The development of the N1 and N2 components in auditory oddball paradigms: a systematic review with narrative analysis and suggested normative values

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Abstract Auditory event-related potentials (AERPs) are widely used in diverse fields of today's neuroscience, concerning auditory processing, speech perception, language acquisition, neurodevelopment, attention and cognition in normal aging, gender, developmental, neurologic and psychiatric disorders. However, its transposition to clinical practice has remained minimal. Mainly due to scarce literature on normative data across age, wide spectrum of results, variety of auditory stimuli used and to different neuropsychological meanings of AERPs components between authors. One of the most prominent AERP components studied in last decades was N1, which reflects auditory detection and discrimination. Subsequently, N2 indicates attention allocation and phonological analysis. The simultaneous analysis of N1 and N2 elicited by feasible novelty experimental paradigms, such as auditory oddball, seems an objective method to assess central auditory processing. The aim of this systematic review was to bring forward normative values for auditory oddball N1 and N2 components across age. EBSCO, PubMed, Web of Knowledge and Google Scholar were systematically searched for studies that elicited N1 and/or N2 by auditory oddball paradigm. A total of 2,764 papers were initially identified in the database, of which 19 resulted from hand search and additional references, between 1988 and 2013, last 25 years. A final total of 68 studies met the eligibility criteria with a total of 2,406 participants from control groups for N1 (age range 6.6–85 years; mean 34.42) and 1,507 for N2 (age range 9–85 years; mean 36.13). Polynomial regression analysis revealed that N1 latency decreases with aging at Fz and Cz, N1 amplitude at Cz decreases from childhood to adolescence and stabilizes after 30–40 years and at Fz the decrement finishes by 60 years and highly increases after this age. Regarding N2, latency did not covary with age but amplitude showed a significant decrement for both Cz and Fz. Results suggested reliable normative values for Cz and Fz electrode locations; however, changes in brain development and components topography over age should be considered in clinical practice.

Keywords Event-related potentials · Auditory oddball paradigm · N1 wave · N2 wave · Aging · Normative values

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Introduction

As a researcher of the third generation of neuroscientists, we are in the course of explaining the possession of psychological attributes by human beings, probably obsessively ascribing such attributes not to the mind but to the brain or parts of the brain. This is a rising issue among neurophilosophers, we still cannot explain how we perceive or think by reference to the brain or some part of the brain, for it makes no sense to ascribe such psychological attributes to anything less than a human being as a whole (Bennett and Hacker 2007).

Thereupon, systematic reviews are required to update, appraise and synthesize all quality research evidence relevant to a question. Such method is necessary and essential

in today's neuroscience, cognitive psychology and psychophysiological research.

Keeping the above in mind, ever since Berger (1929) demonstrated that it is possible to record the electrical activity of the brain by placing electrodes on the surface of the scalp and with ¾ of century of knowledge since the first investigation of sound-evoked changes in the electroencephalogram (EEG) in the waking human brain (Davis 1939), there has been always considerable interest in the relationship between these recordings of neurophysiological activity and psychological processes in various fields and types of studies (e.g., experimental, translational, clinical, case study). We will review one of the most prominent human auditory event-related potential and field (ERP and ERF, respectively)—the N1/N1 m.

The electrical activity occurring on the scalp consists of changing electrical voltages that are caused by action potentials summed over large numbers of neurons, synapses, neuronal pathways and systems. There are two non-invasive measures for this electrical brain activity, the EEG and ERPs, both in time course. The EEG measures the spontaneous electrical brain activity and the ERPs are derived by averaging EEG changes over experimental or cognitive events. The averaging process attenuates the spontaneous activity in the EEG and results in electrical potential changes related to specific events (Eysenck and Keane 2003).

ERPs are induced exogenously by environmental events (such as sensory stimuli) or endogenously by processes such as decision making, eliciting a characteristic series of waves labeled according to their latency and polarity (Davis and Zerlin 1966). Parallel streams of neuronal activity originate overlapping ERP deflections composed of several components. Therefore, a component can be defined as a voltage contribution to the ERP which reflects a functionally discrete stage of neuronal processing occurring in a restricted cerebral area (Näätänen and Picton 1987), also are hypothesized to be linked and reflected by psychological and cognitive processes (Hillyard and Kutas 1983; Fabiani et al. 2007).

In auditory novelty processing experiments, the experimental paradigm that seems the most feasible is the oddball paradigm. The oddball paradigm, as a signal-detection paradigm was first described by Ritter and Vaughan (1969). In the auditory modality, it was first used in 1975 by Squires, Squires and Hillyard at the University of California, San Diego, USA (Squires et al. 1975). In this paradigm, physically deviant stimuli (silence included) are randomly presented among repetitive streams of homogenous sounds (standard stimuli; Näätänen 1975; Näätänen et al. 1978; Donchin 1981; Jääskeläinen 2012).

The electrically recorded N1 or N100 in auditory oddball stimulation is a negative wave response, which peaks about 100 ms after stimulus onset and lasts for approximately 100 ms (Näätänen and Picton 1987; Tiitinen et al. 1994; May and Tiitinen 2010). Multiple sources have been identified to generate N1 wave.

In the classical review of Näätänen and Picton (1987), it was concluded that at least six different cerebral processes, occurring in different cerebral locations and subserving different psychophysiological functions, can contribute to a negative wave recorded from the scalp peaking between 50 and 150 ms: (1) a component generated bilaterally in the auditory cortex by vertically oriented sources in the supratemporal plane (STP); (2) a component generated in the association cortex on the lateral aspect of the superior temporal gyrus (STG) and parietal cortex, also termed by T-complex (Wolpaw and Penry 1975); (3) a vertex negative component generated in the motor and premotor cortices, its widespread and nonspecific neuronal networks with thalamo-reticular system facilitates stimulus detection, analysis and response (Crowley and Colrain 2004); (4) the mismatch negativity (MMN); (5) a temporal component of the processing negativity; and a (6) frontal component of the processing negativity. The first three are considered the true N1 components. Thus, N1 is a fronto-centrally component with a peak latency of 100 ms after stimulus onset generated bilaterally mainly in the auditory cortices. Its magnetic equivalent (N1 m/N100 m; Elberling et al. 1980; Hari et al. 1980) originates deep within the Sylvian fissure in tonotopically organized areas (Yamamoto et al. 1988; Cansino et al. 1994), but also comprises secondary areas such as Heschl's gyrus, STG and planum temporale providing the major source (Papanicolaou et al. 1990; Pantev et al. 1995; Jääskeläinen et al. 2004; Inui et al. 2006).

The auditory ERP N1, does not reflect a single underlying cerebral process, but it appears to contain both stimulus-specific and stimulus-nonspecific components, closely following the P1 component (Yvert et al. 2001; Hamm et al. 2013). These ERP components reflect sensory and perceptual processes. Further, N1 seems to reflect early synchronization between primary and secondary auditory cortices in the lateral and STP (Yvert et al. 2005; Liasis et al. 2006).

The generation source location of N1 depends on stimulus frequency. Woods et al. (1993) have found that N1 is more frontally distributed following 4,000 Hz than 250 Hz tone burst stimuli. Numerous studies have shown that N1 amplitude and latency have a large variation according to type of deviant auditory stimuli. For example, in speech oddball paradigms, the first stages of detection, graphemic analysis and stimuli recognition correspond to the appearance of a N1 with latency between 150 and 200 ms (Bentin and Carmon 1984). Subsequently, the N2 indicates an attentional allocation and stage of phonological type analysis of the information, related to the sounds of the language, both in the auditory and visual modalities of

presentation (Sams et al. 1985; Reinvang et al. 2000). Two components contribute to forming N2, usually labeled MMN (N2a) and N2b (Novak et al. 1990, 1992). The first component (MMN) has been related to the automatic detection of stimulus changes (Ritter et al. 1979; Näätänen 1982), the later component (N2b) is interpreted as a correlate of controlled detection of stimulus changes and phonological categorization (Ritter et al. 1979; Näätänen 1982; Amenedo and Díaz 1998; Näätänen et al. 2007).

In auditory oddball, N1 amplitude is strongly modulated by attentional context and interstimulus interval (ISI; Hillyard et al. 1973; Rosburg et al. 2008; May and Tiitinen 2010). During the process of falling asleep, N1 gradually declines in amplitude, possibly because of the decrease in the level of attention, and during REM sleep it is approximately 25–50 % of its waking amplitude (Crowley and Colrain 2004). According to Hamm et al. (2013), this indexes the earliest cognitively influenced auditory neuronal event reliably measureable with EEG (Hamm et al. 2013).

The effects of aging on ERPs vary among studies. With respect to changes of the N2 component from childhood to adulthood, most studies showed a decrease in the N2 amplitude and latency (Johnstone et al. 1996; Mueller et al. 2008). Regarding N1 latency, only Iragui et al. (1993) reported significant age-related increases at Cz, while the common finding has been that N1 latency remains unchanged with advancing age (Barrett et al. 1987). Ladish and Polich (1989) found an increase in N1 amplitude and a decrease in N1 latency with increasing age from 5 to 19 years, but other studies reported variations of N1 amplitude from childhood to adolescence (Ladish and Polich 1989; Ponton et al. 2000; Čeponienė et al. 2002, 2003, 2008). As for findings regarding adult development and aging, N1 amplitude increased significantly with age and increased N1 latency was only significant in the posterior region (Anderer et al. 1996).

This wide spectrum of results, findings and variety of auditory stimuli used, has possibly delayed the translation of N1 and N2 to clinical practice. Without linking auditory N1 and N2 to a restricted neuropsychological meaning, we are undoubtedly and objectively assessing the first stages of cognitive auditory processing.

Aims and objectives

The clinical significance and application of auditory ERPs such as N1 and N2 also, have remained marginal in the current practice of audiology, neurophysiology, neurology or psychiatry. Particularly the N1 wave, as an exogenous and robust auditory ERP may be used to assess central auditory processing.

This review will address the following aspects of N1 and N2: latency, amplitude, electrode location (most prominent—Cz and Fz), development and maturation, aging, data published by year and country, type of stimuli in auditory oddball and research areas. We purpose that this review may lead to the first human normative values for N1 and N2 components when elicited by auditory oddball, being beneficial for a standard, controlled and wide accepted application to assess central auditory processing.

Methods

For this review, we searched EBSCO, PubMed, Web of Knowledge and Google Scholar to identify potential studies, without pre-defined time-window. Further studies were identified through references and citations. As inclusion criteria, we searched the term "N1" limited by the term "auditory oddball" in title, abstract, keyword or topic. Papers with no control group and animal research were excluded.

A search between ending November and early December 2013 found 2764 articles. Duplicates were removed and articles with no full text available were kindly asked by mail to authors, leaving a selection of papers for assessment of eligibility upon reading the full text article.

Data extraction, quality and relevance assessments

Data extracted from each full text article for eligibility assessment included: Authors; Year Publication; Aim; Country; Research areas; Control group (*N*), age (mean); Type of stimuli (frequency, intensity, speech/consonant-vowel, pure-tone); Electrodes recording for target/deviant stimuli (amplitude and latency of N1 and N2); Findings and suggestions. The complete systematic process of deduction can be found below in Fig. 1.

Polynomial regression was applied as a descriptive method to data analysis that better fits a nonlinear model.

Results

A total of 2764 papers were initially identified in the database search from which 19 were hand searched and additional references. After, 2680 papers that did not meet inclusion criteria or duplicated were removed through title and abstract screening, leaving a total of 84 papers. Sixteen papers could not be accessed, 14 of which were not archived or we did not receive answer from authors and two were foreign language articles. A final total of 68 full text articles were assessed for eligibility, none of which required translation. Study's authors, country, mean age of control groups, number of participants, results for amplitude and latency across Fz and Cz electrodes for deviant

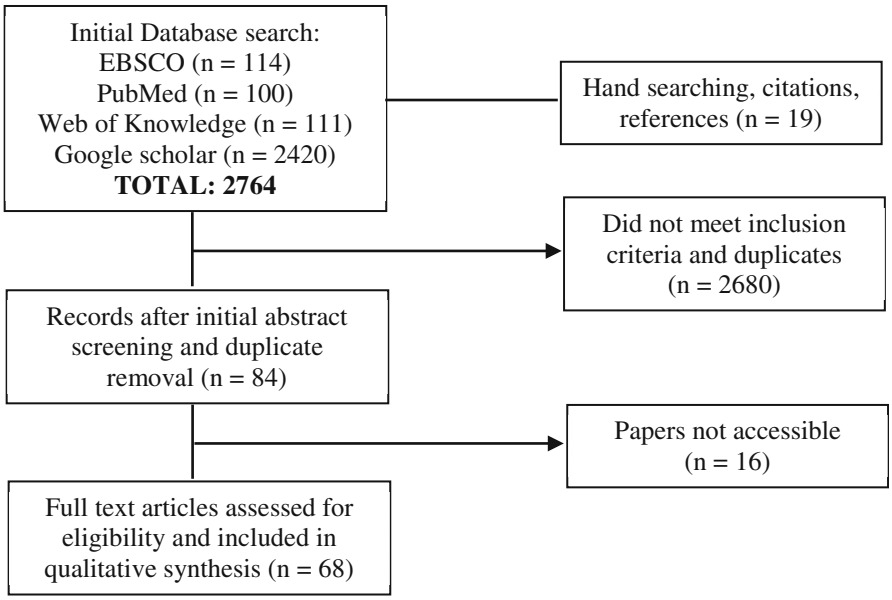

Fig. 1 PRISMA systematic search flow diagram (Moher et al. 2009)

stimuli and subject's article (notes) are summarized in Tables 1 and 2, for N1 and N2, respectively. Many studies have results for different electrode locations. We considered Fz and Cz since they are the most common locations and were maximal amplitude for N1, N2 and MMN is achieved (Näätänen and Picton 1987; Winkler 2007; Duncan et al. 2009; May and Tiitinen 2010). Studies with mean global field power were considered as Cz data, representing the standard deviation across electrodes at a given time point indicating the presence of a specific underlying neuronal component (Murray et al. 2008; Altieri 2013).

The results revealed papers between 1988 and 2013, last 25 years. The majority of the studies were published between 1995 and 1999, also it was when most of the studies elicited and analyzed both N1 and N2 (presented in Fig. 2). The five most published research areas were neurosciences/neurology, psychology, physiology, psychiatry and engineering. The five countries/territories with more papers published were USA, Australia, Germany, Japan and Austria.

Data analysis revealed a total of 3934 participants from control groups (Table 3), 2427 for N1 studies (male = 1247, female = 1180), ranging in age from 6.6 to 85.0 years with a mean of 34.4 (SD = 18.6). Regarding N2 studies, a total of 1507 participants were obtained from all control groups (male = 769, female = 738), ranging in age from 9.0 to 85.0 years with a mean of 36.1 (SD = 20.7). All participants from control groups were reported to have normal hearing and vision, no neurological, psychiatric or other disorder. The majority of studies applied a frequency auditory oddball. The most standard stimuli used was a pure-tone of 1000 Hz at an intensity level range from 55 to 109 dB above hearing threshold, with at least 200 Hz of difference to the deviant stimuli, except in Demiralp et al. (1999), Wang et al. (2005) and Čeponienė et al. (2008) studies. Others used duration auditory oddball (Shelley et al. 1999; Segalowitz et al. 2001; Bortoletto et al. 2011; Wetzel et al. 2011; Neuhaus et al. 2013) and few intensity auditory oddball (Anderer et al. 1996, 1998a, b; Wang and Wang 2001; Wang et al. 2005; Barry et al. 2006). Only two papers elicited N1 and N2 with speech stimuli (Henkin et al. 2002; Toscano et al. 2010), due to the scarce data and type of stimuli they were not considered in the regression analysis.

Orthogonal polynomial regression equations were fit to the data, including degrees 4 through 6 to significantly increase predictability.

For N1, results revealed at Cz a mean amplitude of 7.3 μV (SD = 3.0) ranging from 2.0 to 15.1 μV (data from 69 cells) and a mean latency of 107.6 ms (SD = 15.0) ranging from 82.0 to 164.0 ms (data from 75 cells). At Fz, mean amplitude was 7.0 μV (SD = 3.2) ranging from 1.1 to 14.4 (data from 48 cells) and a mean latency of 118.2 ms (SD = 31.5) ranging from 88.9 to 260.0 ms (data from 45 cells).

Regarding N2, results revealed at Cz a mean amplitude of 4.9 μV (SD = 2.6) ranging from 1.0 to 14.2 μV (data from 40 cells) and a mean latency of 231.4 ms (SD = 33.9) ranging from 200.0 to 371.0 ms (data from 43 cells). At Fz the mean amplitude was 5.7 μV (SD = 3.9) ranging from 0 to 16.3 μV (data from 34 cells) and a mean latency of 231.8 ms (SD = 18.9) ranging from 200.0 to 277.0 ms (data from 32 cells). Table 3 summarizes the results for N1 and N2 by four age ranges, suggested as normative values.

Table 1 Studies with auditory oddball N1 elicitation (control groups data from deviant stimuli at Fz and Cz)

References	Country	*N*	Age	Cz (amp)	Fz (amp)	Cz (lat)	Fz (lat)	Stimuli	Notes
Hegerl et al. (1988)	Germany	27	34.1	12.2		91.4		Std: 1600 Hz, Dev: 800 Hz/65 dB	Schizophrenia prognosis
Verma et al. (1989)	USA	11	63.5			103.7		Std: 1000 Hz, Dev: 2000 Hz/60 dB	Dementia
Iragui et al. (1993)	USA	28	29	6.6		94		Std: 1000 Hz, Dev: 1500 Hz/70 dB	Aging; correlations; reaction time
		16	49	8.1		95			
		27	71	6.6		97			
Lembreghts et al. (1995)	Belgium	86	34.1	6.6	6.7	94	95	Std: 800 Hz, Dev: 1470 Hz/ 70 dB	Age, gender, intervariability
Tarter et al. (1995)	USA	56	13.2	9.5		119.9		Std: 1000 Hz, Dev: 2000 Hz/64.5 dB	Adolescence; substance abuse
Winter et al. (1995)	Netherlands	13	21.4	5.3	6.7	105	103	Std: 1000 Hz, Dev: 1200 and 2000 Hz/65 dB	Sleep stage 2
Siedenberg et al. (1996)	USA	10	36.5			103		Std: 1000 Hz, Dev: 2000 Hz/65 dB	ERP vs ERF
Anderer et al. (1996, 1998a, b)	Austria	58	25	8.6		95		Std: 90 dB Dev: 70 dB/ 1000 Hz	Aging; ERPs; LORETA
		19	35	8.3		100			
		13	45	8.7		99			
		33	55	9.2		95			
		29	65	9		96			
		12	75	8.5		93			
		8	85	8.1		96			
Wright et al. (1996)	Australia	28	62.8	13.6		164		Std: 1000 Hz, Dev: 2000 Hz/60 dB + back noise	Parkinson's disease
Johnstone et al. (1996)	Australia	10	9.1	9.6	12.1	132	133	Std: 1000 Hz, Dev: 1500 Hz/60 dB	Child and adolescent; waves morphology; ERPs
		10	11.2						
		10	12.8	8.3	9.1	127	133		
		10	15						
		10	16.8	6.3	8.3	123	124		
Hirata et al. (1996)	Japan	14	66.8	6.9		82		Std: 2000 Hz, Dev: 1000 Hz/80 dB	Stroke
Akaho (1996)	Japan	47	21.9	9.1		100.2		Std: 1000 Hz, Dev: 2000 Hz/70 dB	Cognition; effects of AED
Kazis et al. (1996)	Greece	53	45.6	10.3		98.9		Std: 1000 Hz, Dev: 2000 Hz/70 dB	Myotonic dystrophy
Brigham et al. (1997)	USA	29	11.5	5.9	8.1	157.9	168.4	Std: 1000 Hz, Dev: 2000 Hz/64.5 dB	Polysubstance and alcohol
Haig et al. (1997)	Australia	25	27.3	8.6		99		Std: 1000 Hz, Dev: 1500 Hz/60 dB	Gaussian component; schizophrenia
Potts et al. (1998a)	USA	24	39	3.8		100		Std: 1000 Hz, Dev: 1500 Hz/97 dB	Schizophrenia
Potts et al. (1998b)	USA	20	21.1	2	6.7	100	110	Std: 440 Hz, Dev: 1245 Hz	Various ERPs
Amenedo and Díaz (1998)	Spain	20	30.5	5.7	5.2	112	108	Std: 1000 Hz, Dev: 2000 Hz/90 dB	Aging; ERPs
		20	50	7.5	7	105	104		
		33	71.5	7.1	6.5	110	108		
Gonsalvez et al. (1999)	Australia	12	28.3	10.5	8.4	110	105	Std: 1000 Hz, Dev: 1200 Hz/60 dB	Target-to-target hypothesis (P3)
Shelley et al. (1999)	USA	17	37.4		8.0		102.4	Std: 1000 Hz, Dev: 1200 Hz/75 dB (ISI: variable)	Schizophrenia; cortical dysfunction

Table 1 continued

References	Country	*N*	Age	Cz (amp)	Fz (amp)	Cz (lat)	Fz (lat)	Stimuli	Notes
Demiralp et al. (1999)	Turkey	10	21.5	11.6	10.2	125	125	Std: 2000 Hz, Dev: 1950 Hz/60 dB	Wavelet transform; cognitive processes
Gölgeli et al. (1999)	Turkey	38	20.6	9.2	9	107	113	Std: 2000 Hz, Dev: 1500 Hz	Gender differences
Barry et al. (2000)	Australia	14	30.5	6		117.6		Std: 1000 Hz, Dev: 1500 Hz/60 dB	Alpha activity
Sumi et al. (2000)	Japan	39	68.5		Pz			Std: 2000 Hz, Dev: 1000 Hz/70 dB	Alzheimer
Reinvang et al. (2000)	Norway	27	29.7	6.4	4.9	99	97	Std: 800 Hz, Dev: 1200 Hz/ 80 dB	Head injury
Johnstone et al. (2001)	Australia	50	12.5		7.8			Std: 1000 Hz, Dev: 1500 Hz/60 dB	Two sub types AD/HD
Ford et al. (2001)	USA	32	38	9.3	7.3	105	100	Std: 500 Hz, Dev: 1000 Hz/ 80 dB	Epilepsy and schizophrenia
Segalowitz et al. (2001)	Canada	12	20.7		8.2		119.3	Std: 800 Hz, Dev: 1500 Hz/ 100 ms	Mild head injury
Ullsperger et al. (2001)	Germany	15	23.8	4.5	6.3	133.3	133.3	Std: 1000 Hz, Dev: 2000 Hz/55 dB	Mental workload changes
Wang and Wang (2001)	China	39	26.6	11.4	10.2	120.1	118.6	Std: 0 dB, Dev: 60 dB/ 2000 Hz	Sensation seeking
Brown et al. (2002)	Australia	40	36.7	11.4	10.2	103.1	108.3	Std: 1000 Hz, Dev: 1500 Hz/60 dB	1st episode schizophrenia
		40	19.7	10.6	9.3	104.5	110.4		
Cohen et al. (2002)	USA	39	24.7	4.9	5.5	117	117.6	Std: 600 Hz, Dev: 1600 Hz/ 60 dB	Alcohol
Henkin et al. (2002)	Israel	20	14.4	8	7.5	103.9		Std: 1400 Hz, Dev: 1000 Hz	AERPs; phonetic and semantic processing
Valkonen-Korhonen et al. (2003)	Finland	19	29	5.0		100		Std: 800 Hz, Dev: 560 Hz/ 55 dB	Auditory processing; psychotic
Lucchesi et al. (2003)	Brazil	12	24.8	10	11.2	100	100	Std: 800 Hz, Dev: 1500 Hz/ 70 dB	Flunitrazepam effect
Barry et al. (2003)	Australia	16	28	6.3		90		Std: 1000 Hz, Dev: 1500 Hz/80 dB	EEG brain states; effects ERPs
Barry et al. (2004)	Australia	14	31	8.7	9.6	114	114.5	Std: 1000 Hz, Dev: 1500 Hz/60 dB	EEG alpha phase
Mulert et al. (2004)	Germany	9	24.2	10.2		119.3		Std: 800 Hz, Dev: 1300 Hz/ 95 dB	fMRI; source localization
Chao et al. (2004)	USA	15	44.1	6.5		90.9		Std: 1000 Hz, Dev: 2000 Hz/55 dB	Suppressed HIV patients
Wang et al. (2005)	USA	6	32.3		1.1		145	Std: 440 Hz, 80 dB, Dev: 494 Hz 80 dB, e 440 Hz 65 dB	TWI; development of auditory processing; MMN
		9	10.8		5.3		222		
		11	6.6		5.6		260		
Gilmore et al. (2005)	USA	14	43.6		2.1	102		Std: 1000 Hz, Dev: 2000 Hz/76 dB	Schizophrenia
Chunhau et al. (2005)	Japan	16	23.5	7	7.7	101	109	Std: 1000 Hz, Dev: 2000 Hz/60 dB	40 min oddball
Brown et al. (2006)	Australia	20	24.8	2.7	3.7	105	105	Std: 1000 Hz, Dev: 2000 Hz/60 dB	Inter-modal attention
Barry et al. (2006)	Australia	20	36	2.3	4.2	139	144	Std: 50 dB, Dev: 80 dB/ 1000 Hz	Narrow band EEG phase effects
van Harten et al. (2006)	Netherlands	53	73.6	8.3	12	100.8	100.8	Std: 1000 Hz, Dev: 2000 Hz/80 dB	Vascular cognitive impairment; peak-to-peak ERPs

Table 1 continued

References	Country	*N*	Age	Cz (amp)	Fz (amp)	Cz (lat)	Fz (lat)	Stimuli	Notes
Dixit et al. (2006)	India	40	21.5	6.1	7.4	110.7	100.4	Std: 1000 Hz, Dev: 2000 Hz/60 dB	Caffeine users
Čeponienė et al. (2008)	USA	19	25.5	2.4	2.4	119		Std: 500 Hz, Dev: 550 Hz/ 63 dB	Aging; N1–N2–P2
		19	71.3	2.1	2.0	118			
Kreukels et al. (2008)	Netherlands	23	53.2	8.7		101		Std: 1000 Hz, Dev: 2000 Hz/75 dB	Chemotherapy; N1 independent from P3
Zhu et al. (2008)	China	15	23	4.9	7.7	110	120	Std: 1000 Hz, Dev: 2000 Hz/80 dB	Music; mandarin lexical tones
Wise et al. (2009)	Australia	98	35.6	7.7	7.3	113.8	111.7	Std: 500 Hz, Dev: 1000 Hz/ 75 dB	Panic disorder
Guney et al. (2009)	Turkey	32	37.1	11.6	12.2	97.7	97.2	Std: 1000 Hz, Dev: 2000 Hz/80 dB	Tobacco smokers
Dassanayake et al. (2009)	Sri Lanka	38	49			99.1		Std: 1000 Hz Dev: 2000 Hz/75 dB	Pesticides
Gilmore et al. (2009)	USA	12	25	3.2	2.6	101	107.8	Std: 1000 Hz, Dev: 2000 Hz/76 dB	Hemispheric differences
Ogawa et al. (2009)	Japan	19	64.5	5.7		85.9		Std: 1000 Hz, Dev: 2000 Hz/80 dB	Amyotrophic lateral sclerosis
Cahn and Polich (2009)	USA	16	45.5	3.1	3.3	105	105	Std: 500 Hz, Dev: 1000 Hz/ 80 dB	Meditation; different mental states
Sakamoto et al. (2009)	Japan	11	30.9	5.5	6.7			Std: 1000 Hz, Dev: 2000 Hz/55 dB	Mastication
Whelan et al. (2010)	Ireland	21	40.3		1.6		90.6	Std: 500 Hz, Dev: 1000 Hz	Multiple sclerosis
Boucher et al. (2010)	Canada	99	11.3	4.7		127.1		Std: 1000 Hz, Dev: 2000 Hz/70 dB	MeHg; PCBs; neurodevelopment
Gandelman-Marton et al. (2010)	Israel	18	35.6			113.2	113.4	Std: 1000 Hz, Dev: 2000 Hz/70 dB	Immediate and short-term retest
Bortoletto et al. (2011)	Italy	20	25	5.1		133		5 pair of Hz and 3 different ISI	Sleep deprivation
Wetzel et al. (2011)	Germany	18	25	4.5	2.2	110	110	Std: 500 Hz, Dev: 200 and 500 ms	Novel; duration; better in children
Tsai et al. (2012)	Taiwan	63	9		Pz			Std: 3000 Hz, Dev: 2000 Hz/60 dB	ADHD; age-related; correlations;
		51	9	13.6	14.4	119.5	126.2		
Tanaka et al. (2012)	Japan	14	43	4.8		99		Std: 1000 Hz, Dev: 2000 Hz/80 dB	Myotonic dystrophy type 1
Ho et al. (2012)	Taiwan	15	23.7			91	88.9	Std: 2000 Hz Dev: 1000 Hz/80 dB	Normal aging
		15	70.1			95	90		
Neuhaus et al. (2013)	Germany	144	32.4	15.1	11.4	111.1	127.8	Std: 500 ms 109 dB, Dev: 40 ms 83 dB	Schizophrenia; gatings; AUC e ROC
Schneider et al. (2013)	USA	40	13.9	3.4		125		Std: 1000 Hz, Dev: 2000 Hz/70 dB	Fragile X syndrome treatment
Hamm et al. (2013)	USA	70	38.4	2.2	2.2	92	92	Std: 1500 Hz, Dev: 1000 Hz/75 dB	Bipolar disorder

Polynomial regression curves showed that latency of N1 component slightly decreases with aging at Cz ($r = 0.50$; Fig. 3) and Fz ($r = 0.78$; Fig. 4). As for amplitude, N1 seems to decrease from birth until 30 years and stabilize after 40 years old at Cz ($r = 0.20$), but at Fz a similar decrement only finishes by 60 years and starts to highly increase after ($r = 0.35$). Regarding N2 component (Figs. 5, 6), latency did not covary with age for both Cz ($r = 0.36$) and Fz ($r = 0.44$) locations. In contrast, N2 amplitude at Cz ($r = 0.68$) and Fz ($r = 0.81$) showed a significant decrement with age, with a curious slight increment between 20 and 45 years at Cz.

Table 2 Studies with auditory oddball N2 elicitation (control groups data from deviant stimuli at Fz and Cz)

References	Country	*N*	Age	Cz (amp)	Fz (amp)	Cz (lat)	Fz (lat)	Stimuli	Notes
Verma et al. (1989)	USA	11	63.5			237.8		Std: 1000 Hz, Dev: 2000 Hz/60 dB	Dementia
Iragui et al. (1993)	USA	28	29	2.26		211		Std: 1000 Hz, Dev: 1500 Hz/70 dB	Aging; correlations; reaction time
		16	49	3.9		220			
		27	71	5.5		231			
Lembreghts et al. (1995)	Belgium	41	34.1	4.7	3.6	207	212	Std: 800 Hz, Dev: 1470 Hz/70 dB	Age, gender, intervariability
Anderer et al. (1996)	Austria	58	25	6.7	6.5	210	212	Std: 90 dB, Dev: 70 dB/ 1000 Hz	Aging; ERPs
		19	35	5.6	4	213	218		
		13	45	7.1	5	224	225		
		33	55	8.1	3.8	221	220		
		29	65	4.2	1.5	222	217		
		12	75	3.1	1.9	212	212		
		8	85	2	0	254	253		
Siedenberg et al. (1996)	USA	10	36.5			216		Std: 1000 Hz, Dev: 2000 Hz/65 dB	ERP vs ERF
Johnstone et al. (1996)	Australia	10	9.1		14.6		253.3	Std: 1000 Hz, Dev: 1500 Hz/60 dB	ERPs; child and adolescent; morphology
		10	11.2						
		10	12.8						
		10	15	6.7	12.9	240	253.3		
		10	16.8		5.8		253.3		
Akaho (1996)	Japan	47	21.9	3.5		223.2		Std: 1000 Hz, Dev: 2000 Hz/70 dB	Cognition; effects of AED
Kazis et al. (1996)	Greece	53	45.6	8.4				Std: 1000 Hz, Dev: 2000 Hz/70 dB	Myotonic dystrophy
Brigham et al. (1997)	USA	29	11.5	7.3	16.3	221	221.1	Std: 1000 Hz, Dev: 2000 Hz/64.5 dB	Polysubstance and alcohol
Haig et al. (1997)	Australia	25	27.3	4		201		Std: 1000 Hz, Dev: 1500 Hz/60 dB	Gaussian component; schizophrenia
Amenedo and Díaz (1998)	Spain	20	30.5	2.5	4.5	237	242	Std: 1000 Hz, Dev: 2000 Hz/90 dB	Age-related ERPs
		20	50	3.4	3.1	250	252		
		33	71.5	3.8	3.7	271	277		
Demiralp et al. (1999)	Turkey	10	21.5	6.8	3.4	270	270	Std: 2000 Hz, Dev: 1950 Hz/60 dB	Wavelet transform; cognitive processes
Gölgeli et al. (1999)	Turkey	38	20.6	7.1	8	225	240	Std: 2000 Hz, Dev: 1500 Hz	Gender differences
Sumi et al. (2000)	Japan	39	68.5		Pz			Std: 2000 Hz, Dev: 1000 Hz/70 dB	Alzheimer
Reinvang et al. (2000)	Norway	27	29.7	2	1.5	207	207	Std: 800 Hz, Dev: 1200 Hz/80 dB	Head injury
Johnstone et al. (2001)	Australia	50	12.5		8.5		238.5	Std: 1000 Hz, Dev: 1500 Hz/60 dB	Two sub types AD/HD
Segalowitz et al. (2001)	Canada	12	20.7			235.3		Std: 800 Hz, Dev: 1500 Hz/100 ms	Mild head injury
Wang and Wang (2001)	China	39	26.6		2.9	245.1	236.8	Std: 0 dB, Dev: 60 dB/ 2000 Hz	Sensation seeking
Brown et al. (2002)	Australia	40	36.7	7.5	5.3	207.2	209.8	Std: 1000 Hz Dev: 1500 Hz/60 dB	1st episode Schizophrenia
		40	19.7	3.2	9.9	209.3	214.1		
Cohen et al. (2002)	USA	39	24.7	2.3	5.1	217.6	235.3	Std: 600 Hz, Dev: 1600 Hz/60 dB	Alcohol

Table 2 continued

References	Country	*N*	Age	Cz (amp)	Fz (amp)	Cz (lat)	Fz (lat)	Stimuli	Notes
Henkin et al. (2002)	Israel	20	14.4	4.9	8.8		246.3	Std: 1400 Hz, Dev: 1000 Hz	AERPs; phonetic processing in children
Valkonen-Korhonen et al. (2003)	Finland	19	29	2.4		200		Std: 800 Hz, Dev: 560 Hz/55 dB	Auditory processing; psychotic
Lucchesi et al. (2003)	Brazil	12	24.8	5	3	200	200	Std: 800 Hz, Dev: 1500 Hz/70 dB	Flunitrazepam effect; N2b
van Harten et al. (2006)	Netherlands	53	73.6	5.6	5.6	232.5	232.5	Std: 1000 Hz, Dev: 2000 Hz/80 dB	Vascular cognitive impairment; peak-to-peak ERPs
Dixit et al. (2006)	India	40	21.5	3.9	5.5	209.6	215.3	Std: 1000 Hz, Dev: 2000 Hz/60 dB	Caffeine
Čeponienė et al. (2008)	USA	19	25.5	2.5	2.4	341		Std: 500 Hz, Dev: 550 Hz/63 dB	Aging; N1–N2–P2
		19	71.3	1.0	1.1	371			
Zhu et al. (2008)	China	15	23		3.1		225	Std: 1000 Hz, Dev: 2000 Hz/80 dB	Music; mandarin lexical tones
Wise et al. (2009)	Australia	98	35.6	5.5	4.9	231.6	238	Std: 500 Hz, Dev: 1000 Hz/75 dB	Panic disorder
Guney et al. (2009)	Turkey	32	37.1	8	10.0	216.3	225.8	Std: 1000 Hz, Dev: 2000 Hz/80 dB	Tobacco smokers
Dassanayake et al. (2009)	Sri Lanka	38	49			226.3		Std: 1000 Hz, Dev: 2000 Hz/75 dB	Pesticides
Ogawa et al. (2009)	Japan	19	64.5	4.7		210		Std: 1000 Hz, Dev: 2000 Hz/80 dB	Amyotrophic lateral sclerosis
Whelan et al. (2010)	Ireland	21	40.3	1.7		211.7		Std: 500 Hz, Dev: 1000 Hz	Multiple sclerosis
Gandelman-Marton et al. (2010)	Israel	18	35.6	7.1	7.1	221.2	223.1	Std: 1000 Hz, Dev: 2000 Hz/70 dB	Immediate and short-term retest
Tsai et al. (2012)	Taiwan	63	9		Pz			Std: 3000 Hz, Dev: 2000 Hz/60 dB	Age-related; correlations ADHD
		51	9	14.2	10.8	240.7	239.2		
Tanaka et al. (2012)	Japan	14	43	5.9		215		Std: 1000 Hz, Dev: 2000 Hz/80 dB	Myotonic dystrophy type 1
Schneider et al. (2013)	USA	40	13.9	1.6		285		Std: 1000 Hz, Dev: 2000 Hz/70 dB	Fragile X syndrome treatment

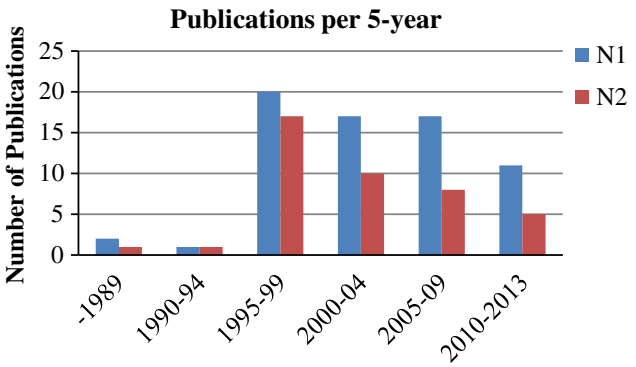

Fig. 2 Number of publications per 5-year for auditory oddball N1 (N2 included in N1 studies)

Discussion

Most of the studies assessed have elicited N1 and several N2 as well, in clinical groups, mainly in Neurology, Psychiatry and Psychology areas. This fact, suggests that in the last decades N1 and N2 components have been studied as possible endophenotypes or bio-markers for different disorders and in some cases for recovery index: namely, in schizophrenia (Hegerl et al. 1988; Haig et al. 1997; Potts et al. 1998a; Shelley et al. 1999; Ford et al. 2001; Brown et al. 2002; Gilmore et al. 2005; Neuhaus et al. 2013), dementia (Verma et al. 1989), Alzheimer (Sumi et al. 2000), epilepsy and AED effects (Akaho 1996; Ford et al. 2001; Lucchesi et al. 2003), alcohol (Brigham et al. 1997; Cohen et al. 2002) and substance abuse (Tarter et al. 1995; Brigham et al. 1997), psychosis (Valkonen-Korhonen et al. 2003), panic disorder (Wise et al. 2009), Parkinson's disease (Wright et al. (1996), ADHD (Johnstone et al. 2001; Tsai et al. 2012), stroke (Hirata et al. 1996) and vascular cognitive impairment (van Harten et al. 2006), myotonic dystrophy (Kazis et al. 1996; Tanaka et al. 2012), head injury (Reinvang et al. 2000; Segalowitz et al. 2001),

Table 3 Suggested normative values for N1 and N2 by age (control groups data from deviant stimuli at Fz and Cz)

Component	Age (years)	*N* (3934)	Participants age (mean/SD)	Amplitude (mean/SD) (μV)		Latency (mean/SD) (ms)	
				Cz	Fz	Cz	Fz
N1	(6–20)	518	12.3/3.3	8.0/3.0	8.8/2.8	124.0/15.1	160.0/54.0
	(21–40)	1326	28.8/5.7	7.1/3.2	6.9/2.9	107.2/11.8	111.0/13.6
	(41–60)	276	47.0/4.4	7.4/2.3	3.5/2.4	99.1/4.3	99.9/8.1
	(61–85)	307	69.9/6.0	7.6/2.9	6.8/5.0	103.5/21.4	99.6/9.1
N2	(9–20)	343	12.9/3.3	6.3/4.4	11.0/3.5	239.2/28.8	239.9/15.1
	(21–40)	706	28.3/5.8	4.7/2.1	4.7/2.2	224.8/30.9	225.6/17.5
	(41–60)	208	47.1/4.6	5.5/2.6	4.0/1.0	224.0/12.5	232.3/17.2
	(61–85)	250	70.9/6.3	3.7/1.6	2.3/2.0	249.0/49.7	238.3/26.9

suppressed HIV patients (Chao et al. 2004), chemotherapy (Kreukels et al. 2008) and pesticides exposure (Dassanayake et al. 2009), smoking (Guney et al. 2009), amyotrophic lateral sclerosis (Ogawa et al. 2009) and multiple sclerosis (Whelan et al. 2010), more recently fragile X syndrome treatment (Schneider et al. 2013), and bipolar disorder (Hamm et al. 2013).

In several respects, our data replicate the findings of others with regard to the effects of age on the amplitude and latency of N1 and N2. Regarding N1 latency, only Iragui et al. (1993) reported significant age-related increases at Cz. The common finding has been that N1 latency remains unchanged or slightly decreases with advancing age for target stimuli at this electrode, from where it has usually been recorded (Goodin et al. 1978; Brown et al. 1983; Picton et al. 1984; Barrett et al. 1987; Anderer et al. 1996; Amenedo and Díaz 1998; Tsai et al. 2012). Further, Anderer et al. (1996, 1998a) found a latency increase over age only for standard stimuli. But at Fz, N1 latency decrement is particularly pronounced during childhood and adolescence, this result confirms the assumptions that the maturation of the frontal N1 is not yet completed at the age of 9–12 years due to incomplete frontal myelination (Bruneau et al. 1997). Overall, the decrease in N1 latencies probably result from an increase in neural transmission speed due to age-related changes in myelination of underlying neural generators as well as increases in synaptic synchronization and efficacy (Huttenlocher 1979; Eggermont 1988; Courchesne 1990; Ponton et al. 1999).

A paradoxical but astonishing phenomenon seems to take place from childhood to adulthood. In 1979, Huttenlocher observed that a decline in synaptic density between ages 2 and 16 years was accompanied by a slight decrease in neuronal density. This is consistent with our results if we consider that synaptic density is related to N1 and N2 amplitudes. Both components have an amplitude decrement at Fz and Cz (Figs. 3, 4, 5, 6). Human cerebral cortex is one of a number of neuronal systems in which loss of neurons and synapses appears to occur as a late developmental event. The relationship of N1/N2 to the adult N1/N2 is unclear. Maturational changes in the central auditory system are complex and extend well into the second decade of life (Sharma et al. 1997; Gilley et al. 2005). We believe that the effects of age, stimulation rate and education influence components morphology and amplitude, the latter is usually taken as an exclusion or inclusion criteria. The scarce literature on possible education effects on N1 and N2 does not enable us to achieve further conclusions. However, a profound and comprehensive revision on these topics should be made in a future article. In a recent systematic review and meta-analysis regarding P3, no education effects were found (van Dinteren et al. 2014). This was possible because P3 is one of the most studied components with over 12,000 publications in 50 years of intensive research (van Dinteren et al. 2014). Despite the interesting literature review and discussion made by van Dinteren et al. (2014), we cannot compare results since they analyzed data only for the Pz electrode site. Although, it definitely seems that N1/N2 and P3 have different development across the lifespan, due to different generators and underlying psychophysiological processes.

The range age between 41 and 60 years is the one with fewer participants, both to N1 and N2 (Table 3). This seems to be related with scarce literature and less investigation oriented to clinical populations in this range.

Regarding N1 amplitude, results are inconsistent among authors, possibly due to short age range comparison. Our results reveal that N1 amplitude at Cz remains stable after adolescence as seen in Fig. 3 (Tsai et al. 2012), but at Fz there is a linear decrease from birth to 60 years old and then an exponential increment (Fig. 4). This result is consistent with the common finding in the literature, that young, adult and elderly differ in their ability to inhibit the processing of task irrelevant information resulting in a higher level of general attention during oddball task (Ford and Pfefferbaum 1991; Friedman et al. 1993; Anderer et al. 1998a; Ford et al. 2001). Furthermore, if we analyze the

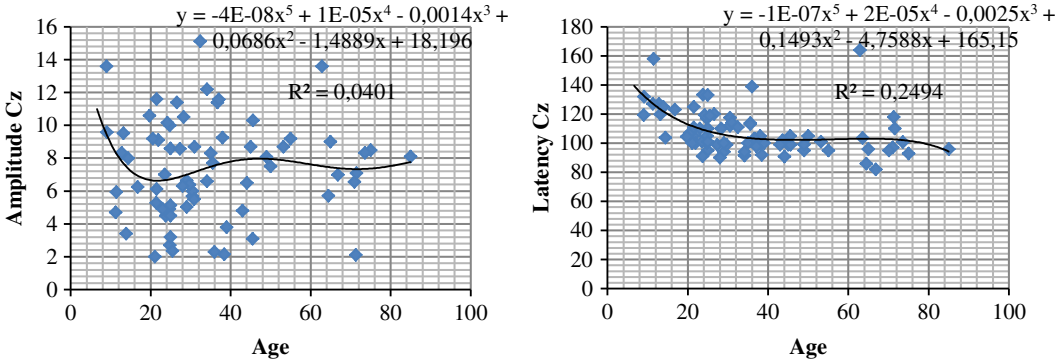

Fig. 3 Scatter plots of the relationship between N1 amplitude (μV; *left*), latency (ms; *right*) and age (years) at Cz (*curved lines* are the fifth degree polynomial regression over the age range 0–100 years)

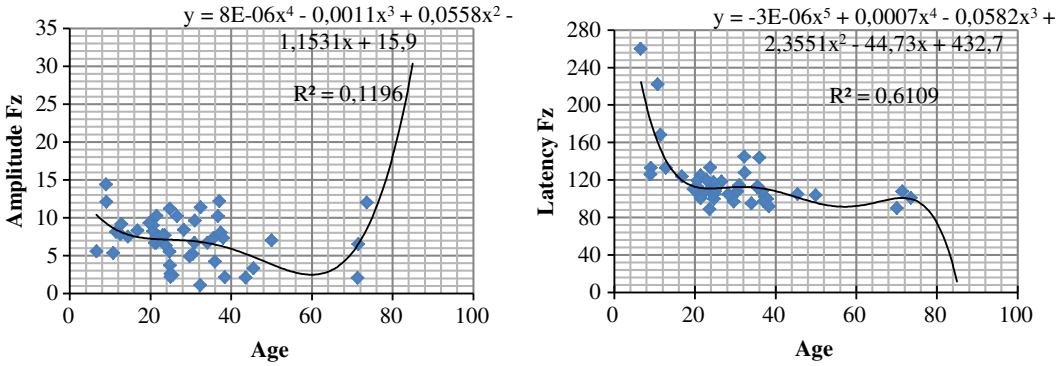

Fig. 4 Scatter plots of the relationship between N1 amplitude (μV; *left*), latency (ms; *right*) and age (years) at Fz (*curved lines* are the fourth and fifth degree, for amplitude and latency, respectively, polynomial regression over the age range 0–100 years)

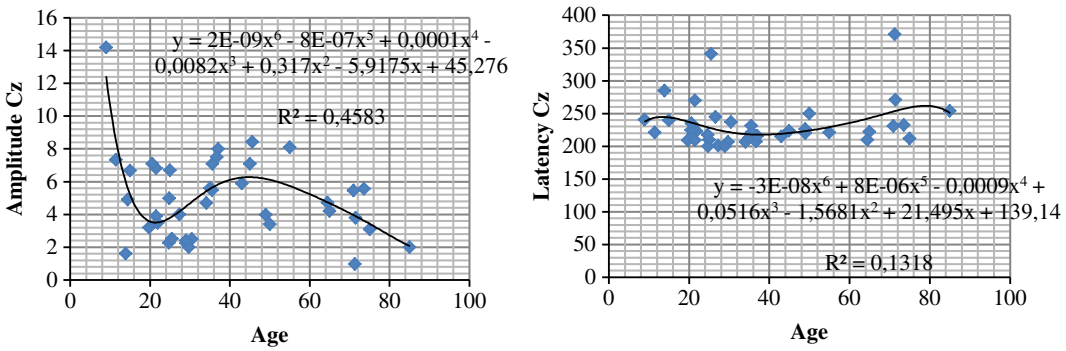

Fig. 5 Scatter plots of the relationship between N2 amplitude (μV; *left*), latency (ms; *right*) and age (years) at Cz (*curved lines* are the sixth degree polynomial regression over the age range 0–100 years)

differences between deviant and standard N1 sources of low resolution electromagnetic tomography (LORETA) studies, younger subjects show a pronounced difference, while it is hard to distinguish deviant N1 from standard N1 source in elder subjects (Pascual-Marqui et al. 1994; Anderer et al. 1998a, b).

This is called the reduction or decline of frontal inhibitory control, due to an ineffective top–down modulation of primary auditory responses by prefrontal cortex (Hasher and Zacks 1988; Čeponienė et al. 2008; for neuro-anatomical evidence see Raz et al. 1997; Chao and Knight, 1997b; Dustman et al. 1996; Kok 1999). In addition, it has been recently found that aging is related with a decrease in event-related spectral power activity, a low phase locking in N1 theta (4–7 Hz) band over the parietal/frontal regions and with a decrease of functional connections in the alpha

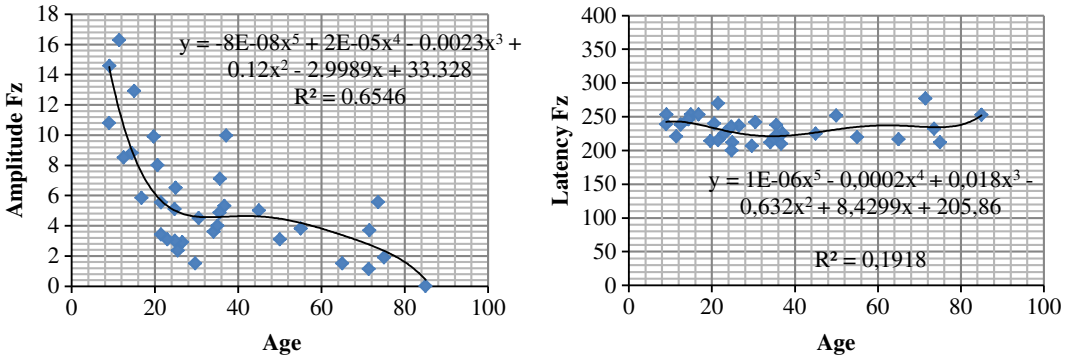

Fig. 6 Scatter plots of the relationship between N2 amplitude (μV; *left*), latency (ms; *right*) and age (years) at Fz (*curved lines* are the fifth degree polynomial regression over the age range 0–100 years)

(7–13 Hz) and beta (13–30 Hz) bands (Ho et al. 2012); this might indicate a higher neuroplasticity of younger brains and easy engagement of attentional resources.

Lastly, if we consider and combine results on P3 amplitude decrement and topography change to two distinct foci of activity over age and neuropsychological deficits pointed by several studies, it may be concluded that aging is associated with a general difficulty to distinguish relevant stimulation from irrelevant stimuli, in other words difficulties in selection, categorization and storing in working memory (Picton et al. 1984; Iragui et al. 1993; Lembreghts et al. 1995; Friedman et al. 1997; Amenedo and Díaz 1998; Polich 2007; Martin et al. 2008).

These findings are consistent with N2 common results. The N2 component is responsible for the classification or categorization of deviant stimuli (Mueller et al. 2008), a previous and required process before storing in working memory. Most studies showed a decrease in the N2 amplitude and latency from childhood to adulthood (Ladish and Polich 1989; Iragui et al. 1993; Lembreghts et al. 1995; Johnstone et al. 1996; Bertoli and Probst 2005; Mueller et al. 2008; Čeponienė et al. 2008; Tsai et al. 2012), our results are consistent with literature at Fz and Cz but only a slight decrement in latency until 40 years old (Figs. 5, 6). Similarly, Goodin et al. (1978) have already found a decrease in N2 latency with age in children of 6–15 years, but an increase in this latency activated or passed as early as during the N1 and P2 components. Regarding adulthood, Picton et al. (1984) reported a significant increase in N2 peak latency with age at a rate of 0.65 ms per year in adults of 20–79 years, for similar results have been achieved by several authors (Anderer et al. 1996; Mueller et al. 2008). Our results also revealed a slight increment in latency after 40 years old for both Fz and Cz (Figs. 5, 6). The increase in N2 latency seems to be due to linear increases in one of its components' latency (N2b) at Fz, Cz and Pz, indicating that the aging-related slowing begins at controlled memory comparison between non-target and target stimuli (Amenedo and Díaz 1998).

Keeping the above in mind, N2 amplitude decrement over age and latency increment after adulthood are the only auditory ERP findings that are in agreement with age-related decrease in auditory acuity (Baltes and Lindenberger 1997; Chao and Knight 1997a, b; Tremblay et al. 2003; Čeponienė et al. 2008). However, other studies have found no changes in N2 amplitude with advancing age (Brown et al. 1983; Picton et al. 1984; Barrett et al. 1987) and even increases at central and parietal scalp sites (Iragui et al. 1993; Enoki et al. 1993) which might explain N1 and N2 amplitude increment at Cz from 20 to 45 years in our scatter-plot (Figs. 3, 5). N1 and N2 amplitude increase in this range (20–45 years) might correlate with growth of synaptic density, paralleled by improving synaptic efficiency and spatiotemporal synchronization.

Further, Čeponienė et al. (2008) consider that N2 diminution with age could represent cyto-architectonic derangement in sensory regions, including diminished synaptic synchronization causing decreased processing speed (Peters 2002).

Finally, to consider using N1 and N2 components in clinical practice, it must be kept in mind that ERPs change topography over age, due to senescent-related changes and individual profound cortical reorganization. Regarding N1 and N2, a change from frontal to central–parietal topography peak with aging is the most common finding (Anderer et al. 1996, 1998a; Amenedo and Díaz 1998; Mueller et al. 2008) because frontal areas have also been reported to be more affected by aging than others (Goodin et al. 1978; West 1996; Amenedo and Díaz 1998; Mueller et al. 2008). Anderer et al. (1996) reported that N1 latency increases with advancing age were dependent on electrode position, as they only increased at posterior scalp sites. However, gender is a variable that should be taken notice of. Some authors have reported differences across electrode location and amplitude (Gölgeli et al. 1999). Very few studies reported control groups by gender precluding us to include it in our statistical analyses. In addition, a

comprehensive review regarding possible effects of IQ and years of education is of particular importance, in a forthcoming article. Also, despite means and standard deviations for N1 and N2 in Table 3, it is still a challenge to compare results of a single participant, and does not exempt of establishing normative values in the local laboratory, department or service.

It is important to conduct systematic reviews and normative values regarding P1 and P2 components and P1–N1–P2 complex (Zheng et al. 2011) also with auditory oddball paradigms and considering putative gender differences. The objective measurement of central auditory processing and integrity of final auditory pathway should not depend on just one component but on the presence of several ERPs (complex), reflecting progressive central sound processing and representation (McGee et al. 1997; Näätänen et al. 2007; Nikjeh et al. 2009) and also preservation of neuronal synchrony encoding temporal information (Rance et al. 2002; Tomé et al. 2012).

Summary and conclusion

The clinical use of N1 and N2 remains minimal because recording requires special equipment and knowledge to process and extract ERPs. But also because normative data are lacking and responses vary widely between subjects.

Results from this systematic review suggest that Fz and Cz are the electrode locations to clinically consider the normative values presented in results section. However, to accept N1 and N2 components as brain markers of pre-attentive and early cognitive function, it should be regarded parallel changes in brain development and components topography over age, mainly in childhood, adolescence and after 60 years. But certainly, using auditory oddball paradigms in an attended or unattended condition, N1 and N2 components seem a promising clinical indexing measure in the assessment of central auditory processing among several clinical conditions and populations.

Conflict of interest The authors have declared that there are no conflicts of interest in relation to the subject of this study. The contribution of Kamila Nowak was supported by NCBR grant No. INNOTECH-K1/IN1/30/159041/NCBR/12

References

Akaho R (1996) The effects of antiepileptic drugs on cognition in normal volunteers. Psychiatry Clin Neurosci 50:60–61

Altieri N (2013) Neural dynamics of audiovisual integration for speech and non-speech stimuli: a psychophysical study. Open Neurosci J 7:5–18

Amenedo E, Díaz F (1998) Aging-related changes in processing of non-target and target stimuli during an auditory oddball task. Biol Psychol 48:235–267

Anderer P, Semlitsch HV, Saletu B (1996) 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 99:458–472

Anderer P, Pascual-Marqui RD, Semlitsch HV, Saletu B (1998a) Differential effects of normal aging on sources of standard N1, target N1 and target P300 auditory event-related brain potentials revealed by low resolution electromagnetic tomography (LORETA). Electroencephalogr clin Neurophysiol 108:160–174

Anderer P, Pascual-Marqui RD, Semlitsch HV, Saletu B (1998b) Electrical sources of P300 event-related brain potentials revealed by low resolution electromagnetic tomography. 1. Effect of normal aging. Neuropsychobiology 37:20–27

Baltes PB, Lindenberger U (1997) Emergence of a powerful connection between sensory and cognitive functions across the adult life span: a new window to the study of cognitive aging? Psychol Aging 12(1):12–21

Barrett G, Neshige R, Shibasaki H (1987) Human auditory and somatosensory event-related potentials: effects of response condition and age. Electroencephalogr Clin Neurophysiol 66:409–417

Barry RJ, Kirkaikul S, Hodder D (2000) EEG alpha activity and the ERP to target stimuli in an auditory oddball paradigm. Int J Psychophysiol 39:39–50

Barry RJ, de Pascalis V, Hodder D, Clarke AR, Johnstone SJ (2003) Preferred EEG brain states at stimulus onset in a fixed interstimulus interval auditory oddball task, and their effects on ERP components. Int J Psychophysiol 47:187–198

Barry RJ, Rushby JA, Johnstone SJ, Clarke AR, Croft RJ, Lawrence CA (2004) Preferred EEG brain states at stimulus onset in a fixed interstimulus interval auditory oddball task, and their effects on ERP components. Clin Neurophysiol 115:2593–2601

Barry RJ, Rushby JA, Smith JL, Clarke AR, Croft RJ (2006) Dynamics of narrow-band EEG phase effects in the passive auditory oddball task. Eur J Neurosci 24:291–304

Bennett M, Hacker P (2007) Philosophical foundations of neuroscience. In: Bennett M, Dennett D, Hacker P, Searle J (eds) Neuroscience and philosophy: brain, mind and language, 1st edn. Columbia University Press, New York, pp 3–49

Bentin S, Carmon A (1984) Verbal information processing and hemispheric AEP asymmetry. Ann N Y Acad Sci 425:166–170

Berger H (1929) Uber das Elektrenkephalogramm das menchen. Archiv fur Psychiatrie 87:527–570

Bertoli S, Probst R (2005) Lack of standard N2 in elderly participants indicates inhibitory processing deficit. NeuroReport 16(17):1933–1937

Bortoletto M, Tona G, Scozzari S, Sarasso S, Stegagno L (2011) Effects of sleep deprivation on auditory change detection: a N1-Mismatch Negativity study. Int J Psychophysiol 81:312–316

Boucher O, Bastien C, Saint-Amour D, Dewailly E, Ayotte P, Jacobson J et al (2010) Prenatal exposure to methylmercury and PCBs affects distinct stages of information processing: an event-related potential study with Inuit children. Neurotoxicology 31:373–384

Brigham J, Moss HB, Murrelle EL, Kirisci L, Spinelli JS (1997) Event-related potential negative shift in sons of polysubstance- and alcohol-use disorder fathers. Psych Res 73:133–146

Brown WS, Marsh JT, La Rue A (1983) Exponential electrophysiological aging: P300 latency. Electroencephalogr Clin Neurophysiol 55:277–285

Brown KJ, Gonsalvez CJ, Harris AWF, Williams LM, Gordon E (2002) Target and non-target ERP disturbances in first episode vs. chronic schizophrenia. Clin Neurophysiol 113:1754–1763

Brown CR, Clarke AR, Barry RJ (2006) Inter-modal attention: ERPs to auditory targets in an inter-modal oddball task. Int J Psychophysiol 62:77–86

Bruneau N, Roux S, Guérin P, Barthélémy C, Lelord G (1997) Temporal prominence of auditory evoked potentials (N1 wave) in 4–8 year-old children. Psychophysiology 34:32–38

Cahn B, Polich J (2009) Meditation (Vipassana) and the P3a event-related brain potential. Int J Psychophysiol 72:51–60

Cansino S, Williamson SJ, Karron D (1994) Tonotopic organization of human auditory association cortex. Brain Res 663:38–50

Čeponienė R, Rinne T, Näätänen R (2002) Maturation of cortical sound processing as indexed by event-related potentials. Clin Neurophysiol 108:870–882

Čeponienė R, Lepistö T, Alku P, Aro H, Näätänen R (2003) Event-related potential indices of auditory vowel processing in 3-year-old children. Clin Neurophysiol 114:652–661

Čeponienė R, Westerfield M, Torki M, Townsend J (2008) Modality-specificity of sensory aging in vision and audition: evidence from event-related potentials. Brain Res 1215:53–68

Chao LL, Knight RT (1997a) Age-related prefrontal alterations during auditory memory. Neurobiol Aging 18(1):87–95

Chao LL, Knight RT (1997b) Prefrontal deficits in attention and inhibitory control with aging. Cereb Cortex 7(1):63–69

Chao LL, Lindgren JA, Flenniken DL, Weiner WW (2004) ERP evidence of impaired central nervous system function in virally suppressed HIV patients on antiretroviral therapy. Clin Neurophysiol 115:1583–1591

Chunhau P, Shimono M, Iwanaga M, Hasegawa R, Honda Y, Shirahata A et al (2005) Analysis of P3 in a continuous 40-min auditory oddball task. Int Cong Series 1278:105–108

Cohen HL, Ji J, Chorlian DB, Begleiter H, Porjesz B (2002) Alcohol-related ERP changes recorded from different modalities: a topographic analysis. Alcohol Clin Exp Res 26(3):303–317

Courchesne E (1990) Chronology of postnatal human brain development: event-related potential, positron emission tomography, myelinogenesis, and synaptogenesis studies. In: Rohrbaugh JW, Parasuraman R, Johnson R (eds) Event-related brain potentials: basic issues and applications. Oxford University Press, New York, pp 210–241

Crowley KE, Colrain IM (2004) A review of the evidence for P2 being an independent component process: age, sleep and modality. Clin Neurophysiol 115:732–744

Dassanayake T, Gawarammana IB, Weerasinghe V, Dissanayake PS, Pragaash S, Dawson A, Senanayake N (2009) Auditory event-related potential changes in chronic occupational exposure to organophosphate pesticides. Clin Neurophysiol 120:1693–1698

Davis PA (1939) Effects of acoustic stimuli on the waking human brain. J Neurophysiology 2:494–499

Davis H, Zerlin S (1966) Acoustic relations of the human vertex potential. J Acoust Soc Am 39:109–116

Demiralp T, Yordanova J, Kolev V, Ademoglu A, Devrim M, Samar VJ (1999) Time-frequency analysis of single-sweep event-related potentials by means of fast wavelet transform. Brain Lang 66:129–145

Dixit A, Vaney N, Tandon OP (2006) Evaluation of cognitive brain functions in caffeine users: a P3 evoked potential study. Indian J Physiol Pharmacol 50(2):175–180

Donchin E (1981) Presidential address, 1980. Surprise!. Surprise? Psychophysiology 18:493–513

Duncan C, Barry R, Connolly J, Fischer C, Michie P, Näätänen R et al (2009) Event-related potentials in clinical research: guidelines for eliciting, recording, and quantifying mismatch negativity, P300, and N400. Clin Neurophysiol 120:1883–1908

Dustman RE, Emmerson RY, Shearer DE (1996) Life span changes in electrophysiological measures of inhibition. Brain Cogn 30(1):109–126

Eggermont JJ (1988) On the rate of maturation of sensory evoked potentials. Electroencephalogr Clin Neurophysiol 70(4):293–305

Elberling C, Bak C, Kofoed B, Lebech J, Saermark K (1980) Magnetic auditory responses from the human brain. A preliminary report. Scand Audiol 9:185–190

Enoki H, Sanada S, Yoshinaga H, Oka E, Ohtahara S (1993) The effects of age on the N200 component of the auditory event-related potentials. Cogn Brain Res 1:161–167

Eysenck MW, Keane MT (2003) (eds) Cognitive psychology: a student's handbook, 4th edn. Psychology Press, East Sussex, pp 1–27

Fabiani M, Gratton G, Federmeier (2007) Event-related brain potentials: methods, theory, and applications. In: Cacioppo J, Tassinary LG, Berntson GG (eds) The Handbook of Psychophysiology, 3rd edn. Cambridge University Press, New York, pp 94–119

Ford JM, Pfefferbaum A (1991) Event-related potentials and eyeblink responses in automatic and controlled processing: effects of age. Electroencephalogr Clin Neurophysiol 78:361–377

Ford JM, Mathalon DH, Kalba S, Marsh L, Pfefferbaum A (2001) N1 and P300 abnormalities in patients with schizophrenia, epilepsy, and epilepsy with schizophrenia like features. Biol Psychiatry 49:848–860

Friedman D, Simpson GV, Hamberger M (1993) Age-related changes in scalp topography to novel and target stimuli. Psychophysiology 30:383–396

Friedman D, Kazmerski V, Fabiani M (1997) An overview of age-related changes in the scalp distribution of P3b. Electroencephalogr Clin Neurophysiol 104:498–513

Gandelman-Marton R, Theitler J, Klein C, Rabey JM (2010) The effects of immediate and short-term retest on the latencies and amplitudes of the auditory event-related potentials in healthy adults. J Neurosci Methods 186:77–80

Gilley PM, Sharma A, Dorman M, Martin K (2005) Developmental changes in refractoriness of the cortical auditory evoked potential. Clin Neurophysiol 116:646–657

Gilmore CS, Clementz BA, Buckley PF (2005) Stimulus sequence affects schizophrenia–normal differences in event processing during an auditory oddball task. Cogn Brain Res 24:215–227

Gilmore CS, Clementz BA, Berg P (2009) Hemispheric differences in auditory oddball responses during monaural versus binaural stimulation. Int J Psychophysiol 73:326–333

Gölgeli A, Süer C, Özesmi Ç, Dolu N, Aşcioğ lu M, Şahin Ö (1999) The effect of sex differences in young adults on event-related potentials. Int J Neurosci 99:69–77

Gonsalvez CJ, Gordon E, Grayson S, Barry RJ, Lazzaro I, Bahramali H (1999) Is the target-to-target interval a critical determinant of P3 amplitude? Psychophysiology 36:643–654

Goodin DS, Squires KC, Henderson BH, Starr A (1978) Age-related variations in evoked potentials to auditory stimuli in normal human subjects. Electroencephalogr Clin Neurophysiol 44:447–458

Guney F, Genc B, Kutlu R, Ilhan B (2009) Auditory P300 event-related potential in tobacco smokers. J Clin Neurosci 16:1311–1315

Haig AR, Rennie C, Gordon E (1997) The use of Gaussian component modelling to elucidate average ERP component overlap in schizophrenia. J Psychophysiol 11:173–187

Hamm JP, Ethridge LE, Shapiro JR, Pearlson GD, Tamminga CA, Sweeney JA, Keshavan MS, Thaker GK, Clementz BA (2013) Family history of psychosis moderates early auditory cortical response abnormalities in non-psychotic bipolar disorder. Bipolar Disord 15:774–786

Hari R, Aittoniemi K, Järvinen M-L, Katila T, Varpula T (1980) Auditory evoked transient and sustained magnetic fields of the human brain. Localization of neural generators. Exp Brain Res 40:237–240

Hasher L, Zacks RT (1988) Working memory, comprehension, and aging: a review and new view. In: Bower GH (ed) The Psychology of Learning and Motivation, 22. Academic Press, San Diego, pp 193–225

Hegerl U, Gaebel W, Gutzman H, Ulrich G (1988) Auditory evoked potentials as possible predictors of outcome in schizophrenic outpatients. Int J Psychophysiol 6:207–214

Henkin Y, Kishon-Rabin L, Gadoth N, Pratt H (2002) Auditory event-related potentials during phonetic and semantic processing in children. Audiol Neurootol 7:228–239

Hillyard SA, Kutas M (1983) Electrophysiology of cognitive processing. Ann Rev Psychol 34:33–61

Hillyard SA, Hink RF, Schwent VL, Picton TW (1973) Electrical signs of selective attention in the human brain. Science 182:177–180

Hirata K, Katayama S, Yamazaki K, Fujikane M, Katayama K (1996) Electric field distribution of event-related potentials in stroke patients. Brain Topogr 8(3):279–284

Ho M-C, Chou C-Y, Huang C-F, Lin Y-T, Shih C-S, Han S-Y, Shen M-H et al (2012) Age-related changes of task-specific brain activity in normal aging. Neurosci Lett 507:78–83

Huttenlocher PR (1979) Synaptic density in human frontal cortex—developmental changes and effects of aging. Brain Res 163(2):195–205

Inui K, Okamoto H, Miki K, Gunji A, Kakigi R (2006) Serial and parallel processing in the human auditory cortex: a magnetoencephalographic study. Cereb Cortex 16:18–30

Iragui VJ, Kutas M, Mitchiner MR, Hillyard SA (1993) Effects of aging on event-related brain potentials and reaction times in an auditory oddball task. Psychophysiology 30:10–22

Jääskeläinen IP (2012) Introduction to cognitive neuroscience. Ventus Publishing ApS, Denmark, pp 92–112. http://pt.scribd.com/doc/102335497/Introduction-to-Cognitive-Neuroscience

Jääskeläinen IP, Ahveninen J, Bonmassar G, Dale AM, Ilmoniemi RJ, Levänen S et al (2004) Human posterior auditory cortex gates novel sounds to consciousness. Proc Nat Acad Sci (USA) 101:6809–6814

Johnstone SJ, Barry RJ, Anderson JW, Coyle SF (1996) Age-related changes in child and adolescent event-related potential component morphology, amplitude and latency to standard and target stimuli in an auditory oddball task. Int J Psychophysiol 24:223–238

Johnstone SJ, Barry RJ, Anderson JW (2001) Topographic distribution and developmental timecourse of auditory event-related potentials in two subtypes of attention-deficit hyperactivity disorder. Int J Psychophysiol 42:73–94

Kazis A, Kimiskidis V, Georgiadis G, Kapinas K (1996) Cognitive event-related potentials and magnetic resonance imaging in myotonic dystrophy. Neurophysiol Clin 26:75–84

Kok A (1999) Varieties of inhibition: manifestations in cognition, event-related potentials and aging. Acta Psychol (Amst) 101(2–3):129–158

Kreukels BPC, Hamburger HL, de Ruiter MB, van Dam FS, Ridderinkhof KR, Boogerd W, Schagen SB (2008) ERP amplitude and latency in breast cancer survivors treated with adjuvant chemotherapy. Clin Neurophysiol 119:533–541

Ladish C, Polich J (1989) P300 and probability in children. J Exp Child Psychol 48:212–223

Lembreghts M, Crasson M, EL Ahmadi A, Timsit-Berthier M (1995) Éude de la variabilité interindividuelle des potentiels évoqués auditifs exogènes et endogènes en condition d'attention volontaire. Neurophysiol Clin 25:203–223

Liasis A, Bamiou DE, Boyd S, Towell A (2006) Evidence for a neurophysiologic auditory deficit in children with benign epilepsy with centro-temporal spikes. J Neural Transm 113:939–949

Lucchesi L, Pompéia S, Manzano G, Kohn A, Galduroz J, Bueno O, Tufik T (2003) Flunitrazepam-induced changes in neurophysiological, behavioural, and subjective measures used to assess sedation. Prog Neuro-Psychopharmacol Biol Psychiatry 27:525–533

Martin BA, Tremblay KL, Korczak P (2008) Speech evoked potentials: from the laboratory to the clinic. Ear Hear 29:285–313

May P, Tiitinen H (2010) Mismatch negativity (MMN), the deviance-elicited auditory deflection, explained. Psychophysiology 47:66–122

McGee T, Kraus N, Nicol T (1997) Is it really a mismatch negativity? An assessment of methods for determining response validity in individual subjects. Electroencephalogr Clin Neurophysiol 104:359–368

Moher D, Liberati A, Tetzlaff J, Altman DG (2009) Preferred reporting items for systematic reviews and meta-analyses: the PRISMA statement. Ann Intern Med 151(4):264–269

Mueller V, Brehmer Y, von Oertzen T, Li S-C, Lindenberger U (2008) Electrophysiological correlates of selective attention: a lifespan comparison. BMC Neurosci 9:18

Mulert C, Jäger L, Schmitt R, Bussfeld P, Pogarell O, Möller H-J et al (2004) Integration of fMRI and simultaneous EEG: towards a comprehensive understanding of localization and time-course of brain activity in target detection. Neuroimage 22:83–94

Murray MM, Brunet D, Michel CM (2008) Topographic ERP analyses: a step-by-step tutorial review. Brain Top 20(4):249–264

Näätänen R (1975) Selective attention and evoked potentials in humans—a critical review. Biol Psychol 2:237–307

Näätänen R (1982) Processing negativity: an evoked-potential reflection of selective attention. Psychol Bull 92:605–640

Näätänen R, Picton T (1987) The N1 wave of the human electric and magnetic response to sound: a review and an analysis of the component structure. Psychophysiology 24:375–425

Näätänen R, Gaillard AWK, Mäntysalo S (1978) Early selective attention effect on evoked potential reinterpreted. Acta Psychol (Amst) 42:313–329

Näätänen R, Paavilainen P, Rinne T, Alho K (2007) The mismatch negativity (MMN) in basic research of central auditory processing: a review. Clin Neurophysiol 118:2544–2590

Neuhaus AH, Popescu FC, Rentzsch J, Gallinat J (2013) Critical evaluation of auditory event-related potential deficits in schizophrenia: evidence from large-scale single-subject pattern classification. Schizophr Bull doi:10.1093/schbul/sbt151

Nikjeh DA, Lister JJ, Frisch SA (2009) Pre-attentive cortical-evoked responses to pure tones, harmonic tones, and speech: influence of music training. Ear Hear 30:43–46

Novak GP, Ritter W, Vaughan HG Jr, Wiznitzer ML (1990) Differentiation of negative event-related potentials in an auditory discrimination task. Electroencephalogr Clin Neurophysiol 75:255–275

Novak GP, Ritter W, Vaughan HG Jr (1992) Mismatch detection and the latency of temporal judgements. Psychophysiology 29:399–411

Ogawa T, Tanaka H, Hirata K (2009) Cognitive deficits in amyotrophic lateral sclerosis evaluated by event-related potentials. Clin Neurophysiol 120:659–664

Pantev C, Bertrand O, Eulitz C, Verkindt C, Hampson S, Schuierer G et al (1995) Specific tonotopic organizations of different areas of the human auditory cortex revealed by simultaneous magnetic and electric recordings. Electroencephalogr clin Neurophysiol 94:26–40

Papanicolaou AC, Baumann S, Rogers RL, Saydjari C, Amparo EG, Eisenberg HM (1990) Localization of auditory response sources

using magnetoencephalography and magnetic resonance imaging. Arch Neurol 47:33–37
Pascual-Marqui RD, Michel CM, Lehmann D (1994) Low resolution electromagnetic tomography: a new method for localizing electrical activity in the brain. Int J Psychophysiol 18:49–65
Peters A (2002) Structural changes in the normally aging cerebral cortex of primates. Prog Brain Res 136:455–465
Picton TW, Stuss DT, Champagne SC, Nelson RF (1984) The effects of age on human event-related potentials. Psychophysiology 21:312–325
Polich J (2007) Updating P300: an integrative theory of P3a and P3b. Clin Neurophysiol 118:2128–2148
Ponton CW, Moore JK, Eggermont JJ (1999) Prolonged deafness limits auditory system developmental plasticity: evidence from evoked potentials study in children with cochlear implants. Scand Audiol Suppl 51:13–22
Ponton CW, Eggermont JJ, Kwong B, Don M (2000) Maturation of human central auditory system activity: evidence from multichannel evoked potentials. Clin Neurophysiol 111:220–236
Potts G, Hirayasu Y, O'Donnell B, Shenton M, McCarley R (1998a) High-density recording and topographic analysis of the auditory oddball event-related potential in patients with schizophrenia. Biol Psychiatry 44:982–989
Potts G, Dien J, Hartry-Speiser A, McDougal L, Tucker D (1998b) Dense sensor array topography of the event-related potential to task-relevant auditory stimuli. Electroencephalogr clin Neurophysiol 106:444–456
Rance G, Cone-Wesson B, Wunderlich J, Dowell R (2002) Speech perception and cortical event related potentials in children with auditory neuropathy. Ear Hear 23:239–253
Raz N, Gunning FM, Head D, Dupuis JH, McQuain J, Briggs SD et al (1997) Selective aging of the human cerebral cortex observed in vivo: differential vulnerability of the prefrontal gray matter. Cereb Cortex 7(3):268–282
Reinvang I, Nordby H, Nielsen CS (2000) Information processing deficits in head injury assessed with ERPs reflecting early and late processing stages. Neuropsychologia 38:995–1005
Ritter W, Vaughan HG (1969) Averaged evoked responses in vigilance and discrimination: a reassessment. Science 164:326–328
Ritter W, Simson R, Vaughan H, Friedman D (1979) A brain event related to the making of a sensory discrimination. Science 203:1358–1361
Rosburg T, Boutros NN, Ford JM (2008) Reduced auditory evoked potential component N100 in schizophrenia—a critical review. Psychiatry Res 161:259–274
Sakamoto K, Nakata H, Kakigi R (2009) The effect of mastication on human cognitive processing: a study using event-related potentials. Clin Neurophysiol 120:41–50
Sams M, Paavilainen P, Alho K, Näätänen R (1985) Auditory frequency discrimination and event-related potentials. Electroencephalogr Clin Neurophysiol 62:437–448
Schneider A, Leigh MJ, Adams P, Nanakul R, Chechi T, Olichney J, Hagerman R, Hessl D (2013) Electrocortical changes associated with minocycline treatment in fragile X syndrome. J Psychopharmacol 27(10):956–963
Segalowitz S, Bernstein D, Lawson S (2001) P300 event-related potential decrements in well-functioning university students with mild head injury. Brain Cogn 45:342–356
Sharma A, Kraus N, McGee TJ, Nicol TG (1997) Developmental changes in P1 and N1 central auditory responses elicited by consonant-vowel syllables. Electroencephalogr clin Neurophysiol 104:540–545
Shelley A-M, Silipo G, Javitt D (1999) Diminished responsiveness of ERPs in schizophrenic subjects to changes in auditory stimulation parameters: implications for theories of cortical dysfunction. Schizophr Res 37:65–79
Siedenberg R, Goodin DS, Aminoff MJ, Rowley HA, Roberts TPL (1996) Comparison of late components in simultaneously recorded event-related electrical potentials and event-related magnetic fields. Electroencephalogr clin Neurophysiol 99:191–197
Squires NK, Squires KC, Hillyard SA (1975) Two varieties of long-latency positive waves evoked by unpredictable auditory stimuli in man. Electroencephalogr clin Neurophysiol 38(4):387–401
Sumi N, Nan'no H, Fujimoto O, Ohta Y, Takeda M (2000) Interpeak latency of auditory event-related potentials (P300) in senile depression and dementia of the Alzheimer type. Psychiatry Clin Neurosci 54(6):679–684
Tanaka H, Arai M, Harada M, Hozumi A, Hirata K (2012) Cognition and event-related potentials in adult-onset non-demented myotonic dystrophy type 1. Clin Neurophysiol 123:261–269
Tarter R, Blackson T, Brigham J, Moss H, Caprara G (1995) The association between childhood irritability and liability to substance use in early adolescence: a 2-year follow-up study of boys at risk for substance abuse. Drug Alcohol Depend 39:253–261
Tiitinen H, May P, Reinikainen K, Näätänen R (1994) Attentive novelty detection in humans is governed by pre-attentive sensory memory. Nature 372:90–92
Tomé D, Marques-Teixeira J, Barbosa F (2012) Temporal lobe epilepsy in childhood—a study model of auditory processing. J Neurol Neurophysiol 3(2). doi:10.4172/2155-9562.1000123
Toscano J, McMurray B, Dennhardt J, Luck S (2010) Continuous perception and graded categorization: electrophysiological evidence for a linear relationship between the acoustic signal and perceptual encoding of speech. Psychological Sci 21(10):1532–1540
Tremblay KL, Piskosz M, Souza P (2003) Effects of age and age-related hearing loss on the neural representation of speech cues. Clin Neurophysiol 114(7):1332–1343
Tsai M-L, Hung K-L, Tung W, Chiang T-R (2012) Age-changed normative auditory event-related potential value in children in Taiwan. J Form Med Assoc 111:245–252
Ullsperger P, Freude G, Erdmann U (2001) Auditory probe sensitivity to mental workload changes—an event-related potential study. Int J Psychophysiol 40:201–209
Valkonen-Korhonen M, Purhonena M, Tarkka I, Sipilä P, Partanen J, Karhu J et al (2003) Altered auditory processing in acutely psychotic never-medicated first-episode patients. Cogn Brain Res 17:747–758
van Dinteren R, Arns M, Jongsma MLA, Kessels RPC (2014) P300 development across the lifespan: a systematic review and meta-analysis. PLoS One 9(2):e87347
van Harten B, Laman DM, van Duijn H, Knol DL, Stam CJ, Scheltens P, Weinstein HC (2006) The auditory oddball paradigm in patients with vascular cognitive impairment: a prolonged latency of the N2 complex. Dement Geriatr Cogn Disord 21:322–327
Verma N, Nichols C, Greiffenstein M, Singh R, Hurst-Gordon D (1989) Waves earlier than P3 are more informative in putative subcortical dementias: a study with mapping and neuropsychological techniques. Brain Topogr 1(3):183–191
Wang W, Wang Y-H (2001) Sensation seeking correlates of passive auditory P3 to a single stimulus. Neuropsychologia 39:1188–1193
Wang W, Datta H, Sussman E (2005) The development of the length of the temporal window of integration for rapidly presented auditory information as indexed by MMN. Clin Neurophysiol 116:1695–1706
West RL (1996) An application of prefrontal cortex function theory to cognitive aging. Psychol Bull 120:272–292

Wetzel N, Widmann A, Schröger E (2011) Processing of novel identifiability and duration in children and adults. Biol Psychol 86:39–49

Whelan R, Lonergan R, Kiiski H, Nolan H, Kinsella K, Bramham J, O'Brien M et al (2010) A high-density ERP study reveals latency, amplitude, and topographical differences in multiple sclerosis patients versus controls. Clin Neurophysiol 121:1420–1426

Winkler I (2007) Interpreting the mismatch negativity. J Psychophysiol 21(3–4):147–163

Winter O, Kok A, Kenemans J, Elton M (1995) Auditory event-related potentials to deviant stimuli during drowsiness and stage 2 sleep. Electroencephalogr clin Neurophysiol 96:398–412

Wise V, McFarlane AC, Clark CR, Battersby M (2009) Event-related potential and autonomic signs of maladaptive information processing during an auditory oddball task in panic disorder. Int J Psychophysiol 74:34–44

Wolpaw JR, Penry JK (1975) A temporal component of the auditory evoked response. Electroencephalogr clin Neurophysiol 39(6):609–620

Woods DL, Alho K, Algazi A (1993) Intermodal selective attention: evidence for processing in tonotópica auditory fields. Psychophysiology 30:287–295

Wright MJ, Geffen GM, Geffen LB (1996) ERP measures of stimulus processing during an auditory oddball task in Parkinson's disease: evidence for an early information processing deficit. Parkinsonism Rel Disord 2(1):13–21

Yamamoto T, Williamson SJ, Kaufman L, Nicholson C, Llinás R (1988) Magnetic localization of neuronal activity in the human brain. Proc Nat Acad Sci (USA) 85:8732–8736

Yvert B, Crouzeix A, Bertrand O, Seither-Preisler A, Pantev C (2001) Multiple supratemporal sources of magnetic and electric auditory evoked middle latency components in humans. Cereb Cortex 11:411–423

Yvert B, Fischer C, Bertrand O, Pernier J (2005) Localization of human supratemporal auditory areas from intracerebral auditory evoked potentials using distributed source models. Neuroimage 28:140–153

Zheng Y, Zhao F, Liang M, Bardsley B, Yang H, Zhang Z (2011) Toward an understanding of auditory evoked cortical event-related potentials: characteristics and classification. Audiol Med 1–10. doi:10.3109/1651386X.2010.537910

Zhu W, Zhang J, Liu H, Ding X, Ma Y, Zhou C (2008) Differential cognitive responses to guqin music and piano music in Chinese subjects: an event-related potential study. Neurosci Bull 24(1):21–28