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Analysis of Auditory Evoked Late Latency Potentials in Stuttering

Kekemelerde İşitsel Uyarılmış Geç Latans Potansiyellerinin Değerlendirilmesi

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ABSTRACT

Objective: Our aim is to evaluate and compare the cortical responses of stutterers and fluents by using auditory late latency responses with the hypothesis of stuttering may result with the auditory feedback delay. The relationship between the auditory late latencies and stuttering durations were also investigated. **Material and Methods:** Fifteen male right-handed stutterers aged between 18 and 43 years without any hearing loss and neurological problems, and 15 male fluents participated in this study. Immitansmetric evaluation, distortion product otoacoustic emission, and pure tone audiometry were performed to all fluent and stutterer individuals after otoscopic examination. Auditory late latency responses were obtained for those with normal audiological findings. Click stimulus was given to their right ears. Silent images on the computer were shown to the subjects during the test. **Results:** P1 wave of the auditory evoked late latency potential latencies were found as 56.68±7.37 msec for stutterers, and 57.36±7.74 msec for fluents. P1 amplitudes were 0.71±0.53 μV for stutterers, and as 0.73±0.51 μV for the control group. Stuttering duration of stutterer subjects ranged from 11 to 33 years (mean 20.2 years), and was not correlated to P1 latencies. **Conclusion:** Stutterers and fluents were not significantly different regarding the P1 latency or amplitude of the auditory evoked late latency potentials. This shows that stutterers have no problem of realization of sounds. Absence of any relationship between stuttering duration and auditory late latency responses is another result of our study.

Keywords
Auditory evoked potentials; speech disorders; stuttering

ÖZET

Amaç: Kekemeliğin işitsel geri bildirimde gecikmeye sebep olabileceği hipoteziyle, işitsel uyarılmış geç latans potansiyeller kullanılarak kekemelerde ve akıcı konuşanlarda kortikal cevapların değerlendirilmesi ve karşılaştırılması amaçlanmıştır. Ayrıca işitsel geç latanslar ve kekemelik süresi arasındaki ilişki de incelenmiştir. **Gereç ve Yöntemler:** Çalışmaya 18-43 yaş arasında sağ elini kullanan, işitme kaybı ve nörolojik problemi olmayan 15 kekeme ve 15 normal konuşan (kontrol grubu) erkek birey alındı. Akıcı konuşan ve kekeme bireylere otoskopik muayene sonrasında immitansmetrik inceleme, distorsiyon ürünü otoakustik emisyon ve saf ses işitme testleri yapıldı. Normal odyolojik bulgular elde edildikten sonra geç latans potansiyel kayıtları alındı. Katılımcıların sağ kulaklarından klik uyaran verildi. Bireylere test esnasında bilgisayar aracılığı ile sessiz resimler gösterildi. **Bulgular:** İşitsel uyarılmış geç latans potansiyellerinden P1 dalga latansı kekemelerde 56,68±7,37 msn, akıcı konuşanlarda ise 57,36±7,74 msn olarak bulunmuştur. P1 dalga amplitüdü kekemelerde 0,71±0,53μV, akıcı konuşanlarda ise 0,73±0,51 μV olarak bulundu. Kekeme bireylerin kekemelik süreleri 11 ile 33 yıl arasında (ortalama 20,2 yıl) değişmekte olup P1 latansları ile korelasyon göstermemektedir. **Sonuç:** Kekeme ve akıcı konuşanlar arasında işitsel uyarılmış geç latans potansiyellerinden P1 dalgasının latans ve amplitüdleri açısından istatistiksel anlamlı fark yoktur. Bu bulgu, kekemelerin sesleri anlamalarında herhangi bir problemleri olmadığını göstermektedir. Kekemelik süresi ve işitsel geç latans cevapları arasında herhangi bir ilişki bulunmayışı ise çalışmamızın bir diğer sonucudur.

Anahtar Sözcükler
İşitsel uyarılmış potansiyeller; konuşma bozukluğu; kekemelik

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INTRODUCTION

Stuttering is a speech disorder characterized by involuntary stoppages in the stream and rhythm of person's speech, not in compliance with the age. It usually begins between the ages of 3 and 8 years with an uncertain cause, and can recuperate frequently in the preadolescence.[1-4]

In stuttering, there is psychological, physiological and neurological rhythm disturbances such as repetitions of sounds and syllables, prolonging sounds, hesitations, exclamation, substituting other words instead of jawbreakers, and stuttering words with physical strain.[5-7] Despite a large number of brain and behavioral research, the cause of the stuttering has not been proven, and it is not known whether it is environmental or anatomical. Psychological, neurological, genetic and biomechanical causes, language and speech are the research topics for this interesting entity.[3,4,8]

Auditory evoked potentials are the wave forms that rises from pathway along cochlear to cerebral cortical area as an electrophysiological response of central nervous system to different stimuli such as click, tone burst, tone bip.[9,10] The auditory evoked late latency responses (ALR) are the first auditory electrical responses that have been obtained from central nervous system, thalamic and auditory cortical areas.[11-13] P1 wave of the ALR take source from secondary auditory cortex (lateral Hechl's Gyrus), and N1 wave from several distinct generators (lateral supratemporal) located in primary auditory cortex, frontal lobe and midbrain.[14]

Auditory processes that are in cerebral cortex are screened by ALR.[15] The waves which occur between 50-300 msec are defined as P1, N1, P2 and N2. The waves are observed are P1 between 50 and 80 msec, N1 between 80 and 100 msec, P2 between 180 and 200 msec, and N2 between 200 and 300 msec. The titles express positive and negative voltage polarity of the responses recorded from vertex.[10,14,16-18] Exposure to repetitive stimuli result in decrease of the negative (N1) and positive (P1) components' amplitudes.[19]

In our study, we aimed is to investigate and compare the cortical responses of stutterers and fluents by using auditory late latency potentials, and hypothesized that stuttering may result from an auditory feedback delay.

MATERIAL AND METHODS

This study was performed in the Audiology Unit of a tertiary academic center. The study protocol was approved by Institutional Clinical Research and Ethics Board (Feb 09, decree no: 9). All individuals participated in the study were informed about the study, and they provided their verbal and written informed consents. The study population composed of 15 right-handed stutterers between the ages of 18-41 years (mean age 30.6±3.2 years) without any hearing loss or neurological problems; and 15 fluent right-handed males between the ages of 19-43 years (mean age 27±4.6 years) without any hearing loss or neurological problems. After all of the participants have ear, nose and throat examinations, the immitansmetric study, distortion product otoacoustic emissions (DP-OAE) and pure tone audiometry were performed. The individuals with normal audiological examinations were, included in the study.

Stimulus type and properties used for late latency recordings

Medelec Synergy T-EP system (Medelec Synergy, Oxford Instruments Medical, Surrey, UK) ABR device was used to give click stimuluses, through TDH-49 headphones, and to record cortical auditory evoked late latency responses. The repetition of stimulus was determined as 1.1 pulse per second (pps).

Recording the late latencies

The cortical potentials were recorded in a silent chamber in the audiology unit. During the test, the subjects were exposed to soundless pictures on the computer screen in a sitting position. P1 and N1 waves of the auditory evoked late latencies are to be expected in the first 100 msec of the recording. Therefore, the analysis interval was determined as -100 msec and +100 msec. The electrodes were placed according to the international 10-20 system to record evoked potentials. Vertex for (Cz) noninverting electrode, forehead for (Fpz) ground electrode and right mastoid for (M1) inverting electrode were the installation points (Table 1).

The testing period for each patient including audiological evaluation, electrode assembly and recording of the evoked potentials was approximately 60 minutes. Verbal and written descriptive information was presented to the subjects about the test.

Table 1. Parameters used in late latency recordings.

	Parameters
Analysis interval	100 msec before the stimulus, 100 msec after stimulus
Frequency	Click
Stimulus intensity level	75 dB SPL
Filtering	1-30 Hz
Number of channels	One
Averaging	250 sweep
Repetition of stimulus	1 pps
Electrode assembly	Vertex (Cz), Forehead (Fpz), right mastoid (M1)
Artifacts Rejection	+/- 100 ∞V

Statistical Analysis

SPSS for Windows 12.0 (SPSS Inc., Chicago, IL, USA) was used for statistical analysis. Student's t test was used to compare normally distributed parameters between the groups while Mann-Whitney U test was used to compare abnormally distributed parameters. The results were evaluated at 95% confidence interval, and considered as statistically significant if $p<0.05$. In addition, correlation between the P1 and N1 wave latency and duration of stuttering were analyzed with Pearson (r) test.

RESULTS

Latencies and amplitudes of P1 wave of the fluent subjects are shown in Table 2, and of the stutter subjects are shown in Table 3.

P1 wave latencies of fluents were found between 42.00 and 66.00 msec, and the mean latency was 57.36±7.74 msec while the latencies of stutterers were found between 41.70-72.00 msec with a mean latency of 56.68±7.37 msec. No statistically significant difference was found between P1 wave latencies of stutterers and their fluent peers ($p>0.05$).

P1 wave amplitudes of fluents were found between 0.96 and 1.90 μV with a mean amplitude of 0.73±0.51 μV; and the amplitudes of stutterers were between 0.16 and 2.20 μV with a mean amplitude of 0.71±0.53 μV. Similarly, no statistically significant difference was found between P1 wave amplitudes of stutterers and fluents ($p>0.05$).

Mean stuttering duration of the stutterer subjects was 20.2 years, and ranged between 11 and 33 years. The stuttering durations and P1 wave latencies of stutters were compared. No correlation was detected between stuttering time and P1 wave latencies (r= -0.07).

Table 2. The latencies and amplitudes of the fluent subjects.

Fluents (Name)	Age	P1 latency (msec)	P1 amplitude (μV)
KF	19	64.20	0.19
BM	20	49.80	0.19
FU	25	60.20	0.51
OK	27	62.80	0.26
UT	27	64.80	1.10
VD	29	42.00	0.54
GK	29	48.60	1.90
CG	31	66.20	0.43
AC	31	60.20	1.00
SU	35	53.40	0.31
BE	35	62.00	1.00
NY	35	60.80	1.50
MEC	36	44.40	0.96
RD	37	62.20	0.23
MO	43	58.80	0.77

DISCUSSION

Despite a great number of neurologic and behavioral studies, the cause for stuttering is still not known.[20] Various theories have been proposed about causes of stuttering including cerebral dominance theory, functional disability of the basal ganglion which provides motor coordination, theory of learning, diagnosogenic theory, psycholinguistic theory, and psychological and organic theories.[3,4]

In our study, we aimed to investigate and compare the cortical responses of stutterers and fluents by using auditory late latency potentials, and hypothesized that stuttering might result from an auditory feedback delay.

Table 3. The latencies and amplitudes of the stutterer subjects.

Stutters (Name)	Age	P1 latency (msec)	P1 amplitude (μV)
HA	18	58.60	0.16
AC	18	49.20	0.22
HG	19	49.60	0.44
AA	23	61.00	0.56
SM	23	41.40	1.20
SA	24	66.40	0.48
MŞ	25	55.80	1.30
DO	25	52.40	0.38
AS	25	53.60	2.20
MOD	27	59.60	0.70
EA	30	58.20	0.72
CG	35	61.20	0.93
IT	35	53.80	0.65
ES	37	72.00	0.54
TY	41	57.40	0.20

Genetic studies reported that stuttering was more common among males, and it occurred more frequently if there is a positive family history.[21] It has been shown that stuttering is observed in 10% of the daughters and 20% of the sons of stutterer individuals.

The results of imaging studies indicated abnormalities in the primary auditory cortexes of the stutterers.[21-23] It has been supposed that insufficiency in speech and motor skills arise from the left hemisphere. Those were tried to demonstrated with positron emission tomography (PET) and magnetic resonance imaging (MRI). The brain imaging studies of the stutterers pointed out the difference of right and left hemispheric areas. Both hemispheres of brain take charge in production of speech. Left hemisphere adjusts the transition of the sounds while the right hemisphere is associated with integrality, music and emotions.

According to the theory of cerebral dominance, left hemisphere that provides fast pass of signs during conversation is not dominant enough in stutterers. In addition, decreased dominance of the left hemisphere indicates the cause of the increase in emotional activity is associated with stuttering. Insufficient activation of the left hemisphere's temporal lobe, which involves motor and language areas, and the abnormalities in right fronto-temporal network anatomy have been shown in functional magnetic resonance imaging (f-MRI) and other neuroimaging studies performed on stutterers.[21-24] In contrast, MRI, f-MRI and PET studies suggest that there are failure of activation in those areas of brain, and biochemical failure.

Sharma et al found mean P1 wave latency 59 msec, and the mean amplitude of P1 wave as 1.2 μV, with using /ba/ sound on 10 fluent adults between the ages of 21-27 years with normal hearing.[25] Poulsen et al. reported that P1-N1 wave latencies decreased with age in normal-hearing adults aging between 19 and 45 years.[18] In that study, they reported the P1 wave latency as 58 msec. They also found mean P1 amplitude as 0.76 μV. Hung Jang et al. studied the individuals between the ages of 2-17 years with normal hearing, found P1 wave latency as 122 61 msec, and indicated that the wave length decreased with maturation.[26]

In our study, P1 wave latency was 57.36 msec in individuals having normal speech, and 56.68 msec in stutterers. P1 amplitude was as 0.73 μV in fluents, and as 0.71 μV in stutterers.

Auditory evoked cortical potential studies on male adults with normal hearing found mean P1 latency as 50 msec.[27,28] The results of the auditory evoked late latency potential records in stutterers and fluents were consistent with the literature in our study.

Click, tone burst and speech stimulus have been used in cortical potential recordings. In our study, we found that the results of cortical potentials obtained with click stimuluswere similar to the results obtained with tone burst and speech stimulus.[15,16,19,28] Click stimulus can also be used reliably for recording cortical potentials.[17]

We designed a study to investigate the thalamocortical and primary auditory areas of male stutterers using late latency potentials. We compared the latencies and amplitudes of auditory evoked late latency potentials of 15 male stutterers and fluent subjects. We did not find any statistically significant difference between normal speakers and stutterers. The results of our study did not show any difference arising from hearing and speaking centers in the responses of auditory evoked late latency potentials.

Both our results and results of late latency potentials in Weber-Fox and Hampton et al.'s study indicated normal responses in primary and secondary auditory areas in stutterers.[29,30] In contrast MRI, f-MRI and PET studies suggested failure of activation in those areas of brain, and biochemical failure.[21-24]

The differences between the results of those studies may be due to analysis of one particular problem affecting obvious parts of brain with different methods. Therefore, further studies on neuroimaging and auditory evoked potentials are needed.

No statistically significant difference was detected between the auditory evoked late latency potentials of stutterers and fluents. Our results may be interpreted as such the stutterers have the same functions of the hearing and speaking centers with the fluents.

Stuttering duration was compared with P1 wave latency in stutterers. No correlation was detected between stuttering duration and P1 wave latency. This is the first study that compared stuttering duration and P1 wave latencies.

Studies with more participants and different stuttering times are needed. No studies n the literature have investigated P1 component of auditory evoked late potentials in stutters. This study is particularly important for indicating no abnormalities in the P1 wave of secondary auditory cortex (lateral Heschl's Gyrus) of the stutterers. Stutterers realize the sounds as good as fluents. Therefore, further studies on the upper cortical pathways and areas are needed. We may suggest that auditory late latencies do not have any relationship with stuttering duration.

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