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Functional Impairment of the Vestibular End Organ Resulting From Impulse Noise Exposure

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Objectives/Hypothesis: **To assess the effect of exposure to impulse noise, known to cause damage to the cochlea, on the vestibular part of the inner ear using short latency vestibular evoked potentials (VsEPs), which is a direct and objective test for evaluating the function of the vestibular end organs.** ***Study Design:*** **Prospective animal study.** ***Methods:*** **Sand rats (*Psammomys obesus*) underwent baseline measurements of VsEPs in response to linear and angular acceleration stimuli and measurement of the auditory nerve and brainstem evoked response (ABR). The animals were then exposed to 10 gunshots generating impulse noise at an intensity of approximately 160 dB sound pressure level (SPL). Repeat measurements of the evoked potentials were conducted 2 to 4 hours, 1 week, and 6 weeks after the exposure. The amplitude and latency of the first wave of VsEPs in response to linear and angular acceleration stimuli, reflecting the function of the otolith organs and semicircular canals respectively, were compared between baseline and post-exposure measurements, as were ABR thresholds.** ***Results:*** **The amplitude of the first wave of the VsEPs in response to linear acceleration was significantly (P <.001) reduced and the latency significantly (P <.005) prolonged 2 to 4 hours after the exposure in comparison to baseline measurements. The latency prolongation persisted in follow-up measurements, whereas the amplitude showed partial recovery. The first wave of VsEPs in response to angular acceleration was unchanged long-term. ABR thresholds were elevated in the long-term by 60 dB.** ***Conclusion:*** **It seems that impulse noise not only damages the cochlea, but also causes clear functional impairment to the vestibular end organs, mainly the otolith organs.** ***Key Words:*** **Impulse noise, vestibular end organs, vertigo, vestibular evoked potentials, auditory brainstem evoked response.**

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INTRODUCTION

Individuals exposed to intense impulse noise occasionally experience vertigo. Over the years, a number of studies have been directed to the possible association between exposure to noise and vestibular dysfunction.[1–7] A small proportion of these studies focused specifically on the effect of intense impulse noise.[4–7] The results in these reports were contradictory; while some studies concluded that impulse noise damaged the vestibular system,[4,5,7] others concluded that it did not have any effect.[6] The assessment of possible functional vestibular damage in the studies on humans was conducted using indirect clinical tests such as electronystagmography and posturography. In addition, an animal study[7] described varying degrees of histologic damage to vestibular end organs of guinea pigs after exposure to impulse noise. Thus, functional damage to the vestibular end organ resulting from exposure to impulse noise has not yet been confirmed, probably because of the lack of an objective and direct test for assessment of vestibular function.

In recent years, an objective method for directly evaluating vestibular function has been developed: short latency vestibular evoked potentials (VsEPs). These evoked potentials have been recorded in response to angular[8,9] and linear[10–12] acceleration impulses to the head of both laboratory animals[8–13] and human subjects.[14,15] This electrical activity, in the form of 5 to 6 waves, reflects the neural activity of the vestibular pathways similar to the auditory nerve and brainstem evoked response (ABR)[16] reflecting the auditory pathways. It has been shown that the first wave of the VsEPs is the compound action potential of the vestibular nerve fibers synchronously activated by the stimulus[17] and therefore reliably reflects the function of the vestibular end organs.[18] It has also been specifically shown that the first wave of VsEPs in response to impulses of angular acceleration stimuli is initiated in the semicircular canals and that the same wave in response to impulses of linear acceleration stimuli is generated in the otolith organs.[11]

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The purpose of the present study was to assess the effect of exposure to impulse noise, known to cause damage to the cochlea, on the vestibular part of the inner ear using vestibular evoked potentials (VsEPS), which is a direct and objective test for evaluating the function of the vestibular end organs.

METHODS

General Description

The study was conducted on 18 adult sand rats (*Psammomys obesus)* with a mean weight ± standard deviation (SD) of 190 ± 20 g. The sand rat is a rodent species found in the deserts of the Middle East and Northern Africa, and this laboratory has extensive experience in induction and recording of short latency auditory and vestibular evoked potentials in this animal.[11,13,19] For experimentation, the animals were anesthetized using an intraperitoneal injection of 25 mg/kg pentobarbital and additional doses were given intraperitoneally as needed. While under anesthesia, rectal temperature was monitored using a thermistor probe (Yellow Spring Instruments, Yellow Springs, OH) and maintained at 37 ± 0.5°C (using heating pads).

Initially, the animals underwent baseline measurements of all three types of evoked potentials. All 18 animals underwent ABR recordings, 16 of these animals underwent VsEPs in response to linear acceleration stimuli (L-VsEPs), and 10 of them underwent VsEPs in response to angular acceleration stimuli (A-VsEPs). Subsequently, the awake animals in a cage 50 cm from the rifle were exposed to 10 gunshots at an intensity of approximately 160 dB per sound pressure level (SPL). The experimenter was equipped with ear protectors during the exposure. The intensity of the impulse noise was measured using a Bruel & Kjaer, type 2218, precision integrating sound level meter (Naerum, Denmark). It was necessary to extend the range of the sound level meter. A cover for the microphone was fashioned from Mack's earplugs (McKeon Products, Inc., Madison Heights, MI) material, providing a 20-dB sound attenuation. Recordings of all of the evoked potentials were conducted again in the laboratory 2 to 4 hours, 1 week, and 6 weeks after the exposure. At the conclusion of the final recording session, a lethal dose of pentobarbital was injected intraperitoneally and 5 minutes after respiratory arrest; postmortem recordings of VsEPs were performed to rule out possible electromagnetic or electromechanically induced artifacts in the measurements.

The amplitude and latency of the first wave of VsEPs in response to linear and angular acceleration stimuli, reflecting the function of the otolith organs and semicircular canals, respectively, were compared between the baseline and post-exposure measurements using a non-parametric test (Wilcoxon paired rank test). ABR thresholds were also compared between the different recordings. $P < .05$ was taken to be statistically significant.

All experimental procedures were authorized by the Hebrew University–Hadassah Medical School Animal Care and Use Committee.

Techniques for Induction and Recording of Evoked Potentials

Linear vestibular evoked potentials. A detailed description of the stimulating apparatus has been reported in previous publications.[10,11] Briefly, the linear acceleration stimulator consisted of a solenoid that repeatedly delivered acceleration impulses to a sliding device restricted to moving the head of the animal in one axis. The head of the animal was attached to the moving sliding device by a head holder that firmly gripped the upper jaw in a plane that is optimal for utricle stimulation in rodents (head forward).[20] The magnitude of the acceleration was measured with a Bruel and Kjaer 4393 accelerometer mounted on the sliding device. The stimuli were accelerations of 3 g with a short rise time of 1 to 1.5 ms and displacements of approximately 50 μm. This 3-g intensity is in the midregion of the VsEP input–output function.[11] After each stimulus, the head was returned to its original position using a much lower acceleration. Acceleration impulse stimuli were given at a rate of 2.06 per second.

Angular vestibular evoked potentials. A complete technical description of the stimulator has also been previously reported.[8,9] Generally, the apparatus consisted of a drum, which was accelerated by a stepper motor. The animal was placed in the drum and the acceleration stimuli were transferred to the animal's head by a head holder, which firmly gripped the upper jaw in a plane that is optimal for maximal stimulation of the lateral semicircular canals (the head flexed down at approximately 15°). The resultant stimuli were clockwise and counterclockwise acceleration impulses at a peak intensity of 15,000°/second^2 with a rise time of 1–3 ms (8 stimuli in one direction and then 8 stimuli in the other direction). Acceleration stimuli were given at a rate of 2.06 per second.

Auditory evoked potentials. The response was elicited by alternating polarity click stimuli at a rate of 20.6 per second from an intensity of 120 dB pe SPL down to threshold in 5-dB steps. Threshold was defined as the lowest intensity which elicited repeatable responses in at least three repeat measurements. If no response could be recorded at 120 dB pe SPL, an intensity of 135 dB pe SPL was used. The earphone was placed 0.5 cm from the left ear without deflecting the pinna, which could obstruct the external meatus.

Recording apparatus. The electrical activity in response to the different stimuli was recorded by needle electrodes (Grass Instruments, Astro-Med, Inc., RI) inserted subdermally into the vertex referred to the left pinna with the right pinna serving as ground. The activity was band-pass filtered (300–1500 Hz), amplified, and averaged (n = 128) by standard evoked potential equipment (Microshev 4000, Efrath, Israel) and displayed "vertex positive up." Each response was obtained at least three times to ensure reproducibility.

RESULTS

Linear Vestibular Evoked Potentials

The mean (± SD) amplitude of the first wave of L-VsEPs before exposure to impulse noise (baseline) was 1.61 ± 0.48 μV. Two to 4 hours after exposure, it was significantly reduced ($P < .001$) to 1.06 ± 0.23 μV. The significantly reduced amplitude persisted in the measurement conducted 1 week after the exposure ($P < .005$) and showed partial recovery after 6 weeks (Table I).

The mean latency (± SD) of the first wave of the L-VsEPs before exposure was 2.03 ± 0.32 ms. Two to 4 hours after the exposure, it was significantly prolonged ($P < .005$) to 2.29 ± 0.36 ms. The prolongation of the latency persisted in the following measurements and was statistically significant even after 1 week ($P < .02$) and 6 weeks ($P < .005$) (Table I). Figure 1 demonstrates typical recordings from a sand rat before, 2 hours, and 6 weeks after exposure.

Angular Vestibular Evoked Potentials

The mean amplitude of the first wave of A-VsEPs before the exposure was 0.59 ± 0.32 μV. It was unchanged in the measurement 2 to 4 hours after the exposure. There

TABLE I.
Comparison of First Wave Peak-to-Peak Amplitudes and Peak Latencies (average ± SD) of Linear and Angular VsEPs and ABR Thresholds Between Baseline and Following Measurements.

	Linear VsEPs (n = 16)		Angular VsEPs (n = 10)		ABR (n = 18)
	P1 Amplitude (μV)	P1 Latency (msec)	P1 Amplitude (μV)	P1 Latency (msec)	Threshold (dB pe SPL)
Before exposure	1.61 ± 0.48	2.03 ± 0.32	0.59 ± 0.32	3.02 ± 0.36	55 ± 3.5
2–4 hr after	1.06 ± 0.23*	2.29 ± 0.36*	0.67 ± 0.23	3.30 ± 0.23*	>135*
1 wk after	1.16 ± 0.43*	2.23 ± 0.28*	0.46 ± 0.26	3.13 ± 0.45	115 ± 9*
6 wk after	1.42 ± 0.68	2.25 ± 0.38*	0.86 ± 0.51	3.14 ± 0.39	112 ± 8*

*Statistically significant ($P < .05$) in comparison to baseline measurements.
SD = standard deviation; VsEPs = vestibular evoked potentials; ABR = auditory nerve and brainstem evoked response.

were also no significant changes in amplitude in the following measurements (Table I).

The mean latency of the first wave of A-VsEPs before exposure was 3.02 ± 0.36 ms. Two to 4 hours after exposure it was significantly prolonged ($P < .02$) to 3.30 ± 0.23 μs. This statistically significant prolongation did not persist and there was recovery in the following measurements (Table I).

Auditory Brainstem Response

The ABR thresholds before exposure (baseline) were between 50 to 60 dB pe SPL (mean, 53 ± 3.5 dB). Two to 4 hours after the exposure, ABR could not be recorded even in response to clicks at an intensity of 135 dB pe SPL. One week after exposure, the mean ABR threshold was 115 ± 9 dB and 6 weeks after the exposure it was 112 ± 8 dB (Table I). Figure 2 demonstrates ABR recordings from a sand rat before, 2 hours, and 6 weeks after exposure (stimulus intensity 120 dB per SPL).

DISCUSSION

This study has shown that exposure of sand rats to intense impulse noise causes clear functional damage to their vestibular end organs, mainly the otolith organs. This is demonstrated by the significant decrease of the amplitude and irreversible prolongation of the latency of the first wave of L-VsEPs, which reflects the function of the otolith organs after exposure to impulse noise. In contrast, the amplitude and latency of first wave of A-VsEPs, which reflects the function of the semicircular canals, was not affected in the

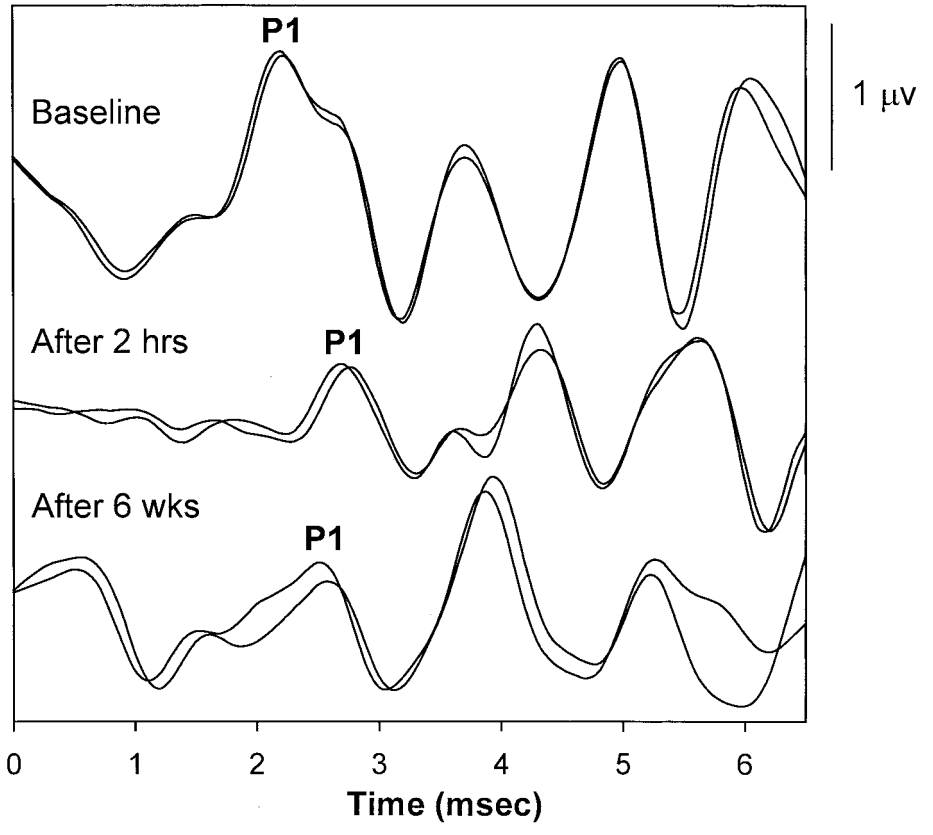

Fig. 1. Recordings of linear vestibular evoked potentials from a typical sand rat. The upper traces were recorded before exposure (baseline), the middle traces 2 hours after, and the bottom traces 6 weeks after the exposure (the decreased amplitude and prolonged latency in the traces after the exposure are evident). The stimulus trigger was at the onset of the trace and P1 shows the first wave of the response.

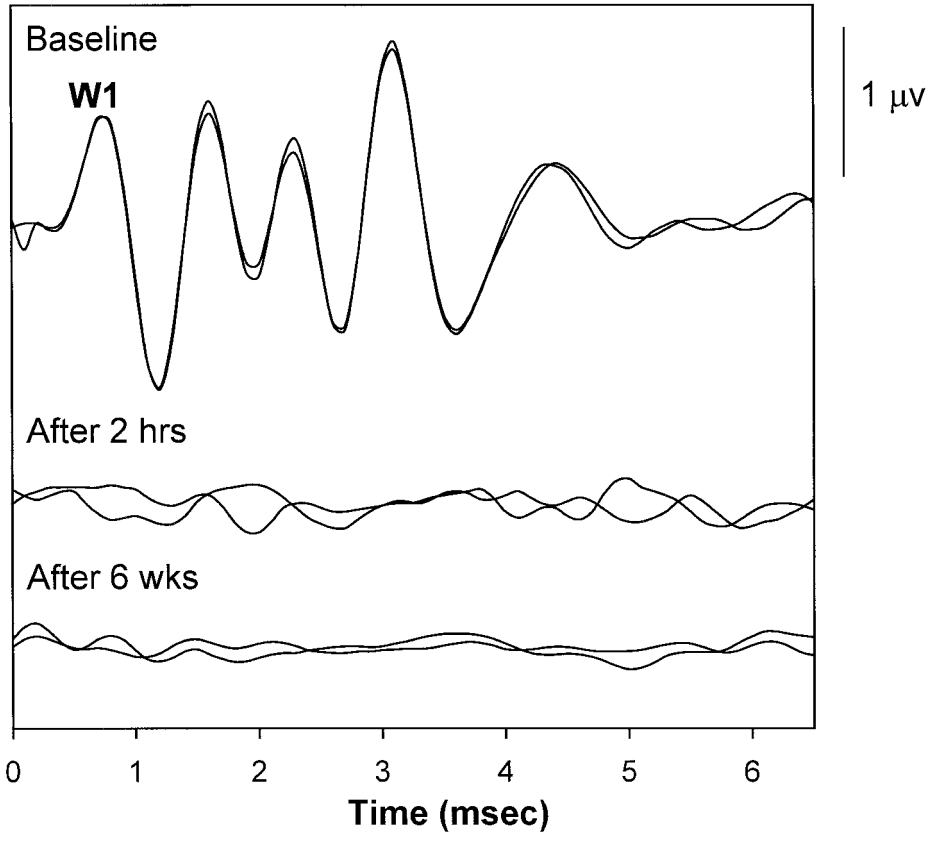

Fig. 2. Recordings of ABR in response to 120 dB pe SPL clicks from the same typical sand rat. The upper traces were recorded before exposure (baseline), the middle traces 2 hours after, and the bottom traces 6 weeks after exposure.

long-term by the exposure. As expected, ABR thresholds were significantly elevated after the noise exposure.

A number of clinical and animal studies examined the effect of impulse noise on the vestibular end organ. As mentioned earlier, the conclusions of these studies were contradictory. Juntunen et al.[4] and Ylikoski et al.[5] reported that subjects exposed to impulse noise showed significantly more body sway than controls. They suggested that exposure to impulse noise causes subclinical vestibular disturbances. Pyyko et al.,[6] using posturography for assessment of vestibular damage, reported that no significant differences in body sway were found, suggesting that impulse noise may not cause functional changes in the vestibular system. Of note is a recently published clinical report by Golz et al.[1] who showed that when intense noise induced asymmetric hearing loss, there was also evidence of vestibular dysfunction assessed by electronystagmography. In an animal study conducted by Ylikoski et al.,[7] guinea pigs were exposed to rifle shots and histologic analysis of their inner ears was conducted. Damage was seen in their ampullary cristae and utricular and saccular maculae. It appeared that the damage was primarily mechanical. Our study, using a direct test for functional assessment of the vestibular end organ, correlates with most of these studies. Nevertheless, there is disagreement with the reported histologic damage to the ampullary cristae of the guinea pigs described in Ylikoski's animal study, because in our study there was little effect on the response of the semicircular canals (no long-term changes in A-VsEPs).

When clinically applying the results of the present animal study to human subjects, one must be careful. First, it seems that the sand rats' inner ears are more vulnerable to the impulse noise than those of humans. The evidence for this is the dramatic effect of the impulse noise on their cochlear response. It seems that during the first hours after the exposure, the animals suffered total hearing loss. This is usually not the case in humans. Secondly, we would rarely expect clinical vestibular symptoms in humans because of the existence of compensation mechanisms. While this study has shown clear damage to the vestibular end organ of the sand rat, the range of appearance of vestibular symptoms in humans exposed to impulse noise is probably variable and might depend on individual vulnerability and compensation ability.

The vulnerability of the vestibular end organs to noise exposure may depend on several additional factors:

1) *the exposure regimen*—in a previous study[3] conducted in this laboratory, rats were exposed to different regimens of noise duration and intensity (between 113 and 135 dB) and for periods from a few minutes to 3 weeks. These noise exposures did not cause a significant long-term effect on the VsEPs, in contrast to the clear affect in the present study (about 160 dB SPL), implying that the appearance of damage and its extent are related to the intensity of the noise. Another possibility is that the impulsive nature of the noise (repeat onset gunshots) to which the animals were exposed in the present study is more "destructive" than the steady state[21] and may play a significant role in inflicting vestibular end organ damage.

2) *The state of the ear*—Exposure of the normal ear to 113 dB SPL broad band noise for 60 minutes did not have an effect on the vestibular end organs, whereas after fenestration of the semicircular canal, the same noise exposure caused significant vestibular dysfunction.[22] This is similar to the findings in patients with superior canal dehiscence syndrome[23] in whom vertigo or oscillopsia is seen after exposure to loud noise. It has been suggested that these findings may be the result of the round window serving as a pressure release in the cochlear perilymphatic channel in the normal ear. Therefore, the sound pressures induced in the cochlear perilymph by the stapes footplate are preferentially transmitted to the cochlear channels and not to vestibular channels. By inducing a fenestration in the semicircular canal, presumably "a round window" is created in the vestibular part of the inner ear, allowing additional sound energy to reach the vestibular channel.

The finding in the present study that the semicircular canals are less sensitive to impulse noise than the otolith organs is interesting and may also have implications for the possible mechanism of the vestibular damage by noise. This may be the result of a possible symmetric spread of energy through the semicircular canals causing a smaller pressure differential across the cristae, whereas there is greater pressure differential across the maculae.

CONCLUSION

It appears that impulse noise not only damages the cochlea, but also causes clear functional impairment to the vestibular end organs, mainly the otolith organs. When clinically applying these conclusions, it should be taken into consideration that this is an animal study and that the range of vestibular symptoms in human subjects exposed to impulse noise may be variable.

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