

---

# Some Effects of Aging on Auditory Event-Related Potentials

---

**I. Griškova-Bulanova**  
**N. Kazlauskienė**

*Department of Neurobiology  
and Biophysics,  
Faculty of Natural Sciences,  
Vilnius University*

**Summary.** Event-related potentials provide an objective way to evaluate activity of the brain at different stages of stimulus processing-sensory, preattentive and cognitive. In Lithuania several types of auditory event-related potentials are being applied: P300 potential, mis-match negativity, P1-N1-P2 potential, P50 potential and auditory steady-state response. Potentials are applied in psychiatry and neurology to evaluate disease-related changes, to monitor effects of both medicative and non-medicative treatment, and to search for the optimal recording conditions. Obviously, when studies are performed in healthy subjects, it is easy to collect homogenous groups in respect to age. However, it is well known that patients who might benefit from event-related potential examination are of very different age. And thus, it is tempting to know the effect of aging on the routinely applied event-related potentials to provide their objective evaluation.

**Keywords:** auditory, event-related potential, age.

Neurologijos seminarai 2013; 17(56): 132–137

---

Auditory event-related potentials (ERPs) can be used to examine both exogenous (acoustic representation) and endogenous (attention and learning) aspects of sound processing [1, 2]. ERPs are comprised of positive and negative waves reflecting activation from neural resources that are involved in the processing of sensory information [1, 2].

Currently, in the research and clinical practice in Lithuania several types of auditory event-related potentials (ERPs) are being applied. These are so called P300 potential, mis-match negativity (MMN), P50 potential (and its gating), and auditory steady-state responses (ASSR). Mostly, these techniques are adapted for use in psychiatry and neurology. P300 potential have been implemented in the studies of Alzheimer disease [3, 4], schizophrenia [5], and during testing of clinical effects of both chemical medications and non-medicative therapies [4, 6–9]. MMN was applied in the studies of schizophrenia [10] and study of clinical effects of olanzapine [11]. Both P50 potential [12, 13] and ASSRs [14] were used to investigate optimal experimental conditions for ERP recordings, and ASSR was used in the study of schizophrenia [15].

When studies are performed in healthy subjects, it is easy to collect homogenous groups in respect to age and gender and overcome unwanted effects. However, it is well known that patients who might benefit from ERP examination are of very different age. And thus it is tempting

to know the effect of aging on the routinely evaluated components of the above-mentioned ERPs to provide objective evaluation. Herein, we present a brief review on the effects of age on classical and commonly used ERPs.

## P1-N1-P2 COMPLEX

Sounds must be detected before they can be discriminated, grouped, and ultimately perceived. The P1-N1-P2 complex is an ERP that signifies the physiological detection of audible stimulus energy. From this perspective, it can be seen as an index of the capacity for perception of the sound. The P1-N1-P2 response is considered as the onset response and classified as “obligatory” response because it represents the physiological detection of a sound. It can be elicited by a variety of stimuli. P1-N1-P2 responses are generated by multiple sources, including thalamo-cortical projections as well as primary and association areas of each auditory cortex, thus reflecting synchronous neuronal activation of structures in the thalamic-cortical segment of the central auditory system. The presence of a P1-N1-P2 response implies that a sound has reached the level of the cortex and is now available for further processing. Moreover, the P1-N1-P2 complex can be used to indirectly assess the integrity of the central auditory system, up to the cortex [16, 17]. The schematic representation of P1-N1-P2 complex or ERP is presented in Figure 1.

Pfefferbaum et al (1980) found no age-related differences on N1 amplitude or latency [18]. In the later study by Laffont et al (1989) increased P1-N1 amplitudes with increasing stimulus intensity were found to be more pronounced in older persons. P2 was larger and later in the

---

### Adresas:

*Inga Griškova-Bulanova, PhD, Associate professor  
Department of Neurobiology and Biophysics  
Faculty of Natural Sciences, Vilnius University  
Čiurlionio 21/ 27, Vilnius, LT-03101 Lithuania  
Phone +370 5 239 8227, fax: +370 5 239 8216  
E-Mail: inga.griskova-bulanova@gf.vu.lt*

aged subjects as well [19]. By analogy, pronounced effects of aging have also been reported for P1-N1-P2 response latencies and amplitudes: P1, N1 and P2 latencies increased with age, N1 and P2 amplitudes were enhanced [20], and N2 amplitudes were reduced [20, 21]. However, Czigler et al (1992) and Lijffijt et al (2009) showed that P2 amplitude was larger in the younger group [22, 23].

### P50 POTENTIAL AND SENSORY GATING

One of the most frequently investigated auditory evoked potentials – P50 potential – is widely applied in research practice. During classical P50 paradigm, two identical brief stimuli are presented 500 ms apart (ISI) in a pair with inter-pair interval (ITI) of 8–10 s [23]. In a sense, P50 is the same as P1, however this term is used when stimuli are presented repetitively and P50 auditory sensory gating is measured as the degree of reduction in P50 amplitude in response to stimulus repetition [24]. P50 auditory sensory gating is believed to reflect a preattentive filter mechanism [24]. Gating of P50 wave is known to be affected in diseased state [24–26], by medication or chemical compounds [27, 28] and by the level of arousal [12, 29]. A schematic representation of P50 wave and its gating are presented in Figure 2.

There are few studies investigating effect of aging on P50 potential and the relationship between age and sensory gating remains unclear. Some studies reported no changes in P50 gating with age [23, 30, 31], whereas others reported a low positive correlation between gating and age [32]. Rasco et al (2000) tested several inter-stimulus intervals and found a significant decrease in sensory gating of the P50 potential in the adolescent group compared to older age groups at the 250 msec ISI, but not at the 500 or 1000 msec ISI [33].

### P300 POTENTIAL

The most widely used ERP is P300. Generally, it is elicited by a typical oddball task, when a series of two different tone stimuli is presented with the rare tone occurring less frequent than the standard [1, 2]. The subject is required to note the occurrence of the target and to not respond to the standard. In the response to target tones late positive wave peaking at around 300 ms appears. A schematic representation of P300 wave is presented in Figure 3. Thus, P300 potential is closely reflecting cognitive functions such as stimulus evaluation and task relevance. Hence, both their potential clinical application for detecting cognitive disturbances [3, 4] and an increasing interest in the aging of cognitive human brain functions resulted in a growing number of studies on age-related P300 changes.

Effects of age on P300 potential are relatively well studied and consistent. In a number of studies P300 component showed systematic latency prolongation and am-

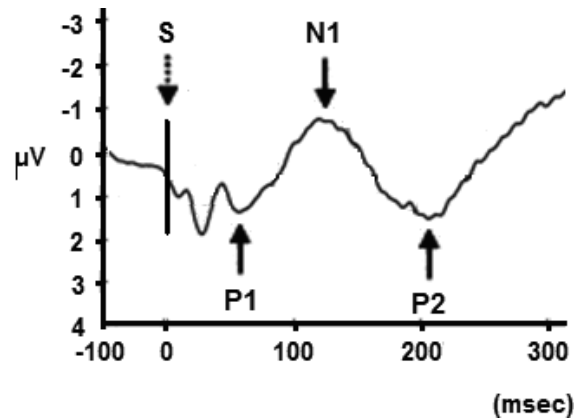


Fig. 1. A schematic representation of P1-N1-P2 complex. S marks the occurrence of the stimulus.

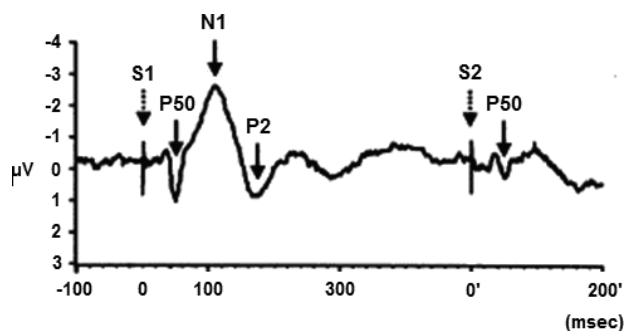


Fig. 2. A schematic representation of P50 wave and gating effect.

S1 marks the occurrence of the first stimulus; S2 marks the occurrence of the second stimulus.

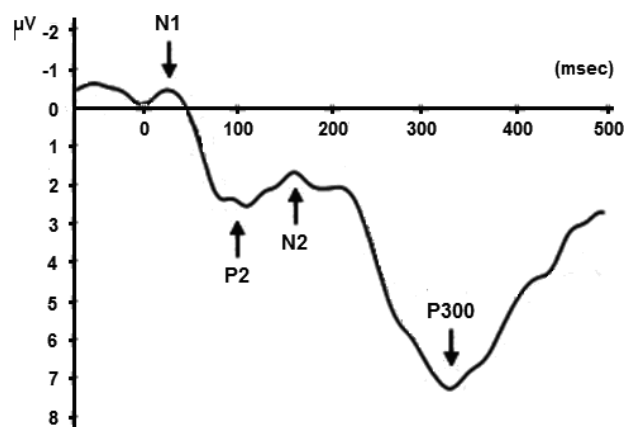


Fig. 3. A schematic representation of P300 wave.

plitude decrease with advancing age [3, 34–37]. This was confirmed by a very recent study of Juckel et al (2012), in a classical P300 paradigm [38]. However, there are some deviating effects. In a study of Woods (1992), elderly subjects showed a trend toward smaller P300 amplitudes and delayed P300 latencies, but group differences did not reach statistical significance [39]. Pfefferbaum et al (1980) observed only prolonged P300 latencies with no effect on P300 amplitudes [18]. This was partly supported by Gaal et al (2006), who found no effect on P300 amplitude in the oddball paradigm [40].

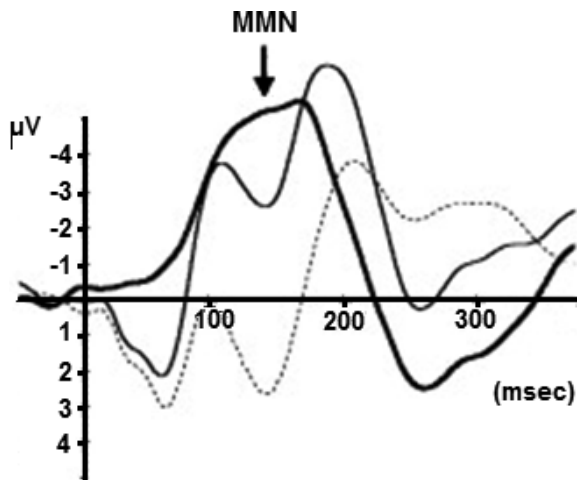


Fig. 4. A schematic representation of MMN wave.

Grey solid line – response to deviant tone, grey dashed line – response to standard tone. Black solid line indicates difference (standard-deviant), where MMN wave is evident.

Similar results were obtained using auditory deviant paradigm, frequent stimuli are presented with rare non-targets and rare “deviant” targets. Knight et al (1987) showed systematic latency prolongation and amplitude decrease of the P300 component with advancing age [35]. Similarly, Gaal et al (2006) showed the decrease of the amplitude of P300 with age in a novelty task [40]. However, in the study of Gaeta et al (1998), where P300 component was observed in the ERPs of the young to both large tonal and novel deviants, a robust P300 component was evident only to the novel deviants in the ERPs of the old [41].

## MMN

Deviant tones that are randomly embedded in a sequence of standard tones elicit an event-related potential called the mismatch negativity (MMN). MMN is partly overlapped by other ERP components at 100–200 msec latency (N1 and P2) and its shape varies, MMN peak latency and amplitude may be ambiguous. The MMN appears as negativity in response to the deviant stimulus, approximately 100 to 300 ms post stimulus onset and MMN is computed as a difference between response to the standard and to the deviant. A schematic representation of MMN is presented in Figure 4. “Obligatory” P1-N1-P2 responses are evoked simultaneously with the MMN, as the deviants must first be detected before they can be discriminated. The processes, involved in MMN generation, are assumed to initiate involuntary auditory change detection [42]. The mechanism of change detection, in turn, is known to work through the memory comparison process. Therefore, it is suggested that MMN provides an index of the auditory sensory (“echoic”) memory [43]. The MMN is described as a discriminative re-

sponse, because it presumably reflects the physical mismatch between a memory trace of the frequently occurring stimuli and the distinguishing acoustics of the deviant stimuli.

General finding presented in the literature is MMN amplitude decrease with age [22, 39, 44]. Pekkonen et al (1995) showed that aging effect on MMN is ISI-dependent: MMN area was stable regardless of age with 1 sec ISI, whereas with 3 sec ISI, MMN area was significantly smaller in the old than in the young subjects [45]. This was confirmed by the later study, where the group showed that aging affected neither frequency nor duration of MMN at interstimulus intervals of 1.5 ms, but with a 4.5-s ISI the MMN attenuated significantly more in the older than younger subjects [46]. Gaeta et al (1998) showed that at each level of deviance, MMN amplitude was smaller in the older relative to younger adults [41]. This was confirmed by Bertoli et al (2005). Moreover, in both studies older adults were less sensitive to the smallest frequency change, as indicated by an absent MMN to the smallest deviant stimuli [34]. However, in a study by Amenedo and Diaz (1998), MMN latency and amplitude were quite stable regardless of age [47].

## AUDITORY STEADY-STATE RESPONSE

The auditory steady-state response (ASSR) is observed when stimuli are presented periodically resulting in electroencephalographic entrainment [48]. The frequency of the ASSR is close to the frequency of stimulation and the greatest magnitude is observed when stimuli are presented at 40 Hz [49]. The source of ASSR has been localized in the primary auditory cortex, supratemporal gyrus, and brainstem with additional activity arising from cerebellum [50–52]. Predominately, ASSR is used for testing hearing sensitivity or as a marker of the state of consciousness during anaesthesia [48]. But the gamma range ASSR (especially in the case of 40 Hz ASSR) has also been used as an index of the ability for gamma band frequency generation in local cortical networks in neuropsychiatric disorders [53]. Stimuli, used for ASSR generation vary: they can be amplitude-modulated tones, frequency-modulated tones and discrete clicks [48]. A schematic representation of ASSR to 40 Hz click train is presented in Figure 5.

Muchnik et al (1993) have analyzed first N1 responses to 40 Hz periodic stimuli and showed latency prolongation and amplitude enhancement in the older subjects [55]. This finding nicely corresponds to the effects of age on classical P1-N1-P2 component, as discussed above.

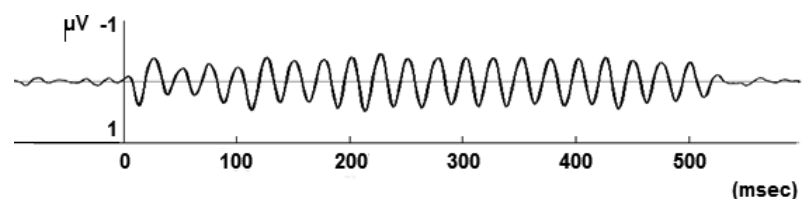


Fig. 5. A schematic representation of 40 Hz ASSR.

The literature contains divergent findings with regard to the effect of age on steady-state responses. Johnson et al (1988) did not show any significant differences in phase or amplitude of the 40 Hz ERP between the age groups, indicating that the normal aging process does not have an effect on this response [54]. However, a trend that age effects are more likely to be present at higher modulation frequencies was observed. For example, no age effect was found by Boettcher et al (2001) at stimulation rates 50 Hz [56]. Similarly, Purcell et al (2004) found no age effect in the amplitude of the ASSR response when the frequency ranged from 30–50 Hz. In contrast, the stimulation frequencies at which the largest ASSR age effect occurred were over 100 Hz [57]. This was supported by findings of Grose et al (2006): ASSR amplitudes diminished in older listeners relative to young listeners for a high modulation rate but not for a low modulation rate [58].

### FUNCTIONAL RELEVANCE OF AGE-RELATED CHANGES

In order to reveal functional significance of the findings presented above, it is crucial to evaluate functional processes that are leading to the ERP generation. Cortical evoked responses result from stimulus-locked postsynaptic potentials within apical dendrites of pyramidal neurons in the cerebral cortex [16]. The number of recruited neurons and the extent of neuronal activation are believed to be reflected by the amplitude of ERPs. And thus, the amplitude can be used as an index of the strength of the response in microvolts ( $\mu\text{V}$ ) [1, 2, 16]. However, synchrony of the neural response also crucially contributes to the resulting ERP pattern. It is also partially reflected by the amplitude [16]. The latency of the ERP wave refers to the amount of time, in milliseconds (ms), that it takes to generate the bioelectrical response following stimulus presentation. And thus, latency is related to the neural conduction time and site of excitation. In a sense, it reflects the time it takes for the sound to travel through the peripheral auditory system to the place of excitation in the central auditory system [1, 2, 16]. Thus, amplitude reduction with increasing age that occurs for most of the waves discussed, is a sign of reduced number of neurons and their synchronisation. This process is evident in both obligatory sensory pre-attentive components and in cognitive components, indicating that general ability of neural populations to work synchronously diminishes with age. Similarly, prolonged latencies of the most waves indicate age-induced slowing of the conduction of information at most levels in the auditory-cortical system. With respect to above listed information, when ERPs are introduced into clinical practice, it is absolutely necessary to collect healthy control data from various age groups for all the component of interest.

Gauta:  
2013 03 26

Priimta spaudai:  
2013 04 11

### References

1. Korostenskaja M, Dapys K, Mačiulis V. Kognityvinis klausos sukeltas potencialas P300 ir jo panaudojimas psichiatrije praktikoje. *Biologinė psichiatrija ir psichofarmakologija* 2000; 2(2): 171–6.
2. Vaitkevičius A, Kaubrys G, Klimašauskienė A, et al. Kognityvinių sukeltųjų potencialų tyrimai: P300 potencialo svarba klinikinei praktikai. *Neurologijos seminarai* 2007; 12(32): 86–94.
3. Vaitkevičius A, Kaubrys G, Budrys V. P300 kognityvinio potencialo reikšmė, vertinant sergančiųjų Alzheimerio liga kognityvines funkcijas. *Neurologijos seminarai* 2007; 11(34): 261–8.
4. Vaitkevičius A, Kaubrys G, Budrys V. Elektrofiziologiniai sergančiųjų Alzheimerio liga kognityvinių funkcijų skirtumai: P300 kognityvinio potencialo tyrimas ir gydymas cholinerginiais vaistais. *Neurologijos seminarai* 2008; 12(35): 26–34.
5. Korostenskaja M, Dapšys K, Šiurkutė A, et al. Evaluation of new parameters of auditory evoked potentials (AEPs) in patients with schizophrenia spectrum disorders. *Psichologija* 2006; 33: 76–88.
6. Dapšys K, Griškova-Bulanova I, Kaukėnas R, et al. Elektros impulsų terapijos poveikis klausos sukeltajam potencialui P300 nuotaikos sutrikimų ir šizofrenijos spektro sutrikimų atveju. *Sveikatos mokslai* 2012; 3(27): 14–7.
7. Griskova I, Dapsys K, Andruskevicius S, et al. Does electroconvulsive therapy (ECT) affect cognitive components of auditory evoked P300? *Acta Neurobiol Exp (Wars)* 2005; 65(1): 73–7.
8. Korostenskaja M, Dapsys K, Siurkute A, et al. The effect of quetiapine on auditory P300 response in patients with schizoaffective disorder: preliminary study. *Ann Clin Psychiatry* 2009; 21(1): 49–50.
9. Korostenskaja M, Dapsys K, Siurkute A, et al. Effects of risperidone on auditory information processing in neuroleptic-naive patients with schizophrenia spectrum disorders. *Acta Neurobiol Exp (Wars)* 2006; 66(2): 139–44.
10. Korostenskaja M, Dapsys K, Mačiulis V, et al. Evaluation of new MMN parameters in schizophrenia. *Acta Neurobiol Exp (Wars)* 2003; 63(4): 383–8.
11. Korostenskaja M, Dapsys K, Siurkute A, et al. Effects of olanzapine on auditory P300 and mismatch negativity (MMN) in schizophrenia spectrum disorders. *Prog Neuropsychopharmacol Biol Psychiatry* 2005; 29(4): 543–8.
12. Griskova-Bulanova I, Paskevicius J, Dapsys K, et al. The level of arousal modulates P50 peak amplitude. *Neurosci Lett* 2011; 499(3): 204–7.
13. Griskova-Bulanova I, Ruksenas O, Dapsys K, et al. P50 potential-associated gamma band activity: modulation by distraction. *Acta Neurobiol Exp (Wars)* 2012; 72(1): 102–9.
14. Griskova-Bulanova I, Ruksenas O, Dapsys K, et al. Distraction task rather than focal attention modulates gamma activity associated with auditory steady-state responses (ASSRs). *Clin Neurophysiol* 2011; 122(8): 1541–8.
15. Griskova-Bulanova I, Dapsys K, Mačiulis V, et al. Closed eyes condition increases auditory brain responses in schizophrenia. *Psychiatry Res* 2012; 211(2): 183–5.
16. Alain C, Tremblay K. The role of event-related brain potentials in assessing central auditory processing. *J Am Acad Audiol* 2007; 18(7): 573–89.
17. Harris KC, Mills JH, Dubno JR. Electrophysiologic correlates of intensity discrimination in cortical evoked potentials of younger and older adults. *Hear Res* 2007; 228(1–2): 58–68.



18. Pfefferbaum A, Ford JM, Roth WT, et al. Age-related changes in auditory event-related potentials. *Electroencephalogr Clin Neurophysiol* 1980; 49(3-4): 266-76.
19. Laffont F, Bruneau N, Roux S, et al. Effect of age on auditory evoked responses (AER) and augmenting-reducing. *Neurophysiol Clin* 1989; 19(1): 15-23.
20. Anderer P, Semlitsch HV, Saletu B. Multichannel auditory event-related brain potentials: effects of normal aging on the scalp distribution of N1, P2, N2 and P300 latencies and amplitudes. *Electroencephalogr Clin Neurophysiol* 1996; 99(5): 458-72.
21. Bertoli S, Probst R. Lack of standard N2 in elderly participants indicates inhibitory processing deficit. *Neuroreport* 2005; 16(17): 1933-7.
22. Czigler I, Csibra G, Csontos A. Age and inter-stimulus interval effects on event-related potentials to frequent and infrequent auditory stimuli. *Biol Psychol* 1992; 33(2-3): 195-206.
23. Lijffijt M, Moeller FG, Boutros NN, et al. P50, N100, and P200 sensory gating: relationships with behavioral inhibition, attention, and working memory. *Psychophysiology* 2009; 46(5): 1059-68.
24. Boutros N, Zouridakis G, Rustin T, et al. The P50 component of the auditory evoked potential and subtypes of schizophrenia. *Psychiatry Res* 1993; 47(3): 243-54.
25. Brenner CA, Kieffaber PD, Clementz BA, et al. Event-related potential abnormalities in schizophrenia: a failure to "gate in" salient information? *Schizophr Res* 2009; 113(2-3): 332-8.
26. Ghisolfi ES, Margis R, Becker J, et al. Impaired P50 sensory gating in post-traumatic stress disorder secondary to urban violence. *Int J Psychophysiol* 2004; 51(3): 209-14.
27. Ghisolfi ES, Prokopiuk AS, Besker J, et al. The adenosine antagonist theophylline impairs p50 auditory sensory gating in normal subjects. *Neuropsychopharmacology* 2002; 27(4): 629-37.
28. Knott V, Millar A, Fisher D. Sensory gating and source analysis of the auditory P50 in low and high suppressors. *Neuroimage* 2009; 44(3): 992-1000.
29. Woods AJ, Philbeck JW, Chelette K, et al. Cold pressor stimulation diminishes P50 amplitude in normal subjects. *Acta Neurobiol Exp (Wars)* 2011; 71(3): 348-58.
30. de Wilde OM, Bour LJ, Dingemans PM, et al. Failure to find P50 suppression deficits in young first-episode patients with schizophrenia and clinically unaffected siblings. *Schizophr Bull* 2007; 33(6): 1319-23.
31. Wang J, Miyazato H, Hokama H, et al. Correlation between P50 suppression and psychometric schizotypy among non-clinical Japanese subjects. *Int J Psychophysiol* 2004; 52(2): 147-57.
32. Patterson JV, Hetrick WP, Boutros NN, et al. P50 sensory gating ratios in schizophrenics and controls: a review and data analysis. *Psychiatry Res* 2008; 158(2): 226-47.
33. Rasco L, Skinner RD, Garcia-Rill E. Effect of age on sensory gating of the sleep state-dependent P1/P50 midlatency auditory evoked potential. *Sleep Res Online* 2000; 3(3): 97-105.
34. Bertoli S, Smurzynski J, Probst R. Effects of age, age-related hearing loss, and contralateral cafeteria noise on the discrimination of small frequency changes: psychoacoustic and electrophysiological measures. *J Assoc Res Otolaryngol* 2005; 6(3): 207-22.
35. Knight RT. Aging decreases auditory event-related potentials to unexpected stimuli in humans. *Neurobiol Aging* 1987; 8(2): 109-13.
36. Korostenskaja M, Dapys K, Maciulis V. First clinical research on cognitive auditory P300 potential in Lithuania: normative data. *Health Sciences* 2003; 1: 85-8.
37. Polich J, Howard L, Starr A. Effects of age on the P300 component of the event-related potential from auditory stimuli: peak definition, variation, and measurement. *J Gerontol* 1985; 40(6): 721-6.
38. Juckel G, Karch S, Kawohl W, et al. Age effects on the P300 potential and the corresponding fMRI BOLD-signal. *Neuroimage* 2012; 60(4): 2027-34.
39. Woods DL. Auditory selective attention in middle-aged and elderly subjects: an event-related brain potential study. *Electroencephalogr Clin Neurophysiol* 1992; 84(5): 456-68.
40. Gaal ZA, Csehaj R, Molnar M. Age-dependent changes of auditory evoked potentials - Effect of task difficulty. *Biol Psychology* 2007; 76(3): 196-208.
41. Gaeta H, Friedman D, Ritter W, et al. An event-related potential study of age-related changes in sensitivity to stimulus deviance. *Neurobiol Aging* 1998; 19(5): 447-59.
42. Naatanen R, Michie PT. Early selective-attention effects on the evoked potential: a critical review and reinterpretation. *Biol Psychol* 1979; 8(2): 81-136.
43. Naatanen R, Alho K. Generators of electrical and magnetic mismatch responses in humans. *Brain Topogr* 1995; 7(4): 315-20.
44. Schroeder MM, et al. Event-related potential correlates of early processing in normal aging. *Int J Neurosci* 1995; 80(1-4): 371-82.
45. Pekkonen E, Rinne T, Naatanen R. Variability and replicability of the mismatch negativity. *Electroencephalogr Clin Neurophysiol* 1995; 96(6): 546-54.
46. Pekkonen E, Rinne T, Reinikainen K, et al. Aging effects on auditory processing: an event-related potential study. *Exp Aging Res* 1996; 22(2): 171-84.
47. Amenedo E, Diaz F. Aging-related changes in processing of non-target and target stimuli during an auditory oddball task. *Biol Psychol* 1998; 48(3): 235-67.
48. Picton TW, John MS, Dimitrijevic A, et al. Human auditory steady-state responses. *Int J Audiol* 2003; 2(4): 177-219.
49. Galambos R, Makeig S, Talmachoff PJ. A 40-Hz auditory potential recorded from the human scalp. *Proc Natl Acad Sci USA* 1981; 78(4): 2643-7.
50. Makela JP, Hari R. Evidence for cortical origin of the 40 Hz auditory evoked response in man. *Electroencephalogr Clin Neurophysiol* 1987; 66(6): 539-46.
51. Pantev C, Roberts LE, Elbert T, et al. Tonotopic organization of the sources of human auditory steady-state responses. *Hear Res* 1996; 101(1-2): 62-74.
52. Pastor MA, Thut G, Pascual-Leone A. Modulation of steady-state auditory evoked potentials by cerebellar rTMS. *Exp Brain Res* 2006; 175(4): 702-9.
53. Herrmann CS, Demiralp T. Human EEG gamma oscillations in neuropsychiatric disorders. *Clin Neurophysiol* 2005; 116(12): 2719-33.
54. Johnson BW, Weinberg H, Ribary U, et al. Topographic distribution of the 40 Hz auditory evoked-related potential in normal and aged subjects. *Brain Topogr* 1988; 1(2): 117-21.
55. Muchnik C, Katz-Putter H, Rubinstein M, et al. Normative data for 40-Hz event-related potentials to 500-Hz tonal stimuli in young and elderly subjects. *Audiology* 1993; 32(1): 27-35.
56. Boettcher FA, Poth EA, Mills JH, et al. The amplitude-modulation following response in young and aged human subjects. *Hear Res* 2001; 153(1-2): 32-42.

57. Purcell DW, John SM, Schneider BA, et al. Human temporal auditory acuity as assessed by envelope following responses. *J Acoust Soc Am* 2004; 116(6): 3581-93.
58. Grose JH, Mamo SK, Hall JW 3<sup>rd</sup>. Age effects in temporal envelope processing: speech unmasking and auditory steady state responses. *Ear Hear* 2009; 30(5): 568-75.

**I. Griškova-Bulanova, N. Kazlauskienė**

### **SENĖJIMO POVEIKIAI SU ĮVYKIU SUSIJUSIEMS KLAUSOS POTENCIALAMS**

#### **Santrauka**

Sukeltieji potencialai yra objektyvus būdas įvertinti smegenų veiklą skirtinguose stimulo apdorojimo etapuose – sensori-

niuose, ikidėmesiniuose ir kognityviniuose. Lietuvoje yra taikomos skirtingos sukeltųjų potencialų rūšys: klasikinis P300 potencialas, nesutapimo negatyvumas, P1-N1-P2 potencialas, P50 potencialas ir girdimasis nuostovus atsakas. Potencialai naudojami klinikinėje praktikoje vertinant ligos sukeltus pažeidimus, medikamentinio ir nemedikamentinio gydymo poveikį, ieškant optimalių registravimo sąlygų. Aki-vaizdu, kad, dirbant su sveikais tiriamaisiais, galima nesunkiai parinkti vienalytes pagal amžių ir lytį grupes. Tačiau pacientų, kuriems ištyrimas sukeltaisiais potencialais gali būti naudingas, amžiaus išsibarstymas yra labai platus. Todėl svarbu žinoti, kaip sukeltieji potencialai ir jų parametrai priklauso nuo amžiaus.

**Raktažodžiai:** su įvykiu susiję klausos potencialai, amžius.