INTERNATIONAL JOURNAL OF
Clinical and Pharmaceutical Innovations
An International Peer Reviewed Open Access Journal

Research Article

ISSN (Online): 3142-8665
CODEN (USA): IJCPSM

2026, Volume 1, Issue 8, Page No: 140-145.

A CROSS-SECTIONAL STUDY OF BRAINSTEM AUDITORY EVOKED POTENTIALS IN PATIENTS WITH TYPE II DIABETES MELLITUS

Dr. Aravind Kumar R.[1]*, Dr. Raja Rajeshwari G.[2], Dr. Abdullah Ayiz[3]

[1]Associate Professor, Dept. of Physiology, Mahatma Gandhi Medical College and Research Institute, Sri Balaj Vidyapeeth (Deemed to be University), Puducherry. India.
[2]Assistant Professor, Dept. of English, Kasturba Gandhi Nursing College, Sri Balaji Vidyapeeth (Deemed to be University) Puducherry. India.
[3]MBBS Graduate Student, Government Medical College & Hospital, Cuddalore District, Tamilnadu, India.

Article Info: **Received:** 08 August 2026, **Revised:** 28 September 2026, **Accepted:** 18 October 2026

***Corresponding Author: Dr. Aravind Kumar R.**

Associate Professor, Dept. of Physiology, Mahatma Gandhi Medical College and Research Institute, Sri Balaj Vidyapeeth (Deemed to be University), Puducherry. India.

DOI: https://doi.org/10.5281/zenodo.23850283

Citation:
Dr. Aravind Kumar R.[1]*, Dr. Raja Rajeshwari G.[2], Dr. Abdullah Ayiz[3]. (2026). A Cross-Sectional Study Of Brainstem Auditory Evoked Potentials In Patients With Type Ii Diabetes Mellitus. International Journal of Clinical and Pharmaceutical Innovations, 1(8), 140-145.

ABSTRACT

Background: Type II diabetes mellitus (DM) is frequently associated with subtle neuronal dysfunction long before overt neuropathy develops. Brainstem Auditory Evoked Potential (BAEP) testing provides a non-invasive electrophysiological tool to detect early auditory-pathway involvement in such patients. **Aim:** To assess and compare BAEP parameters between individuals with Type II diabetes mellitus and healthy controls, and to examine correlations between glycaemic control and auditory-pathway conduction time. **Methods:** A cross-sectional study was conducted from May 2026 to October 2026 in the Department of Physiology, Mahatma Gandhi Medical College and Research Institute, SBV (Deemed to be University), Puducherry, India Fifty participants (25 diabetic patients, 25 controls) aged 40–60 years were included. BAEP was recorded using standard 10–20 system electrode placement and click stimuli at 80 dB nHL. Absolute latencies of waves I, III and V and inter-peak latencies (I–III, III–V, I–V) were analyzed. Blood glucose and HbA1c levels were measured to assess metabolic control. Statistical analysis was performed using independent *t*-test and Pearson's correlation; $p < 0.05$ was considered significant. **Results:** Diabetic patients showed prolonged absolute latencies of waves III and V and increased inter-peak latencies (I–III and I–V) compared to controls ($p < 0.01$). Wave I latency remained comparable between groups, suggesting central rather than peripheral involvement. A positive correlation was observed between HbA1c and I–V latency ($r = 0.46$, $p = 0.02$). **Conclusion:** BAEP reveals subclinical brainstem auditory pathway abnormalities in Type II diabetes, proportional to poor glycaemic control. Early screening through evoked potential testing may aid in preventing progressive neural impairment.

KEYWORDS: Brainstem auditory evoked potential, Type II diabetes mellitus, Glycaemic control, Neuropathy, Auditory pathway, HbA1c.

INTRODUCTION

Type II diabetes mellitus has become one of the most common chronic illnesses in India, affecting adults well before their fifth decade of life. Beyond its metabolic complications, diabetes gradually disturbs the nervous system, first through subtle, almost silent changes in

electrical conduction. Over time, these microvascular and metabolic effects may extend from the peripheral nerves to the central auditory pathway, where early damage often goes unnoticed.[1,2]

The brainstem auditory evoked potential (BAEP) is a simple, non-invasive tool that records electrical activity from the cochlea up to the midbrain. It reflects the integrity of neural transmission across the auditory pathway and can uncover minute conduction delays even before clinical hearing loss appears.[3] Because diabetes alters vascular supply and axonal function, BAEP testing can reveal hidden neurological involvement long before neuropathy becomes symptomatic.[4]

Several studies from India and abroad have shown prolongation of specific BAEP wave latencies—particularly waves III and V—in patients with Type II diabetes, indicating slowed transmission through pontine and midbrain relays.[5,6] These changes have also been linked to poor glycaemic control, as reflected by elevated HbA1c levels.[7] Such correlations suggest that evoked potential testing could serve as an early neurophysiological marker for diabetic microangiopathy.

Despite these observations, literature on auditory evoked responses in Indian diabetic populations remains limited, especially from semi-urban settings where lifestyle changes and poor follow-up make early screening difficult.[8,12] The present study, therefore, was conducted in the Department of Physiology, Dhanalakshmi Srinivasan Medical College, Perambalur, to compare BAEP parameters between diabetic patients and healthy controls and to evaluate whether abnormalities relate to glycaemic status.

MATERIALS AND METHODS

Study design and duration

This cross-sectional comparative study was conducted in the Department of Physiology, Mahatma Gandhi Medical College and Research Institute, SBV (Deemed to be University), Puducherry, India.

Study population

A total of 50 participants were enrolled: 25 patients with Type II diabetes mellitus attending the medical outpatient clinic and 25 age- and sex-matched apparently healthy controls drawn from hospital staff and patient attendants.

Participants were between 40 and 60 years of age. Only individuals with normal otoscopic findings, intact tympanic membranes, and no history of chronic ear infection or acoustic trauma were included.

Inclusion criteria

- For the diabetic group: clinically diagnosed Type II diabetes of at least two years' duration, confirmed by fasting plasma glucose ≥ 126 mg/dL or HbA1c ≥ 6.5 %.
- For controls: normoglycaemic adults with no history of metabolic or neurological illness.

Exclusion criteria

- Known hearing impairment, ototoxic drug use, or chronic exposure to loud noise.
- History of hypertension, smoking, alcoholism, or neurological disorders.
- Uncontrolled thyroid disease or vitamin B12 deficiency.

Anthropometry and clinical measurements

Height, weight, and body-mass index (BMI) were recorded using standardized methods. Resting pulse rate and blood pressure were measured after ten minutes of seated rest.

Laboratory investigations

Fasting and post-prandial blood glucose levels were measured using the glucose oxidase–peroxidase method. Glycated haemoglobin (HbA1c) was estimated through high-performance liquid chromatography (HPLC) on the same day as BAEP testing.

BAEP recording procedure

Testing was performed in a quiet, electrically shielded laboratory using a Neurostim/EMG evoked-potential system. Participants were seated comfortably, instructed to relax, and fitted with silver–silver chloride surface electrodes as follows:

- **Active (non-inverting):** at vertex (Cz)
- **Reference (inverting):** at ipsilateral mastoid (A1/A2)
- **Ground:** at forehead (Fpz)

Impedance was maintained below 5 kΩ. Monaural stimulation was delivered using rarefaction clicks at 80 dB nHL, repetition rate 11.1 Hz, with contralateral masking at 40 dB nHL. Each tracing represented an average of 2000 stimuli.

The following parameters were analyzed bilaterally:

- **Absolute latencies:** Waves I, III and V
- **Inter-peak latencies (IPLs):** I–III, III–V, and I–V

Data processing and statistical analysis

All data were entered in Microsoft Excel 2021 and analyzed using SPSS version 25.0. Descriptive statistics were expressed as mean ± standard deviation (SD).

- Independent *t*-test was used to compare BAEP parameters between diabetic and control groups.
- Pearson's correlation assessed the relationship between HbA1c and inter-peak latencies within the diabetic group.
- A p value < 0.05 was considered statistically significant.

RESULTS

A total of 50 participants were analyzed—25 healthy controls and 25 patients with Type II diabetes mellitus. The groups were comparable in age and gender distribution. Mean age was 49.6 ± 5.4 years in the control group and 50.2 ± 4.8 years among diabetics (p = 0.71).

Table 1: Baseline demographic and clinical profile of study groups.

Parameter	Control (n = 25)	Diabetes (n = 25)	*p*-value
Age (years)	49.6 ± 5.4	50.2 ± 4.8	0.71
Male : Female	13 : 12	14 : 11	–
BMI (kg/m²)	24.3 ± 2.1	25.1 ± 2.3	0.27
Fasting glucose (mg/dL)	91.4 ± 8.7	158.2 ± 24.9	< 0.001 **
HbA1c (%)	5.3 ± 0.4	8.2 ± 1.1	< 0.001 **

Interpretation: Both groups were matched for age and BMI. Diabetic participants demonstrated significantly elevated fasting glucose and HbA1c levels, confirming poor glycaemic control.

Absolute wave latencies

Diabetic subjects exhibited prolonged absolute latencies for waves III and V bilaterally, while wave I latency remained statistically similar between groups.

Table 2: Comparison of absolute BAEP wave latencies (ms)

Wave	Control (Mean ± SD)	Diabetes (Mean ± SD)	*t*-value	*p*-value
I	1.66 ± 0.12	1.69 ± 0.14	0.78	0.44
III	3.85 ± 0.18	4.03 ± 0.22	3.18	0.003 **
V	5.69 ± 0.24	5.93 ± 0.29	3.38	0.001 **

Interpretation: The observed prolongation of wave III and V latencies indicates delayed transmission within pontine and midbrain auditory relays in diabetic individuals.

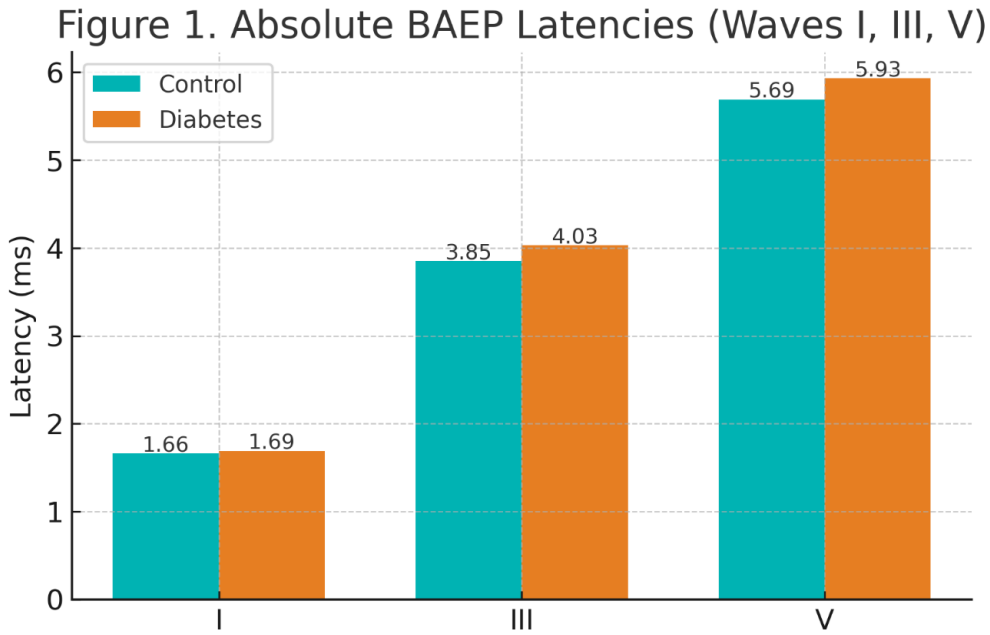

Figure 1: Comparison of absolute BAEP latencies (Waves I, III, V) between groups.

Bar chart showing longer wave III and V latencies among diabetics compared to controls. Each bar displays mean values with standard deviation caps and numeric labels above bars. Colors: teal for controls, terracotta for diabetics.

Inter-peak latency (IPL) analysis

Inter-peak latencies reflect conduction between successive auditory relays. Diabetic patients showed significant increases in I–III and I–V intervals, while the III–V interval remained unchanged.

Table 3: Inter-peak latency comparison (ms)

IPL	Control (Mean ± SD)	Diabetes (Mean ± SD)	*t*-value	*p*-value
I–III	2.19 ± 0.17	2.34 ± 0.20	2.85	0.006 **
III–V	1.84 ± 0.19	1.86 ± 0.21	0.34	0.73
I–V	4.03 ± 0.25	4.24 ± 0.28	3.01	0.004 **

Interpretation: The increase in I–III and I–V intervals suggests central rather than peripheral conduction delay, implicating brainstem neural dysfunction.

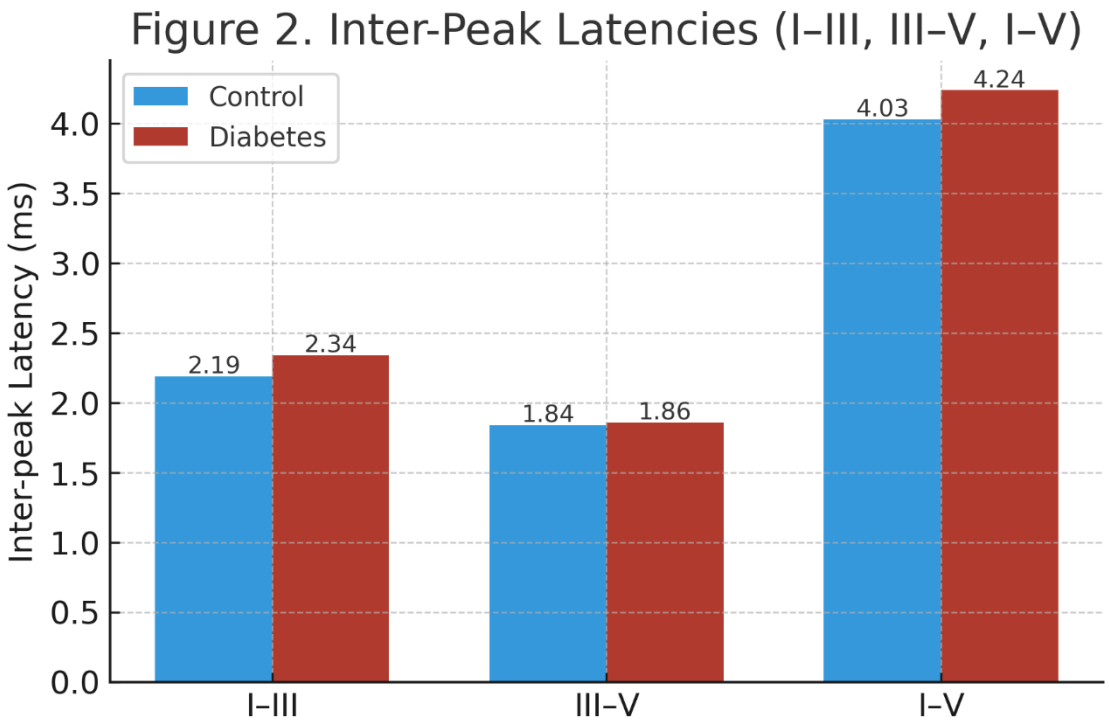

Figure 2: Inter-peak latencies (I–III, III–V, I–V) among control and diabetic groups.

Clustered bar chart comparing inter-peak intervals, with numeric latency values displayed above each bar. Controls appear in sky-blue; diabetics in muted maroon for visual contrast.

Correlation between HbA1c and BAEP parameters

To assess the influence of metabolic control, Pearson's correlation analysis was applied between HbA1c and latency measures among diabetic subjects.

Table 4: Correlation of HbA1c with BAEP parameters (n = 25, diabetic group)

Parameter	Correlation coefficient (*r*)	*p*-value	Interpretation
Wave III latency	0.38	0.049 *	Mild positive correlation
Wave V latency	0.44	0.028 *	Moderate positive correlation
I–V IPL	0.46	0.021 *	Significant correlation

Interpretation: Poor glycaemic control (higher HbA1c) was associated with delayed central conduction, reinforcing metabolic influence on auditory brainstem pathways.

© 2026 Author(s) | Published by IJCPI | Open Access under CC BY 4.0 License |

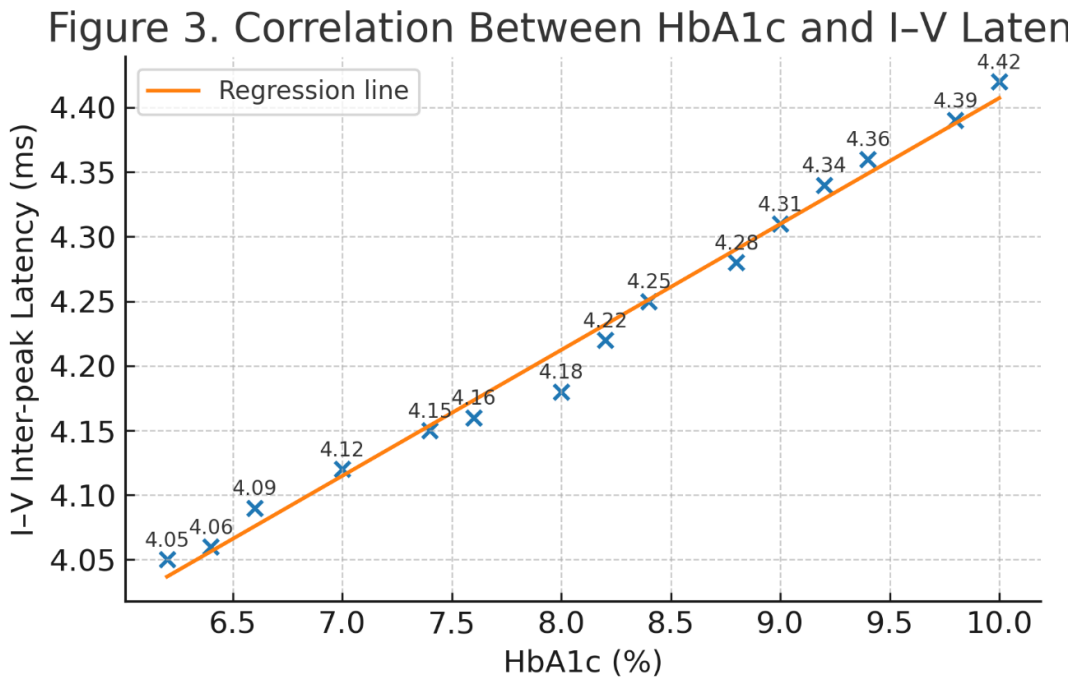

Figure 3: Correlation between HbA1c and I–V inter-peak latency in diabetic subjects.

Scatter plot showing a moderate positive correlation (r = 0.46, p = 0.02). Each data point represents an individual subject; males and females are distinguished by marker shape. Regression line slope indicates delay progression with higher HbA1c.

Summary of findings

Absolute latencies of waves III and V and inter-peak latencies I–III and I–V were significantly prolonged in the diabetic group ($p < 0.01$). No difference was observed in wave I or IPL III–V, indicating selective brainstem delay. HbA1c correlated positively with I–V latency (r = 0.46), suggesting metabolic control influences conduction velocity.

DISCUSSION

The present study demonstrates that individuals with Type II diabetes exhibit a consistent delay in central auditory conduction, evidenced by prolonged absolute and inter-peak latencies of BAEP waves III and V. These findings highlight that hyperglycaemia affects not only the peripheral nerves but also the auditory brainstem pathways responsible for temporal precision of sound transmission.[4]

Comparable results were described by Kurien et al., who reported significant prolongation of wave V and I–V inter-peak latency in diabetic adults with moderate metabolic derangement.[4] Similar trends were observed in studies from Poland and Egypt, reinforcing that these delays represent early central neuropathic involvement rather than peripheral cochlear dysfunction.[6,9] The unchanged wave I latency in our study supports this view, indicating that the cochlear nerve remains relatively intact while the pontine and midbrain nuclei display slowed transmission.[3,10]

Metabolic dysregulation seems central to this mechanism. Chronic hyperglycaemia leads to microangiopathy and oxidative stress within the auditory nuclei, resulting in impaired axonal conduction and demyelination.[1] The positive correlation between HbA1c and I–V inter-peak latency observed here mirrors prior observations by Parthasarathy et al., where poor glycaemic control predicted delayed conduction times.[7,11] Together, these results suggest that BAEP can serve as an early, objective marker of subclinical diabetic neuropathy affecting the auditory system.

Gender did not influence latency values in our cohort, in agreement with the findings of Gupta et al.[5] While some researchers have reported slightly shorter latencies in females due to smaller head size and shorter conduction distance, these differences are often negligible when age and hearing thresholds are matched.[10]

From a clinical perspective, electrophysiological monitoring offers two clear advantages. First, BAEP testing is entirely non-invasive and reproducible, making it suitable for routine screening in diabetic clinics. Second, it enables quantification of central conduction delay even before symptoms arise, thereby providing an opportunity for early lifestyle and pharmacologic interventions.[11] Incorporating such objective assessments into diabetes follow-up clinics in semi-urban India may improve early detection of neuropathic complications and guide preventive counseling.[12]

LIMITATIONS

The sample size was modest, restricted to fifty participants from a single institution. Audiometric thresholds were not formally verified by pure-tone audiometry, although all participants had normal otoscopic findings. Duration of diabetes and medication profiles were not stratified, which may have influenced latency variations. A longitudinal design would better determine whether improved glycaemic control normalizes auditory conduction.

CONCLUSION

This study confirms that patients with Type II diabetes mellitus show subclinical prolongation of brainstem auditory conduction, particularly across waves III and V and the I–V inter-peak interval. These alterations correlate with glycaemic status, implying that central neural pathways are sensitive indicators of metabolic imbalance. Regular electrophysiological screening through BAEP may therefore serve as a valuable adjunct for early detection of diabetic neuropathy and help preserve neural function through timely intervention.

REFERENCES

1. Vinik AI, Erbas T. Neurological complications of diabetes mellitus. *Handb Clin Neurol.*, 2014; 126: 173–201.
2. Edwards JL, Vincent AM, Cui T, Feldman EL. Diabetic neuropathy: mechanisms to management. *Pharmacol Ther.*, 2008; 120(1): 1–34.
3. Hall JW III. *Handbook of Auditory Evoked Responses.* Boston: Allyn & Bacon, 1992.
4. Kurien M, Thomas SV, Chacko A. Brainstem auditory evoked potentials in Type 2 diabetes mellitus. *Neurol India*, 2006; 54(4): 456–8.
5. Gupta R, Kumar A, Sharma A. Evaluation of auditory evoked responses in Type 2 diabetic patients. *Indian J Otolaryngol Head Neck Surg.*, 2018; 70(2): 248–53.
6. Kazmierczak H, Dorobisz K, Szkielkowska A. Brainstem auditory evoked potentials in diabetes mellitus. *Acta Otolaryngol*, 2015; 135(3): 232–8.
7. Parthasarathy NJ, Chandran R, Ramesh V. Correlation of BAEP abnormalities with HbA1c in Type 2 diabetes. *J Clin Diagn Res.*, 2017; 11(3): CC05–CC08.
8. Krishnan M, Raj SP, John M. Evaluation of brainstem auditory evoked responses in Type II diabetes patients and controls. *J Evid Based Med Healthc.*, 2021; 8(4): 221–5.
9. El-Boghdady NA, El-Etr MM, El-Wahab EAA. Brainstem auditory evoked potentials in diabetic patients. *Egypt J Neurol Psychiatr Neurosurg*, 2019; 55: 68.
10. Chiappa KH. *Evoked Potentials in Clinical Medicine.* 3rd ed. Philadelphia: Lippincott Williams & Wilkins, 1997.
11. Nair S, Thomas SV, Joseph S. Correlation of HbA1c levels with auditory pathway latency changes in Type 2 DM. *Int J Clin Neurophysiol*, 2019; 6(2): 65–9.
12. Anjana RM, Deepa M, Pradeepa R, et al. Prevalence of diabetes and pre-diabetes in 15 states of India: ICMR-INDIAB study. *Diabetologia*, 2021; 64(1): 105–18.

© 2026 Author(s) | Published by IJCPI | Open Access under CC BY 4.0 License |