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Electrophysiological-Cortical Encoding of Central Auditory Processing in Adults with Temporal Lobe Epilepsy

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ABSTRACT

Background: Temporal lobe epilepsy (TLE) is the most common focal type of epilepsy that originates near the auditory cortex and hence may impair auditory cortical processing. The functional integrity of the auditory cortex could be assessed electrophysiologically using slow cortical response (SCR).

Objective: The current study was designed to predict the validity of SCR in evaluating central auditory processing (CAP) in TLE.

Subjects and Methods: This observational, case-control study involved 38 right-handed participants; the TLE group included 19 right-handed patients (International League Against Epilepsy criteria was used for diagnosis), and the control group included 19 right-handed adults matching the TLE group in both gender and age.

Results: TLE group exhibited a significant delay of the left ear's N1 and P2 latency and a significant reduction of the N1P2 complex amplitude (in both ears). The effect of different variables revealed negative correlations between SCR latencies and each of the age, age of onset of TLE, and time of last seizure; positive correlations between SCR amplitudes and each of the age, age of onset of TLE, and time of last seizure; a significant effect of gender on P2 and N2 latencies; and a significant effect of seizure frequency on P1 latency. Validity measures and the ROC curve revealed a better accuracy for the left ear's N1 (82%) and then P2 (73%) latencies and for the N1P2 amplitude (right=81% and left=76%).

Conclusions: The current results indicate altered neural activities within the temporal lobes of patients with TLE, which can be documented electrophysiologically by SCR. N1 and P2 waves' measures showed the highest accuracy, hence providing useful information about possible functional deterioration and impaired CAP in this population.

Keywords: Central auditory processing, Temporal lobe epilepsy, Slow cortical response.

INTRODUCTION

Epilepsy is a widespread neurological disorder that affects people of different ages, races, and socioeconomic statuses. The International League Against Epilepsy (ILAE) has defined epilepsy as having one or more of following symptoms: 1) at least 2 unprovoked (or reflex) seizures, at least twenty-four hours apart; 2) a single unprovoked (or reflex) seizure with a 10-year recurrence risk of at least 60% after two unprovoked seizures; and 3) verification of a specific epilepsy syndrome [(1)].

Focal seizures are the commonest seizure type. Temporal lobe epilepsy (TLE) is the most common type of localization-related epilepsies [(2)]. TLE patients who suffer from epileptic seizures may have their auditory cortex processing and neuronal functional coordination disrupted. Central auditory processing (CAP) and neuro-cognitive abilities, all of which are crucial for speech intelligibility and effective communication, may thereafter become impaired [(3)].

When talking about how different sounds are perceived and processed, it is the CAP. Central auditory processing disorder (CAPD) occurs when sounds are not adequately processed due to a deficit in CAP, leading to erroneous data perception. CAPD could be presented by difficulty in one or more of the sound localization and lateralization, auditory discrimination, auditory pattern identification, auditory performance in challenging situations, and temporal processing[(4)].

Early assessment of CAPD would help for early intervention and hence improve speech perception and communication. The integrity of the central auditory pathway can be evaluated with the help of electrophysiological testing. Cortical auditory evoked potentials, such as the slow cortical response (SCR; P1-N1-P2-N2), are useful tools for assessing CAP[(5)]. Little is known about CAPD in TLE. Therefore, the current research was conducted to predict the validity of SCR in the diagnosis of CAPD in patients with TLE.

SUBJECTS AND METHODS

Subjects:

This observational, case-control study was performed at the Audio-Vestibular Medicine Unit, ENT Department, Zagazig University Hospitals. The sample size for this study was estimated to be 38 participants, using Open-Epi at 80% test power and 95% confidence limits (CL). This sample was divided into control and patient groups:

The patient group:

Included 19 right-handed patients with TLE. Their ages ranged between 18-45 years, of both genders. These participants were referred from the Epilepsy Unit, Neurology Department, Faculty of Medicine, Zagazig University. The diagnosis and lateralization of the seizure focus in the TLE patients were established following the ILAE criteria by a comprehensive evaluation, including detailed seizure history and seizure semiology. All patients exhibited normal hearing sensitivity (pure tone average (PTA) ≤ 25 dB HL).

They were excluded from the study if they had:

- Medical and/or mental disability or symptomatic epilepsy (Symptomatic epilepsy is defined as

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epilepsy in which convulsions are due to structural disease of the brain e.g.: intracranial tumors, chronic inflammatory condition of meningitis).

- Other neurological disorders such as stroke, infection, encephalitis, tumors, and trauma.
- Other types of epilepsy, including generalized epilepsy and other focal epilepsy (frontal, occipital, or parietal).
- Epileptic syndrome.

The control group:

Involved 19 right-handed adults matching the study group in both gender and age. They had normal hearing sensitivity (PTA ≤ 25 dB HL) with no history of neurological or systemic disorders.

Procedure:

This study was conducted during the years 2021 and 2022. Electrophysiologic testing with SCR and tone stimuli was performed, along with a thorough history taking and otoscopic examination to all participants. These examinations required two hours to be completed. In addition, the TLE group was subjected to neurological examination, ictal/interictal electroencephalography (EEG) and/or video EEG monitoring, and neuroimaging using magnetic resonance imaging of the brain, with or without contrast.

1- **Full history taking** as regards personal, past medical history for otological and neurological diseases, and family history. The TLE participants were asked for a history of the frequency of the seizures (Low <5, Moderate 5-10, Severe >10), family history of epilepsy, age of onset of the disease, and the time from the last seizure before the examination.

2- **Neurological examination (of the patient group):** to exclude symptomatic epilepsy.

3- **Otoscopic examination:** to exclude external ear diseases and confirm intact tympanic membrane.

4- **Basic audiological evaluation** using the two-channel diagnostic audiometer; Madsen, Model "Orbiter 922", version 2, and an immittancemeter model "Zodiak 902, Denmark". Air-conducted stimuli and speech audiometry were conducted through TDH-39 supra-aural headphones. This evaluation included:

a) **Pure-tone audiometry** involving air conduction stimulation at the octave frequencies 250 Hz through 8000 Hz and bone conduction stimulation at the octave frequencies 500 Hz through 4000 Hz.

b) **Speech audiometry** including speech reception threshold (SRT) using the Arabic bisyllabic words for adults and word recognition score (WRS) using the Arabic phonetically-balanced words for adults[6]. Pure-tone and speech audiometry were performed in a sound-treated booth.

c) **Immittancemetry** involving both tympanometry and contra-lateral acoustic reflex, which were elicited using the pure tones of 500, 1000, 2000, and 4000 Hz.

5- **Slow cortical response (P1-N1-P2-N2):**

A. **Task:** The participants were asked to keep their eyes open, remain awake, and ignore the stimuli by reading during the recording.

B. **Electrode montage:** To reduce electrical resistance between the electrode and the skin, the contact area was scrubbed with an abrasive paste and wiped down with alcohol. Below 2k Ohms was the limit set for the impedance. When the impedance is too high, noise levels rise. In this research, we used a set of four disposable adhesive electrodes coated in a conductive gel to pick up the response. The 10-20 International Electrode System was followed to place the electrodes as follows: (a) Non-inverting electrode: at the middle of the high forehead (Fz). (b) Two inverting electrodes: on the right and left mastoids. (c) Ground electrode: below the non-inverting electrode with a space of about 2 cm between both.

C. **Stimulus and recording parameters:** The auditory evoked potential system, Oto access, model Eclipse 25, version 1.3 was used to conduct a 2000 Hz tone burst to induce SCR in a sound-isolated room. It has a 15-ms rise/fall time and a 50-ms plateau. An insert earphone was used to deliver the stimulus to the ear being evaluated at 70 dB nHL. A stimulus rate of 0.5/s was used to present a total of 100 stimuli of alternating polarity. The low pass filter had a sample rate of 6/octave and a band-pass filter of 0.1-100 Hz. The time window extended from 150 ms pre-stimulus up to 750 ms post-stimulus. An artifact rejection level of ± 80 μV was used during the recording.

D. **Response analysis and measurement:** The waveform morphology of P1, N1, P2, and N2 was evaluated. In each ear, two traces were obtained to ensure replicability. The latency measure of these waves was estimated as time in ms between the stimulus beginning and the center of the peak or trough. In addition, the amplitude measure was obtained for the amplitude complexes P1N1, N1P2, and P2N2[7].

Ethical approval:

All participants provided written consent prior to examinations after an explanation of the objective and procedure. The Institutional Research Board (IRB) of Zagazig University (IRB#:7087-15-8-2021) has approved this study. The Helsinki Declaration was upheld throughout the course of the investigation.

Statistical analysis

Statistical Package for the Social Sciences (SPSS), version 20, was utilized to analyze the collected data. Frequencies and percentages were used to represent qualitative data. Chi-square (X^2) test

determined the relationship between different qualitative variables. After the Shapiro-Wilk test was run to ensure that the data were normally distributed, the mean, standard deviation (SD), range, and confidence limit (CL) were displayed for the numerical data. Two sets of numbers were compared using a parametric test (the independent sample t-test).

Pearson's correlation coefficient was used to analyse the associations between the variables. Finally, the validity of SCR measures was estimated by calculating the sensitivity, specificity, positive predictive value, and accuracy. The diagnostic accuracy of the test was shown by the receiver operating characteristic (ROC) curve was also demonstrated. To be statistically significant, the p-value has to be less than 0.05.

RESULTS

Two groups were examined in this study; control and TLE patients (n=19 each) with a mean Age±SD of 27.74±6.62 and 29.89±11.23 years, respectively. The gender distribution was homogenous among the control and TLE groups (males = 26.32% versus 63.12%, females = 73.68% versus 36.84%, X^2 = 5.43, p = 0.07, respectively). The history characteristics of the TLE group revealed a positive family history in 31.58%, a mean age of onset of 19.07±10.34 years, a predominance of the cases with a frequency of seizures of less than five (89.47%), a range of time of last seizure before examination of one day to two years, and a mean duration of TLE of 10.78±7.32 years.

All participants in this study had bilateral normal middle ear functions as implicated with bilateral type (A) tympanogram with preserved ipsilateral acoustic reflexes at 0.5, 1, 2, and 4 kHz in both ears. They also exhibited bilateral normal hearing sensitivity in the frequency range of 0.25 to 8 kHz with excellent WRS% that was matching in the two groups.

Analysis of SCR waveforms distribution revealed a homogenous distribution among the control versus TLE groups in both right (χ^2=0.34, p=0.95) and left ears (χ^2=0.38, p=0.94). However, waves N1 and P2 were the most predominant in both ears of the two groups followed by P1 and N2 waves as shown in **Figure (1)**.

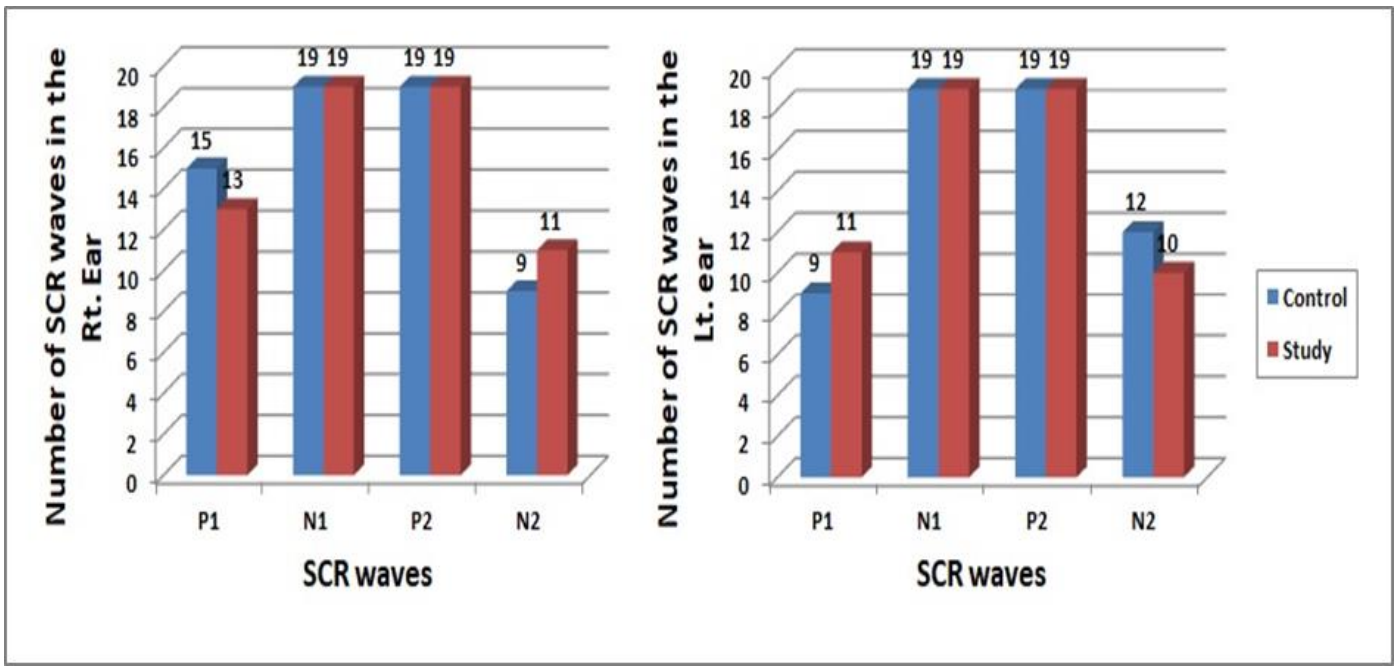

Figure (1): Waveform identification of tonal-evoked SCR in the control versus TLE groups

The comparison of SCR measures in the control versus TLE group as a response to tonal stimuli revealed a significant delay of the left ear's N1 and P2 latency in the TLE patients' group as shown in **(Table 1)**. In addition, there was a significant bilateral reduction of the N1P2 complex amplitude **(Table 2)**.

Table (1): Comparison of tonal-evoked SCR wave's latency in the control versus TLE groups

Tonal-evoked SCR latency		Control group (N=19)			TLE group (N=19)			*t*	*p*
		Mean (±SD)	Range	95%CL	Mean (±SD)	Range	95%CL		
P1	**Rt**	44.33 (4.17)	40-50	42.02-46.64	46.67 (5.77)	40-60	43.54-50.31	1.22	0.23
	Lt	46.67 (7.07)	40-60	41.23-52.10	43.18 (7.17)	35-60	38.37-48.00	1.09	0.29
N1	**Rt**	88.05 (11.85)	70-110	82.34-93.76	94.11 (13.17)	70-112	86.65-98.93	1.49	0.14
	Lt	90.05 (7.19)	80-106	86.59-93.52	101.47 (13.28)	90-120	95.09-106.48	3.30	**0.002**
P2	**Rt**	160.47 (14.27)	124-185	153.59-167.35	172.95 (28.13)	134-215	161.52-187.54	1.72	0.09
	Lt	170.32 (13.22)	150-190	163.95-176.69	187.11 (28.19)	130-225	176.91-202.15	2.35	**0.02**
N2	**Rt**	243.33 (15.81)	230-270	231.18-255.48	239.18 (44.65)	186-285	215.04-268.78	0.27	0.79
	Lt	250.83 (13.79)	230-270	242.07-259.59	239.30 (24.05)	210-270	232.27-257.73	1.41	0.17

Rt= Right; Lt= Left; SD=Standard deviation; CL=Confidence limits; *p*=significance value; SCR=slow cortical response; *t*=independent sample *t*-test.

Table (2): Comparison of tonal-evoked SCR wave's amplitude in the control versus TLE groups

Tonal-evoked SCR amplitude		Control group			TLE group			*t*	*p*
		Mean (±SD)	Range	95% CL	Mean (±SD)	Range	95% CL		
P1N1	**Rt**	3.71 (1.38)	2-6	2.95-4.48	2.74 (0.64)	1.50-3.70	2.15-3.11	2.07	0.05
	Lt	3.66 (0.49)	3-4	3.28-4.03	3.18 (0.55)	2-4.10	2.75-3.60	2.84	0.007
N1P2	**Rt**	5.93 (1.82)	2.10-11	5.05-6.80	3.42 (1.82)	0.60-5.60	2.48-4.35	4.13	**<0.001**
	Lt	5.78 (1.05)	3.40-8.20	5.28-6.29	3.86 (2.07)	0.80-7.20	3.25-5.18	3.57	**0.001**
P2N2	**Rt**	2.94 (0.87)	1.50-4	2.27-3.62	2.17 (1.45)	0.50-4.20	1.15-3.21	1.39	0.18
	Lt	2.72 (0.70)	1.90-3.60	2.27-3.16	1.91 (1.86)	0.60-5.80	0.75-3.68	1.37	0.19

SD=Standard deviation; CL=Confidence limits; p=significance value; SCR=slow cortical response; t=independent t-test sample; Rt=right; Lt=left.

Moreover, the effect of different variables (personal and history-related) on the outcomes of SCR measures in TLE patients was studied **(Tables 3 and 4)**. Age was negatively associated with latency measurements (weak to strong) and positively associated with amplitude measures (weak to strong). Gender exhibited a significant effect mainly on wave P2 and N2 latency, with a relatively non-significant effect on other waves' latency or amplitude measures. Generally, a family history of epilepsy showed a non-significant effect on most of the SCR measures. The age of onset of TLE and time of last seizure had negative, weak-to-moderate relationships with the latency measures and positive, weak-to-moderate relationships with the amplitude measures. In addition, higher seizure frequency was found to be associated with delayed P1 latency.

Table (3): Effect of different variables on tonal-evoked SCR waves' latency in the TLE group

Different variables	Tonal evoked SCR							
	P1		N1		P2		N2	
	Rt	Lt	Rt	Lt	Rt	Lt	Rt	Lt
Age (*r*(*p*))	-0.427 (0.17)	-0.541 (0.08)	-0.429 (0.67)	-0.480 **(0.03)**	-0.151 (0.54)	-0.176 (0.47)	-0.474 (0.14)	-0.352 (0.32)
Gender (*t*(*p*))	0.16 (0.88)	2.32 **(0.04)**	0.98 (0.30)	0.81 (0.43)	3.87 **(0.001)**	3.09 **(0.007)**	3.01 **(0.01)**	3.04 **(0.02)**
FH (*t*(*p*))	0.48 (0.64)	1.60 (0.14)	0.50 (0.62)	0.88 (0.39)	2.59 **(0.02)**	1.10 (0.29)	2.15 (0.06)	0.09 (0.93)
Age of onset (*r*(*p*))	-0.592 (0.05)	-0.575 (0.082)	-0.470 (0.066)	-0.281 (0.29)	-0.045 (0.87)	-0.093 (0.73)	-0.270 (0.45)	-0.274 (0.48)
Freq. of seizures (F(*p*))	6.50 **(0.02)**	13.83 **(0.005)**	0.62 (0.55)	1.13 (0.35)	1.03 (0.38)	0.01 (0.99)	1.66 (0.23)	0.20 (0.67)
Time of last seizure (*r*(*p*))	-0.408 (0.21)	-0.235 (0.51)	0.563 **(0.02)**	-0.195 (0.47)	-0.003 (0.99)	-0.141 (0.60)	-0.141 (0.70)	-0.630 (0.06)
Duration of TLE (*r*(*p*))	0.354 (0.26)	0.083 (0.81)	0.405 (0.06)	0.204 (0.40)	0.115 (0.64)	0.222 (0.36)	0.220 (0.52)	0.408 (0.07)

FH=family history; TLE=temporal lobe epilepsy; SCR=slow cortical response; Rt=right; Lt=left. *p*=significance value; *t*=independent sample *t*-test; *r*=correlation value; F=One-Way-ANOVA value.

Table (4): Effect of different variables on tonal-evoked SCR wave`s amplitude complexes in the TLE group

Different variables	Tonal evoked SCR					
	P1N1 Amplitude		N1P2 amplitude		P2N2 Amplitude	
	Rt	Lt	Rt	Lt	Rt	Lt
Age (*r*(*p*))	0.411 (0.24)	0.139 (0.72)	0.193 (0.46)	0.036 (0.89)	0.314 (0.38)	0.572 (0.14)
Gender (*t*(*p*))	0.19 (0.86)	0.04 (0.97)	1.44 (0.17)	1.15 (0.27)	0.57 (0.58)	0.10 (0.93)
FH (*t*(*p*))	2.23 (0.05)	1.48 (0.18)	1.13 (0.28)	1.82 (0.08)	0.28 (0.79)	0.27 (0.80)
Age of onset (*r*(*p*))	0.368 (0.33)	0.233 (0.58)	0.219 (0.45)	0.140 (0.63)	0.167 (0.67)	0.530 (0.22)
Freq. of seizures (F(*p*))	0.22 (0.81)	12.21 **(0.01)**	1.63 (0.23)	1.35 (0.29)	1.14 (0.37)	0.18 (0.69)
Time of last seizure (*r*(*p*))	0.036 (0.93)	0.472 (0.24)	0.176 (0.55)	0.186 (0.52)	0.488 (0.18)	0.209 (0.65)
Duration of TLE (*r*(*p*))	-0.448 (0.19)	-0.584 (0.09)	-0.217 (0.40)	-0.215 (0.41)	-0.092 (0.80	-0.320 (0.44)

FH=family history; TLE=temporal lobe epilepsy; SCR=slow cortical response; Rt=right; Lt=left. *p*=significance value; *t*=independent sample *t*-test; *r*=correlation value; F=One-Way-ANOVA value.

Depending on validity measures and ROC curve analysis, the sensitivity of tonal-evoked SCR wave latencies ranged from 47.4% to one hundred percent while the specificity ranged from 42.1% to 83.3%. The best accuracy found was for N1 and then P2 latencies (in the left ear; 82% and 73%, respectively) **(Table 5 and Figure 2)**. Moreover, the amplitude measures exhibited a sensitivity ranged between 50% to 94.7% and a specificity ranged between 63.2% to 92.3%. The best accuracy was for the N1P2 complex amplitude in the right ear (81%) **(Table 6 and Figure 3)**.

Table (5): Validity of tonal-evoked SCR latency in CAPD diagnosis in TLE patients

Tonal SCR measures		Sensitivity (%)	Specificity (%)	Positive predictive value (%)	Accuracy (%)
P1	Rt	73.3%	53.8%	61.3%	61%
	Lt	55.6%	83.3%	76.9%	68%
N1	Rt	47.4%	78.9%	69.2%	62%
	Lt	84.2%	78.9%	78%	82%
P2	Rt	100%	42.1%	63.3%	61%
	Lt	84.2%	63.2%	69.6%	73%
N2	Rt	100%	45.5%	64.7%	55%
	Lt	83.3%	55.6%	65.2%	72%

Rt=right, Lt=left, SCR=slow cortical response.

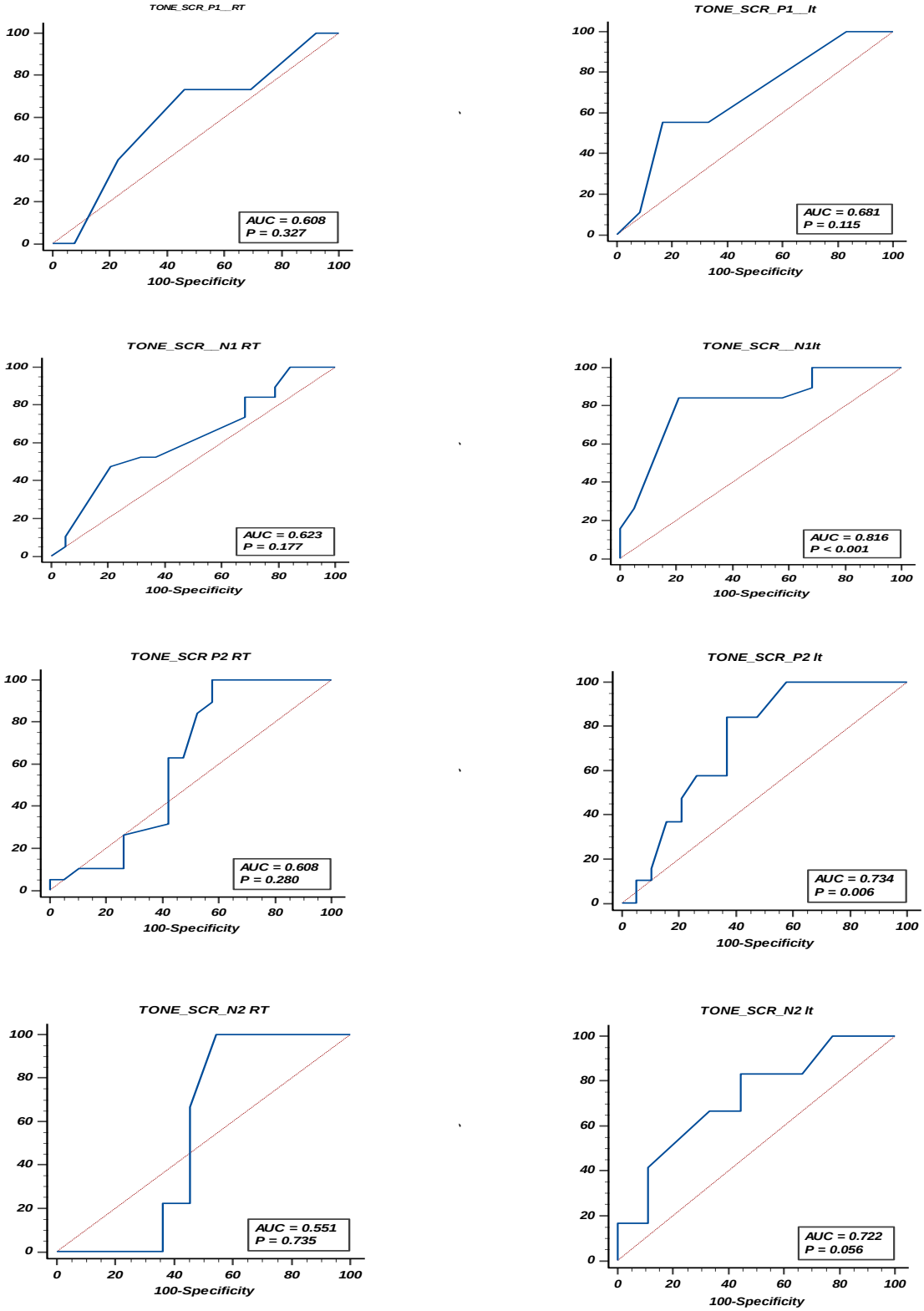

Figure (2): ROC curve analysis of tonal SCR latency measures in predicting CAPD in TLE patients.

Table (6): Validity of tonal-evoked SCR amplitude measures in CAPD diagnosis in TLE patients

Tonal SCR amplitude tests		Sensitivity (%)	Specificity (%)	Positive predictive value (%)	Accuracy (%)
P1N1	**Rt**	53.3%	92.3%	87.4%	72%
	Lt	66.7%	91.7%	88.9%	74%
N1P2	**Rt**	63.2%	89.5%	85.8%	81%
	Lt	94.7%	63.2%	72%	76%
P2N2	**Rt**	77.8%	72.7%	74%	61%
	Lt	50%	77.8%	69.3%	57.5%

Rt=right, Lt=left, SCR=slow cortical response.

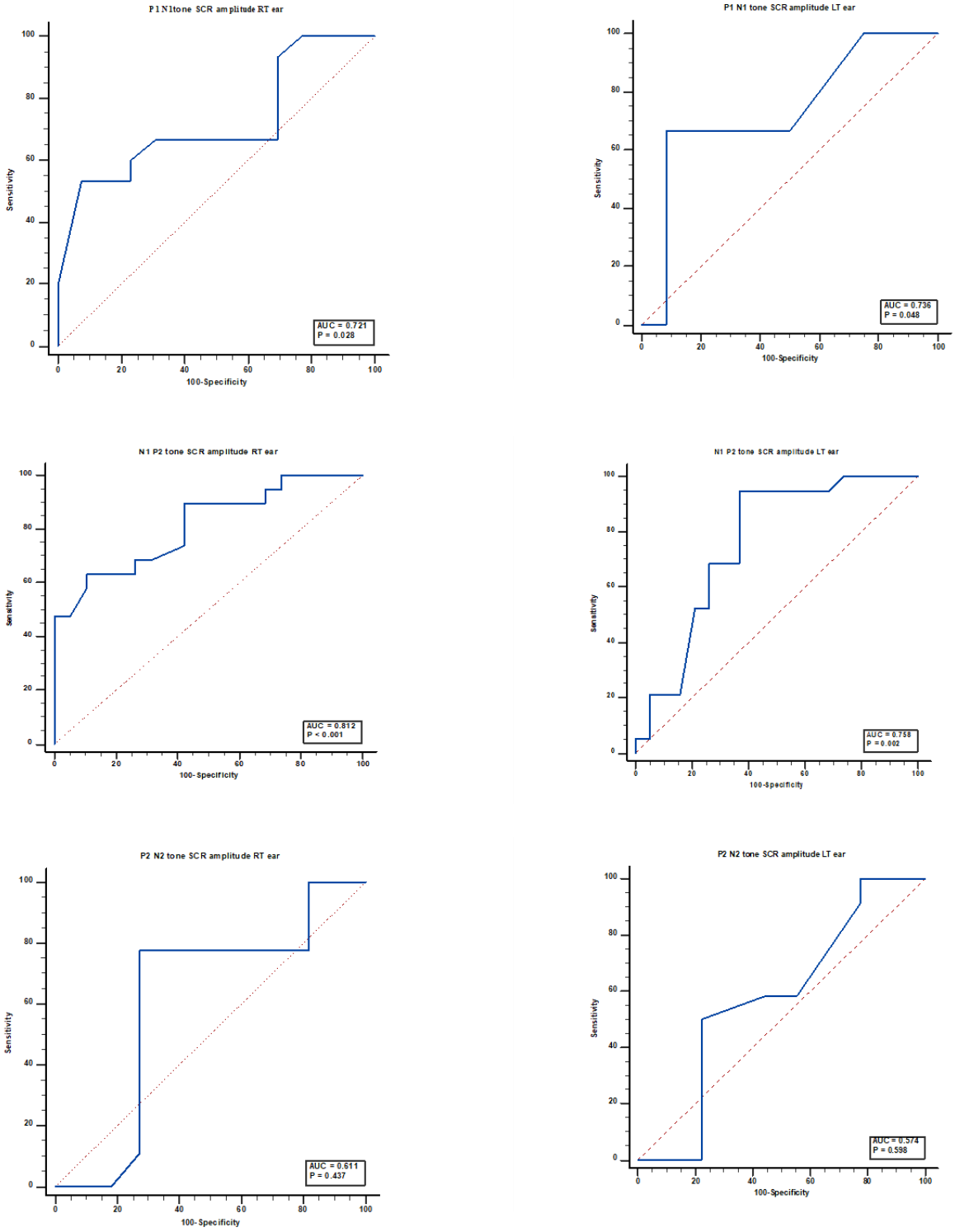

Figure (3): ROC curve analysis of tonal SCR amplitude measures in predicting CAPD in TLE patients

DISCUSSION

The SCRs have been used in this study as electrophysiological correlates to evaluate the effect of TLE on auditory processing and to predict its value in the diagnosis of CAPD in TLE patients. SCRs are obligatory exogenous evoked potentials that can be elicited by different stimuli. The most identifiable SCR waves were N1 and P2 in the two groups, followed by P1 and N2 waves. Likewise, **Didoné *et al.*** [8] studied SCR in 30 normal adults of both genders with a mean age of 23.3 ± 3.5 years. The N1 and P2 waves were visualized in all patients, whereas the P1 and N2 waves were less commonly detected. In congruent with this outcome, the N1 and P2 components were found to dominate and their morphology matures in adults and older children [7].

Furthermore, the comparison of SCR measures in the control versus TLE group as a response to tonal stimuli revealed a significant delay of the left ear's N1 latency and P2 latency in the patient group. In addition, there was a significant bilateral reduction of the N1P2 complex amplitude in the TLE patients.

Relatively similar results were demonstrated in the literature [9-12]. **Knight *et al.*** [9] reported that N1 amplitudes were significantly reduced (by about 63%) in ten TLE cases, their mean age was 53 years, using 800 Hz tone-burst stimuli. **Japaridze *et al.*** [10] examined 43 normal-hearing subjects (27 males and 16 females) having partial epilepsy (22 patients were diagnosed with temporal epilepsy and 21 with extra-temporal epilepsy) with an age range of 14-44 years, using 1 kHz tone-burst stimuli. They found no statistically significant difference between the patients and the control group in either ABRs or MLRs. Similar to our results, P2 peak latency was prolonged but conversely, amplitudes of P1N1 and N1P2 were enlarged. It has been hypothesised, in light of ABR and MLR processes, that the individual components of the central auditory system up to the primary cortex have no decisive role in the etiology of partial epilepsy. In addition, the cortical regions responsible for generating SCRs are thought to play a crucial role in epilepsy mechanisms despite their largely generic nature. They explained their results by the fact that it is common for the SCR parameters to exhibit an inverse beahviour: when the amplitude is increased, the peak latency is decreased, and vice versa. Rarely do peak latencies and amplitudes of SCRs change in tandem. Specifically, these occur as a function of ISI, with longer SCR latencies and larger amplitudes being observed at lower stimulus rates and shorter latencies and smaller amplitudes being observed at higher rates. Separate SCR deflections were thought to indicate a mixture of many components, each one corresponding to the discharge of a different neuronal population, and this was proposed as an explanation for the observed behavior.

Furthermore, **Brodtkorb *et al.*** [11] recorded SCRs to binaural tones in the left and right hemispheres from eight patients with lateral TLE, aged from 19-75 years. Patients with TLE were observed to have decreased N1P2 amplitude over the left hemisphere. Possible explanations for their findings were insufficient cortical excitation, excessive cortical inhibition, and a smaller-than-average left auditory cortex.

The functional integrity of the auditory regions was better reflected in the auditory-evoked magnetic fields measured in a prior investigation utilising monaural tone bursts in 17 right-handed individuals with mesial TLE (ages 23 to 58, among eleven females) [12]. The M100 component was measured, and it is the magnetoencephalographic analogue to the auditory N100 that originates in the planum temporale and the Heschl's gyrus on hemispheres [13]. The M100 amplitudes were negatively correlated with the volume of Heschl's gyrus in the mesial TLE region [12]. Their findings are consistent with the hypothesis that TLE compromises the normal temporal lope's structural and functional integrity and hence affects auditory processing.

Limited data are available in the literature about SCR outcomes in TLE patients and the effect of different variables on them. The effect of different variables on the electrophysiological tonal SCR measures revealed a negative, weak-to-strong relationship between age and latency measures. Meanwhile, there was a positive, weak-to-strong relationship between age and amplitude measures. Despite structural and functional changes in epilepsy, this did not interfere with the normal sequence of SCR evolution that involves latency reduction and amplitude enhancement with age increase. Most of the patients' epilepsy was under control, thus neuroplasticity may have aided in the remodeling of their auditory pathways. This can be thought of as the central nervous system undergoing physiological changes in reaction to external stimuli [14].

Gender exhibited a significant effect mainly on the tonal wave P2 and N2 latency, with a relatively non-significant effect on other waves' latency or amplitude measures. Similarly, **Japaridze *et al.*** [10] denoted a significant effect of gender on tonal-evoked P2 latency with the latency in males being longer than that in females, owing to anatomical and skull size variations.

Generally, a family history of epilepsy showed a non-significant effect on most of the SCR measures. The age of onset of TLE and time from the last seizure had negative, weak-to-moderate relationships with the latency measures and positive, weak-to-moderate relationships with the amplitude measures. This suggests that the earlier onset of TLE and the later time of the last seizure are associated with electrical discharges that could lead to extensive functional changes in the temporal lobe.

Another factor is the frequency of seizures. Greater seizure frequency showed an association mainly with a delay in tonal-evoked P1 latency but did not

affect other waves' latency or amplitude measures. In addition, the duration of TLE displayed a positive, weak-to-moderate relationship with the latency measures and a negative, weak-to-moderate relationship with the amplitude measures. These results are consistent with the location of TLE's aberrant electrical discharges at the auditory pathway's final destinations. Dysfunction in various regions may be caused by such discharges [(14)]. However, the results of **Japaridze *et al.*** [(10)] revealed no significant dependence of tonal-evoked SCR upon the rate of seizures nor the duration of the disease in patients with partial epilepsy. The difference between the two studies could be related to that the sample in the latter one involved both temporal and extra-temporal epilepsy.

Upon performing validity statistics and reading the ROC curve analysis, the sensitivity of SCR wave latencies ranged from 47.4% to 100% while the specificity ranged from 42.1% to 83.3%. The best accuracy was for N1 and then P2 latencies (in the left ear; 82% and 73%, respectively). In addition, the amplitude measures exhibited a sensitivity ranging from 50% to 94.7% and a specificity ranging from 63.2% to 92.3%. The best accuracy appeared for the N1P2 complex amplitude in the right ear (81%). It has been denoted that the latency and amplitude measures of N1 and P2 waves were the most affected in central auditory lesions and their abnormalities have the highest accuracy in predicting these lesions. Probably, this is because N1 and P2 are the most predominant waves in adults; the main age of our TLE group. **Luauté *et al.*** [(15)] have concluded that the SCRs (especially N1) are strong predictors of functional integrity in adults.

CONCLUSION

Electrophysiological assessment with SCR revealed altered neural activities within the temporal lobes of patients with TLE. Consequently, SCRs, especially N1 and P2 waves' measures, help to provide useful information about possible functional deterioration and impaired CAP in this population. Alteration in the latency and amplitude measures of N1 and P2 waves provided the highest accuracy values and seemed to be the most accurate in predicting CAPD in TLE patients. Therefore, evaluation of SCR in patients with TLE would increase the awareness of auditory processing abnormalities and hence earlier treatment can be implemented. Proper treatment and management for these individuals are crucial and could help improve overall quality of life.

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