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Subjective Visual Horizontal During Follow-up After Unilateral Vestibular Deafferentation with Gentamicin

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The subjective visual horizontal (SVH) was measured by means of a small, rotatable, luminous line in darkness in the upright head and body position and at 10, 20 and 30° of tilt to the right and left before, and repeatedly during a follow-up period of 1 year after intratympanic gentamicin instillations in 12 patients with recurrent vertigo attacks. This treatment caused a loss of the bithermal caloric responses on the diseased side. Shortly after treatment there was a significant tilt of SVH towards the treated side (group mean = 10.6°). Repeated testing made it possible to characterize mathematically the changes with time for SVH. For the group of patients as a whole this otolithic component of vestibular compensation was best described by a power function, $SVH = 8.65t^{-0.16}$ degrees, where t is time in days after maximum tilt of SVH. After 1 year, SVH was still significantly tilted towards the treated side (group mean = 3.16°). Gentamicin treatment also caused a significant reduction in the perception of head and body tilt towards the deafferented side, while the perception of tilt towards the healthy side did not show any significant changes. During follow-up there was a gradual improvement in the perception of tilt towards the treated side. However, a significant asymmetry in roll-tilt perception was still present 1 year after deafferentation. There was no correlation between SVH in the upright position and roll-tilt perception, suggesting that these parameters are to some extent dependent on different afferent input from the vestibular organ. They were also found to be complementary for the detection of vestibular disturbance. *Key words*: *CNS plasticity*, *gravity*, *otolith*, *roll-tilt perception*, *utricle*, *vestibular compensation*.

INTRODUCTION

Most available data on vestibular lesions and compensation processes have been gained from studying semicircular canal function. In recent years, however, dysfunction of the otolith organs has been a matter of growing interest.

Clinical studies of otolith function have mainly considered the utricle. The utricular macula is divided by the striola into a medial and a lateral area with oppositely polarized hair cells. Sensory cells excited by ipsilateral head tilt are located in the medial area and those excited by contralateral tilt are located in the lateral area (1, 2). In the squirrel monkey the medial area has been found to be larger than the lateral (3). Electrophysiological studies on the cat and squirrel monkey have revealed a predominance of sensory afferents in the vestibular nerve excited by ipsilateral as opposed to contralateral tilt (3–5). Because of such findings, in the diagnosis of vestibular disturbances the human utricle has also been considered as an asymmetric sensor with preference for ipsilateral tilt (6–8), which is a prerequisite for the feasibility of localizing unilateral hypofunction by means of otolithic tests.

One method for testing otolith function is to ask the subject to indicate, by setting a luminous line in complete darkness, the subjective visual horizontal (SVH) or vertical at different angles of head and body tilt (9). Normal subjects, while in the upright position, are able to set the luminous line with great accuracy close to the true horizontal (8, 10). By testing in tilted positions, the ability to perceive head and body tilt can be calculated. In most clinical investigations SVH has been studied after an acute loss of peripheral vestibular function on one side. These patients show a significant tilt of SVH towards the side with the lesion (6, 10). In addition, a reduced perception of head and body tilt towards the lesioned side but a normal perception of tilt towards the healthy side has been demonstrated, suggesting an asymmetric tilt sensitivity of the single remaining utricle on the intact side (6, 11).

The perceptual and ocular motor findings, such as tilt of SVH and spontaneous nystagmus, after unilateral vestibular deafferentation are caused by a loss of activity in the ipsilesional vestibular nucleus, resulting in an imbalance in neural resting activity between the two vestibular nuclei [for references see (12)]. Vestibular compensation denotes the gradual reduction of symptoms and signs that occurs even when the loss of vestibular function is permanent. It has been shown that the decay of spontaneous nystagmus parallels the restoration of neuronal tone balance between the ipsilesional and contralesional medial vestibular nuclei (12). A reduction in tilt of SVH found by Curthoys et al. (10) is probably an analogue of the diminution of spontaneous nystagmus. Serial measurements of SVH would then be one possible method for studying the process of vestibular compensation.

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Intratympanic instillations of gentamicin are a well-established treatment in patients with disabling peripheral vestibular disorders (13–15). In the present investigation the SVH for different body positions was measured before and repeatedly during a follow-up period of 1 year after gentamicin treatment. The major aim was to establish whether changes in SVH after gentamicin treatment are comparable to those seen after unilateral vestibular nerve section (10, 11) and in the acute stage of vestibular neuritis (6). In particular, we wanted to index the otolithic components of vestibular compensation by means of repetitive measurements of SVH.

MATERIALS AND METHODS

Patients

Fourteen consecutive patients undergoing unilateral intratympanic instillations of gentamicin for treatment of recurrent disabling vertigo or drop attacks were initially recruited for the study. One of these was not available for follow-up. In another there was no reduction in vestibular function after treatment, as reflected by the caloric test. The material therefore comprises 12 patients (seven men and five women, aged 21–76 years; mean 53 years). All were diagnosed as having a unilateral peripheral vestibular disturbance. In nine cases the diagnosis was Meniere's disease. In the remaining three the vestibular disorder was preceded by hearing loss (in one case a sudden deafness) on the same side and there was not a typical history of Meniere's disease. All patients had been subjected to careful neuro-otological examinations to rule out any affection of the central nervous system. Eight patients were treated on the right side and four on the left. Although the patients were all considered to have an active disease when treatment was started, none had any attack of vertigo during the days of treatment.

Measurements of the subjective visual horizontal

The test procedure and treatment of test data have been described in detail in an earlier paper (8). In brief, at different angles of head and body tilt in the roll plane the patient has to rotate, by remote control, a narrow luminous bar in an otherwise completely darkened room so that it looks horizontal. Eight settings of the light-bar are done in the upright position and at 10, 20 and 30° of tilt to the right and to the left. The SVH is measured as the deviation in degrees from the true gravitational horizontal and for simplicity it is called B, for bias. Parameters considered are SVH in the upright position, B_0, and the ability to perceive head and body tilt to the right and to the left. As a measure of tilt perception the correction for tilt, K, is calculated as the mean of SVH in the three tilted positions with respect to B_0. This measure of tilt perception is independent of any deviation of SVH in the upright body position. An ideal correction for tilt, i.e. SVH in the three tilted positions not differing from SVH in the upright position, gives $K(\text{right}) = 0$, or $K(\text{left}) = 0$. A reduced ability to perceive roll tilt gives a negative K-value, while a tendency to overestimate tilt gives a positive K-value. The normal ranges [95% of normal subjects (8)] are: $-2.5° < B_0 < +2.5°$; $K(\text{right})$ or $K(\text{left}) > -3.0°$. The test–retest variability in normal subjects was less than 1.2° for B_0, and less than 3.7° for K (8).

Electronystagmography

Eye movements were recorded by means of electrooculography, and the slow-phase velocity of spontaneous and calorically induced nystagmus was analysed with a computerized technique (16). Bithermal caloric testing (30°C and 44°C) was performed according to standardized procedures at the department. The side difference was calculated using Jongkees' formula (17) and a side difference of 20% or more (caloric ratio ≥ 0.2) was considered pathological.

Gentamicin injections

Patients received two to six intratympanic instillations of a sodium bicarbonate-buffered (pH 6.4) solution of gentamicin (30 mg/ml), daily or every second day until an ototoxic effect was manifested by symptoms of dizziness or by spontaneous nystagmus.

Test occasions

Before treatment, measurements of SVH, as well as of spontaneous and calorically induced nystagmus, were performed in all patients. During follow-up, testing times for SVH were related to the day when symptoms or spontaneous nystagmus, indicating ototoxicity, first appeared, and this was denoted as day 1. Tests could not be performed on the same number of days after treatment for the different patients, and for purposes of analysis measurement occasions are grouped in intervals as follows. First follow-up during week 1; second, week 2; third, week 3–4; fourth, week 5–8; fifth, week 9–16; sixth, week 17–32; and seventh follow-up after approximately 1 year. If a patient was tested twice or more during one period, where appropriate the mean value and mean time for that individual were calculated. A caloric test was performed at the sixth or seventh follow-up.

RESULTS

The results presented here were obtained from 12 patients with a complete loss of the caloric responses on the treated side (bithermal testing; 30°C and 44°C), as confirmed at long-term follow-up. Not all patients were available for testing in every interval. The number of patients tested in the different time intervals was: before treatment, $n = 12$; first follow-up, $n = 12$; second, $n = 11$; third, $n = 12$; fourth, $n = 10$; fifth, $n = 10$; sixth, $n = 11$; seventh, $n = 12$.

Most of the patients tolerated the treatment well. In the course of treatment, one patient was confined to bed rest due to vertigo symptoms accompanied by nausea and vomiting. In long-term follow-up five patients reported short-lasting dizziness or unsteadiness after rapid head movements but in none of the patients were there any vertigo attacks or drop attacks during the follow-up period. All patients were pleased with the treatment.

Hearing was measured before treatment and after cessation of vestibular symptoms induced by treatment. Regarding the pure tone average (mean of hearing thresholds at 0.5, 1.0 and 2.0 kHz), there was no change in hearing after treatment. The group means were 65 ± 16 dB before treatment and 66 ± 18 dB after treatment. Only one patient showed a reduction in pure tone average of more than 15 dB. There was no relation between hearing level before treatment and change in hearing caused by the treatment.

Subjective visual horizontal in the upright body position, B_0

Individual measures of B_0 before treatment, at maximum deviation in each patient after treatment, and at the 1-year follow-up are shown in Fig. 1. Before treatment, four patients had a B_0 that was pathologically tilted (more than 2.5°) away from the diseased side, while only one had a pathological tilt of B_0 towards the diseased side. After treatment, a pathological tilt of B_0 towards the treated side was found in all patients. The individual maximum value of B_0 was found at a mean of 18 days (range 7–41) after treatment. The mean for the entire group of this maximum was $10.6 \pm 6.51°$.

Clearly, there was considerable interindividual variability in the tilt of B_0 induced by the treatment. A correlation was found between the caloric ratio (CR) before treatment and the maximum tilt of B_0 after treatment: $r = 0.80$, $n = 12$, $p < 0.01$ (linear regression). Two patients, who before treatment had the weakest caloric responses on the diseased side (CR = 0.86 and 0.88), also showed the smallest tilts of B_0 ($+5.5°$ and $+3.0°$) after treatment. The only patient with side-like caloric responses (CR = 0.15) before treatment showed $B_0 = +26.9°$ at day 7 after treatment. Hence, the magnitude of tilt after treatment was related to the functional level of the diseased vestibular organ before treatment. As a comparison, no correlation was found between CR and B_0 before treatment (coefficient of correlation, $r = 0.38$, $p = 0.18$). Although a measure of semicircular canal function, CR was chosen as a well-established indicator of a side difference in functional level.

Group means of B_0 at the different testing times are shown in Fig. 2. Tilt of the light-bar towards the diseased side (ipsilesional end down) is assigned a positive value. In this way data from patients receiving right and left gentamicin treatment are combined.

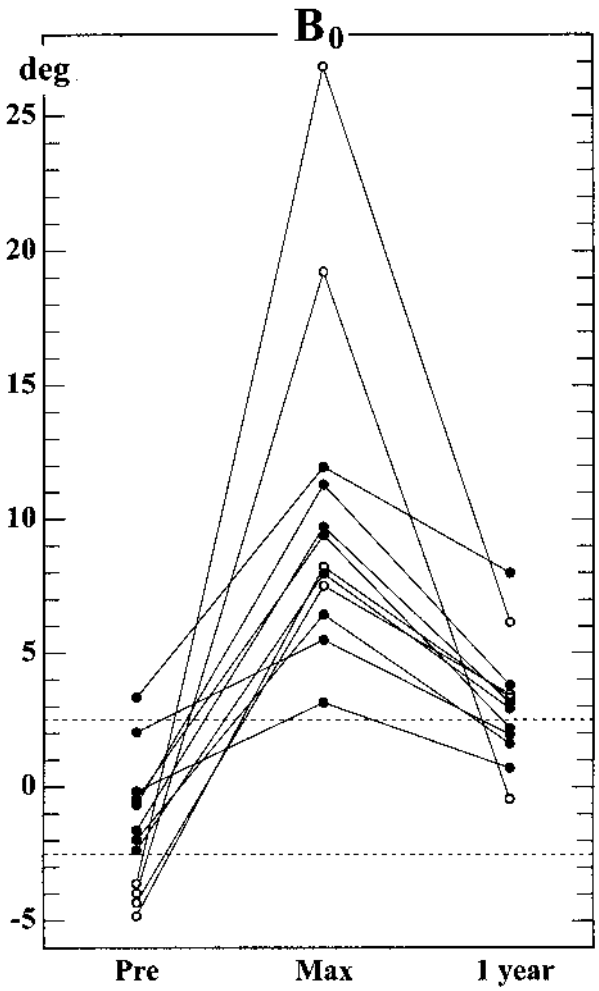

Fig. 1. SVH in the upright body position (B_0) before treatment (Pre), at maximum deviation after treatment (Max) and at the 1-year follow-up. Tilt of B_0 towards the diseased side is assigned a positive value. The lines join the data points for each patient. Normal range is indicated by *dashed lines*. The four patients who had a pathological tilt of B_0 towards the healthy side before treatment are represented by *open symbols*.

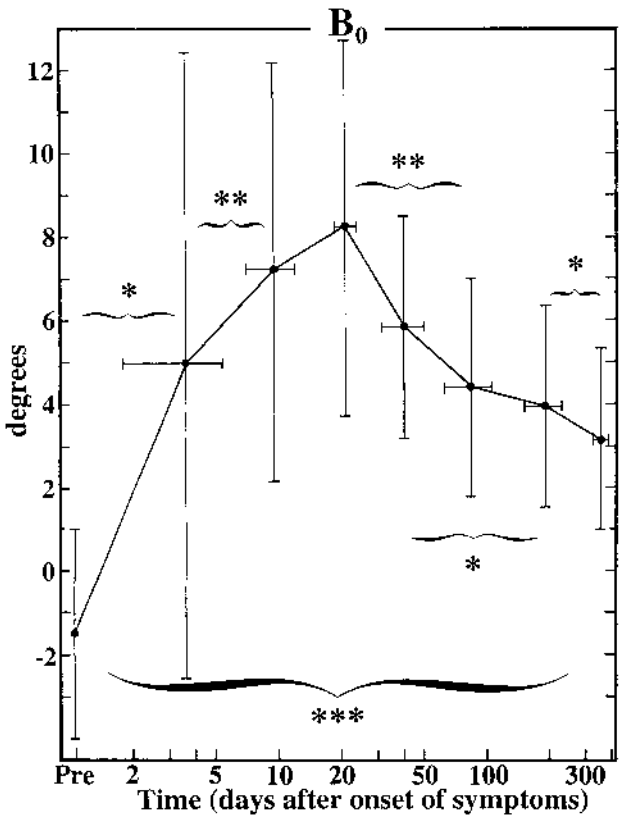

Fig. 2. Group means of B_0 in the different test intervals. Tilt of B_0 towards the diseased side is assigned a positive value. Data points represent both the mean of B_0 and the mean testing time for each interval. Error bars denote 1 SD. The time (logarithmic scale) is days from end of treatment. Asterisks indicate a significant change in B_0: $*p < 0.05$; $**p < 0.01$; $***p < 0.001$.

Before treatment the group mean of B_0 was not significantly different from zero: mean of $B_0 = -1.52 \pm 2.53°$, $n = 12$, $p > 0.05$ (two-tailed t-test). During follow-up, B_0 was significantly different from the pretreatment value. In the third follow-up the group mean was: $+8.26 \pm 4.45°$, $n = 12$, $p < 0.001$ (paired t-test). During the remaining follow-up period there was a gradual reduction in tilt of B_0. In the last testing the group mean was $+3.16 \pm 2.25°$, which was significantly different from the value in the third follow-up: $n = 12$, $p < 0.001$ (paired t-test). It was, however, still significantly different from zero ($p < 0.001$).

Changes in B_0 for each individual after the maximum tilt are shown in Fig. 3. Although differences in the maximum value were considerable, most patients showed a pattern characterized by a very rapid decay in the early stage followed by slow changes during the remaining observation period. In one patient, a 50-year-old woman, the vestibular compensation as reflected by changes in SVH seemed to be almost absent; at the last testing B_0 was still tilted by 8° towards the treated side. She did not, however, show any signs of impaired semicircular canal compensation; nor were there any problems with the subjective functional recovery.

In order to define a function characterizing the time-course of compensatory changes in B_0 for the group of patients as a whole, the data were treated as follows. First, for each patient the time when maximum deviation of B_0 was found was denoted day 1. Then, to correct for the large interindividual variability in the maximum tilt of B_0, the data from each patient were normalized, i.e. the value of B_0 at every testing time was divided by the individual total range (B_0(max)–B_0(min)) of the entire period of compensation. Finally, we corrected for the interindividual variability in the value of B_0 found at long-term follow-up: for each patient the difference between the individual minimum value and the group mean minimum (found at the last follow-up) was subtracted from every value of B_0, i.e. all data were transformed so that the individual minimum value was made equal to the minimum of the group mean. This treatment of the data provided the opportunity to establish the pure time-course of the central nervous compensatory process, irrespective of either individual differences in maximum deviation of B_0 (dependent on the functional level of the diseased vestibular organ before treatment) or individual differences in B_0 at long-term follow-up. The change in B_0 with time was found to be best represented by a power function: $B_0(\text{normalized}) = 1.55t^{-0.15}$, where t is time in days after maximum deviation of B_0. With both axes logarithmic this function gives a straight line with a coefficient of correlation $r = 0.81$, $p < 0.01$. Adaptation (least-square fit; Fig. 3) of this type of function to the real group means of B_0 in the third to seventh follow-up gives $B_0 = 8.65t^{-0.16}$ degrees.

Correction for body tilt, K_d and K_h

After gentamicin treatment there were also changes in the perception of roll tilt, as represented by the correction for tilt towards the diseased side (K_d) and towards the healthy side (K_h). Individual data for K_d and K_h before treatment, at the time when K_d reached its minimum value in each patient and in the 1-year follow-up, are shown in Fig. 4. Before treatment three of the four patients who had a pathological tilt of B_0 *away* from the diseased side also showed the largest values of K_d. In addition, K_d was greater than K_h ($K_d > K_h$) for these patients. This combination of findings in B_0, K_d and K_h is opposite to that seen after a unilateral loss of function. In addition, it was noted that the patient who had the most pronounced reduction in tilt perception before treatment showed the

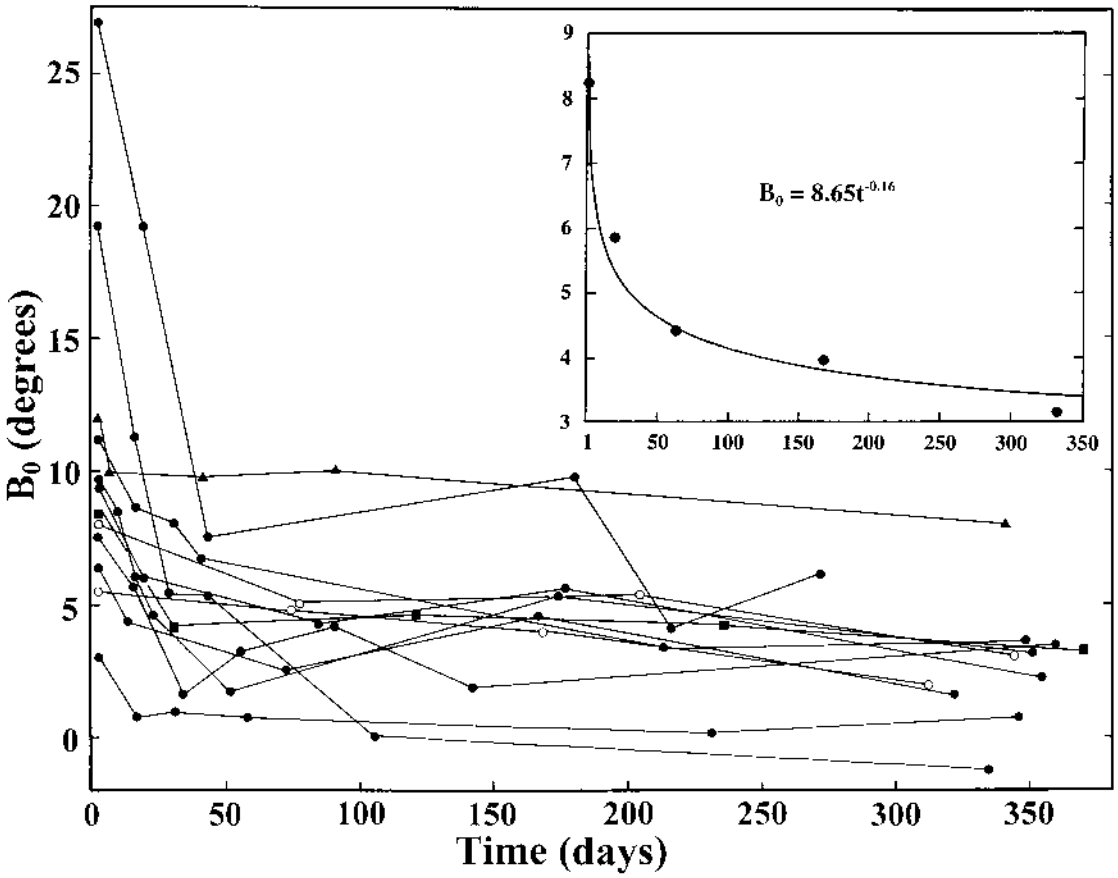

Fig. 3. Changes in B_0 for each patient after the maximum tilt. The time of maximum tilt is denoted day 1. For clarity a few different symbols are used. One patient, for whom changes in B_0 during follow-up were very small, is represented by *filled triangles*. In two patients (*open circles*) data could not be gained in the early stage after maximum tilt. The insert shows the group means of B_0 during the period of compensation and the power function defining the best-fitting line.

smallest tilt of B_0 after treatment, while the patient with the highest values of K_d and K_h before treatment showed a B_0 amounting to 19° shortly after treatment.

In 10 patients the individual minimum of K_d was found during the period from day 5 to day 48 (mean 16 days) after treatment. The two others showed the minimum at day 207 and day 399, respectively. However, they reached pathological values for K_d as early as during the fourth week and changes in K_d after that did not exceed the test–retest variability in normal subjects. The group mean of the individual minimum of K_d was $-11.4 \pm 4.5°$ ($n = 12$).

The group means of K_d and K_h on all test occasions are shown in Fig. 5. Before treatment there was no difference between the group means of K_d ($-1.86 \pm 5.06°$) and K_h ($-1.67 \pm 4.23°$). However, in the second follow-up K_d was significantly reduced: mean of $K_d = -8.68 \pm 5.86°$, $n = 11$, $p < 0.001$ (paired t-test), while K_h remained unchanged: mean of $K_h = -1.74 \pm 4.78°$, $p > 0.5$. After the second follow-up there was a gradual improvement in K_d but, with the exception of the sixth follow-up (0.5 years after treatment), it remained significantly different from pre-treatment values at all testing times (paired t-tests). No significant changes were seen in K_h during this period. Consequently, the treatment induced an asymmetry in roll-tilt perception; from the second follow-up there was a significant difference between K_d and K_h at all testing times (paired t-tests). In the last follow-up the means were: $K_d = -5.63 \pm 4.19°$ and $K_h = -2.01 \pm 7.06°$, $n = 12$, $p < 0.05$. Owing to the large differences between individuals it was not possible to define reliably the time-course of compensatory changes in K_d. However, as seen in Fig. 5, this did not appear to be similar to the time-course of B_0.

Detection of pathology

Although both the tilt of B_0 and the asymmetry in tilt perception were systematically related to the deafferented side, there was no correlation between K_d and B_0 (coefficient of correlation ranged from 0.07 to 0.59 during follow-up), nor was there any systematic relationship between the time when B_0 and K_d, respec-

tively, reached maximum abnormal deviation. As a consequence, measuring the SVH not only in the upright body position but also in tilted positions increases the possibility of detecting an abnormality in the vestibular system, especially on later test occasions.

The number of patients with pathological results in each test parameter at the different testing times is shown in Fig. 6. In the early stage after treatment, B_0 seems to be the most sensitive parameter for detecting unilateral otolith hypofunction; in the third follow-up all 12 patients had a pathological tilt of B_0 towards the treated side. During the entire period, K_d was more frequently pathological than K_h. In the last test, K_h was also pathological in a majority of the patients.

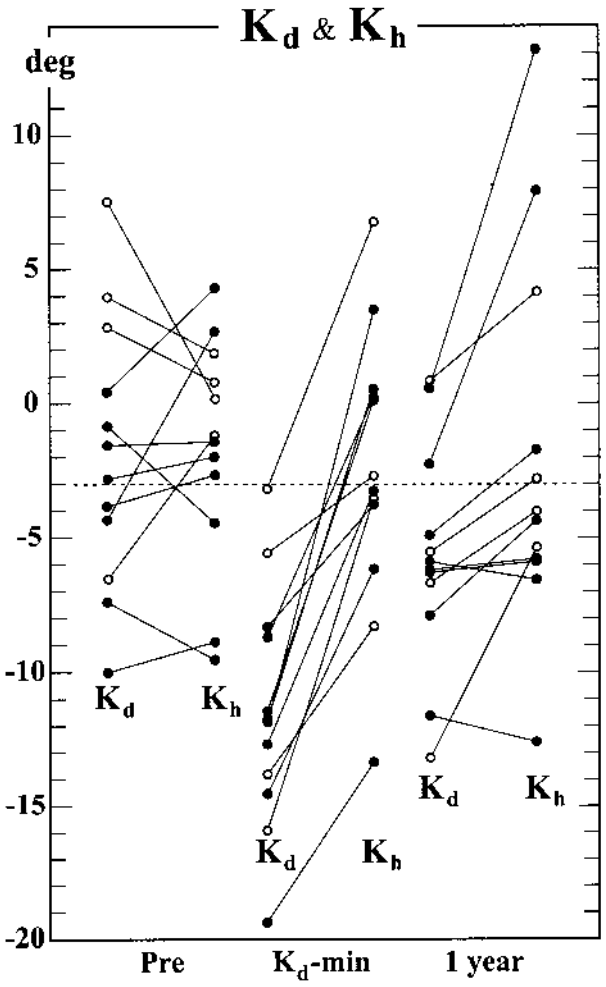

Fig. 4. Individual data of K_d and K_h before gentamicin treatment (Pre), at the testing when K_d reached its lowest value in each patient (K_d-min), and at the 1-year follow-up. Each line joins the values of K_d and K_h for one patient. The limit of the normal range is indicated by the *dashed line*. The four patients who showed a tilt of B_0 away from the diseased side before treatment (see Fig. 1) are represented by *open symbols*.

When considering only the findings of roll-tilt perception (K_d and K_h), at the second follow-up when K_d reached its group mean minimum, nine of the 11 patients tested had a pathological value for K_d and in eight of these the value for K_d was considerably (4–14°) more abnormal than the value for K_h. Findings were similar at the third follow-up, but later there was a tendency for this asymmetry in tilt perception to be reduced.

DISCUSSION

Subjective visual horizontal in the upright body position, B_0

The present study has shown that chemical labyrinthectomy causes changes in B_0 that are comparable to changes reported in the acute stage after unilateral vestibular neurectomy and vestibular neuritis (6, 10). The group mean of the individual maximum tilt of B_0 presented here was $10.6 \pm 6.5°$ (range 3–27°) at a mean time of 18 days after the last injection. Curthoys et al. found a mean tilt of SVH of $11.7 \pm 5.6°$ (range 3–25°) in 23 patients 1 week after neurectomy (10).

Patients showed the maximum tilt of B_0 at different times after treatment. Individual differences in the number of injections necessary, and in the time until maximum effect is reached, may depend on anatomical conditions as well as on differences in the sensitivity of the vestibular organ to gentamicin. The present findings suggest that measuring SVH might be a simple way to monitor ototoxic effects in gentamicin treatment. Further, it is of interest that the peak value for B_0 appears as late as 2–3 weeks after treatment. This is in line with previous findings on the delayed effects of gentamicin (15), suggesting that instalments should not necessarily be continued until vestibulotoxic effects are observed.

After gentamicin treatment it cannot be known with certainty for each individual that the loss of vestibular function on the treated side is complete. In the present material, however, none of the patients had any caloric reaction at long-term follow-up, and there were no reports of symptoms suggesting a fluctuating remnant of function. The group mean of B_0 and the interindividual variability were comparable with findings after vestibular neurectomy. The interindividual variability may be explained by differences in the functional level of the diseased vestibular organ *before* treatment (see *Vestibular compensation*, below). Hence, as to the function of the peripheral vestibular system after treatment, the present material may be regarded as a homogeneous group suitable for the study of otolith function.

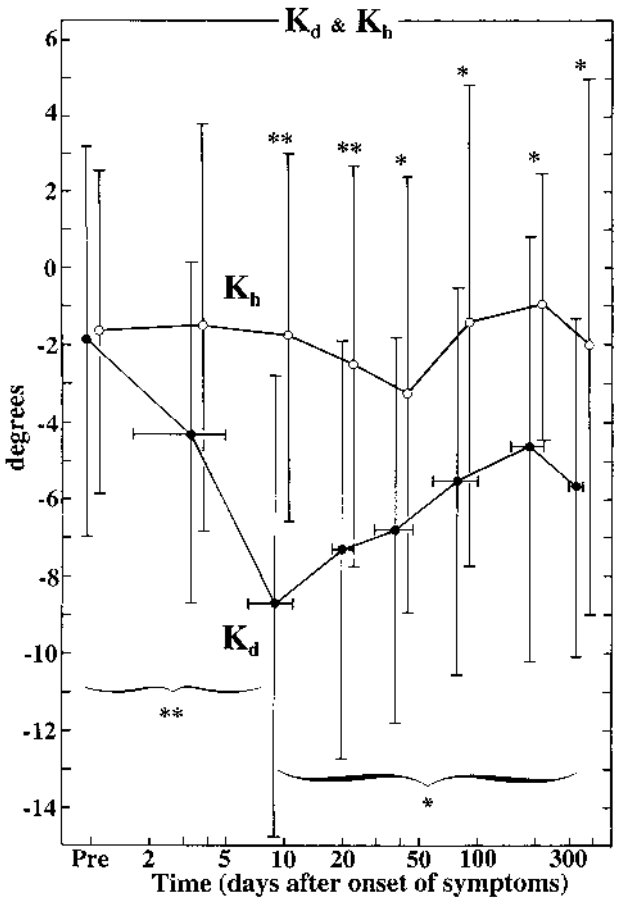

Fig. 5. Group means of K_d (correction for tilt to the diseased side) and K_h (correction for tilt to the healthy side) on all test occasions. The time-scale is logarithmic. Error bars represent interindividual variability (1 SD). Asterisks denote a significant difference between K_d and K_h or a significant change in K_d: $*p < 0.05$; $**p < 0.01$; $***p < 0.001$.

Roll-tilt perception, K_d and K_h

The present study also demonstrated a significant reduction in the perception of body tilt towards the deafferented side, causing an asymmetric roll-tilt perception. This confirms suggestions that the human utricle is an asymmetric sensor with a preference for ipsilateral tilt (7, 11).

No correlation was found between the values for B_0 and K_d. This is consistent with recent findings in normal subjects (8) and in patients who had undergone stapedotomy (18), and suggests that these parameters are to some extent dependent on different afferent information from the vestibular organ. Consequently, measuring SVH not only in the upright position but also at moderate lateral tilts increases the possibility of detecting and lateralizing a lesion. As seen in Fig. 6, at the 1-year follow-up five of the 12 patients had normal values for B_0 but were pathological in K_d or in both K_d and K_h; when considering the combined results of the three test parameters, all 12 patients were found to have pathological results, which in 10 of them also indicated the lesioned side.

An irritative state of the otolith organs

Before treatment four patients showed a B_0 that was pathologically tilted away from the diseased side. Three of these four patients had the largest values of K_d before treatment; they were the only patients showing an overcorrection ($K > 0°$) for tilt to the diseased side. In addition, they also had $K_d > K_h$. These findings are the opposite of what is generally seen after unilateral loss of vestibular function. They are consistent with changes in B_0, K_d and K_h found after stapedotomy (18), and suggest an increased resting activity in otolithic sensory afferents. This is probably an analogue of the irritative nystagmus sometimes seen in patients with Meniere's disease (19), with the fast phase beating towards the affected side. Thus, knowledge about the possibility of abnormal excitation of sensory or neural structures in the vestibular organ is of importance for correctly interpreting deviations in SVH.

Vestibular compensation

The gradual reduction in tilt of SVH is one manifestation of vestibular compensation. Serial measurement of SVH is, thus, one possibility for studying the course of vestibular compensation. In spite of the interindividual variability in the material and an uncertainty in the co-ordinate for the maximum tilt of B_0, which may differ in time and magnitude from the real maximum, it was possible to characterize the time-course of compensation for the loss of tonic otolithic input on the diseased side. The rapid decay of B_0 in the early stage, followed by very slow changes later during the observation period, does not fit well with an exponential decay ($B_0 = ae^{-bt}$). It is better represented by a power function ($B_0 = 8.65t^{-0.16}$ degrees). It must be borne in mind that this formula is valid only for group data; to obtain normative data for compensation, valid for individuals, it would be necessary to study larger materials, grouped according to differences in maximum deviation of B_0. However, the formula seems to give a fairly good prediction of the mean tilt of SVH at long-term follow-up. In 39 patients with unilateral loss of vestibular function, Tabak et al. (20) found a mean tilt of the subjective visual vertical towards the deafferented side of 2.22° (right-sided deafferentation) and 2.55° (left-sided) at a mean time of 4.5 years after deafferentation. Our formula gives a prediction of 2.65°.

It remains an open question as to which processes on neural level are reflected in the decay of B_0.

Compensation is generally believed to depend on a restoration of the resting neural tone balance between the right and left vestibular nucleus, due to a reappearance of activity on the deafferented side. However, electrophysiological studies in the cat have shown that after unilateral vestibular deafferentation there is, in the early stage, a reduced resting activity in the lateral vestibular nucleus on the *intact* side as well (21). Such a mechanism seems likely to contribute to a reduction in tilt of SVH during the early stage, by reducing the side difference in resting activity in the central otolithic system. It is, of course, a matter of definition whether such depression of vestibular activity initially after lesions should be regarded as a neural correlate of compensation, or whether changes in a parameter such as B_0 reflect several factors, not only compensation.

A relationship was found between the functional level of the diseased labyrinth before treatment and the maximum deviation in SVH shortly after treatment. It can reasonably be assumed that the mechanism by which the functional level of the diseased labyrinth influences SVH after treatment involves vestibular compensation. In cases where compensation has already developed due to a partial loss of function, complete deafferentation will result in a smaller deviation in SVH. The single patient in the present material who had normal caloric responses before treatment showed the largest change in SVH, while two patients with a pronounced loss of caloric responses showed the smallest changes. A large tilt (25°) of SVH after deafferentation in a patient with normal caloric responses before treatment has also been reported by Curthoys et al. (10). In the present study it was also noted that the patient with the most pronounced reduction in tilt perception before treatment showed the smallest tilt of SVH after treatment, while the patient with the highest tilt perception before treatment showed a very large tilt of SVH after treatment. It is significant that even in patients who, with respect to function of the peripheral vestibular system shortly after deafferentation, constitute a homogeneous group, interindividual differences in SVH may be considerable, owing to differences in the degree of vestibular compensation that has already developed before treatment.

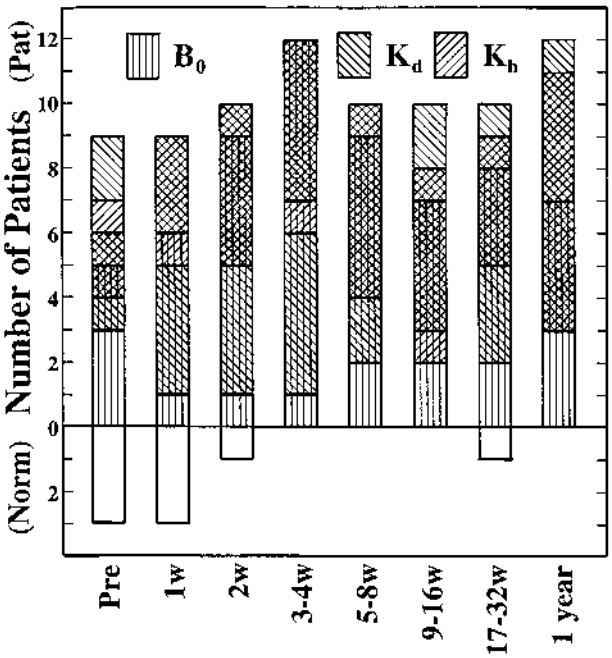

Fig. 6. Number of patients with pathological values (Pat) in one, two or all three test parameters in the different testing intervals.

In contrast to the tilt of B_0, which represents a resting asymmetry in the otolith system, the difference between K_d and K_h represents an asymmetry in the responsiveness to stimuli of the utricle on the healthy side. Changes with time for the group means of K_d and K_h show that compensation for asymmetry in roll-tilt perception mainly involves a gradual improvement in K_d. This suggests a relatively selective increase in the gain for contralateral tilt of the remaining utricle. However, there was a large individual variability in K_h at the end of follow-up and the number of patients showing pathological values for K_h tended to increase. It seems that in some patients a reduction in responsiveness for tilt towards the healthy side is one component of a restoration of symmetric roll-tilt perception. Further studies will show whether patients can be grouped according to different patterns of compensation.

A diagnostic consequence of otolith compensation may be the problem of detecting and localizing long-standing lesions. For the otoliths there is still nothing similar to the caloric test, which provides the opportunity to stimulate the vestibular organ on one side.

Finally, it may be of general interest, considering CNS plasticity, that there are advantages in studying the recovery of a function that can easily be observed and quantified repeatedly in the same individual. It would be of major interest to relate the time-course of otolith compensation to the chemical, morphological and electrophysiological changes seen in CNS regions after deafferentation, as well as to the time-course of recovery of other functions.

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