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Early Auditory Evoked Potentials (EAEP) in Vertebral Basilar Insufficiency

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Summary. Stimulation with a short tone pip elicits an acoustic nerve compound action potential (I) and different waves (II—VII) in the initial 10 ms. Seven waves have been studied in 40 control subjects and five waves in 12 patients with vertebral-basilar insufficiency. Abnormalities of the different waves were observed at levels such as cochlea and/or acoustic nerve, medulla, caudal pons, rostral pons, and midbrain. The recording of early auditory evoked potentials (EAEP) is a noninvasive method of confirming impairment of the auditory pathway caused by a reduced vascular supply of vertebral and basilar arteries.

Key words: Early auditory evoked potentials – Vertebral-basilar insufficiency.

Zusammenfassung. Nach akustischer Reizung mit einem kurzen Tonimpuls (tone pip) lassen sich in den ersten 10 ms das Summenaktionspotential des N. acusticus (I) und weitere 6 Wellen (II—VII) ableiten. 7 Komponenten wurden bei 40 Normalpersonen und 5 Komponenten (I—V) bei 12 Patienten mit typischen Symptomen einer vertebro-basilären Insuffizienz einer genauen Analyse unterzogen. Veränderungen der frühen akustisch evozierten Potentiale (FAEP) lassen eine topische Zuordnung gefäßbedingter Schädigungen dorso-lateraler Hirnstammanteile zu. Es handelt sich um eine nicht-invasive Untersuchungsmethode, die durch Verlaufskontrollen prognostische Aussagen ermöglicht.

Schlüsselwörter: Frühe akustisch evozierte Potentiale – Vertebro-basiläre Insuffizienz.

Introduction

The diagnostic value of early auditory evoked potentials (EAEP) in the assessment of acoustic nerve and brain stem dysfunction is well-known (Starr and Achor, 1975; Thornton and Hawkes, 1976; Stockard and Rossiter, 1977; Chiappa

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et al., 1977) and has been described by our group previously (Maurer et al., 1978 and 1979a, b, c). It is generally accepted that waves I through V originate from different levels along the auditory pathway: cochlea and/or acoustic nerve (I), medulla (II), caudal pons (III), rostral pons (IV), and midbrain (V). Acoustic route impairment due to tumors, demyelination and in this context, disturbances of vascular supply in the vertebral and basilar arteries can be identified by abnormalities of latency and amplitude of the various components. Besides the description of neurologic defects and corresponding wave form abnormalities, otologic and psychiatric symptoms, if present, will be mentioned.

Methods and Patients

Detailed information for recording EAEP is given elsewhere (Maurer et al., 1979c). The choice of stimulus was determined by conditions such as short duration and optimal synchronization of auditory units. This condition seems to be best achieved by a short tone pip. The acoustic pattern of our stimulus consists mainly of three half-cycles of about 1 ms duration (Fig. 1). The acoustic waveform corresponds to the gaussian elementary signal or 'logon' described in detail by Gabor (1947) and Davis (1976).

Tone pips were presented monaurally with a repetition rate of 10/s and an intensity of 70 and 80 dBHL (above hearing level of normal listeners). The recording procedure was carried out at least twice and only reproducible components were accepted for clinical evaluation.

Latencies were determined between the onset of the stimulus and the upward positive peak of each wave, or between peaks of two waves, and calculated to the nearest 0.05 ms (Table 1). Amplitudes were measured from positive peaks to following negative troughs (post-I, -II, -III, -V) or vice versa (pre-IV). Latency and amplitude abnormalties could be defined by comparing values to corresponding means and standard deviations of a control group (40 normal listeners). A prolongation of latency or reduction of amplitude by more than two standard deviations from these normal values was considered significant. The higher degree of variability of amplitudes between individuals made it advisable to compare abnormalities with preceding normal waves from the same side or to superimpose corresponding waves from both auditory pathways (Fig. 1).

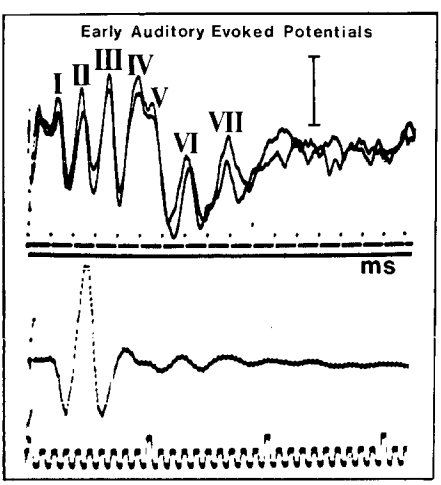

Fig. 1. *Upper part:* Scalp recordings of cochlear nerve (I) and brain stem evoked potentials in a neurologically and audiometrically normal subject. Each of the two tracings is the average of 1500 responses evoked by 10/s 80 dBHL monaural tone pips and recorded from vertex to ipsilateral mastoid. In this and all subsequent figures, posivity at the vertex produces upward deflections. Calibration 200 nV. *Lower part:* Acoustical pattern of the tone pip used in this study delivered by a specially shielded earphone

Table 1. Normal latencies of acoustic nerve and brain stem responses. Mean of 40 normal subjects; values given in milliseconds

Intensity	I	II	III	IV	V	VI	VII
70 dB	1.55 ± 0.1	2.7 ± 0.15	3.7 ± 0.15	4.9 ± 0.12	5.7 ± 0.13	7.2 ± 0.34	8.95 ± 0.37
80 dB	1.4 ± 0.06	2.6 ± 0.14	3.65 ± 0.14	4.8 ± 0.15	5.6 ± 0.1	7.1 ± 0.32	8.6 ± 0.55

All our patients underwent neurologic, otologic, and psychiatric examinations. The diagnostic procedure was completed in all cases by neuroradiologic means such as computerized axial tomography and brain perfusion to confirm the site of the vascular lesion. Only components I through V were taken into consideration, waves which could be recorded in 100% of our control group.

Results

Twelve patients (Table 2) had intermittent or permanent symptoms due to an impaired blood supply in the distribution area of the vertebral or basilar arteries. The main complaints were drop seizures, episodes of confusion, vertigo, transitory blindness, diplopia, ophthalmoplegia, and dysarthria. Ataxia, weakness, and numbness were occasionally apparent on one or both sides of the body. In some cases a depressive syndrome could be explored by psychiatric examination with typical diagnostic criteria of an exogenous cyclothymia (Marneros and Philipp, 1978; Marneros, 1979).

In two patients (Table 2, cases 1 and 2) results obtained by acoustic stimulation were normal regarding latency and amplitude. The clinical signs had a transitory character and there were no complaints or symptoms in the interval. Eight patients had abnormalities pointing to a lesion at the medullary, pontine, and mesencephalic levels of the acoustic route (Table 2, cases 3 to 10). Two patients (Table 2, cases 11 and 12) showed poor reproducibility of wave I and succeeding waves.

The following section deals with typical cases showing lesions at different levels of the auditory pathway. Anamnestic data and main symptoms are mentioned to complete the picture of the underlying circulatory disturbances. Figure 2 illustrates the main vessels and branches supplying the brain stem.

Lesions Affecting Wave II (Table 1, Case 3)

A 29-year-old man rapidly developed symptoms indicative of ischemia in the right dorsolateral part of the medulla supplied by the posterior inferior cerebellar artery (Wallenberg's syndrome). After turning his head, this patient complained of sudden pain in the neck and in the right eye followed by symptoms such as vertigo, gait ataxia, dysarthria, dysphagia, and facial palsy on the right side. A neurologic examination also revealed ipsilateral Horner's syndrome, rotating nystagmus, and impaired ipsilateral facial sensory perception to pain, with impairment of pain sensation on the contralateral (left) half of the body

Table 2. Tabulation of 12 patients with vertebral-basilar insufficiency. Age, sex, main clinical data, and EAEP-abnormalities are indicated

Case	Age	Sex	Neurological defect	EAEP	
				Left ear	Right ear
1	52	M	Drop seizures	Normal	Normal
2	60	M	1–2 paroxysmal symptoms such as vertigo and diplopia	Normal	Normal
3	29	M	Wallenberg's syndrome	Normal	Medullary lesion
4	63	M	Drop seizures, gait ataxia	Medullopontine lesion	Medullopontine lesion
5	60	M	Drop seizures, ophthalmoplegia, vertigo, occipital headache	Medullopontine lesion	Medullopontine lesion
6	57	M	Drop seizures, reduced optokinetic nystagmus, hypacusis on both sides	Pontomesencephalic lesion	Pontomesencephalic lesion
7	58	M	Numbness left trigeminal nerve, left facial palsy	Pontomesencephalic lesion	Medullopontine lesion
8	51	M	Paresis of the right side of the body, hypalgesia 2nd left trigeminal nerve	Pontomesencephalic lesion	Medullopontine lesion
9	63	F	Vertical ophthalmoplegia	Mesencephalic lesion	Mesencephalic lesion
10	38	F	Diplopia, numbness left trigeminal nerve	Mesencephalic lesion	Mesencephalic lesion
11	59	M	Drop seizures	Medullopontine lesion	Poor reproducibility
12	69	M	Generalized cerebral arteriosclerosis, vertebral-basilar symptoms	Poor reproducibility	Poor reproducibility

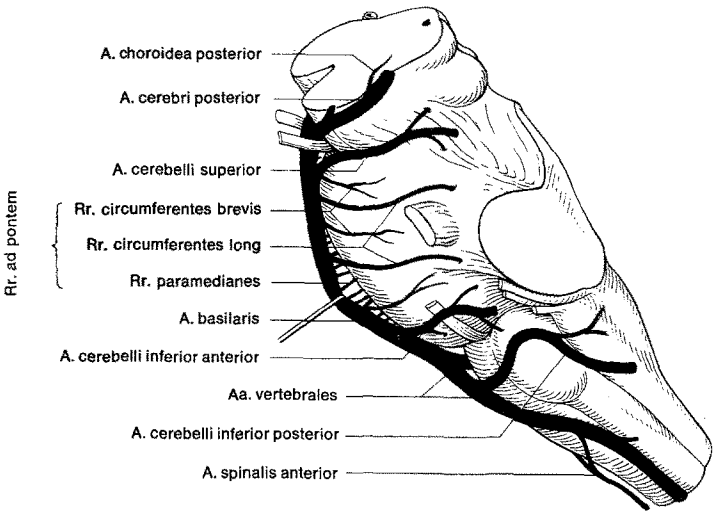

Fig. 2. Vertebral-basilar system, according to Duus (1976)

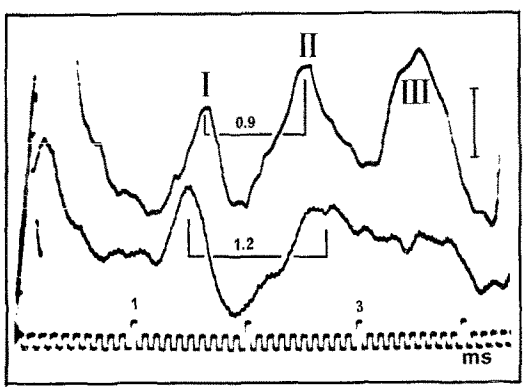

Fig. 3. Records show responses to monaural pips in a patient with Wallenberg's syndrome. Left pip presentation *(upper trace)* contained normal waves I through V. Peripheral conduction time (I—II interwave latency) 0.9 ms. Right stimulation *(lower trace)* yielded a normal acoustic nerve response (I). Wave II was definable but desynchronized; wave III was hard to recognize. Analysis time 4.5 ms, calibration 100 nV. Peripheral conduction time 1.2 ms

below the face. Psychiatric exploration showed a depressive syndrome and organic personality change. Neurologic and psychiatric symptoms underwent a nearly complete remission in the further course of the disease. There were no signs of a peripheral hearing loss (normal tone audiogram and speech discrimination test).

Auditory responses to right pip presentation at 80 dBHL contained a normal I at 1.5 ms (Fig. 3). Succeeding waves had reduced amplitudes of poor reproducibility so that peaks could not be accurately defined. By stimulating the left ear, the EAEP were normal. Six months later during a follow-up the formerly abnormal waves became normal, and an identical pattern could be recorded on both sides (Fig. 4). The normalization of wave form was accompanied by neurologic improvement and by a change in the psychopathologic picture. An arteriography showed stenosis of both vertebral arteries.

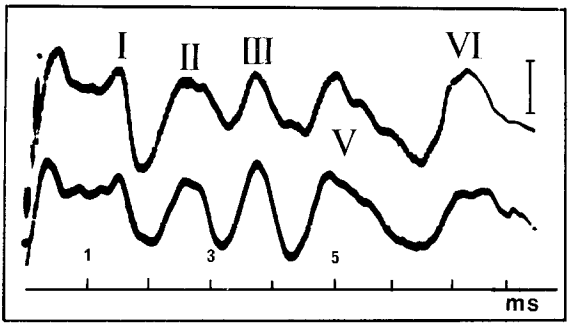

Fig. 4. Responses from the patient in Fig. 3 obtained during a follow-up six months later. The upper trace is from the left and the lower, from the right ear. A normalization of the formerly altered waves took place. Calibration 200 nV

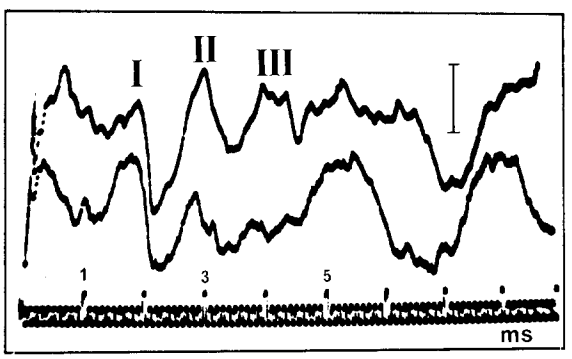

Fig. 5. Responses to left *(upper trace)* and right *(lower trace)* monaural pips. I was delayed on both sides by a conductive hearing loss. Central conduction could still be estimated since waves maintain their interwave correlations. II was normal in amplitude on both sides. There was still a response from the caudal pons (III) by stimulating the left ear. Calibration 200 nV

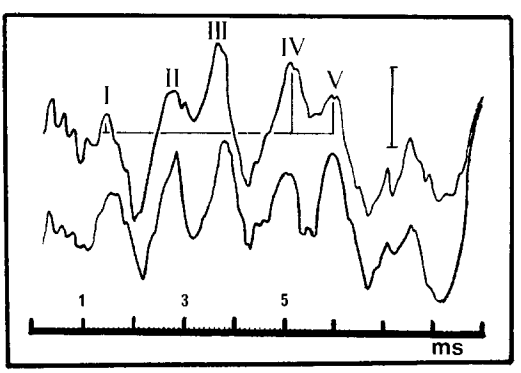

Fig. 6. Responses to left *(upper trace)* and right *(lower trace)* monaural pips. Prolongation of the I—IV and I—V interwave latencies. Normal value for I—IV (3.35 ms ± 0.14) and I—V (4.1 ms ± 0.13). Corresponding values in the patient: I—IV (3.7 ms) and I—V (4.5 ms). Calibration 200 nV

Lesions Affecting Wave III and IV (Table 1, Case 6)

This 57-year-old man had a typical drop attack. During this event a car accident was provoked by extreme vertigo and nystagmus. Neurologic examination showed nystagmus accentuated by horizontal eye movements and reduced vertical optokinetic nystagmus. Weakness and impaired sensory perception were evident on the right side of the upper and lower limbs.

Auditory responses to right and left pips at 80 dBHL contained a late wave I. The delay was caused by a conductive hearing loss on both sides, probably due to

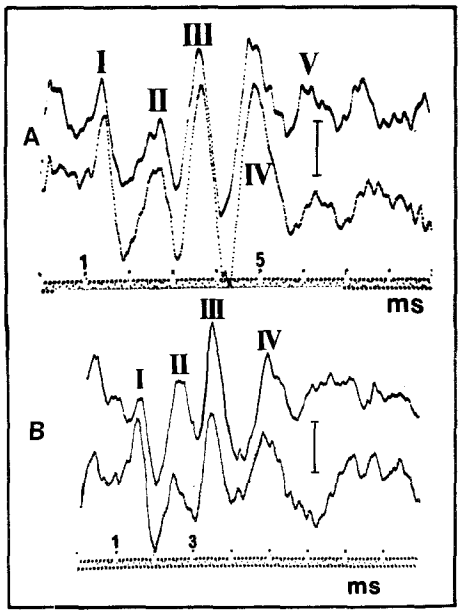

Fig. 7. A. EAEP in response to right pip presentation. Each of the two traces is a separate average of 1500 pips. Waves I through IV are normal and highly reproducible. Calibration 200 nV. B. Potentials obtained by left stimulation. Same recording procedure. There was no recognizable wave V. Calibration 200 nV

otosclerosis. Since the acoustic nerve response, although delayed, was normal in amplitude, an undisturbed cochlear neural output may be assumed, and further brain stem function evaluation was therefore possible. On both sides waves I and II were normal in amplitude (Fig. 5). Peak III could not be adequately defined by right stimulation (lower curve). Left pip presentation (upper curve) contained a response from the caudal pons (III). Waves IV and V were hard to recognize, with poor reproducibility.

A 63-year-old woman (Table 1, case 9) had the typical onset and symptoms of an insufficiency in the vertebral-basilar system. When kneeling down, she complained about sudden loss of vision, dysarthria, difficulties in swallowing, facial palsy, and ophthalmoplegia. All symptoms had a good remission except for vertical eye movement paresis which remained as a symptom of a midbrain malacian defect. In the following course of the disease, an organic personality change and a depressive syndrome were explored by psychiatric examination. The hearing ability was normal.

Auditory brain stem responses to left and right monaural pips of 70 dBHL contained normal waves I through III (Fig. 6). Acoustic stimulation revealed a slightly increased latency of waves IV and V, the only abnormality. The IV—V interwave latency was prolonged.

Lesions Affecting Wave V (Fig. 1, Case 10)

This 38-year-old female noticed the sudden onset of diplopia by turning the eyes to the right side. This symptom persisted over a period of three months. Later she noticed numbness in the left corner of the mouth.

Neurologic examination confirmed this numbness and also revealed a mildly reduced corneal reflex and slightly increased deep reflexes on the right side of the body indicative of a pyramidal tract affection. Symptoms were indicative of a lesion in the pontomesencephalic region of the brain stem. There were no signs of a conductive or sensory-neural hearing loss.

Auditory evoked potentials contained completely normal waves I through IV on the right side. Wave V was definitely delayed and reduced in amplitude. Left pips evoked normal waves I through III; IV was slightly reduced in amplitude compared to the corresponding wave of the opposite auditory pathway. Wave V was hard to recognize (Fig. 7).

Discussion

Results about impairment of circulation in the vertebral-basilar system and corresponding wave abnormalities of EAEP are mentioned in literature. Starr and Hamilton (1976) were the first; they reported the results of two patients on whom autopsia was performed to confirm the distribution of brain stem lesions and their correlation to acoustic response abnormalities. Thornton and Hawkes (1976) described characteristic acoustic abnormalities at the appropriate neurologic brain stem level. In 1977, Chiappa mentioned two patients with 'locked-in' syndrome caused by basilar artery occlusion and two patients with a pontine hemorrhage. All cases showed normal waves originating distally to the suspected lesion. Stockard and Rossiter (1977) saw two patients with lateral pontomedullary junction infarcts, three patients with bilateral infarctions or hemorrhages in the caudal pons, three patients with vascular lesions of the midbrain or rostral pons, and one patient with bilateral temporoparietal infarctions (normal waves I through VII). It was mentioned that most lesions were accurately localized by clinical and radiologic findings and EAEP abnormalities provided a fairly good antemortem localization in only a few cases. In 1977, Gilroy et al. mentioned a patient with 'locked-in' syndrome leaving normal waves I—III and pointing to a rostral pons and midbrain lesion by slowed neural conduction in the part above the superior olivary complexes.

In our control group a 100% stability of components I—V could be guaranteed. Even unstable potentials such as VI and VII appeared in 95% and 55% of the normal cases (Maurer et al., 1979c). This may be due to an optimal recording technique and the use of a tone pip as stimulus. Since there is no evidence at the moment for the generator sites of waves VI and VII, it is difficult to show correlations between abnormalities of these components and sites of auditory pathway involvement. Waves VI and VII seem nevertheless to be of great use for further evaluation of impulse propagation in cases of a midbrain lesion since the proper generation of waves succeeding V is often disturbed.

A series of 12 patients with neurologic and psychiatric symptoms indicating an impairment in vertebral basilar circulation was investigated in this study. It was clear from the review of neurologic and vascular anatomy that a large variety of clinical pictures can be produced. All patients with abnormalities of waves

originating at the medullary, pontine, and/or midbrain level had corresponding clinical signs, and a decision about the level of the area damaged by infarction was possible by regarding the neurologic defect.

An indication of the exact position of the vascular tree involved was possible in a few cases. These conditions have been given eponyms such as Wallenberg's syndrome. One patient (case 3) whose clinical picture was due to an infarction in the distribution area of the right posterior inferior cerebellar artery is therefore worthy of notice. The dorsal segment of the medulla including the vestibular and cochlear nuclei is supplied by this vessel. These patients often complain about hearing disturbances and excessive vertigo. The central genesis (II-abnormality) of auditory pathway invovlement could be confirmed in our patient by a completely normal acoustic nerve response. The undisturbed cochlear function is in accordance with the anatomic fact that the labyrinthine artery which is responsible for the blood supply of the cochlea branches more proximally, i.e., in rare cases, from the inferior anterior cerebellar artery and, in most cases, from the basilar artery itself. Arteriography revealed in this case an advanced stenosis of both vertebral arteries and a normal basilar system. Improvement of neurologic and psychiatric symptoms was accompanied by an amelioration of the formerly pathologic brain stem components.

As mentioned above, the labyrinthine artery branches in the initial part of the basilar system. This may account for the poor reproducibility of auditory responses in two patients (cases 11 and 12). A dysfunction of the highly susceptible cochlear hair cells, combined with a desynchronization of the compound action potential (CAP), may be suspected.

To summarize, there is an extensive mixture of symptoms and signs indicating an overlap in the areas believed to be infarcted with stenosis or occlusion in specific arteries. EAEPs are useful in localizing vascular lesions to peripheral, pontomedullary, pontine, and midbrain levels. Abnormalities of single components are mainly to be expected if the dorsolateral part of the brain stem supplied by long circumferential branches (posterior inferior, anterior inferior, and superior cerebellar arteries) from the vertebral or basilar artery is affected. Infarctions in the ventral medial and in the ventrolateral portion of the brain stem supplied by short paramedian and circumferential branches may not interfere with the auditory tract, and EAEPs may maintain their normal pattern (Fig. 2).

Acoustically evoked waveform abnormalities are similar to those described for tumorous and inflammatory processes (Maurer et al., 1979a, b, c). An aid for differential diagnosis may be the higher percentage of desynchronization of component I, probably due to a reduced blood supply in the distribution area of the labyrinthine artery.

The entity 'vertebral-basilar insufficiency' which comprises various vascular syndromes may be more precisely defined by EAEP. The advantage over other diagnostic procedures such as arteriography resides in the noninvasive character of the method and the possibility of reexaminations as frequently as desired. Prognostic considerations can therefore be drawn by a comparison of the results gained in a series of follow-up examinations.

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