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Effect of noise on the vestibular system - Vestibular evoked potential studies in rats

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Abstract

Studies have shown that in order for sound to affect the vestibular end organs in the inner ear, very high intensities are required. Furthermore, in patients with noise induced hearing loss, vestibular signs, if present, are subclinical. In order to study possible auditory-vestibular interactions in a more controlled fashion, using physiological sound intensities, the present study used short latency vestibular evoked potentials (VsEPs) to impulses of angular (15,000°/sec ^{2} , risetime 1.5 msec) and linear (3-5 g, rise time 1.5 msec) acceleration were used to study the possible effects of sound on peripheral vestibular function in rats. Four different paradigms were used: a - an intense (135 dB pe SPL) click stimulus was presented 5 msec before the linear acceleration impulse and the VsEP to 128 stimuli were recorded with and without this click stimulus. There was no effect of the preceding intense click on the first wave (reflecting end organ activity) of the linear VsEP. b - 113 dB SPL white noise "masking" was presented while the VsEPs were elicited. A 10-20% reduction in the amplitude of the first VsEP wave was seen during the noise exposure, but 5 minutes after this exposure, there was almost complete recovery to pre-exposure amplitude. c - 113 dB SPL noise was presented for one hour and VsEPs were recorded within 15 minutes of cessation of the noise. The auditory nerve-brainstem-evoked response showed a temporary threshold shift while there was no effect on the VsEP. d - 113 dB SPL white noise was presented for 12 hours per day for 21 consecutive days. Auditory nerve-brainstem-evoked responses and vestibular (VsEPs) function were studied one week after the conclusion of the noise exposure. Auditory function was severely permanently depressed (40 dB threshold elevation and clear histological damage) while the amplitude of wave 1 of the VsEP was not affected. It seems therefore that even though intense noise clearly affects the cochlea and may have a "masking" effect on the vestibular end organs, the intensities used in this study (113 dB SPL) are not able to produce a long-term noise induced vestibular disorder in the initially normal ear. These differences between the response of the cochlear and vestibular end organs to noise may be due to dissimilarities in their acoustic impedances and/or their electrical resting potential.

Keywords: Vestibular, Evoked potentials, Noise, Otolith organs, Semi-circular canals

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Introduction

All the sensory end organs in the inner ear, both auditory (cochlea) and vestibular (three semicircular canals and

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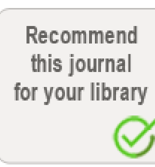

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two otolith organs) contain perilymph outer channels which are in communication, and separate endolymph inner channels which are in communication, through all parts of the inner ear. Thus hydrostatic pressure alterations induced in any way (e.g. sound) in one part of these fluid channels should spread to the other parts, since the fluids are incompressible. Bekesy (1960) has provided evidence that the initiation (and cessation) of an intense tone sets up a streaming of fluid not only in the cochlea but also in the canals.

Although in mammals, the cochlea is the hearing end organ, the semicircular canals (SCCs) are sensitive to angular accelerations and the otolith organs (saccule and utricle) are excited by linear accelerations, in more primitive vertebrates (ray, fish and amphibians) the saccule is the hearing organ. Based on this fact and on the intercommunications between the fluid spaces in the cochlea, in the vestibule and in the SCCs, the possibility that sound stimuli can activate the vestibular end organs has been studied, using several approaches.

Tullio Phenomenon

Intense sound stimuli have been found to induce reflex eye movements in guinea pigs (130-160 dB SPL) and monkeys (120-172 dB SPL) (Parker et al. 1976; 1978) and visual field displacements in humans (125 dB SPL) (Parker et al., 1978). This phenomenon of acoustical activation of vestibular responses has been called the Tullio phenomenon.

Patients with noise induced hearing loss (NIHL) show some signs of vestibular pathology in one or more tests (spontaneous, positional and/or cervical nystagmus) (Oosterveld et al., 1982), significantly more body sway (Ylikoski et al., 1988) and reduced gain of the vestibular ocular reflex (Shupak et al., 1994). These clinical findings have been taken as evidence that intense sound stimuli can induce vestibular disturbances and as confirming the Tullio phenomenon.

In addition, it has been shown that even though amikacin administration in guinea pigs causes total and selective destruction of the cochlea, an evoked potential can be recorded in response to click stimuli which are 60-80 dB greater in intensity than the thresholds of auditory evoked potential in normal animals. The latency of this "auditory" evoked response is shorter than the usual auditory evoked potential in the normal animal (Cazals et al., 1983; Didier and Cazals, 1989). Evidence based on histology (Cazals et al., 1983) and local electrophysiological recordings (Didier and Cazals, 1989) have lead to the conclusion that this "auditory" evoked response of vestibular origin is initiated in the saccule.

Noise Induced Vestibular Histological Damage

Very intense impulse and short duration sound stimuli in animals have also been shown to cause histological damage to the vestibular end organs. Noise at intensities of 136 and 150 dB SPL for 20 minutes was found to have a severe effect on the saccule of guinea pigs, with other vestibular structures remaining normal (McCabe and Lawrence, 1958). Severe damage was also seen in guinea pigs exposed to various single sound frequencies, at 142-163 dB SPL for several minutes. The most common finding seen in the vestibular end organs was severe damage to the saccule and utricle (Mangabeira-Albernaz et al., 1959). With rifle shot impulse noise at 158 dB SPL, all vestibular end organs were damaged, but mostly the ampullary cristae. Some damage was also seen in the utricular and saccular maculae (Ylikoski, 1987).

Vestibular Initiated Myogenic Responses to Click Stimuli

Further evidence that the vestibular system can be excited by sound stimuli comes from studies in which an evoked myogenic response to intense click stimuli could be recorded from over the sternocleidomastoid muscle. This click evoked myogenic potential was lost following selective vestibular nerve section (Cole batch and Halmagyi, 1992). Furthermore a similar response could be recorded in patients with severe sensorineural hearing loss (Colebatch et al., 1994). These findings have been taken as evidence of saccule activation by the click stimuli leading to sacculo-collic reflex activation of the sterno-cleido-mastoid muscle. The findings of Ferber-Viart et al (1998) show that such a myogenic response elicited from the trapezius muscle is initiated in the cochlea as well as in the vestibular end organs. These results therefore contradict those of Colebatch and Halmagyi (1992) and Colebatch et al (1994).

Vestibular Single Unit Studies

Most conclusive evidence that the vestibular end organs can be excited by sound stimuli is based on recordings from single, confirmed vestibular neurons. In monkeys, single units from all 5 vestibular end organs responded to both vibrations and sounds, with lowest thresholds (100-120 dB SPL) in units from the saccule. Also almost all saccule neurons responded to sound while less than half of the semicircular canal afferents responded to sound (Young et al., 1977). In pigeons, single units innervating the horizontal semicircular canal were excited by sound (Wit et al., 1984). In cat, most single units from the saccule responded to sound stimuli with thresholds of 80-90 dB SPL (McCue and Guinan, 1994; 1997). In guinea pigs, short latency responses could be recorded in about 25% of vestibular neurons to click stimuli with thresholds 60-90 dB above the threshold of the ABR in the same animals. Based on their responses to various types of vestibular stimuli and on histological tracing of labeled neurons, it was concluded that the click-responsive vestibular neurons originated in the saccule (Murofushi et al., 1995; Murofushi and Curthoys, 1997).

Thus, it seems that in order for sound to activate the vestibular system, high intensities are required. Furthermore, the vestibular signs seen in patients with noise-induced hearing loss are generally subclinical, requiring more sophisticated clinical tests (e.g., harmonic acceleration) to demonstrate them.

In order to study possible auditory-vestibular interactions in a more controlled way, advantage was taken of the newly-developed ability to record short latency vestibular evoked potentials (VsEPs) to impulses of angular (Elidan et al, 1982) and linear acceleration (Plotnik et al, 1997) in experimental animals. The first wave of the VsEP response is the compound action potential of the activated vestibular neurons. The possible effects of

sound-noise on these VsEPs was studied in four different experimental paradigms: a-click-acceleration impulse pairing - 5 msec apart; b - inducing a temporary threshold shift (TTS) and then eliciting VsEPs; c - inducing a permanent threshold shift (PTS) and then eliciting VsEPs; d - noise "masking" during the vestibular activation.

Methods

These experiments were conducted on young adult (3 months old; about 300 grams), albino, Sabra rats. Rats were chosen for these experiments because the linear acceleration device used to elicit VsEPs is designed for these animals. Furthermore, several rats could be exposed to noise together, leading to better control of the experimental conditions. The care and use of the animals was in accordance with the guidelines published by the Hebrew University - Hadassah Medical School Animal Care and Use Committee. The VsEP recordings were made following anesthesia induced by I.P. injections of 60 mg/kg sodium pentobarbital. Additional doses were given I.P. as required. Body temperature was measured with a rectal thermistor and maintained at 37 ± 0.5°C by a heating pad. The impulses of angular acceleration were elicited with a device based on a stepper motor, a cylindrical drum and a headholder which gripped the rat's head in the plane of the horizontal SCC. The device repeatedly delivered exactly identical impulses of angular acceleration at a rate of 2/sec, an acceleration of 10,000°/sec ^{2} , a risetime of 1.5 msec, maximum displacement 1.8°. A detailed description is given in Elidan et al (1982) and Li et al (1993).

The impulses of linear acceleration were elicited with a solenoid, a sliding device and a headholder. It repeatedly delivered identical impulses of linear acceleration at a rate of 2/sec, an acceleration of 3-6 g, a rise time of 1.5 msec and a displacement of 50 µm. The coupling between the solenoid and the sliding device introduced a short mechanical time delay between the beginning of the recording trace and the actual onset of the acceleration impulse. A more detailed description is given in Plotnik et al (1997). The rats could be stimulated in the optimal plane of the utricle or of the saccule. Acceleration waveform and magnitude were measured with an accelerometer (Bruel & Kjaer 4393) mounted on the sliding device.

Electrical activity was recorded as the potential difference between a vertex needle electrode and a needle electrode in one of the pinnae (usually right ear). The ground needle electrode was in the pinna of the other ear. The recorded activity was digitally filtered (300-1,500 Hz bandpass), amplified and 128 responses were averaged - positivity at the scalp electrode produced an upward deflection.

These VsEPs were elicited during or following the presentation of sound-noise in four different experimental paradigms which are summarized in [Table - 1].

Experiment 1 - Click Stimulus - Acceleration Impulse Stimulus Pairing

Advantage was taken of the time delay purposely introduced between the electrical pulse which activates the solenoid and the actual mechanical acceleration impulse delivered to the head of the animal (see Plotnik et al, 1997). An alternating polarity click stimulus was presented during this delay, so that the click stimulus preceded the acceleration impulse stimulus by about 5 msec. This is similar to studies of the effect on the auditory nerve-brainstem-evoked response (ABR) in response to a second click delivered several milliseconds after a preceding click (paired click adaptation experiments) (Burkard and Deegan, 1984). The amplitude and latency of the first wave of the VsEP (compound action potential of the vestibular nerve fibers synchronously activated by the stimulus) elicited in the absence of a preceding click was compared to that in the presence of the preceding click.

Peak to peak amplitude was measured between the first vertex positive peak and the following vertex negative trough. Latency of this wave was measured from the onset of the acceleration impulse to the first vertex positive peak.

These experiments were conducted on 10 anesthetized rats using 120 dB pe SPL clicks, with the head of the rat in the orientation for optimal activation of the utricle. In 10 other rats, 135 dB pe SPL click were used, with the head held in the saccule orientation.

Experiment 2 - VsEP During a Temporary Threshold Shift (TTS)

After recording VsEP a day earlier in nine anesthetized rats, they were allowed to wake up and then they were exposed to broad band noise, 113 dB SPL, for one hour. This level of broad band noise was chosen because it is intense while still being in the physiological range. Thus the results would be more relevant to possible human exposure conditions. The VsEP in response to 3 g stimuli in the plane of the saccule was again elicited in the anesthetized rats within 15 minutes after termination of the noise, one hour, two hours, six hours and 24 hours later.

The amplitude and latency of the first wave, recorded at the various time intervals after termination of the noise, were compared to those recorded before the noise exposure.

In a second group of five rats, the same noise exposure was used but the ABR was recorded (threshold) at the same time intervals to confirm that a TTS was obtained and of its magnitude.

Experiment 3 - VsEP in the Presence of a Permanent Threshold Shift (PTS)

A permanent threshold shift was induced in 10 adult rats by exposing them to 113 dB SPL broad band noise for 12 hours per day for 21 consecutive days. One week after termination of the exposure, the rats were anesthetized and the VsEP to 3 g and 6 g linear acceleration stimuli in the plane of the utricle was recorded. The amplitude and latency of the first VsEP wave were compared to those in control, non-exposed, litter mates (N =10). The ABR was also recorded in the same animals in order to obtain an estimate of the degree of PTS. Following these

recordings, several of the ears (experimental and control) were histologically processed, by perfusion between the oval and round windows with glutaraldehyde. The status of the inner and outer hair cells was assessed along the entire cochlear duct, by scanning electron microscope surface preparations. The vestibular end organs were also examined.

Experiment 4 - Noise-Masking of Vestibular end organ response

The VsEP was recorded in anesthetized rats in response to 15,000°/sec^2 angular acceleration stimuli, to 3 g linear acceleration stimuli in the plane of the utricle and to 3 g stimuli in the saccular orientation. This VsEP recording was then repeated in the presence of 113 dB SPL broad band noise (masking) delivered during the approximately two minute period required to elicit two repetitive VsEP responses (128 stimuli each, 2/sec). The VsEP was again recorded several times at various intervals (five and fifteen minutes) after termination of the noise exposure to determine recovery. The effect of this same noise on the ABR was also studied. The amplitude of the first wave of the VsEP recorded in each rat during and at several time intervals (minutes) after the noise was statistically compared to that recorded just before the noise.

Results

The experimental findings in each of the four paradigms are summarized in [Table - 2].

Experiment 1- Click and Acceleration Impulse-Pairing

The amplitude of the first wave of the VsEP, when the impulse of linear acceleration (3 g) in the plane of the saccule was presented alone, was 3.73 ± 1.32 µV (N = 10 rats). When a 135 dB pe SPL click was presented 5.64 ± 0.89 msec before the acceleration impulse in the same rats, the amplitude was 3.65 ± 1.31 µV. The difference was not significant (P=0.45; paired t-test). Similarly the latency of wave 1 of the VsEP showed a mean (± SD) latency increase of 0.02 ± 0.1 msec when the acceleration was preceded by the click (P= 0.35; not significant).

In a similar experiment using acceleration impulses in the plane of the utricle and 120 dB pe SPL clicks, changes in the amplitude and latency of the first wave of the VsEP were also not significant.

Experiment 2 - Temporary Threshold Shift and VsEP

Within 15 minutes following the exposure of the rats to broad-band noise of 113 dB SPL for one hour, the mean (± SEM) amplitude of wave 1 of the VsEP (3 g in the plane of the saccule) was found to be slightly (3.51 ± 0.22 µV) depressed (not significantly - ANOVA; P=0.42) compared to the pre-exposure amplitudes (4.30 ± 0.36 µV) within 15 minutes after cessation of the noise. The latency of wave 1 was slightly prolonged (not significant; P=0.44) (from 1.32 ± 0.03 msec pre-exposure to 1.49 ± 0.71 msec 15 minutes post-exposure).

With the progression of time after the exposure, the amplitude and latency of wave 1 returned to the pre-exposure values. Overall (ANOVA) these changes were not significant.

On the other hand, in a second group of rats using an identical noise exposure paradigm, there was a 32 dB threshold elevation (significant) of the ABR recorded during this 15-minute period, an 18 dB elevation six hours later and a 1 dB ABR threshold elevation 24 hours later (not significant).

Experiment 3 - Permanent Threshold Shift

One week following the conclusion of a 21-day, 12-hour-per-day exposure to 113 dB SPL broadband noise, the amplitude of the first wave of the linear VsEP to 6 g stimuli delivered in the plane of the utricle was 4.68 ± 1.41 µV while in a control group of non-exposed litter mates the amplitude was 4.43 ±1.75 µV (not significant; P=0.74). There was also no difference (P=0.61) in the latency of this wave between the exposed (1.97 ± 0.26 msec) and non-exposed (1.91 ± 0.27 msec) animals. Similarly there was no effect on the first VsEP wave (P=0.18 for amplitude and P=0.70 for latency) elicited in response to 3 g acceleration. An example of the VsEPs in a typical experimental and control rat is shown in [Figure - 1]. On the other hand, the ABR threshold in the exposed animals was 86 ± 4.9 dB pe SPL while in non-exposed controls the threshold was better than 60 dB pe SPL.

The cochleograms of the normal (2) ears showed a full complement of inner and outer hair cells. On other hand the noise exposed ears analyzed histologically (2) had loss of both inner and outer hair cells along the middle regions (from 30% of the distance from the base to 80% from the base) of the cochlea. The hair cells at the basal 20% of the cochlea were intact. There were no signs of damage in the vestibular end organs.

Experiment 4 - Noise-Masking of the VsEP

Noise delivered during the period required to elicit a VsEP response had a statistically significant (P=0.003) depressant (12-25%) effect on the first wave of the VsEP. This noise intensity completely masked the ABR. Five minutes later (the noise was no longer being presented), there was already partial recovery of the VsEP and this recovery increased with the further passage of time.

Discussion

In this project, possible effects of sound on the vestibular system were studied using four different experimental paradigms and the effects were evaluated by recording the short latency VsEPs to impulses of either angular or linear acceleration. The angular acceleration VsEPs have been shown to be initiated in the SCCs (Li et al, 1993;

1995) and the linear VsEPs in the otolith organs (Plotnik et al, 1998). The first wave of the VsEPs is the compound action potential of the vestibular neurons activated by the stimulus and would therefore reflect end organ activity (Li et al, 1995; Plotnik et al, 1997).

The only paradigm in which sound-noise was found to affect these VsEPs was that in which noise-masking was presented simultaneously with the elicitation of the VsEP. At that time, the amplitude of the first wave was depressed by 1225%, depending on whether the VsEP was in response to utricular or saccular orientation linear accelerations or horizontal canal angular acceleration (see Freeman et al, 1999 for details). The ABR to 120 dB pe SPL click stimuli was totally depressed by this masking stimulus. However, within five minutes after the end of this noise, the VsEP had already partially recovered. Thus, even though the noise was very effective in masking the ABR, it had only a temporary effect on the VsEP.

In the other paradigms, though the sound-noise stimuli could be shown to have an effect on the cochlea (ABR), no such influence could be demonstrated with respect to the vestibule and the SCCs.

For example, in the click-acceleration impulse pairing experiment (5 msec separation), no effect of the preceding click was apparent in the VsEP elicited 5 msec later. On the other hand, it has been shown that the pairing of two sound stimuli (clicks or tone bursts) presented 5 msec apart, would produce a 37-40% depression of the response to the second stimulus (Sorensen, 1959; Eggermont and Odenthal, 1974; Eggermont and Spoor, 1973; Burkard and Deegan, 1984).

In the TTS experiment, the ABR recorded within 15 minutes after the end of the noise exposure was depressed while no effect was apparent on the VsEP.

Finally, in the PTS paradigm, the ABR threshold was severely elevated and histological damage was apparent in the cochlea. Nevertheless, the VsEPs were not affected.

Thus, overall, even though the sound-noise stimuli clearly affected cochlear function, no such long-term depression could be seen in vestibular function (except for the short-term masking effect).

This preferential effect on the cochlea may be due to several factors. For example, sound stimuli (pressure) entering the inner ear by the oval window would be preferentially shunted to the cochlea due to the presence of the round window. In the absence of an analogous window in the vestibular end organs, the cochlea would have a lower effective acoustic impedance than the vestibule, with a lower effective sound pressure in the vestibule (Wit et al, 1981). However, the introduction of a fistula in one of the SCCs would be able to serve as an "equivalent" round window, allowing sound stimuli to activate the vestibular end organs. In fact, it has been shown that vestibular neurons in pigeons can be made more sensitive to sound by the introduction of such a fenestration (Wit et al, 1981). In addition, single units of the vestibular nerve of deaf mice can be brought to respond to sound following fenestration (Mikaelian, 1964).

In fact, attempts have been made to take advantage of this possibility to activate vestibular end organs by sound stimuli in order to try to provide some hearing capability to profoundly deaf patients. Initially, some types of evoked potentials to sound (bone conduction) stimuli were recorded in profoundly deaf patients, who had normal vestibular function (Ribaric et al., 1984; 1985). Then based on the studies of Wit et al. (1981) who showed that fenestration of a semi-circular canal in pigeons led to increased magnitude of an auditory evoked potential (after surgical removal of the ductus cochlearis), Ribaric et al. (1992a&b) introduced a fenestration in the horizontal semicircular canal in profoundly deaf patients with normal vestibular function. These patients then reported improved perception of bone conducted sounds.

An additional explanation for the greater effect of sound on the cochlea than on the vestibular end organs of the inner ear may be related to the findings that intense sound can produce a reduction in blood flow in the stria vascularis of the cochlea (Goldwyn and Quirk, 1997; Yamane et al, 1995; Quirk et al, 1992; Yamane et al, 1991; Shaddock et al, 1985) and to a reduction in the magnitude of the endocochlear potential (Syka et al, 1981; Vassout, 1984; Wang et al, 1990; Poje et al, 1995; Ide and Morimitsu, 1990). This is thought to be one of the possible mechanisms whereby intense sound leads to a hearing loss. The reduction in strial blood flow would produce a depression of the endocochlear potential, leading to a compromised cochlear transduction mechanism and hearing loss. However, the vestibular end organs do not have a strial-like structure and the potential of the endolymphatic regions of the vestibular end organs is only -1 to +4 mV (Eldredge et al, 1961). Therefore, these vestibular structures would be less affected by sound. This is similar to the suggested explanation for the absence of effect of furosemide, an inhibitor of the strial NaK-2Cl cotransporter, on the VsEPs, while the ABR is severely depressed (Freeman et al, 1998).

In conclusion, the sound intensities used in this study do not produce long-term vestibular disorders in the initially normal ear.

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Figures

[Figure - 1]

Tables

[Table - 1], [Table - 2]

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