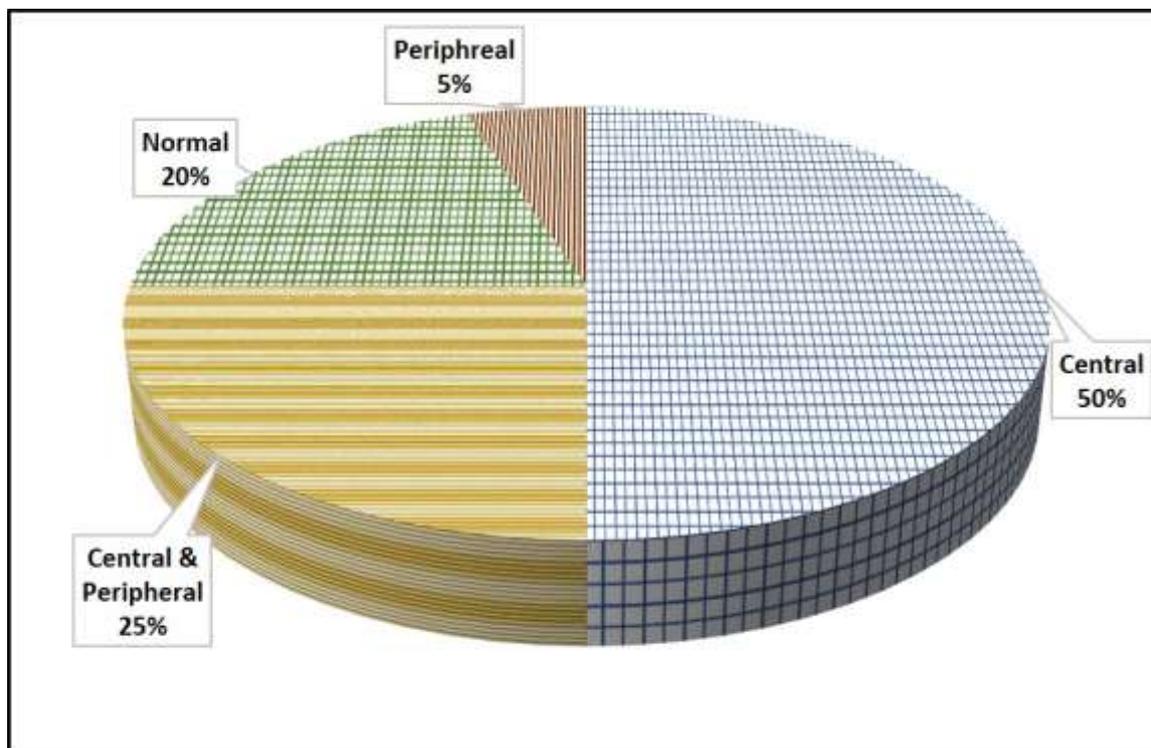


Figure 1. Distribution of central and peripheral abnormalities of BAEPs

Waves IV and V, and III-V IPL were significantly prolonged in patients with stroke as compared to the controls ($p < 0.001$). On the reverse, wave

V amplitude was significantly lower in patients with stroke as compared to the control group ($p < 0.001$) as shown in table 2.

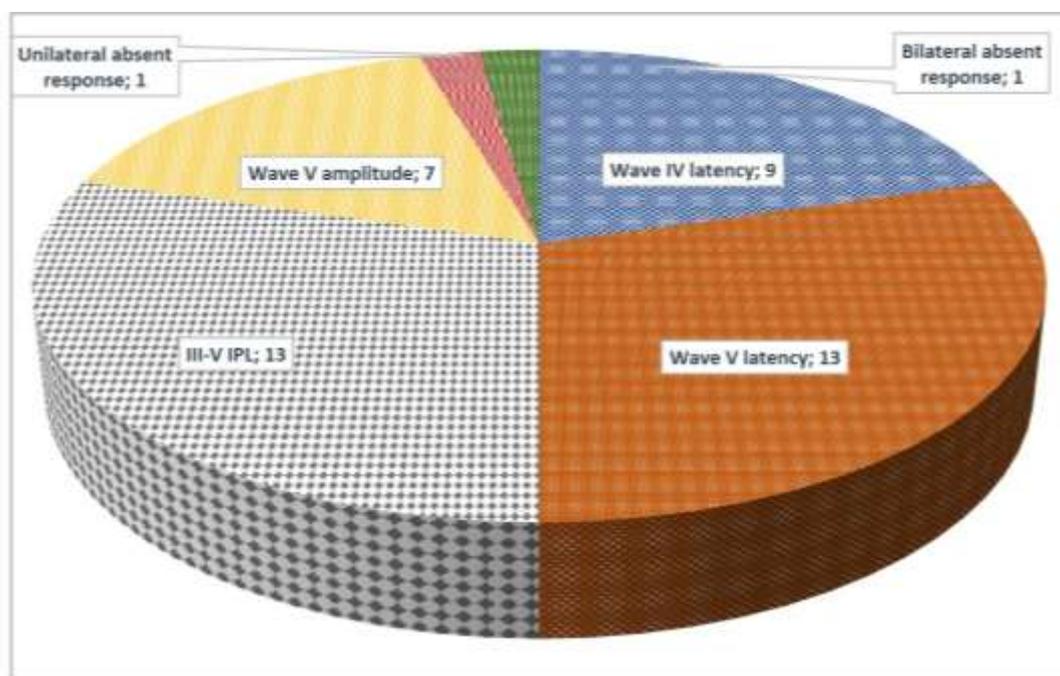
Table 2. Comparison of brainstem auditory evoked potential parameters between the patients with stroke and controls

Variables	Patients N=20 Mean±SD	Controls N=20 Mean±SD	P-value
Wave I latency (ms)	1.46±0.21	1.52±0.2	0.213
Wave II latency (ms)	2.48±0.25	2.47±0.22	0.825
Wave III latency (ms)	3.64±0.32	3.64±0.17	0.988
Wave IV latency (ms)	4.85±0.37	4.41±0.2	<0.001
Wave V latency (ms)	5.78±0.34	5.41±0.19	<0.001
I-III IPL (ms)	2.14±0.41	2.14±0.27	0.930
III-V IPL (ms)	2.12±0.41	1.75±0.24	<0.001
I-V IPL (ms)	4.29±0.38	3.86±0.22	0.089
Wave I amplitude (µV)	0.35±0.11	0.4±0.12	0.089
Wave V amplitude (µV)	0.7±0.12	0.95±0.18	<0.001
V/I ratio	2.2±0.9	2.57±0.83	0.077

IPL = Inter-peak latency

The distribution of BAEP abnormalities among 20 patients was as follows: abnormal wave IV latency in 9; abnormal wave V latency in 13;

abnormal III-V IPL in 13; abnormal wave V amplitude in 7; unilateral absent response in 1; and bilateral absent response in 1 (Figure 2).

**Figure 2. The brainstem auditory evoked potentials abnormality in patients with stroke**

The ROC curve was used to assess the diagnostic significance of wave IV, wave V, III-V ILP, and wave V amplitude in distinguishing stroke patients from healthy volunteers.

The area under the curve (AUC) for wave IV was 0.84, 95% CI= 0.75-0.93, $p < 0.001$. At a cut-off value of wave IV= 4.55 msec, the test's sensitivity and specificity were 0.75 and 0.70, respectively. The AUC for wave V was 0.792,

95% CI= 0.687-0.898, $p < 0.001$. At a cut-off value of wave IV= 5.55 msec, the test's sensitivity and specificity were 0.63 and 0.80, respectively. The AUC for III-V ILP was 0.769, 95% CI= 0.651-0.887, $p < 0.001$. The test's sensitivity and specificity at wave III-V ILP = 1.85 cut-off values were 0.78 and 0.65, respectively (Figure 3).

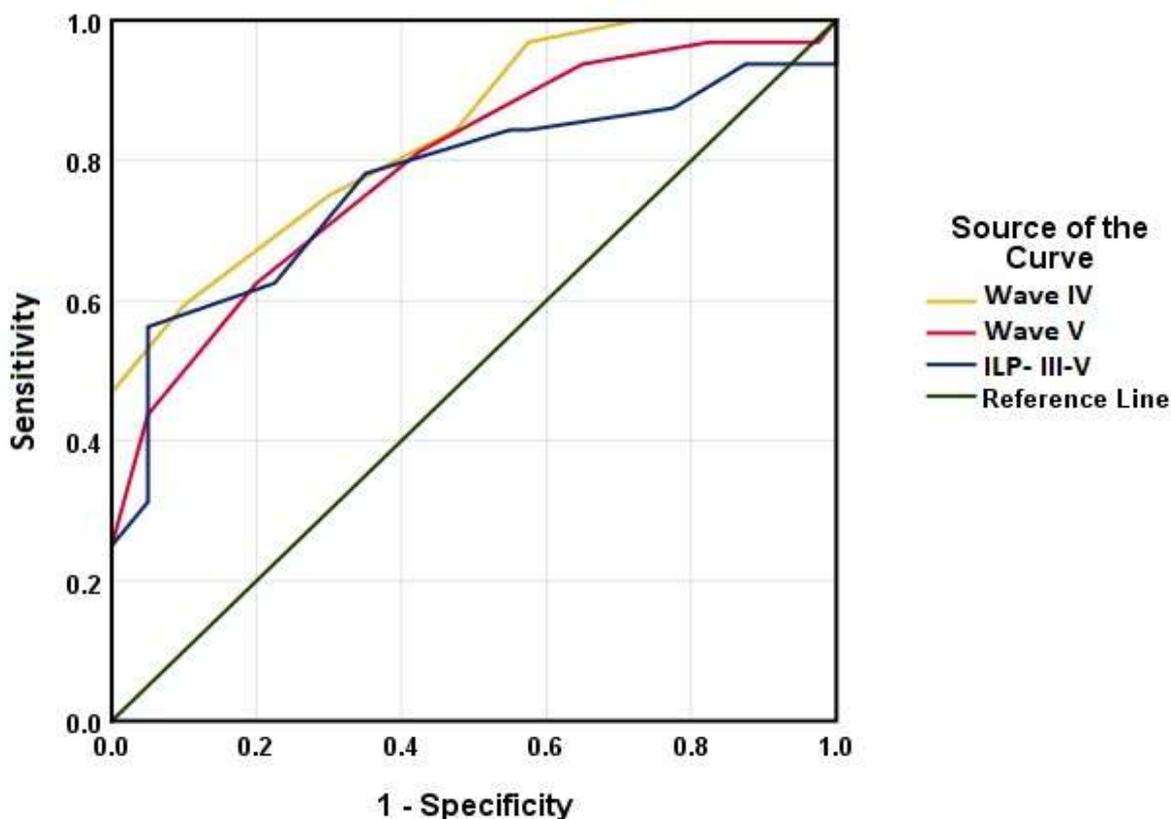


Figure 3. ROC curve for wave IV, wave V and III-V IPL in the context of discrimination between patients with stroke and healthy controls

The AUC for wave V amplitude was 0.888, 95% CI= 0.816-0.959, $p < 0.001$. At a cut-off value of wave V amplitude = 0.75, the test's sensitivity and specificity were 0.90 and 0.70, respectively (Figure 4).

None of the BAEPs parameters was correlated with the age of the subjects. Nonetheless, wave V was significantly correlated ($r = -0.332$, $p < 0.045$) with the duration (Figure 5).

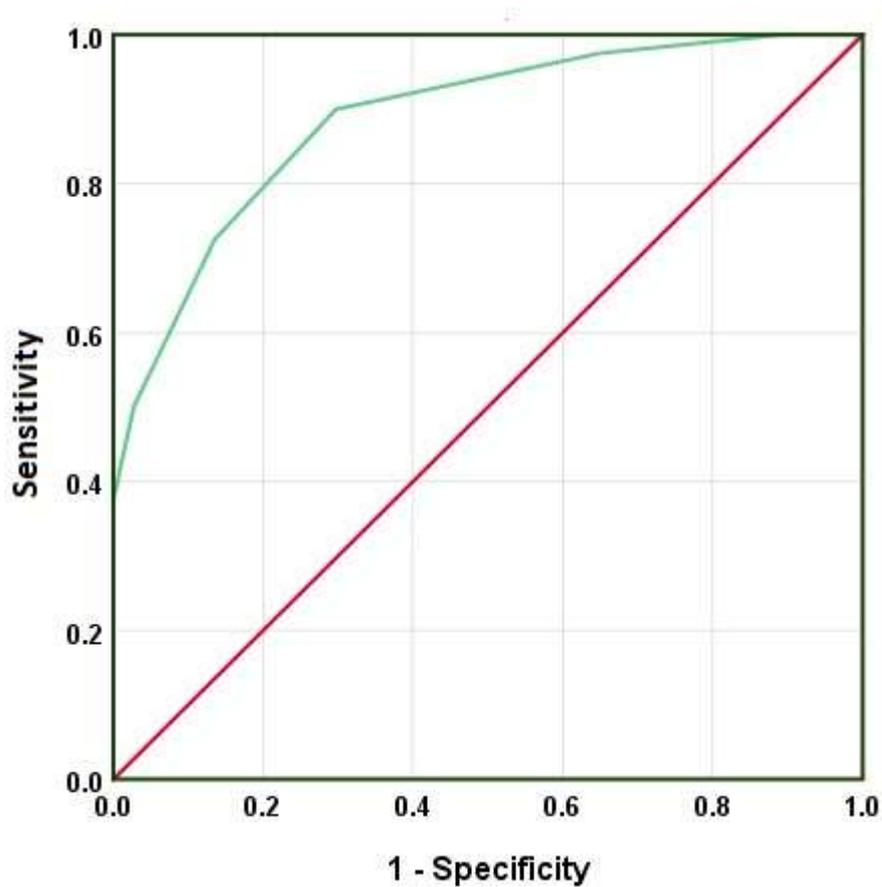


Figure 4. ROC curve for wave V amplitude in the context of discrimination between patients with stroke and healthy controls

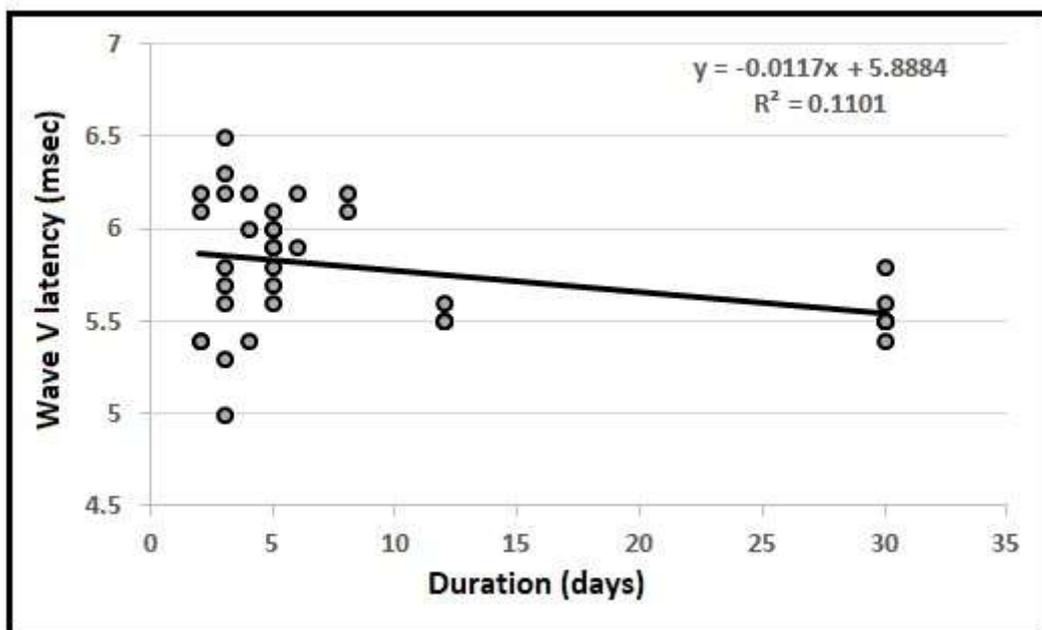


Figure 5. Regression line between wave V latency and onset duration in patients with stroke

Discussion

In this study, 75% (15 cases) of patients with stroke present retrocochlear (central) dysfunction in form of significant prolongation in wave IV and V latencies, III-V IPL, and decreased wave V amplitude. The finding of the current study was in agreement with the results reported by other researchers (7,19-21).

Because the top region of the pons is primarily fed by the short circumflex branch of the pontine artery (a tiny branch from the basilar artery with a right angle), it is more susceptible to ischemia than the lower section, resulting in prolonged III-V IPL (22).

From a pathophysiological standpoint, the aberrant BAEPs came from a transitory lack of blood supply in the relevant functional area (ischemia of the posterior circulation), which causes decreased neuronal metabolism and a decrease in polarization. The large influx of calcium ions into these neurons damages them, slows conduction, and weakens electrical activity (7).

Because the BAEP diagnosis of PCIS can result in a greater abnormal rate, it is commonly employed clinically. In the present study, the BAEPs abnormal rate was as low as 35% (7 cases of low wave V amplitude) to as high as 65% (13 cases of prolonged wave V latency or III-V IPL). Similar findings were demonstrated by other groups (7,23).

The present study demonstrates that wave V latency and amplitude among other BAEPs parameters were the most sensitive and specific indicators of diagnostic utility for stroke. This finding was in agreement with the results reported by other researchers (8,22).

Because wave V of BAEPs as a reliable predictor of brainstem function, any primary or secondary disease that deteriorates and impairs brainstem function must first alter wave V, which originates in the inferior colliculus (9).

Moreover, when vertebrobasilar artery insufficiency leads to auditory pathway ischemia, wave V latency prolongs earlier than the others, or poor morphology is observed. Animal experiments have proved that, when the unilateral vertebral artery was clipped,

wave V latency would prolong more obviously than waves I and III (24).

This study also showed a significant negative correlation between wave V latency and duration from onset of stroke to neurophysiological examination. The shorter the duration, the more abnormal wave V latency. This finding was similar to that reported by others (25,26) in which patients with ischemic stroke have delayed latencies of waveforms I, III, and V of BAEPs which are performed in the early phase of stroke.

Moreover, when an initial examination of evoked potentials is performed within the first week it provides valuable information for a prognostic purpose, however, serial examinations of BAEPs after the first weeks improve the prognostic information only slightly.

In contrast to the findings of other study (27), BAEPs can predict adverse outcomes of stroke patients more reliably when tested 4-7 days after stroke onset than when assessed 1-3 days after stroke onset. Perhaps this disagreement was because outcome evaluation of patients after 6 months by analysis of prognostic authenticity for possible predictors for unfavorable outcomes not included in the present study. Besides, only 6 patients out of the total number were examined within 1-3 days, also day one was not included in the present study.

Brain edema occurs 3-4 days after a stroke, and increased intracranial pressure occurs 4-7 days later (28). During this time, patients frequently deteriorate. The predictive timing of acute stroke examinations at 4-7 days following start is thought to reflect brain function more accurately than assessments at 1-3 days.

The present study showed that lesions involving the pontine tegmentum and midbrain region of the brain stem often cause a bilateral absence of all BAEP waves. This is probably due to vascular territories (anterior inferior cerebellar and posterior inferior cerebellar arteries) that supply these regions of the brainstem which are considered physiological generators of BAEP waves, mainly waves IV and V, so it suggests regional diagnostic validity of BAEPs.

Lesions involving the pontine tegmentum always cause various abnormalities in the BAEPs or SSEPs, so it causes loss of the V wave in BAEPs or loss of N20 in SSEPs. A large lesion involving the bilateral pontine tegmentum causes the disappearance of more than one wave in the BAEPs⁽²⁹⁾.

In conclusions, BAEPs are a useful tool as a biomarker for the clinical evaluation of PCIS. Wave V latency and amplitude are the most specific and sensitive among other parameters in the diagnosis of PCIS. Finally, an early post-stroke BAEPs examination is better in detecting the abnormality and can provide reference values for further evaluation.

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Author contribution

All of the authors contributed directly to the creation of this paper and approved the final version that was submitted. Dr. Al-Hamdani assessed and referred stroke patients clinically. The electrodiagnostic tests were performed by Dr. Tuaimah and Dr. Hamdan likewise the writing of the document. All the three authors participated in the conceptualizing, designing, interpretation and final approval of the paper.

Conflict of interest

The authors declare that they have no conflicts of interest.

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References

1. D'Antoni AV. Ankle and foot. In: Standring S. (ed.) Gray's Anatomy, The anatomical basis of clinical practice, 41st ed. Elsevier Limited; 2016. p. 1418-50.
2. Pai BS, Varma RG, Kulkarni RN, et al. Microsurgical anatomy of the posterior circulation. *Neurol India*. 2007; 55(1): 31-41. doi: 10.4103/0028-3886.30424.
3. Davim ALS, Neto JFS, Albuquerque DF. Anatomical variation of the superior cerebellar artery: A case study. *J Morphol Sci*. 2010; 27(3-4): 155-6.
4. Sacco RL, Kasner SE, Broderick JP, et al. An updated definition of stroke for the 21st century: A statement for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke*. 2013; 44(7): 2064-89. doi: 10.1161/STR.0b013e318296aeca.
5. Nouh A, Remke J, Ruland S. Ischemic posterior circulation stroke: a review of anatomy, clinical presentations, diagnosis, and current management. *Front Neurol*. 2014; 5: 30. doi: 10.3389/fneur.2014.00030.
6. Danton GH, Dietrich WD. Inflammatory mechanisms after ischemia and stroke. *J Neuropathol Exp Neurol*. 2003; 62(2): 127-36. doi: 10.1093/jnen/62.2.127.
7. Zhang XJ, Zhang LJ, Zhu J, et al. Clinical value of brainstem auditory evoked potential in the diagnosis of vertebralbasilar ischemia. *Integr Med Int*. 2015; 1(4): 199-204. doi: http://doi.org/10.1159/000380909.
8. Su YY, Xiao SY, Haupt WF, et al. Parameters and grading of evoked potentials: prediction of unfavorable outcome in patients with severe stroke. *J Clin Neurophysiol*. 2010; 27(1): 25-9. doi: 10.1097/WNP.0b013e3181cb4282.
9. Burghaus L, Liu WC, Dohmen C, et al. Prognostic value of electroencephalography and evoked potentials in the early course of malignant middle cerebral artery infarction. *Neurol Sci*. 2013; 34(5): 671-8. doi: 10.1007/s10072-012-1102-1.
10. Ying T, Thirumala P, Chang Y, et al. Empirical factors associated with Brainstem auditory evoked potential monitoring during microvascular decompression for hemifacial spasm and its correlation to hearing loss. *Acta Neurochir (Wien)*. 2014; 156(3): 571-5. doi: 10.1007/s00701-013-1957-9.
11. Garg S, Sharma R, Mittal S, et al. Alterations in brainstem auditory evoked potentials among drug addicts. A cross-sectional study. *Neurosciences (Riyadh)*. 2015; 20(3): 253-8. doi: 10.17712/nsj.2015.3.20150105.
12. Joo BE, Park SK, Cho KR, et al. Real-time intraoperative monitoring of brainstem auditory evoked potentials during microvascular decompression for hemifacial spasm. *J Neurosurg*. 2016; 125(5): 1061-7. doi: 10.3171/2015.10.JNS151224.
13. Chiappa KH, Ropper AH. Evoked potentials in clinical medicine (second of two parts). *N Engl J Med*. 1982; 306(20): 1205-11. doi: 10.1056/NEJM198205203062004.
14. Polo G, Fischer C, Sindou MP, et al. Brainstem auditory evoked potential monitoring during microvascular decompression for hemifacial spasm: intraoperative brainstem auditory evoked potential changes and warning values to prevent hearing loss--prospective study in a consecutive series of 84 patients. *Neurosurgery*. 2004; 54(1): 97-104;

- discussion 104-6. doi: 10.1227/01.neu.0000097268.90620.07.
15. Youwei H. TCD and BAEP examination for patients of vertebrobasilar ischemia. *Dian Jian Yu Shen Jing Dian Sheng Li Za Zhi* 2010; 19: 188-190.
 16. Peterein JL, Neely JG. Auditory brainstem response testing in neurodiagnosis: structure versus function. *J Am Acad Audiol.* 2012; 23(4): 269-75. doi: 10.3766/jaaa.23.4.5.
 17. Markand ON. Clinical evoked potentials an Illustrated manual. Cham: Springer International Publishing; 2020. p. 25-82.
 18. Husain AM. Illustrated manual of clinical evoked potentials. demosMedical; 2017.
 19. Simonsen CZ, Madsen MH, Schmitz ML, et al. Sensitivity of diffusion- and perfusion-weighted imaging for diagnosing acute ischemic stroke is 97.5%. *Stroke.* 2015; 46(1): 98-101. doi: 10.1161/STROKEAHA.114.007107.
 20. Brušáková Š, Ceé J, Ospalík D, et al. P41-F Reliability of BAEP, MEP and blink reflex (BR) combination in posterior circulation ischemic stroke. *Clin Neurophysiol.* 2019; 130(7): e79. doi: <https://doi.org/10.1016/j.clinph.2019.04.493>
 21. Ran J, Cui Y, Feng XY, et al. Diagnostic value of blink reflex combined with brainstem auditory evoked potential in posterior circulation ischemic stroke. *Revista Argentina de Clínica Psicológica.* 2020; XXIX(3): 471-8. doi: 10.24205/03276716.2020.744.
 22. Mohamed ES, Kaf WA, Rageh TA, et al. Evaluation of patients with vertigo of vertebrobasilar insufficiency origin using auditory brainstem response, electronystagmography, and transcranial Doppler. *Int J Audiol.* 2012; 51(5): 379-88. doi: 10.3109/14992027.2011.652676.
 23. Xiaohua S, Xueling F, Qionxia S, et al. Diagnostic value of brainstem auditory evoked potential in senile vertebrobasilar insufficiency. *J Apoplexy Nerv Dis* 2009; 26: 228-30.
 24. Cai ZL, Zhang ZC, Ni JQ, et al. The changes of brainstem auditory evoked potentials (BAEP) after vertebrobasilar artery ischemia in rabbits. *Neurol Sci.* 2012; 33(5): 1155-60. doi: 10.1007/s10072-012-0930-3.
 25. Haupt WF, Pawlik G, Thiel A. Initial and serial evoked potentials in cerebrovascular critical care patients. *J Clin Neurophysiol.* 2006; 23(5): 389-94. doi: 10.1097/01.wnp.0000223454.04161.cf.
 26. Kim YW, Sohn MK, Jung IY. Relationship between brainstem auditory evoked potentials and clinical function in patients with cerebral infarction. *J Clin Neurophysiol.* 2022; 39(5): 383-9. doi: 10.1097/WNP.0000000000000773.
 27. Zhang Y, Su YY, Xiao SY, et al. Somatosensory and brainstem auditory evoked potentials assessed between 4 and 7 days after severe stroke onset predict unfavorable outcome. *Biomed Res Int.* 2015; 2015: 196148. doi: 10.1155/2015/196148.
 28. Wijdicks EF, Sheth KN, Carter BS, et al. Recommendations for the management of cerebral and cerebellar infarction with swelling: a statement for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke.* 2014; 45(4): 1222-38. doi: 10.1161/01.str.0000441965.15164.d6
 29. Shimbo Y, Sakata M, Hayano M, et al. Topographical relationships between the brainstem auditory and somatosensory evoked potentials and the location of lesions in posterior fossa stroke. *Neurol Med Chir (Tokyo).* 2003; 43(6): 282-91; discussion 292. doi: 10.2176/nmc.43.282.

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