The Egyptian Journal of Otolaryngology

ORIGINAL ARTICLE

Open Access

Cortical auditory evoked potentials in peripheral neuropathy

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Abstract

Background Cortical auditory evoked potentials (CAEPs) display both auditory processing and neurological activity in the auditory cortex. The purpose of this research is to evaluate the importance of CAEPs in identifying auditory processing disorders in patients with peripheral neuropathy (PN) in different pathologies. Sixty cases with PN of different pathologies represented the study group which was classified into two subgroups according to the underlying pathology of PN: those with either axonal PN (44 patients) and those with demyelinating PN (16 patients). The current study also included a control group of 40 healthy volunteers who did not have any peripheral or central auditory neurological disorders. CAEPs were recorded in both groups.

Results The study group's CAEP response showed significantly delayed latencies than the control groups. Comparing the two study subgroups revealed that the axonal PN group had significant delayed latencies of N1 and P2 components in comparison to the demyelinating PN group.

Conclusion Cortical auditory evoked potentials can be used efficiently to diagnosis central auditory processing disorders in patients with PN. CAEP latencies can be employed alone or in conjunction with amplitudes; however, CAEP latencies are more significant than amplitudes for such purpose. Both demyelinating and axonal PN are associated with impaired auditory processing; however, axonal PN patients are more likely to be affected, suggesting that axonal PN has a significantly drastic effect on the central auditory nervous system.

Keywords Cortical auditory evoked potentials, Peripheral neuropathy, Auditory processing impairment, Axonal, Demyelinating

Background

Auditory evoked potentials (AEPs) are an effective method for assessing the health and efficiency of the central auditory nervous system (CANS). They can be used to evaluate working memory, attention, and auditory processing speed [1].

Auditory evoked potentials allow for the assessment of the neural activity involved in most of the stages, beginning with sound detection, to integration abilities, through to discrimination of sounds. This allows for a more thorough assessment of central auditory abilities. AEPs can be used for objective evaluation of these abilities in clinical settings. According to Didoné and colleagues, they are one of the most promising electrophysiological assays for determining CANS dysfunction and/or alterations [2].

One of these powerful potentials is the CAEPs (P1-N1-P2 complex) that displays neurological activity in the brain's auditory cortex [3]. These have been suggested as markers of impaired auditory processing [4]. They are

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obligatory responses that occur beyond 50ms and up to 200ms following the stimulus onset. This complex consists of a series of waves which can be either positive (P) or negative (N) [5]. Longer latency responses in the N1 and P2 components of CAEPs are caused by temporal changes in cortical auditory response characteristics, which delay synchronous firing for the acoustic features perception [6]. Additionally, the evaluation of central processes makes use of distinctive methods for measuring how the nervous system's electrical potentials change in response to auditory stimulation at different levels [7]. Moreover, CAEPs assessment is non-invasive, cost-effective, and reliable and does not call for attention or behavioral reactions [8]. This allows for the recording of brain electrical activity without interference [9].

Peripheral neuropathy (PN) is a painful broad spectrum of diseases that damage and deteriorate peripheral nervous system neurons in different ways [10]. In PN, pain arises as a direct consequence of increased sensitivity of injurious sensory neurons to afferent signals that manifest as hyperexcitability of primary afferent neurons and a decrease in their activation threshold. It could also be a result of drugs that act as neurotoxins that cause axonal degeneration similar to demyelination process [11]

Independent of peripheral hearing loss, a link between central auditory function and auditory processing dysfunction has been established in PN patients [12]. PN induces plastic changes in synaptic transmission at various central system levels including the spinal cord and different brain regions such as the thalamus, brainstem, and cerebral cortex [13].

The majority of those patients also have a higher risk of falling due to peripheral manifestations such as chronic pain and poor sleep. The underlying pathology in PN suggested the possibility of the presence of auditory processing impairment [14].

In this study, we hypothesize that there may be subtle central pathology in PN patients that could be responsible for the presence of auditory processing impairment in PN cases. This assumption did not receive too much attention. So, our principal aim was to evaluate CAEPs' usefulness in identifying patients with PN who are experiencing early auditory processing deterioration. We also investigated the possible effect of the underlying pathology of PN on their central auditory function.

Methods

One hundred adult participants, aged 18 to 45 years, were recruited to participate in this study and were divided into two groups. Sixty cases with PN of different pathologies represented the study group and 40 healthy volunteers (who did not have any peripheral or central auditory neurological disorders) represented the control group. According to Pop-Busui and colleagues [15], when two different aberrant nerve responses occurred in more than one limb and nerve conduction study (NCS) parameters were two standard deviations (SD) above normative values, PN was identified. Two subgroups were created out of the study group: those with axonal PN (44 patients) and demyelinating PN (16 patients). The causes of PN in our study included diabetic mellitus, leprosy, vasculopathy, and chronic inflammatory demyelinating polyradiculoneuropathy (CIDP).

For both groups, those with bilateral normal hearing sensitivity are required for inclusion.

Subjects with a history of head trauma or stroke, ototoxic medication, middle ear disorders, hearing loss, prior ear surgery, psychological disorders, endocrine disorders, or chronic systemic diseases (like uncontrolled diabetes mellitus or hypertension) were excluded from the research.

All cases in this study were recruited from cases attending the Audiovestibular Medicine, Rheumatology, and Internal Medicine clinics at XXX University Hospitals were the participants. Each study participant was given information about the testing procedure before their signed consent was acquired. This study was approved by the Scientific Research Ethics Committee at 8th of April 2020. The approval number is KFSIRB200-2.

A comprehensive physical examination was performed on each participant, which included recording their medical history, performing an otoscopic examination, pure tone audiometry between 0.25 and 8 KHz, and speech audiometry using an Interacoustic AD629 audiometer (Interacoustic, Denmark). An Interacoustic AT235 (Interacoustic, Denmark) was used to measure tympanometry and ipsilateral acoustic reflexes.

In response to a tone burst (TB) at 1000 Hz with a 20-ms rise/fall time and a 50-ms plateau (20-50-20), CAEPs were recorded. An insert-phone ER-3A was used to present TB stimuli at a rate of 0.5 stimuli per second at 70dBSPL with alternate polarity. The low-pass filter was set at 100Hz, the high-pass filter at 1.0Hz 6/octave, and the gain factor was × 10,000. Two hundred sweeps were averaged and the rejection level was ±78 μv. The analysis window was −150ms before the stimulus and 450ms after it. In each recording session, subjects were instructed to perform some other active or passive foreground task in the visual domain. Four electrodes were used for recording CAEPs, based on the side being stimulated: M1 and M2 (mastoids) served as reference electrodes, Fpz (ground electrode), and Fz (active electrode). The electrode impedance was maintained below 5KΩ. The CAEP latency (ms) and the peak to following trough amplitude (μv) were measured. The Interacoustic

Eclipse-EP25 AEPs (Interacoustic, Denmark) was used to record CAEPs.

A statistical analysis of the collected data was conducted using the Statistical Package for the Social Studies (SPSS) version 22 (2013). Since 2013, the United States of America's International Business Machines Corporation (IBM) has been developing SPSS version 22. To describe quantitative data, the terms mean, SD, and median were used. Qualitative data are presented using formats such as numbers and percentages. The significance level was established at $p \leq 0.05$. The Student t test was used to compare quantitative variables with normally distributed data between the two groups, while the Mann-Whitney test was used to compare variables with abnormally distributed data.

Results

This study was done between April 2020 and April 2022, where a total of 100 participants participated in this study: 16 males and 24 females, comprising the control group ($n = 40$), their mean age was 30.50 ± 7.55 years. The study group included 30 males and 30 females ($n = 60$), and their mean age was 31.62 ± 4.11 years. There was no significant difference between the two groups in either age or sex ($p>0.05$).

Only two patients (out of 60) in the study group had absent CAEP response and the rest of cases showed significant delayed latencies of P1, N1, and P2 components in comparison to the control group. In terms of CAEP amplitudes, the study group showed low P1 and N1 amplitudes, but they were statistically non-significant, while P2 amplitude was similar in both groups (Table 1).

The study group was further divided into two subgroups: those with axonal and demyelinating PN (44 and 16 patients respectively). As regards the *axonal PN subgroup*, two cases (out of 44) had absent CAEPs. Meanwhile, the remaining cases showed significantly delayed latencies of P1, N1, and P2 components in comparison to the control group. In terms of CAEP amplitudes, both P1 and N1 components showed significantly reduced amplitude when compared to the control group (Fig. 1, Table 2).

As regards the *demyelinating PN subgroup*, CAEPs were recorded in all cases where P1, N1, and P2 components had significantly delayed latencies in comparison to the control group. However, the CAEP amplitudes showed no statistically significant differences observed in comparison to the control group (Table 3).

Comparing the two subgroups revealed that the axonal PN group had significant delayed latencies of N1 and P2 components in comparison to the demyelinating PN group. However, no statistically significant differences were detected as regard the CAEP amplitudes (Table 4).

Discussion

Central auditory processing deficits in patients with peripheral neuropathy had been reported despite their apparent normal hearing. Slow conduction time or loss of neural synchrony that underlies slow reaction time and perceptual difficulties in challenging acoustic environments had been reported in those patients [16]. Patients with PN frequently have temporal processing deficits

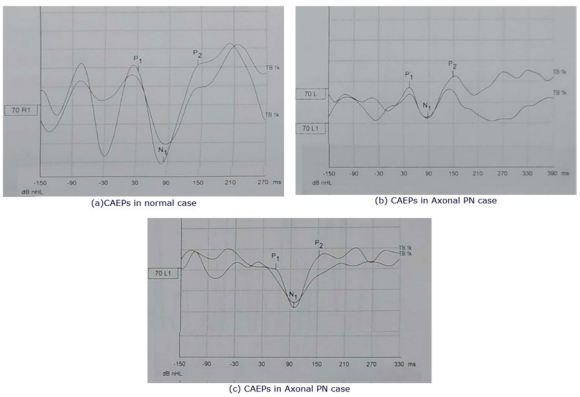

Fig. 1 Examples of CAEPs recorded in normal case (**a**), axonal PN case (**b**) and demyelinating PN case

Table 1 Comparison of CAEPs latencies and amplitudes between the control and the study groups

CAEPs		Control group (n=40) (mean/median) ± (SD/ min–max)	Study group (n=58) (mean/median) ± (SD/ min–max)	t / z	P
Latency (ms)	**P1**	46.95±9.62	51.31±8.80	2.32	**0.022***
	N1	87.05±5.78	93.91±13.38	3.05	**0.003***
	P2	145.95±11.37	151.15±22.84	1.33	**0.011***
Amplitude (μv)	**P1**	1.12 (0.90–2.15)	1.07 (0.38–2.27)	1.406	0.075
	N1	1.97 (1.11–2.95)	1.86 (0.74–4.99)	1.352	0.165
	P2	1.55 (1.00–2.30)	1.55 (0.45–2.70)	1.258	0.478

* Statistically significant at $p \leq 0.05$

CAEPS Cortical auditory evoked potentials, *n* Number of persons, *SD* Standard deviations, *t* Student *t* test, *z* Mann-Whitney test

Table 2 Comparison of CAEPs latencies and amplitudes between the control and Axonal PN groups

CAEPs		Control group (*n*=40) (mean/median) ± (SD/ min–max)	Axonal PN (*n*=42) (mean/median) ± (SD/ min–max)	*t* / *z*	*P*
Latency (ms)	**P1**	46.95±9.62	50.40±7.34	1.83	**0.034***
	N1	87.05±5.78	96.19±13.12	4.05	**ˇ0.001***
	P2	145.95±11.37	156.14±19.38	2.88	**0.005***
Amplitude (μv)	**P1**	1.12 (0.90-2.15)	1.02 (0.38-2.27)	586.0	**0.018***
	N1	1.97 (1.11-2.95)	1.75 (0.74-3.82)	620.0	**0.041***
	P2	1.55 (1.00-2.30)	1.55 (0.45-2.70)	818.0	0.838

* Statistically significant at $p \leq 0.05$

CAEPS Cortical auditory evoked potentials, *n* Number of persons, *SD* Standard deviations, *t* Student *t* test, *z* Mann-Whitney test

Table 3 Comparison of CAEPs latencies and amplitudes between the control and demyelinating PN groups

CAEPs		Control group (*n*=40) (mean/median) ± (SD/ min–max)	Demyelinating PN (*n*=16) (mean/median) ± (SD/ min–max)	*t* / *z*	*P*
Latency (ms)	**P1**	46.95±9.62	53.69±11.76	2.22	**0.031***
	N1	87.05±5.78	87.94±12.55	0.36	**0.005***
	P2	145.95±11.37	138.06±26.50	1.57	**ˇ0.001***
Amplitude (μv)	**P1**	1.12 (0.90–2.15)	1.24 (0.90–2.03)	328.0	0.884
	N1	1.97 (1.11–2.95)	2.08 (1.05–4.99)	348.0	0.611
	P2	1.55 (1.00–2.30)	1.51 (1.06–2.04)	244.0	0.166

* Statistically significant at $p \leq 0.05$

CAEPS Cortical auditory evoked potentials, *n* Number of persons, *SD* Standard deviations, *t* Student *t* test, *z* Mann-Whitney test

Table 4 Comparison of CAEP latencies and amplitudes between axonal PN and demyelinating PN groups

CAEPs		Axonal PN (*n*=42) (mean/median) ± (SD/ min–max)	Demyelinating PN (*n*=16) (mean/median) ± (SD/ min–max)	*t* / *z*	*P*
Latency (ms)	**P1**	50.40±7.34	53.69±11.76	1.28	0.207
	N1	96.19±13.12	87.94±12.55	2.17	**0.035***
	P2	156.14±19.38	138.06±26.50	2.86	**0.006***
Amplitude (μv)	**P1**	1.02 (0.38–2.27)	1.24 (0.90–2.03)	231.0	0.067
	N1	1.75 (0.74–3.82)	2.08 (1.05–4.99)	241.0	0.098
	P2	1.55 (0.45–2.70)	1.51 (1.06–2.04)	393.0	0.321

* Statistically significant at $p \leq 0.05$

CAEPS Cortical auditory evoked potentials, *n* Number of persons, *SD* Standard deviations, *t* Student *t* test, *z* Mann-Whitney test

since diabetes mellitus and vascular abnormalities are so common [17]. It has been postulated that oxidative stress, inflammation, and dyslipidemia, among other things, may be shared pathogenic pathways with PN [18]. PN is associated with deterioration in temporal processing abilities in the presence of normal hearing detection levels regardless of diabetes status and vascular risk factors [19].

It would be great to hypothesize whether patients with PN will also display some degree of central nervous system (CNS) insult given the coexistence of central processing dysfunction in those patients [20]. The examination of the brain activity associated with sound detection, discrimination, and integration skills is made possible by the auditory evoked potentials, which allow the analysis of auditory information processing (such as memory and attention) [2]. The N1 component of the CAEPs can serve as an indicator for the auditory activity involved in acoustic properties decoding, while the P2 component can be utilized to measure discrimination abilities [21].

The P1 and N1 peak latencies of the CAEPs can be used as specific neurophysiological indicators of the time required by the auditory cortex to process

information. The peak latency of P1 or N1 therefore represents the moment at which a particular set of neurons supporting a particular phase of processing in the auditory system are active in a functionally interconnected network [22].

In this study, CAEPs were absent in two cases of the study group. In the remaining cases, P1, N1, and P2 latencies were significantly delayed when compared to those recorded in the control group. This could be the result of the delayed synchronous firing necessary for the acoustic feature perception to occur which is followed by temporal processing delay [6]. This result suggests that these factors are affected by the participant's level of attention on the presented stimulus. It also shows that the sound discrimination process needs more time in patients with PN [23].

In terms of CAEP amplitudes, there were no statistically significant differences of P1, N1, and P2 amplitudes between both groups. Similar findings were reported by Oliveira and colleagues [7]. Additionally, some researchers (Ex. Lai and colleagues [24]) found no significant variations in P2 amplitude between healthy persons and people with mild auditory processing impairment. On the contrary, Lister and colleagues [25] observed that those patients had higher P1 amplitudes. More recently, Mello [26] found that both aging and impaired auditory processing contribute to an increase in the amplitude of evoked potentials as a results of their impact on the cortical and subcortical processes. This discovery revealed the aging might have widespread alterations in the auditory system.

In another study, Lister and colleagues [8] reported a reduction of P2 amplitudes that might reflect additional adjustments to the suppression of neuronal resources in people with impaired auditory processing. Such reduction could also be attributed to deficiency of the available brain resources needed to carry out temporal tasks. In other words, those patients might have brain resources that moved to be utilized in a different region [8].

These contradictory results may suggest that latencies should be added to amplitude in order to provide a more precise assessment of central auditory functions and that amplitude should not be utilized alone as a diagnostic measure for such a purpose. These results suggest that the latencies P1-N1-P2 components of the CAEPs may be valuable markers of early impaired auditory processing, whereas amplitude alterations should be carefully considered in such circumstances [8].

The study group was divided into two subgroups: those with axonal PN and those with demyelinating PN. Both demyelination (the breakdown of the myelin sheath) and axonal degeneration (the destruction of the axon) are examples of neuropathy, which affects the neurons [27].

Using the CAEPs in attempts for differentiating between axonal and demyelinating disease is frequently ambiguous [27] which was true in the current study but not always. As regards the axonal PN subgroup, there were significant delayed latencies of P1, N1, and P2 components in comparison to the control group. In terms of CAEP amplitudes, P1 and N1 components showed significantly reduced amplitude when compared to the control group. On the other hand, the demyelinating PN subgroup showed significant delayed latencies of P1, N1, and P2 components when compared to the control group. These findings of affected latencies and amplitudes in axonal PN in comparison to affected latencies only in demyelinating PN could be explained by axonal loss which is the major outcome of almost all polyneuropathies. Meanwhile, demyelination is typically a subsequent event that is rarely identified as the main pathogenic mechanism in polyneuropathies [28].

Additionally, the findings in clinical presentation can explain that axonal PN group is more affected than demyelinating PN as revealed by our CAEP findings [29]. However, electrophysiological findings revealed that axonal PN often manifests as a decline in the amplitudes of sensory nerve action potentials and compound muscle action potentials (CMAP) and only a mild decrease in conduction velocity. On the other hand, more reduction in motor and sensory nerve conduction velocities, increased temporal CMAP dispersion, and increased distal motor latencies are all indicative of demyelinating PN [30]. These findings make differentiating between axonal and demyelinating disease frequently difficult.

The demyelinating lesions are usually limited either to the CNS or to the peripheral nervous system. It is uncommon for both central and peripheral demyelination to occur at the same time. In some patients, PN and CNS involvement happened at the same time or very quickly after [31].

Conclusion

Before any neurological symptoms manifest, subclinical impaired auditory processing can be identified early with cortical auditory evoked potentials. With regard to central auditory processing function, CAEP latencies can be employed alone or in conjunction with amplitudes; however, CAEP latencies are more significant than amplitudes for such purpose. Both demyelinating and axonal PN affect central auditory processing functions; however, axonal PN patients are more likely to be affected, suggesting that axonal PN has a significantly drastic effect on the central nervous system.

Abbreviations

AEPs Auditory evoked potentials
CAEPs Cortical AEPs

CANS	Central auditory nervous system
CMAP	Compound muscle action potentials
CNS	Central nervous system
CV	Conduction velocity
dBSPL	deciBel Sound Pressure Level
Fpz	Prefrontal midline (high forehead)
Fz	Frontal midline
Hz	Hertz
IBM	International Business Machines Corporation
M1	Left mastoid
M2	Right mastoid
N	Negative
NCS	Nerve conduction studies
P	Positive
PN	Peripheral neuropathy
USA	United States of America
SD	Standard deviation
SPSS	Statistical Package for the Social Studies
TB	Tone burst

Acknowledgements
My special thanks to all the staff in Audiovestibular unit and the volunteers.

Authors' contributions
MM, AI and TG conceived and designed the analysis, collected the data, performed the analysis and wrote the paper.TH and SR collected the data.

Funding
No funding was received for conducting this study.

Availability of data and materials
The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate
This study is approved by the Scientific Research Ethics Committee of Kafrelsheikh University on 8th of April 2020 under the number KFSIRB200-2. Each study participant was given information about the testing procedure before their signed consent was acquired.

Consent for publication
Written informed consent was obtained from the study participants.

Competing interests
The authors declare that they have no competing interests.

Received: 10 February 2024 Accepted: 8 March 2024
Published online: 20 August 2024

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