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Vibration-Induced Nystagmus – A Sign of Unilateral Vestibular Deficit

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Key Words
Vibration · Peripheral vestibular excitability · Nystagmus

Abstract
Vibrations of 60 Hz and 100 Hz were applied on the mastoid of healthy subjects, patients with unilateral peripheral vestibular lesions, with central lesions of different localizations, and patients with benign paroxysmal positioning vertigo (BPPV). In patients with unilateral peripheral deficit a horizontal nystagmus with a small torsional component beating generally to the not affected side could be observed. This nystagmus did not show adaptation during 40 s. The occurrence was more frequent using 60-Hz stimulations. This vibration-induced nystagmus did never occur in healthy subjects, seldom in patients with central vertigo, and only exceptionally in patients with BPPV. It is concluded that the finding of a vibration-induced nystagmus reflects a side difference of peripheral vestibular excitability.

Introduction

Strictly speaking, vibrations are periodic movements within an unlimited frequency frame. Because of their omnipresence, a human being is permanently exposed to mechanical vibrations. In order to perceive vibration stimuli, human beings as well as many animals have mechanosensors in the skin, muscles (muscle spindles), tendons, the capsula of articulations, and in the inner ear. In clinical examination its capacity is tested by placing a tuning fork on certain defined areas or by applying special vibration coils. The sensation of vibration might be impaired by several diseases, e.g. polyneuritis.

The perception of vibration plays a role in the orientation of an individual in space as could be shown by Goodwin et al. [1]. By means of vibrations over muscles illusions of motion were evoked. This observation also proves that there must be a cortical representation of muscle spindles. These findings were confirmed and elaborated in detail by several authors [1–4]. The receptors for vibration stimuli were identified as muscle spindles [1, 2] whose stimulation leads to an activation of Ia fibers [4]. A direct effect on the vestibular system was 'definitively excluded' by Biguer et al. [3].

By vibration it is possible to induce, besides the motion illusion, postural changes that show a direct relation between the stimulated muscle and the direction of body destabilization [4]. In patients suffering from vestibular neuritis a change of horizontal eye position could be induced by 100 Hz vibration of neck muscles [5]. However, a nystagmus-like pattern of eye movements has never been observed. In contrast to these findings Kobayashi et al. [6] detected, by observation with Frenzel's glasses, a

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nystagmus when giving vibration stimuli of 125 Hz to neck muscles not only in 49% of healthy subjects but also in 56% of patients with vestibular lesions. They concluded that cervical afferences participate in vestibular compensation and that unilateral stimulation by vibration leads to decompensation. The possibility of a transduced excitation of the vestibular receptors from the neck muscles was not taken into account.

Lücke [7] was the first to postulate a relation between vibratory stimulation and vestibular disorders. After 2 of his patients had reported vertigo while using an electric shaver, Lücke [7] systematically introduced vibration stimuli produced by a customary vibrator. Applying the vibrations on the mastoid a latent nystagmus could be seen in patients suffering from different unilateral vestibular diseases and sometimes in central vertigo and even exceptionally in persons without vestibular impairment. As a consequence of these observations the occurrence of a vibration nystagmus could be considered as evidence of a peripheral vestibular imbalance.

The aim of this study was to investigate the phenomenon of vibratory nystagmus in more detail, especially its phenomenology, its dependence on different stimulation frequencies, and its correlation to different vestibular disorders.

Methods

For producing vibration a customary vibration stimulator (product of ABC Enterprise) was applied to the mastoid for a maximum duration of 10 s (fig. 1). The patient was in a sitting position. For a first series of observations the stimulus generator yielded a vibratory frequency of 100 Hz. The amplitude at the point of stimulation was about 0.5 mm parallel to the point of application, the actual force depended however on the pressure applied by the examiner. The contact area was 0.9 cm^2. During vibratory stimulation, eye movements were observed under Frenzel's glasses, in some patients recorded by videocamera (see below).

In our study we compared 75 healthy students showing no signs of vestibular disorders, 60 patients suffering from unilateral peripheral vestibular lesion proven by neuro-otological examination (table 1), 20 patients with BPPV (all of them with proven positional nystagmus) as well as 40 patients with vertigo of central origin (table 1). We compared the occurrence of spontaneous nystagmus, head shaking nystagmus or nystagmus during vibratory stimulation.

In the second part of the study varying stimulus frequencies from 40 to 100 Hz were applied in 50 patients with a proven unilateral deficit.

In addition, we figured out whether vibration nystagmus underlies the phenomenon of adaptation by recording the appearance of the nystagmus following constant vibration on the mastoid during at least 40 s by video-oculography. We examined 20 patients with vibratory nystagmus due to peripheral vestibular imbalance caused by vestibular neuritis and Menière's disease. The patients were seated with an indifferent position of the head. We used two types of videorecording systems: MIR 92 electronic box, Delta Electronic power support, MIR 92 D-SYE 100, Euromir 95 OKS Box TRM, PO3 head unity (Kayser-Threde) and Trinitron Video8 together with ESMed Videoglasses and VOG 2 D Video-oculography by Senso Motoric Instruments GmbH, Teltow, Germany. After placing the camera systems on the patient's head we fixed the vibrator manually on the mastoid. The appearance of the nystagmus during vibration stimulus at the same location was recorded for at least 40 s.

Fig. 1. Clinical application of vibrations on the mastoid and synchronous observation of eye movements through Frenzel's glasses.

Table 1. Distribution of patients

Diseases of peripheral vestibular origin	
Vestibular neuritis	31
Menière's disease	22
Acoustic neuroma	2
Otosclerosis	3
Fistula of the lateral canal	1
Traumatic rupture of the vestibular nerve	1
Vertigo of central origin – site of the lesion	
Lateral brain stem	5
Paramedian brain stem	8
Cerebellum	6
Medulla oblongata	1
Pontine region	3
Other localizations (Binswanger's disease, Parkinson's disease, multiple sclerosis, head injury)	14
Phobic vertigo	3

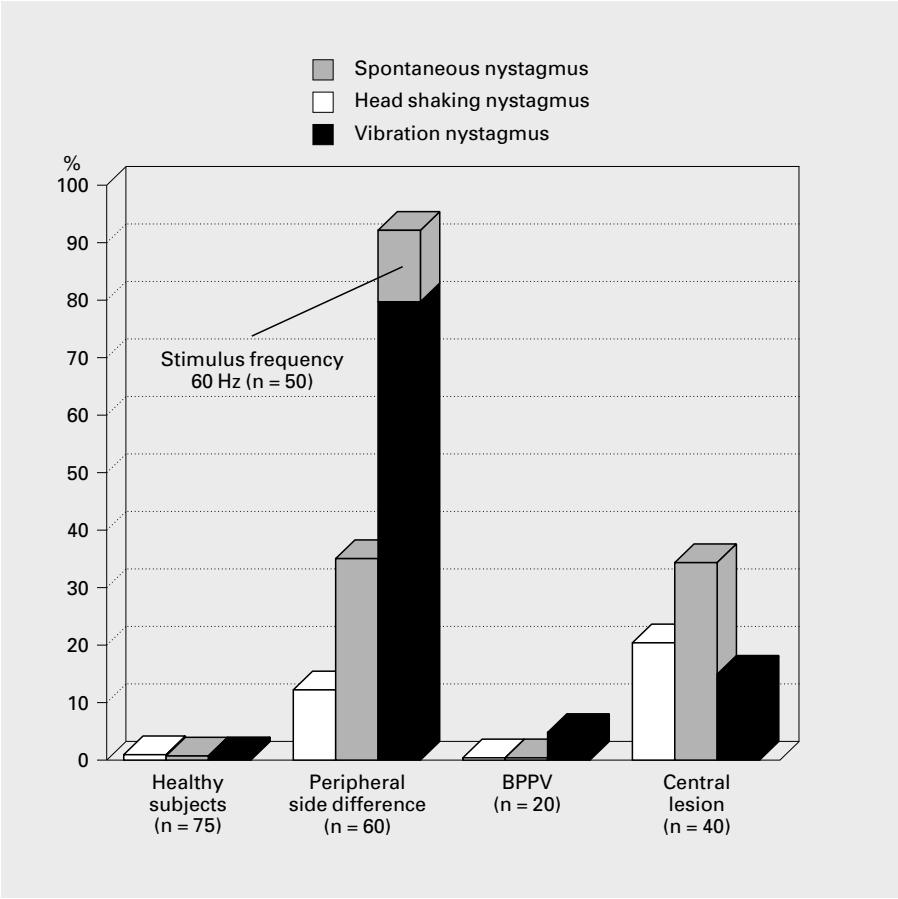


Fig. 2. Occurrence of spontaneous nystagmus, head shaking nystagmus and vibration nystagmus in healthy subjects and patients with different pathologies. The hatched column represents the higher occurrence of vibration nystagmus using a stimulation frequency of 60 Hz.

Results

None of the 75 healthy subjects developed any kind of nystagmus, not even during vibratory stimulation (fig. 2).

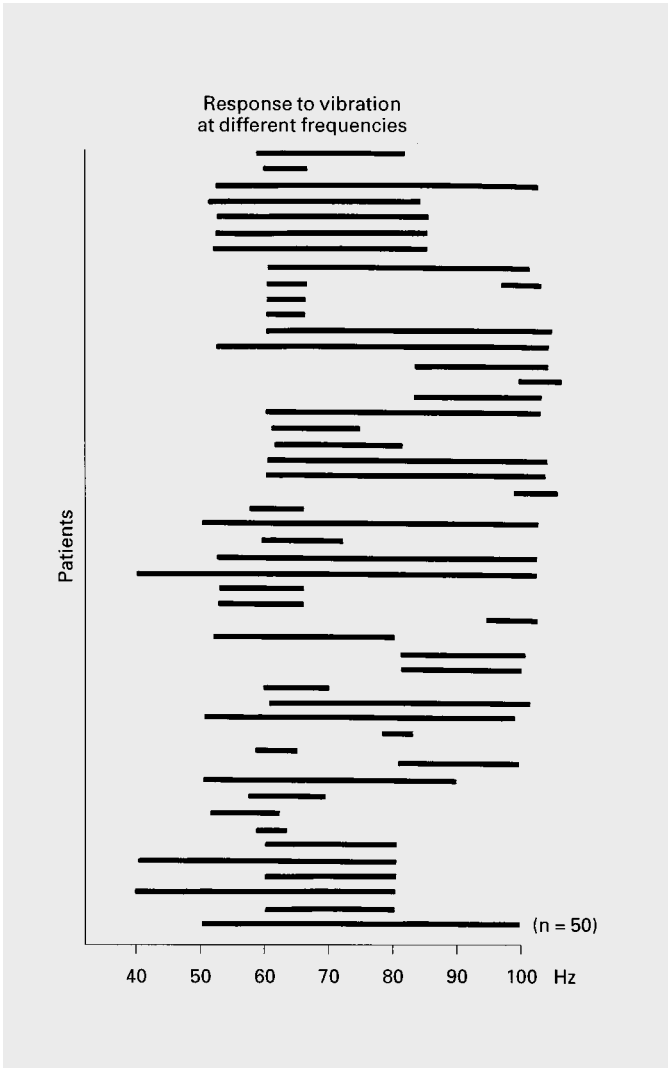
In the group of patients with unilateral peripheral vestibular deficit, in 12% a spontaneous nystagmus occurred, in 35% a head shaking nystagmus and in 80% a vibration nystagmus became visible (fig. 2). In each case, a head shaking nystagmus was observed, a vibration nystagmus could also be proven. In the great majority, the vibration nystagmus beat was always in the direction to the healthy side, only exceptionally in patients with Menière's disease presenting an irritative stage the nystagmus was directed to the affected ear.

In the group of patients with BPPV, neither a spontaneous nor a head shaking nystagmus could be found. A vibrational nystagmus occurred in only 1 of 20 patients (fig. 2).

In 5 of 40 patients suffering from central vertigo a vibratory nystagmus was detected. In these patients the central lesion was located in the cerebellum or the lateral brain stem.

By stimulating each patient at different vibration frequencies an individual frequency range could be determined in which a vibration nystagmus became visible (fig. 3). As shown, the probability of triggering a vibration nystagmus is best at a frequency of 60 Hz. Comparing 60 Hz stimulation to 100 Hz stimulation the rate of positive responses was higher using 60 Hz stimuli (fig. 2).

When examining the appearance of vibratory nystagmus during 30 s we found no significant decrease in the frequency of nystagmus (fig. 4). The nystagmus was horizontal with sometimes a rotatory component; a vertical vibration nystagmus was never observed (fig. 5). The nystagmus nearly showed the same frequency (fig. 4a), amplitude and velocity of the slow component. The frequency individually varied between 18 and 81 beats/min (fig. 4a). The frequency was examined at intervals of 10 s (fig. 4b). During the first 10 s we found 7.25 ± 3.29 beats (n = 20), in the following 10 s 6.75 ± 3.95 beats (n = 20), in the



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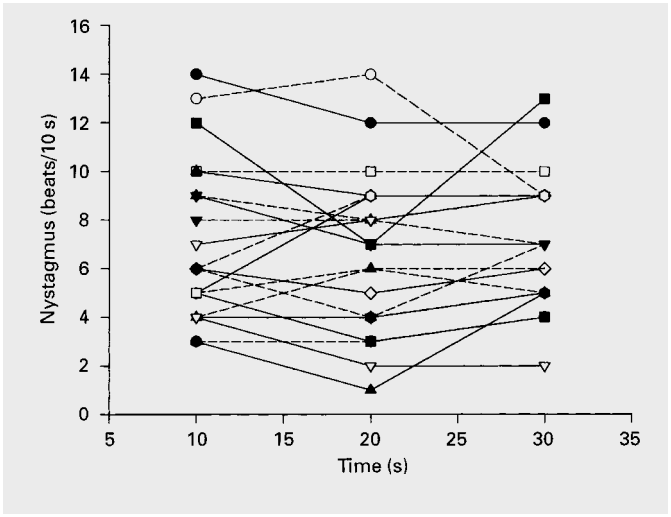

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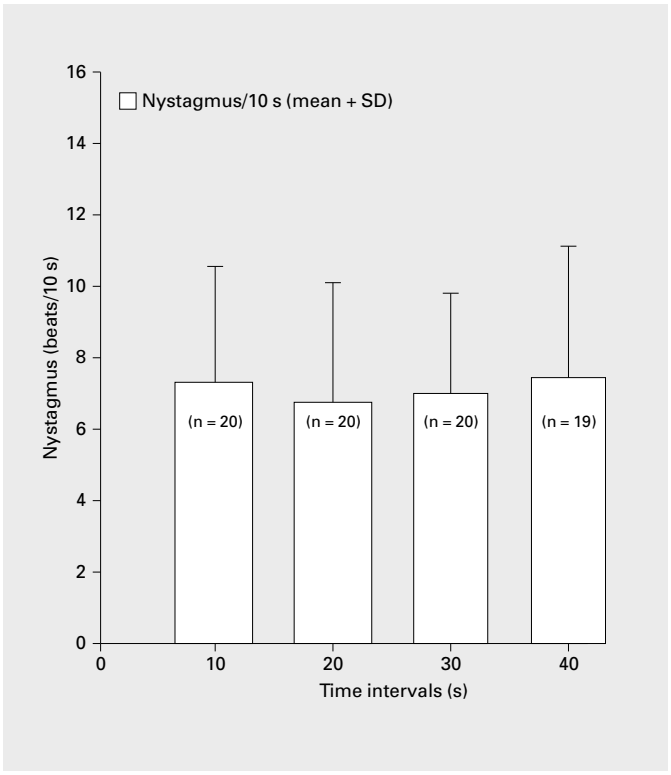

4b

Fig. 3. Occurrence of vibration-induced nystagmus at different stimulation frequencies in 50 patients with a proven difference of peripheral vestibular excitability. Each bar represents the range of frequency in which vibration nystagmus appears.

Fig. 4. a Time course of the vibration-induced nystagmus frequencies during 30 s of stimulation in 20 patients with a proven difference of peripheral vestibular excitability. **b** Mean frequencies of nystagmus beats per 10 s in a time span of 40 s.

third 10 s 7.05 ± 2.78 (n = 20) and in the fourth 7.5 ± 3.63 (n = 19). Obviously, there was a big variability between the individuals, but intraindividually most patients showed constant nystagmus (fig. 4a). It must be pointed out that vibration-induced nystagmus becomes visible only during stimulation and it stops immediately when stimulation is interrupted.

Discussion

In 80% of patients suffering from a unilateral peripheral vestibular lesion a nystagmus can be evoked by vibrations of 100 Hz when applied on the mastoid. Therefore this method proves to be more valid in detecting a peripheral difference of vestibular function than studies relying on head shaking nystagmus or spontaneous nystagmus

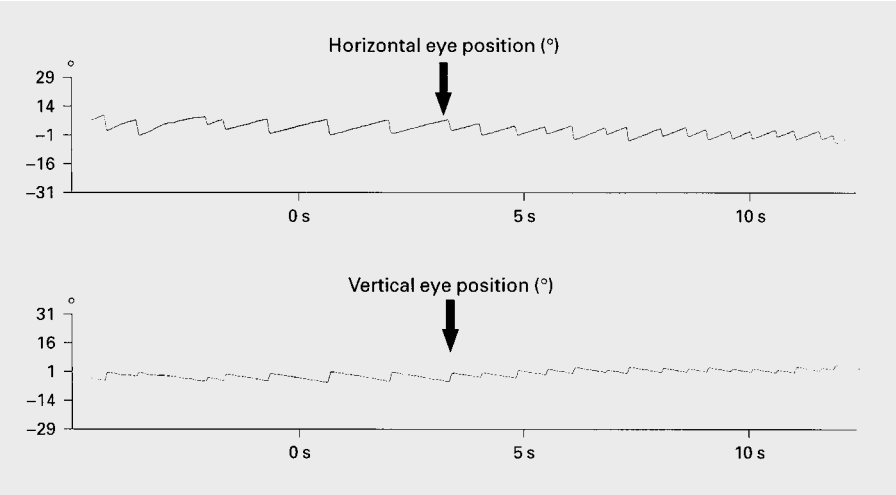

Fig. 5. VOG recording of vibration nystagmus. A preexisting spontaneous nystagmus is enhanced when vibrations are applied (↓).

[8]. The proportion of positive responses can be improved up to 92% by use of 60 Hz vibration. A vibration nystagmus was never seen in healthy subjects, very rarely in patients with central vertigo and only exceptionally in patients with BPPV.

Our observations mainly confirm the experience of Lücke [7], suggesting that vibratory stimuli induce a depolarization at the level of semicircular canals. We support this opinion and postulate that vibration nystagmus is due to selective excitation of vestibular receptors and not to an activation of neck proprioceptors. In contrast to Lücke's data we point out that the finding of a vibration nystagmus reflects the side difference at the level of the vestibular receptors because of its absence in normals and in most patients with central vertigo. So the vibration test gives the same information as caloric testing. On the other hand we must see the difference between caloric unilateral stimulation and vibration nystagmus: as vibration nystagmus does not show an adaptation like continued caloric stimulation, the triggering mechanism must be different [9].

For those cases in which a nystagmus was evoked by stimulating the neck muscles [6] we suggest a mechanical transmission from the muscles to the cranial bone from where vestibular receptors were activated, yet with less intensity. The vibration nystagmus can be triggered from both mastoids, and the transmission over the head bone is hardly reduced by the bone of the skull. This was to be expected as it is well known that acoustic vibrations are also conducted through bones without significant losses.

Actually it cannot be completely determined which of the vestibular receptors is responsible for evoking the vibration nystagmus. The nystagmus beats primarily in a horizontal direction combined with a small torsional component. Therefore it cannot be excluded that otolith receptors are also involved in triggering a vibration nystagmus. It is still unclear if the pulse of the endolymph movement constitutes the sole cause of a vibration-induced nystagmus or if a direct excitation of sensory cells also plays a role.

We explain the occurrence of a vibration-induced nystagmus by a strong supraliminal stimulation of the receptor, inducing an extreme imbalance at the vestibular nuclei, which then triggers the nystagmus. In most cases the imbalance induced by vibrational stimuli is more pronounced than by head shaking because of the evidently less frequent appearance of head shaking nystagmus in the same group of patients.

Provided that this interpretation is correct, in pure central lesions and therefore symmetric caloric excitability, no vibration nystagmus should be detectable. Our results support this hypothesis. It actually occurred solely in patients with centrally but laterally located preponderance of the lesion, i.e. the lesion was located in the cerebellum and often accompanied by caloric hypoexcitability.

Generally, a vibration nystagmus is not to be expected in patients with pure BPPV. This was confirmed by our observations. In case a vibration nystagmus is detected in patients with BPPV, this could be due to peripheral vestibular imbalance as caused by Lindsay-Hemenway syndrome [10].

In contrast to our results, Kobayashi et al. [6] observed a nystagmus also in healthy subjects during vibratory stimulation of the neck muscles. This can be explained by

the fact that these authors used a stimulator with a much larger impact area working at 125 Hz.

We assume that one is more likely to detect a peripheral side difference by using vibratory stimuli than by other conventional methods. As a result, this technique facilitates the distinction between peripheral and central origin of vertigo. At this point of knowledge, the question cannot be answered definitively whether an imbalance at the level of vestibular receptors, vestibular nerves or vestibular nuclei is more likely.

In a future study the choice of different impact points in order to trigger vibration nystagmus will be compared. Another question to be answered is if the occurrence of vibration nystagmus depends on vestibular compensation. Finally the value of the method will be evaluated in special pathologies like acoustic neuroma and well-defined central lesions.

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