On the Functional State of Central Vestibular Structures in Monoaural Symptomatic Tinnitus Patients (BEAM-VbEP Study)

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Key words

Vestibular Evoked Potentials (VbEP), Brain Electrical Activity Mapping (BEAM), monoaural tinnitus, acoustic neuromas patients.

ABSTRACT

Clinical evidence suggests that overexcitation or disinhibition of structures in the brain occurs in tinnitus patients.[1,2] Brain electrical activity mapping of vestibular evoked potentials (BEAM-VbEP method) provides an electrophysiologic approach of quantification of function in brain cortex. The effect of tinnitus on the BEAM-VbEP image was examined in two groups of acoustic tumor patient Group A (n = 24) reported tinnitus and Group B (n = 22) did not.

Statistically significant differences in the VbEP parameters have been identified between the two groups. The amplitude of the III/IV peak-to-peak component elicited by the rotation to the affected side, is higher ($P < 0.05$) in the tinnitus group than in the non-tinnitus group. Latencies of the late VbEP components (III, IV, and V) are shorter.

In subgroup III, latency was 317.9 ± 37.5 ms in the tinnitus group versus 335.5 ± 30.9 ms in non-tinnitus group ($p < 0.05$); subgroup IV's component of 437.1 ± 35.4 ms versus 470.9 ± 43.5 ms ($p < 0.01$), V 622.1 ± 32.6 versus 655.5 ± 46.6 ms ($p < 0.05$). Amplitude mapping of the most prominent VbEP component, subgroup III, demonstrates a well expressed negativity shift of the evoked brain electrical activity.

The character of the electrophysiologic VbEP changes in the group of tinnitus patients is irritative. We consider the above described BEAM-VbEP images in tinnitus patients to reflect an electrophysiologic correlative of a state of cortical disinhibition, caused by either hyperactive or hypersensitive neural structures.

Tinnitus is an aberrant perception of sound unrelated to an external source of acoustic stimulation; a dysynchrony within the auditory system.[3-5]

Does tinnitus originate from hyperactive nerve fibers in the cochlea or is it a consequence of overexcited or disinhibited brain structures?[6,7] This basic question still has no acceptable solution. One working hypothesis is that tinnitus represents periodic or aperiodic excitation in the spontaneous activity of hair cells or nerve fibers originating from a restricted place on the basilar membrane.[8]

In most clinical cases, the complaint of tinnitus is an accompanying symptom of other central or peripheral disorders (cerebrovascular and circulatory diseases, acoustic neuromas, sudden hearing loss, intoxication, side effects of drugs, among others which frequently cause various degrees of hearing loss with a corresponding *decrease in the spontaneous activity of the auditory nerve.)* [9,10] Thus, one could imagine such cases of tinnitus can be caused by some *abnormal form of spontaneous activity in the central nervous system.*

Such considerations influenced us to evaluate the functional state of the brain in patients in whom tinnitus is the chief complaint. The method used in this study is the BEAM-VbEP method. This approach for vestibular stimulation and recording is suggested by the original concept of Shulman and colleagues,[5,11] that in some cases tinnitus can originate at the site of vestibular dysfunction.

Furthermore, the BEAM-VbEP method provides data restricted not only to vestibular brain centers and pathways, but also to a much broader perspective to sensory pathways and associative cortical areas.

METHOD

Forty-six acoustic neurinoma (AN) patients were examined pre-operatively using the BEAM-VbEP technique. Patients were selected from a group with neuroma. Group A (n = 24) suffered from tinnitus at the time of the test. Group B (n = 22) either did not suffer from tinnitus, or had not for at least 3 months before the BEAM-VbEP examination. The AN tumors were verified surgically. All cases completed nuclear magnetic resonance (NMR), audiometry, and equilibriometric tests. Tinnitus was classified by using the guidelines of a neuro-otological questionnaire NODEC. The test group consisted of 29 women and 17 men aged between 30 and 69 years (mean age 49.8 ± 10.9). The VbEP-parameters of latency and amplitude of groups A and B were compared with the VbEP-parameters recorded in a control group, which consisted of 32 neurologically and neuro-otologically healthy volunteers with a similar age range. The BEAM-VbEP images elicited when stimulating the affected ear (e.g., rotating to the ear suffering from tinnitus, hearing loss) were compared with the corresponding image when rotating to the non-affected ear in the same patient.

Clockwise (CW) rotation (ROTR-stimulus) in a position with the head inclined forward by 30° is more effective in stimulating the right horizontal semicircular canal than the left, because the ampulopetal endolymphatic flow is more effective in producing of neuronal discharge firing of hair cells in the crista ampullae, than the ampulofugal endolymphatic flow.[6]

The stimuli applied for eliciting the VbEP were short-lasting repetitive acceleration steps with an intensity of 53.0°/sec^2 and a duration of 1.0 sec. The stimulus profile takes the form of a semisinusoidal impulse. A total of 25 acceleration steps are applied in either CW and counter-clockwise (CCW) rotations. The inter-stimulus interval is 14 sec. Hence, the duration of the stimulation session is about 3 minutes for each CW/CCW rotation. This kind of stimulation is very well tolerated by the patients and no intra-sessional or post-sessional complaints were reported.

The equipment assembly used in this study included a direct drive servo-controlled ServoMed AB Rotation Chair RS/6. Biosignals were transmitted to the main amplifier via an assembly containing 17 slip rings, each with twin sliding contacts. The subject was positioned on the chair with his head inclined forward by 30°. To minimize the effects of electrical eye movement, gaze fixation was introduced through a structured fixation target, fixed at the subject's knee and rotating with him in the chair. Recording electrodes were positioned in accordance to the international 10/20 system, using Medi-CAP electroencephalograph (Picker Int., GmbH) with 19 electrodes. A common bimastoidal reference electrode was used in this study. The responses were monitored on-line and subsequently processed on a Schwarzer Brain Surveyor BS 2400, for spatial and chronological analysis of both spontaneous and evoked brain electrical activity measured by BEAM.[6]

RESULTS

Tumor location

The acoustic tumor was located on the left side in 59% (n = 27) of the patients studied and on the right side in 41% (n = 19). Intrameatal location of the tumor is diagnosed in 53% (n = 24) of the patients and intra- and extrameatal location in another 47% (n = 22). In 20% (n = 10) of the patients, a history of acute or chronic attacks of sudden hearing loss at the same ear was reported. No neurologic sign of brainstem pressure was evidenced in either group.

Analysis of the VbEP-properties

The VbEP patterns in the two patient groups, as well as in controls, recorded a wave complex of 6 to 7 negative/positive deflections within the time interval between 65.0 and 800 ms after onset of the vestibular acceleration stimulus.[11-14] In the present study, statistical analysis was performed of the latency/amplitude properties of the VbEP, obtained from the C3 (left central) and C4 (right central) electrode derivations. Amplitude of the VbEP was analyzed with respect to the voltage of the III/IV component, measured peak-to-peak.

The VbEP-parameters were modified in all AN patients (tinnitus and non-tinnitus

	Ipsilateral Hemisphere			Contralateral Hemisphere	
	Rot/ipsilat.	Rot/contralat.		Rot/ipsilat.	Rot/contralat.
Component	Lat/ms	Lat/ms	t-test	Lat/ms	Lat/ms
I st	80.3 ± 28.3	69.7 ± 20.7	*P < .05*	82.8 ± 28.1	74.4 ± 22.2
II nd	192.5 ± 41.8	181.4 ± 30.2	*P < .05*	186.4 ± 34.9	182.2 ± 33.5
III rd	328.6 ± 34.7	324.2 ± 26.5		313.9 ± 26.3	324.2 ± 33.5
IV th	457.8 ± 43.8	456.4 ± 46.7		475.6 ± 42.4	458.9 ± 40.9
V th	646.4 ± 43.3	656.9 ± 54.5		656.1 ± 50.9	658.1 ± 54.0
VI th	765.6 ± 44.8	771.9 ± 62.3		778.1 ± 52.9	769.4 ± 57.2

Table I
Vestibular evoked potential component latencies in acoustic neuroma patients (n = 46; t-test comparison between latency values elicited when rotating to the side of affected ear versus rotating to the side of non-affected ear).

groups A and B). In AN patients mean VbEP-latencies were significantly longer than corresponding latencies in the normals. Direction-dependent differences were recorded when the rotation was directed to the affected ear, that is, the latencies were increased in relation to the affected ear than when the rotation was directed to the nonaffected ear (Table I, Figure 1). These differences were highly significant for the first ($p < 0.05$) and ($p < 0.005$) VbEP components. This finding was valid for both hemispheres. They are however, more pronounced in the hemisphere ipsilateral to the tumor.

	Ipsilateral Rotation		
Parameter	Tinnitus	t-test	No tinnitus
III/IV amplit.	21.1 ± 11.8	*P < 0.05*	18.8 ± 12.1
Ist. comp.lat.	82.1 ± 16.4	*	79.1 ± 23.7
IInd comp.lat.	187.1 ± 36.4	*	195.9 ± 44.6
IIIrd comp.lat.	317.9 ± 37.5	*P < 0.05*	335.5 ± 30.9
IVth comp.lat.	437.1 ± 35.4	*P < 0.01*	470.9 ± 43.5
Vth comp.lat.	622.1 ± 32.6	*P < 0.05*	655.5 ± 46.6
VIth comp.lat.	754.3 ± 42.3	*	772.7 ± 49.8

Table II
The VbEP component latencies and third and fourth peak-to-peak amplitude in the two subgroups of acoustic neuromas patients: group "A" (suffering from tinnitus) and group "B' (no tinnitus complaint). Derivation: C/ipsilateral to the affected ear; rotation towards the side of the affected ear; t-test comparison.

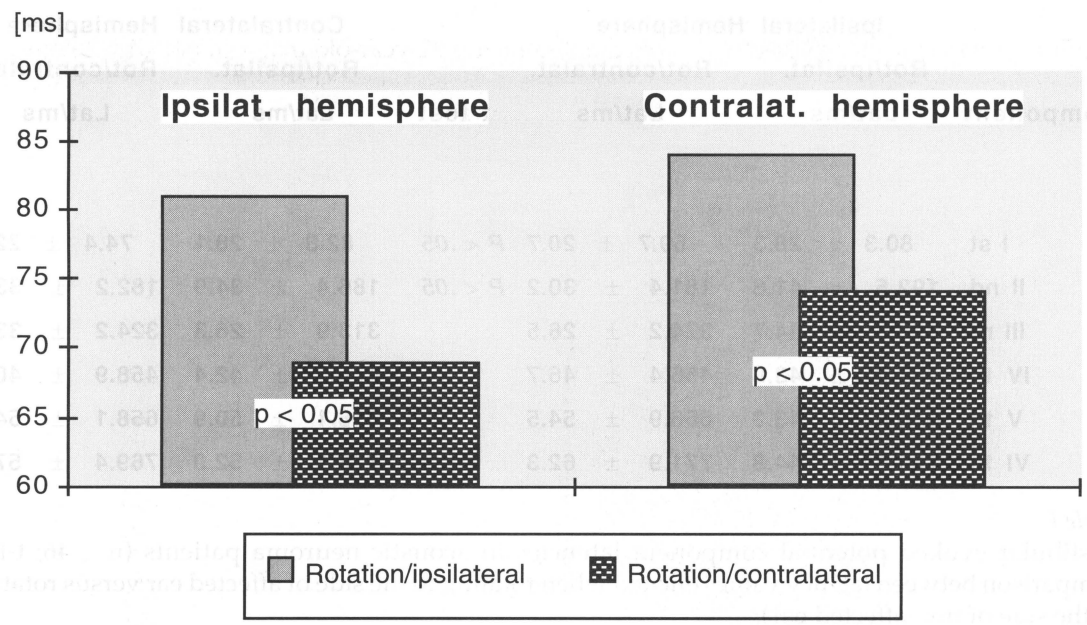


Figure 1
Latency differences of the first component of the vestibular evoked potential in acoustic neuroma patients when rotating to the side of the tumor versus rotation to the contralateral side. Rotation to the tumor side (Rot/ipsil) elicits longer latencies over both hemispheres (C3 and C4-derivations) ($p < 0.05$, $n = 46$).

In AN patients, the VbEP-amplitude (peak-to-peak amplitude of the third and fourth components) is lower than in the normals. Thus, the amplitude elicited when the rotation is directed to the affected ear (18.3 μV) is lower than that obtained when the rotation is directed to the nonaffected ear (21.0 μV) ($p < 0.05$).

In patients with acoustic tumors, differences have been identified. VbEP-amplitude in the tinnitus group (Group A) is higher than that in the non-tinnitus group (Table II). Mapping of VbEP-components in the tinnitus group reveals a well-outlined shift of the BEAM-VbEP image towards the negative electrical pole, when compared with the corresponding BEAM-VbEP image in the non-tinnitus group. On the BEAM display this appears as a high intensity red map, in particular when mapping the most prominent third VbEP-component (Figures 2 and 3).

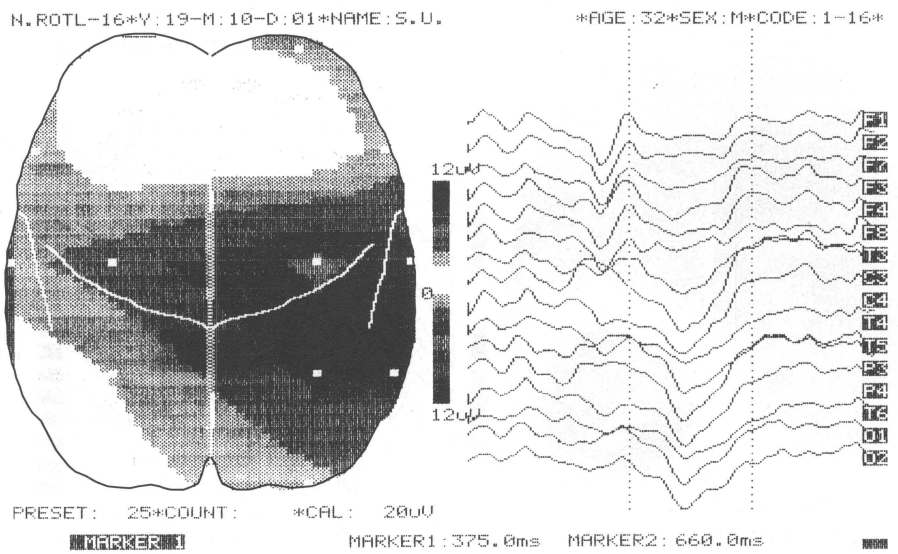
Furthermore, latencies of the later VbEP-components (i.e., 3rd, 4th, and 5th) are significantly shorter in the tinnitus group (Table II).

DISCUSSION

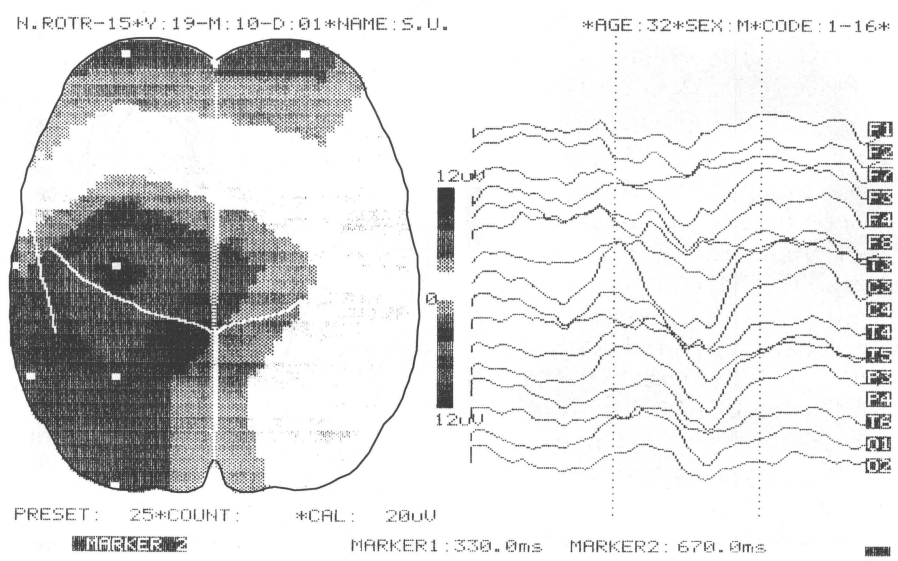
The underlying pathology in both patient groups is the same, that is, a well-defined lesion of the stato-acoustic nerve. The lesion itself caused changes of the BEAM-VbEP image, which were analyzed in the tinnitus (Group A) and non-tinnitus group B. It is hypothesized that modifications of the VbEP-parameters in the tinnitus group when compared with the corresponding parameters of the non-tinnitus group reflect some basic mechanisms of the tinnitus phenomenon.

The character of the electrographic VbEP-changes in the group of patients with tinnitus is principally irritative or excitatory. In this study, we have used a common mastoid as a referential electrode. Consequently, potential differences between the reference point (which is practically *silent* electrically and each one of the scalp electrodes (which is electronegative with respect to the reference point) reflect the gradient of the relative cortical electronegativity during the vestibular stimulation.

Figure 2 A, B
Amplitude mapping of the vestibular evoked potential in a 32-year-old male patient with an acoustic neuroma, located intra- and extrameatal at his left ear. History of discrete progressive hearing loss at the same ear. *No tinnitus at the moment of the investigation.*

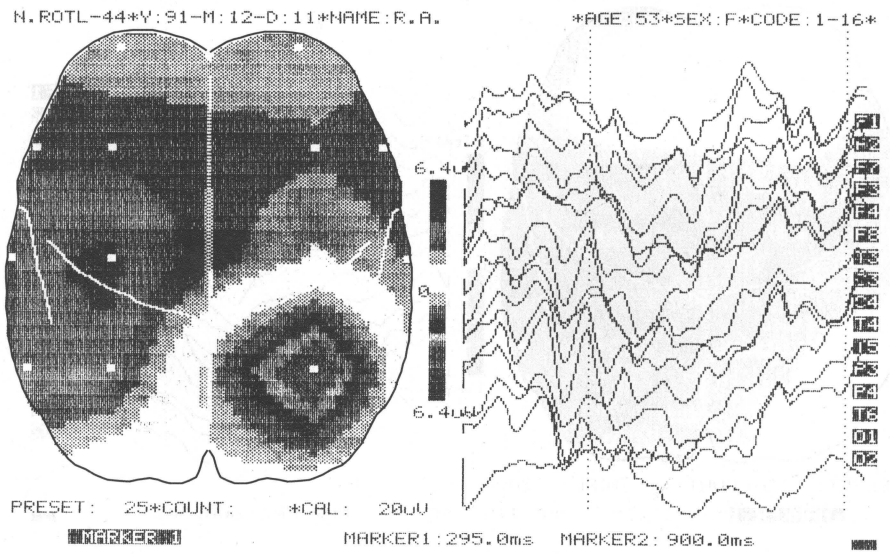


A, Brain electrical map of the vestibular evoked potential (cursor on the third VbEP component) when rotating to the affected side (rotation to the left, ROTL). Delayed latency and lower amplitude of the VbEP can be seen in comparison with the corresponding parameters elicited when rotating to the non-affected ear. Brain mapping of the potential demonstrates a relative deactivation of the cortex during this rotation.

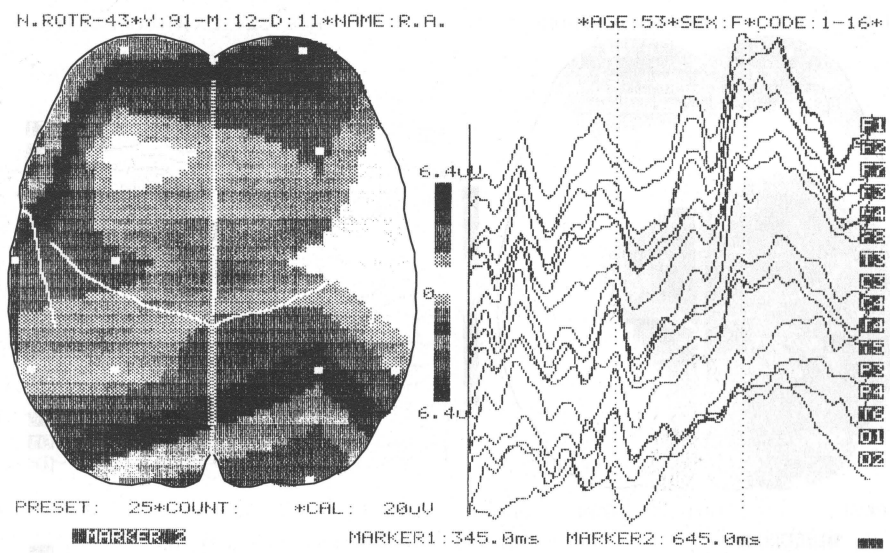


B, Brain electrical map of the same VbEP component when rotating to the side of the non-affected ear. Normal parameters of the VbEP and of the brain map.

Figure 3A, B
Amplitude mapping of the vestibular evoked potential in a 53-year-old female patient with an acoustic neuroma at the left ear (intra- and extrameatal location). The patient suffered from permanent tinnitus at this ear for about 7 months, and episodic vertigo attacks. Audiometric examination revealed a moderate degree of high frequency hearing loss.



A, Brain electrical map of the vestibular evoked potential elicited when rotating to the side of the affected ear (rotation to the left, ROTL). In this patient, a relative negative shift of the brain isoelectric contours can be seen (particularly over the left temporal area) in comparison with the rotation to the non-affected ear. The third component latency is also significantly shorter at the affected side.



B, Beam-VbEP image when rotating to the non-affected ear.

Electronegativity is a fundamental neurophysiologic category. If an afferent influx of impulses occurs in vertically-oriented cortical neurons at a higher frequency for a longer period, then wave-like excitatory post-synaptic potentials (EPSPs) will generated at the superficial dendritic arborization. This produces a broadly extended area of depolarization of the cortical neuronal elements. Prolonged depolarization of the superficial structures caused by sensory afferentiation may lead to a negative shift in the EEG, that is, a negative DC/EEG potential shift.[5]

Discovery of the presence of spontaneous cochlear acoustic emissions has led to the speculation that it may be responsible for some form of a cochlear type of tinnitus. Attempts in some studies, however, to show a relationship between the measured spontaneous acoustical emissions in the external meatus and the subjective pitch matching of tonal tinnitus have not been very successful.[7] Using the simultaneous auditory brainstem response (SABR) method in tinnitus patients[15] suggests the role of either peripheral and central auditory structures for the appearance of the symptom of tinnitus. Auditory brainstem response (ABR) parameters have also been used in tinnitus identifications. In particular, an increase in amplitude of the P3 evoked with ipsilateral stimulation is consistent with the side of the tinnitus.[15]

This higher level of spontaneous activity in the central nervous system, however, seems to be an abnormal form of increased spontaneous activity.[1,9] This could be caused either by hyperactive neurons or by hypersensitive (low-threshold) neural structures. Both hyperactivity or hypersensitivity can be caused by a loss of inhibition.[1] The phenomenon of central disinhibition may become manifest as a very early sign (functional or subclinical phase) of a progressive brain disorder, with many possible causes, such as circulatory, vascular, immunologic, inflammatory, or traumatic. Such a loss of inhibition in auditory neural structures may result in distortion of basic audiologic processes of transduction, coding, transformation, and perception.

The study's data, based on the BEAM-VbEP approach, revealed a modification of the functional state in broad cortical areas in patients suffering from tinnitus. The majority of the patients had a mild to moderate sensorineural hearing loss for high and middle frequencies. According to our experience with BEAM-VbEP, no essential effect of HL on the VbEP-parameters exists. Furthermore, most of the AN patients have no subjective vestibular complaints. This is the basis of our speculation that differences in the BEAM-VbEP images between both groups of patients reflect some central mechanisms related to the tinnitus complaint.

CONCLUSION

The tinnitus symptom in acoustic tumor patients in response to a rotary stimulus, reflected in the BEAM-VbEP image is characterized by an ipsilateral reduction of the typical VbEP component latencies; increased amplitude and a negative shift of the rotationally evoked cortical electrical activity. These findings suggest that in tinnitus patients a state of central disinhibition over broad cortical areas exists.

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